INI CET Biochemistry

Sample Paper – 1

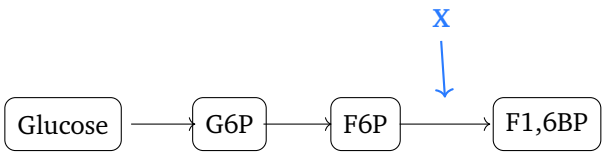
Duration: 14 Minutes **Maximum Marks: 15**

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 pathway shown, the committed rate-limiting step marked **X** (fructose-6-phosphate to fructose-1,6-bisphosphate) is catalysed by:



(A) Hexokinase

(B) Pyruvate kinase

(C) Phosphofructokinase-1

(D) Aldolase

Q2. The net yield of ATP from the anaerobic glycolysis of one glucose molecule to lactate is:

(A) 2 ATP

(B) 4 ATP

(C) 8 ATP

(D) 38 ATP

Q3. Classic galactosemia, with cataracts, hepatomegaly and failure to thrive, is caused by deficiency of:

(A) Galactokinase

(B) Aldose reductase

(C) Galactose-1-phosphate uridyltransferase

(D) UDP-galactose epimerase

Lipid Metabolism

Q4. The rate-limiting enzyme of cholesterol biosynthesis, which is the target of statin drugs, is:

(A) HMG-CoA reductase

(B) HMG-CoA synthase

(C) Acetyl-CoA carboxylase

(D) Squalene synthase

Q5. Beta-oxidation of fatty acids takes place mainly in which subcellular compartment?

(A) Cytoplasm

(B) Mitochondrial matrix

(C) Endoplasmic reticulum

(D) Golgi apparatus

Amino Acid and Protein Metabolism

Q6. Which urea cycle enzyme deficiency is the most common and is inherited in an X-linked manner?

(A) Arginase

(B) Carbamoyl phosphate synthetase I

(C) Argininosuccinate synthetase

(D) Ornithine transcarbamylase

Q7. Transamination reactions of amino acids require which vitamin-derived coenzyme?

(A) Thiamine pyrophosphate (B_1)

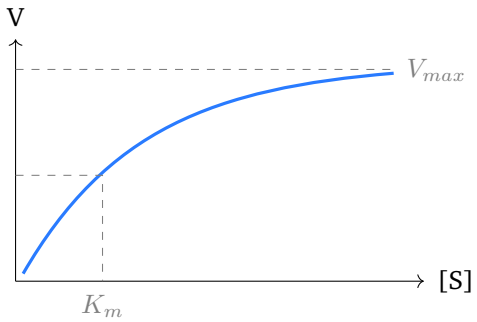
(B) Pyridoxal phosphate (B_6)

(C) Biotin (B_7)

(D) Cobalamin (B_{12})

Enzymes, Vitamins and Coenzymes

Q8. In the Michaelis-Menten plot shown, the Michaelis constant K_m corresponds to the substrate concentration at which the reaction velocity is:



(A) Equal to V_{max}

(B) Zero

(C) At its maximum

(D) Half of V_{max}

Q9. A classic competitive inhibitor alters enzyme kinetics by:

(A) Decreasing V_{max} with K_m unchanged

(B) Decreasing both K_m and V_{max}

(C) Increasing the apparent K_m with V_{max} unchanged

(D) Increasing V_{max}

Q10. Deficiency of thiamine (vitamin B_1) classically produces which disease?

(A) Beri-beri

(B) Pellagra

(C) Scurvy

(D) Rickets

Molecular Biology and Genetics

Q11. During DNA replication, the enzyme that adds nucleotides to synthesise a new strand in the 5′ to 3′ direction is:

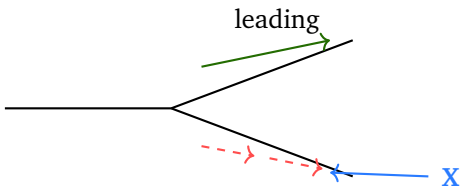
(A) DNA ligase

(B) Primase

(C) DNA polymerase

(D) Helicase

Q12. In the replication fork shown, the strand that is synthesised discontinuously as short Okazaki fragments (marked **X**) is the:



(A) Template strand

(B) Leading strand

(C) Primer strand

(D) Lagging strand

Q13. The synthesis of an RNA molecule from a DNA template is called:

(A) Transcription

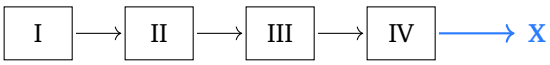
(B) Translation

(C) Replication

(D) Reverse transcription

Bioenergetics, Nucleotides and Clinical Biochemistry

Q14. In the mitochondrial electron transport chain shown, the final acceptor of electrons (marked **X**) is:



(A) NAD^+

(B) Molecular oxygen (O_2)

(C) Cytochrome c

(D) Coenzyme Q

Q15. Lesch-Nyhan syndrome, with hyperuricaemia and self-mutilation, results from deficiency of the purine salvage enzyme:

(A) Adenosine deaminase

(B) Hypoxanthine-guanine phosphoribosyltransferase (HGPRT)

(C) Xanthine oxidase

(D) PRPP synthetase

Detailed Solutions

Q1.

Solution

Concept – regulation of glycolysis: The committed, rate-limiting step is the best control point.

Step 1 – identify the step: The conversion of fructose-6-phosphate to fructose-1,6-bisphosphate is catalysed by **phosphofructokinase-1 (PFK-1)**.

Step 2 – why it is regulatory: PFK-1 is allosterically inhibited by ATP and citrate and activated by AMP and fructose-2,6-bisphosphate.

Why the other options are wrong:

- A, hexokinase: catalyses the first step (glucose to G6P), not step X.
- B, pyruvate kinase: the last irreversible step.
- D, aldolase: splits fructose-1,6-bisphosphate, downstream of X.

Final Answer: Phosphofructokinase-1 $\Rightarrow$ C

Answer: (C) Go Back to Q1

Q2.

Solution

Concept – ATP accounting in glycolysis: Count ATP used in the investment phase and made in the payoff phase.

Step 1 – tally: Two ATP are consumed early; four are produced later.

Step 2 – net figure: Net $= 4 - 2 =$ **2 ATP** per glucose under anaerobic conditions.

Why the other options are wrong:

- B, 4 ATP: the gross, not net, yield.
- C and D, 8 and 38: include aerobic oxidation, which does not occur anaerobically.

Final Answer: 2 ATP $\Rightarrow$ A

Answer: (A) Go Back to Q2

Q3.

Solution

Concept – galactose metabolism: A specific transferase channels galactose into glucose metabolism.

Step 1 – name the enzyme: Classic galactosemia is due to **galactose-1-phosphate uridyltransferase** deficiency.

Step 2 – link to features: Accumulated galactose-1-phosphate and galactitol cause cataracts, liver damage and failure to thrive.

Why the other options are wrong:

- A, galactokinase: its deficiency causes only cataracts (a milder form).
- B, aldose reductase: makes galactitol but is not the classic defect.
- D, epimerase: a rare, usually benign variant.

Final Answer: Galactose-1-phosphate uridyltransferase $\Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – cholesterol synthesis control: The committed step is the drug target.

Step 1 – name the enzyme: HMG-CoA reductase converts HMG-CoA to mevalonate and is rate-limiting.

Step 2 – clinical link: Statins competitively inhibit it to lower plasma cholesterol.

Why the other options are wrong:

- B, HMG-CoA synthase: forms HMG-CoA, an earlier step.
- C, acetyl-CoA carboxylase: rate-limiting for fatty acid, not cholesterol, synthesis.
- D, squalene synthase: a later, non-regulatory step.

Final Answer: HMG-CoA reductase $\Rightarrow$ A

Answer: (A) Go Back to Q4

Q5.

Solution

Concept – site of beta-oxidation: Fatty acids are broken down where the TCA cycle and respiratory chain sit.

Step 1 – name the compartment: Beta-oxidation occurs in the **mitochondrial matrix**.

Step 2 – the carrier: Long-chain fatty acids enter via the carnitine shuttle.

Why the other options are wrong:

- A, cytoplasm: the site of fatty acid synthesis, the opposite pathway.
- C, endoplasmic reticulum: chain elongation and desaturation.
- D, Golgi apparatus: not a beta-oxidation site.

Final Answer: Mitochondrial matrix $\Rightarrow$ B

Answer: (B) Go Back to Q5

Q6.

Solution

Concept – urea cycle defects: One enzyme stands out for frequency and inheritance.

Step 1 – name it: Ornithine transcarbamylase (OTC) deficiency is the commonest and the only X-linked urea cycle disorder.

Step 2 – clue: It causes hyperammonaemia with high orotic acid but normal citrulline.

Why the other options are wrong:

- A, B, C: arginase, CPS-I and argininosuccinate synthetase deficiencies are autosomal recessive and rarer.

Final Answer: Ornithine transcarbamylase $\Rightarrow$ D

Answer: (D) Go Back to Q6

Q7.

Solution

Concept – coenzymes of amino acid metabolism: Transamination moves an amino group using a B-vitamin cofactor.

Step 1 – name the coenzyme: Pyridoxal phosphate, from vitamin B_6, is the coenzyme for transaminases.

Step 2 – reasoning: It forms a Schiff base that carries the amino group between keto acids.

Why the other options are wrong:

- A, thiamine pyrophosphate: for oxidative decarboxylations.
- C, biotin: for carboxylation reactions.
- D, cobalamin: for methylmalonyl-CoA and methionine synthesis.

Final Answer: Pyridoxal phosphate (B_6) $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – meaning of K_m: It is a point defined on the Michaelis-Menten curve.

Step 1 – read the graph: K_m is the substrate concentration giving **half of** V_{max}.

Step 2 – significance: A low K_m means high affinity of the enzyme for its substrate.

Why the other options are wrong:

- A and C: velocity equals or approaches V_{max} only at saturating substrate.
- B, zero: velocity is zero only at zero substrate.

Final Answer: Half of V_{max} $\Rightarrow$ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – competitive inhibition: The inhibitor competes with substrate at the active site.

Step 1 – effect on constants: It **raises the apparent** K_m (more substrate needed) while V_{max} stays the same.

Step 2 – reasoning: Enough substrate can outcompete the inhibitor, so maximal velocity is still reachable.

Why the other options are wrong:

- A, lowered V_{max}: describes non-competitive inhibition.
- B and D: neither pattern fits competitive inhibition.

Final Answer: Increased K_m, V_{max} unchanged $\Rightarrow$ $\boxed{\text{C}}$

Answer: (C) Go Back to Q9

Q10.

Solution

Concept – vitamin deficiency diseases: Each classic deficiency maps to a syndrome.

Step 1 – match B_1: Thiamine deficiency causes **beri-beri** (and Wernicke-Korsakoff syndrome).

Step 2 – mechanism: Thiamine pyrophosphate is needed by pyruvate and alpha-ketoglutarate dehydrogenase.

Why the other options are wrong:

- B, pellagra: niacin (B_3) deficiency.
- C, scurvy: vitamin C deficiency.
- D, rickets: vitamin D deficiency.

Final Answer: Beri-beri $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – enzymes of replication: Different enzymes unwind, prime, extend and seal DNA.

Step 1 – name the polymerising enzyme: DNA polymerase adds nucleotides 5′ to 3′ onto a primed template.

Step 2 – proofreading: It also has 3′ to 5′ exonuclease proofreading activity.

Why the other options are wrong:

- A, ligase: seals nicks between fragments.
- B, primase: lays down RNA primers only.
- D, helicase: unwinds the double helix.

Final Answer: DNA polymerase ⇒ C

Answer: (C) Go Back to Q11

Q12.

Solution

Concept – leading vs lagging strand: Only one new strand can be made continuously toward the fork.

Step 1 – identify X: The strand made in short Okazaki fragments is the **lagging strand**.

Step 2 – why: Because polymerase works only 5′ to 3′, the strand running the other way is built in pieces and later joined by ligase.

Why the other options are wrong:

- A, template: the parental strand being copied, not synthesised.
- B, leading strand: made continuously, not in fragments.
- C, primer strand: primers are short RNA starts, not a whole strand.

Final Answer: Lagging strand ⇒ D

Answer: (D) Go Back to Q12

Q13.

Solution

Concept – the central dogma: Information flows DNA to RNA to protein.

Step 1 – name the step: Making RNA from a DNA template is **transcription**.

Step 2 – the enzyme: RNA polymerase carries it out, needing no primer.

Why the other options are wrong:

- B, translation: RNA to protein at the ribosome.
- C, replication: DNA to DNA.
- D, reverse transcription: RNA to DNA (retroviruses).

Final Answer: Transcription $\Rightarrow$ A

Answer: (A) Go Back to Q13

Q14.

Solution

Concept – the electron transport chain: Electrons flow down the complexes to a terminal acceptor.

Step 1 – identify X: The final electron acceptor is **molecular oxygen (O_2)**, reduced to water at complex IV.

Step 2 – significance: Without oxygen the chain backs up and oxidative phosphorylation stops.

Why the other options are wrong:

- A, NAD^+: an electron donor feeding complex I.
- C and D, cytochrome c and coenzyme Q: mobile carriers within the chain, not the final acceptor.

Final Answer: Molecular oxygen (O_2) $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – purine salvage: A salvage enzyme recycles purine bases.

Step 1 – name the enzyme: **HGPRT** salvages hypoxanthine and guanine; its deficiency causes Lesch-Nyhan syndrome.

Step 2 – link to features: Loss of salvage raises uric acid, giving hyperuricaemia, gout and self-mutilation.

Why the other options are wrong:

- A, adenosine deaminase: its deficiency causes severe combined immunodeficiency.
- C, xanthine oxidase: its inhibition (allopurinol) treats gout.
- D, PRPP synthetase: overactivity, not deficiency, raises purines.

Final Answer: HGPRT $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q15

Answer Key

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