INI CET Biochemistry

Sample Paper – 5

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. The hepatic enzyme that has a high K_m for glucose, is not inhibited by its product glucose-6-phosphate, and thereby acts as a glucose sensor in liver and pancreatic beta cells is:

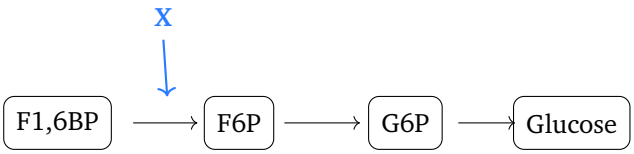
(A) Glucokinase

(B) Hexokinase

(C) Glucose-6-phosphatase

(D) Phosphoglucomutase

Q2. In the gluconeogenic sequence shown, the reaction marked **X** (fructose-1,6-bisphosphate to fructose-6-phosphate), which opposes the action of PFK-1, is catalysed by:



(A) Phosphofructokinase-1

(B) Fructose-1,6-bisphosphatase

(C) Aldolase

(D) Glucose-6-phosphatase

Q3. Deficiency of the lysosomal enzyme acid alpha-glucosidase (acid maltase), causing lysosomal glycogen accumulation with cardiomegaly and hypotonia, is glycogen storage disease type II, also known as:

(A) Von Gierke disease

(B) McArdle disease

(C) Pompe disease

(D) Cori disease

Lipid Metabolism

Q4. In the hepatic ketogenesis pathway shown, the enzyme catalysing step **X**, which cleaves HMG-CoA to release the ketone body acetoacetate, is:

(A) Thiolase

(B) HMG-CoA synthase

(C) HMG-CoA lyase

(D) HMG-CoA reductase

Q5. A 6-month-old infant with progressive neurodegeneration, exaggerated startle response and a cherry-red spot on the macula is found to have hexosaminidase A deficiency. The diagnosis is:

(A) Gaucher disease

(B) Niemann-Pick disease

(C) Fabry disease

(D) Tay-Sachs disease

Amino Acid and Protein Metabolism

Q6. Deficiency of cystathionine beta-synthase, presenting with downward lens dislocation, a marfanoid habitus and a tendency to thromboembolism, causes:

(A) Alkaptonuria

(B) Phenylketonuria

(C) Homocystinuria

(D) Cystinuria

Q7. The universal donor of methyl groups in biological methylation reactions, such as the synthesis of creatine and epinephrine, is:

(A) Tetrahydrofolate

(B) S-adenosylmethionine (SAM)

(C) S-adenosylhomocysteine

(D) Methylcobalamin

Enzymes, Vitamins and Coenzymes

Q8. According to the IUBMB classification into six enzyme classes, an enzyme that joins two molecules together with the concomitant hydrolysis of ATP, such as DNA ligase, belongs to the class of:

(A) Lyases

(B) Isomerases

(C) Transferases

(D) Ligases

Q9. Deficiency of which vitamin classically causes rickets in children and osteomalacia in adults?

(A) Vitamin D

(B) Vitamin C

(C) Vitamin K

(D) Vitamin A

Q10. Which vitamin acts as the coenzyme for carboxylation reactions such as those catalysed by pyruvate carboxylase and acetyl-CoA carboxylase?

(A) Biotin

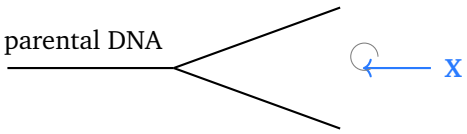
(B) Folate

(C) Niacin

(D) Riboflavin

Molecular Biology and Genetics

Q11. In the replication fork shown, the enzyme that relieves the positive supercoiling generated ahead of the advancing fork (region marked **X**) is:



(A) Helicase

(B) Primase

(C) DNA ligase

(D) Topoisomerase

Q12. The flexibility that allows the third base of a codon to pair with the first base of the anticodon in a non-standard way, so that one tRNA can read several codons, is explained by:

(A) The wobble hypothesis

(B) The central dogma

(C) Chargaff's rule

(D) Semiconservative replication

Q13. Insertion or deletion of a number of nucleotides that is not a multiple of three shifts the reading frame downstream of the mutation. This type of change is called a:

(A) Silent mutation

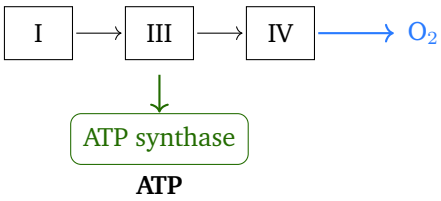
(B) Missense mutation

(C) Frameshift mutation

(D) Point mutation

Bioenergetics, Nucleotides and Clinical Biochemistry

Q14. In oxidative phosphorylation, electrons flow through the respiratory complexes to oxygen while ATP is made, as shown. The number of ATP molecules synthesised per atom of oxygen reduced is termed the:



(A) Respiratory quotient

(B) P/O ratio

(C) Redox potential

(D) Phosphorylation potential

Q15. The committed, rate-limiting step of de novo purine nucleotide synthesis, in which an amino group from glutamine is transferred to PRPP to form 5-phosphoribosylamine, is catalysed by:

(A) PRPP synthetase

(B) Glutamine-PRPP amidotransferase

(C) Adenylosuccinate lyase

(D) IMP dehydrogenase

Detailed Solutions

Q1.

Solution

Concept – glucose-phosphorylating enzymes: Hexokinase and glucokinase both make glucose-6-phosphate but differ in kinetics.

Step 1 – identify the sensor enzyme: Glucokinase has a high K_m (low affinity), so its activity rises as blood glucose rises.

Step 2 – why it senses glucose: It is not inhibited by glucose-6-phosphate, so in liver and pancreatic beta cells its rate tracks the glucose level and helps trigger insulin release.

Why the other options are wrong:

- B, hexokinase: low K_m, saturated at normal glucose and inhibited by its product.
- C, glucose-6-phosphatase: removes phosphate, it does not phosphorylate glucose.
- D, phosphoglucomutase: interconverts G1P and G6P.

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

Answer: (A) Go Back to Q1

Q2.

Solution

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

Step 1 – identify step X: The conversion of fructose-1,6-bisphosphate back to fructose-6-phosphate is catalysed by **fructose-1,6-bisphosphatase**.

Step 2 – why it opposes PFK-1: It removes a phosphate that PFK-1 had added, and the two enzymes are reciprocally regulated by fructose-2,6-bisphosphate.

Why the other options are wrong:

- A, phosphofructokinase-1: runs the opposite, glycolytic direction.
- C, aldolase: splits fructose-1,6-bisphosphate into trioses.
- D, glucose-6-phosphatase: acts later, on glucose-6-phosphate.

Final Answer: Fructose-1,6-bisphosphatase $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q2

Q3.

Solution

Concept – glycogen storage diseases: Each GSD maps to a specific enzyme defect.

Step 1 – name the disease: Deficiency of lysosomal acid alpha-glucosidase (acid maltase) is glycogen storage disease type II, or **Pompe disease**.

Step 2 – link to features: Glycogen builds up inside lysosomes, especially in cardiac and skeletal muscle, giving cardiomegaly and hypotonia.

Why the other options are wrong:

- A, Von Gierke (type I): glucose-6-phosphatase deficiency.
- B, McArdle (type V): muscle glycogen phosphorylase deficiency.
- D, Cori (type III): debranching enzyme deficiency.

Final Answer: Pompe disease $\Rightarrow$ C

Answer: (C) Go Back to Q3

Q4.

Solution

Concept – ketone body synthesis: Ketogenesis in the liver mitochondria makes acetoacetate from acetyl-CoA.

Step 1 – identify step X: HMG-CoA lyase cleaves HMG-CoA into acetoacetate and acetyl-CoA.

Step 2 – the pathway order: Two acetyl-CoA form acetoacetyl-CoA, HMG-CoA synthase makes HMG-CoA, then HMG-CoA lyase releases acetoacetate.

Why the other options are wrong:

- A, thiolase: condenses the first two acetyl-CoA units.
- B, HMG-CoA synthase: forms HMG-CoA, the step before X.
- D, HMG-CoA reductase: belongs to cholesterol synthesis, in the cytosol.

Final Answer: HMG-CoA lyase $\Rightarrow$ C

Answer: (C) Go Back to Q4

Q5.

Solution

Concept – sphingolipidoses: Each lysosomal storage disease reflects a missing degradative enzyme.

Step 1 – match the enzyme: Deficiency of **hexosaminidase A** causes accumulation of GM2 ganglioside, defining **Tay-Sachs disease**.

Step 2 – link to features: GM2 build-up in neurons produces the cherry-red macular spot, the exaggerated startle and progressive neurodegeneration, without hepatosplenomegaly.

Why the other options are wrong:

- A, Gaucher: glucocerebrosidase deficiency, with hepatosplenomegaly.
- B, Niemann-Pick: sphingomyelinase deficiency (cherry-red spot but organomegaly).
- C, Fabry: alpha-galactosidase A deficiency, X-linked, with angiokeratomas.

Final Answer: Tay-Sachs disease $\Rightarrow$ D

Answer: (D) Go Back to Q5

Q6.

Solution

Concept – disorders of sulfur amino acids: A block in transsulfuration raises homocysteine.

Step 1 – name the disease: Deficiency of **cystathionine beta-synthase** causes **homocystinuria**.

Step 2 – link to features: High homocysteine damages connective tissue and vessels, giving lens dislocation (typically downward), a marfanoid build and thromboembolism.

Why the other options are wrong:

- A, alkaptonuria: homogentisate oxidase defect, dark urine and ochronosis.
- B, phenylketonuria: phenylalanine hydroxylase defect.
- D, cystinuria: a transport defect causing cystine kidney stones.

Final Answer: Homocystinuria $\Rightarrow$ C

Answer: (C) Go Back to Q6

Q7.

Solution

Concept – methyl group transfer: Methylation reactions need an activated methyl donor.

Step 1 – name the donor: S-adenosylmethionine (SAM) is the universal methyl donor.

Step 2 – reasoning: Its high-energy sulfonium methyl is passed to acceptors such as guanidinoacetate and noradrenaline, leaving S-adenosylhomocysteine.

Why the other options are wrong:

- A, tetrahydrofolate: carries one-carbon units but is not the universal methyl donor.
- C, S-adenosylhomocysteine: the demethylated product, not the donor.
- D, methylcobalamin: transfers a methyl only in the specific methionine synthase reaction.

Final Answer: S-adenosylmethionine (SAM) $\Rightarrow$ B

Answer: (B) Go Back to Q7

Q8.

Solution

Concept – the six IUBMB enzyme classes: Enzymes are grouped as oxidoreductases, transferases, hydrolases, lyases, isomerases and ligases.

Step 1 – classify the reaction: Joining two molecules using the energy of ATP hydrolysis is the defining action of a **ligase**.

Step 2 – the example: DNA ligase seals two DNA ends together at the expense of ATP (or NAD^+), so it is a ligase.

Why the other options are wrong:

- A, lyases: break bonds without hydrolysis or oxidation, often forming double bonds.
- B, isomerases: rearrange atoms within one molecule.
- C, transferases: move a functional group from one molecule to another.

Final Answer: Ligases $\Rightarrow$ D

Answer: (D) Go Back to Q8

Q9.

Solution

Concept – fat-soluble vitamin deficiency: Vitamin D controls calcium and bone mineralisation.

Step 1 – match the vitamin: Vitamin D deficiency impairs mineralisation, causing rickets in children and osteomalacia in adults.

Step 2 – mechanism: Its active form (calcitriol) raises intestinal calcium and phosphate absorption; without it, bone fails to mineralise.

Why the other options are wrong:

- B, vitamin C: deficiency causes scurvy.
- C, vitamin K: deficiency causes bleeding.
- D, vitamin A: deficiency causes night blindness.

Final Answer: Vitamin D $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q9

Q10.

Solution

Concept – coenzymes of carboxylation: Adding a carboxyl (CO_2) group needs a specific carrier vitamin.

Step 1 – name the coenzyme: Biotin is the coenzyme of carboxylases.

Step 2 – reasoning: It is covalently linked to the enzyme and carries an activated CO_2 for reactions such as pyruvate carboxylase and acetyl-CoA carboxylase.

Why the other options are wrong:

- B, folate: carries one-carbon units other than CO_2.
- C, niacin: forms NAD^+/$NADP^+$ for redox reactions.
- D, riboflavin: forms FAD/FMN for redox reactions.

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

Answer: (A) Go Back to Q10

Q11.

Solution

Concept – topological strain in replication: Unwinding the helix twists the DNA ahead of the fork.

Step 1 – identify X: The enzyme relieving the positive supercoils ahead of the fork is **topoisomerase** (in bacteria, DNA gyrase).

Step 2 – mechanism: It transiently cuts one or both strands, lets the DNA unwind, then reseals it, releasing the torsional strain.

Why the other options are wrong:

- A, helicase: separates the two strands at the fork but does not relieve supercoiling.
- B, primase: lays down RNA primers.
- C, DNA ligase: seals nicks between Okazaki fragments.

Final Answer: Topoisomerase $\Rightarrow$ $\boxed{\text{D}}$

Answer: (D) Go Back to Q11

Q12.

Solution

Concept – codon-anticodon pairing: The genetic code is degenerate, and one tRNA often reads several codons.

Step 1 – name the idea: This flexibility is explained by the **wobble hypothesis**.

Step 2 – reasoning: Only the first two codon bases pair strictly; the third (3′) base can pair loosely with the first anticodon base, so bases like inosine can read several codons.

Why the other options are wrong:

- B, central dogma: the overall DNA to RNA to protein flow.
- C, Chargaff's rule: A=T and G=C base ratios in DNA.
- D, semiconservative replication: describes how DNA copies itself.

Final Answer: The wobble hypothesis $\Rightarrow$ $\boxed{\text{A}}$

Answer: (A) Go Back to Q12

Q13.

Solution

Concept – effect of insertions and deletions: The code is read in non-overlapping triplets.

Step 1 – name the mutation: An insertion or deletion that is not a multiple of three causes a **frameshift mutation**.

Step 2 – reasoning: The reading frame shifts, so every codon downstream is altered and a premature stop codon usually appears.

Why the other options are wrong:

- A, silent mutation: a base change that does not alter the amino acid.
- B, missense mutation: a base change giving a different amino acid.
- D, point mutation: a single base substitution, which keeps the frame.

Final Answer: Frameshift mutation $\Rightarrow$ C

Answer: (C) Go Back to Q13

Q14.

Solution

Concept – efficiency of oxidative phosphorylation: ATP made is related to oxygen consumed.

Step 1 – name the ratio: The ATP synthesised per atom of oxygen reduced is the **P/O ratio**.

Step 2 – typical values: It is about 2.5 for NADH and 1.5 for $FADH_2$, reflecting how many protons each electron pair pumps.

Why the other options are wrong:

- A, respiratory quotient: CO_2 produced over O_2 consumed, a whole-body measure.
- C, redox potential: the tendency of a couple to gain or lose electrons.
- D, phosphorylation potential: the energy state of the ATP/ADP pool.

Final Answer: P/O ratio $\Rightarrow$ B

Answer: (B) Go Back to Q14

Q15.

Solution

Concept – regulation of de novo purine synthesis: The committed step is the main control point.

Step 1 – name the enzyme: Glutamine-PRPP amidotransferase transfers an amino group from glutamine to PRPP to make 5-phosphoribosylamine.

Step 2 – why it is committed: This is the first, rate-limiting and feedback-inhibited step (by IMP, AMP and GMP) of de novo purine synthesis.

Why the other options are wrong:

- A, PRPP synthetase: makes PRPP, a substrate feeding several pathways, so not the committed purine step.
- C, adenylosuccinate lyase: acts later, in AMP synthesis.
- D, IMP dehydrogenase: acts later, in GMP synthesis.

Final Answer: Glutamine-PRPP amidotransferase $\Rightarrow$ $\boxed{\text{B}}$

Answer: (B) Go Back to Q15

Answer Key

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