Biochemistry Unit III Question Bank

Multiple Choice Questions (MCQs)

(10 MCQs, each carrying 1 mark)

1. **How many ATP molecules are produced from the complete β-oxidation of palmitic acid (16 carbons)?**
   a) 106
   b) 108
   c) 129
   d) 131
   Answer: a) 106
   Explanation: Palmitic acid undergoes 7 β-oxidation cycles, producing 8 acetyl-CoA (8 × 10 ATP = 80 ATP via TCA cycle), 7 NADH (7 × 2.5 ATP = 17.5 ATP), and 7 $FADH_2$ (7 × 1.5 ATP = 10.5 ATP). Total = 108 ATP, but 2 ATP are used for activation, yielding a net of 106 ATP.

2. **Ketone bodies are primarily formed in which organ?**
   a) Brain
   b) Liver
   c) Kidney
   d) Muscle
   Answer: b) Liver
   Explanation: The liver produces ketone bodies (acetoacetate, β-hydroxybutyrate, acetone) during fasting or starvation via ketogenesis.

3. **Which enzyme catalyzes the rate-limiting step in de novo fatty acid synthesis?**
   a) Fatty acid synthase
   b) Acetyl-CoA carboxylase
   c) Citrate lyase
   d) HMG-CoA reductase
   Answer: b) Acetyl-CoA carboxylase
   Explanation: Acetyl-CoA carboxylase converts acetyl-CoA to malonyl-CoA, the committed step in fatty acid synthesis.

4. **Cholesterol is a precursor for which of the following?**
   a) Bile acids
   b) Steroid hormones
   c) Vitamin D
   d) All of the above
   Answer: d) All of the above
   Explanation: Cholesterol is converted into bile acids (e.g., cholic acid), steroid hormones (e.g., cortisol), and vitamin D (cholecalciferol).

5. **Which disorder is characterized by high blood cholesterol levels?**
   a) Hypercholesterolemia
   b) Fatty liver
   c) Obesity
   d) Ketoacidosis
   Answer: a) Hypercholesterolemia
   Explanation: Hypercholesterolemia involves elevated blood cholesterol, increasing the risk of atherosclerosis.

6. **The urea cycle primarily occurs in which organ?**
   a) Kidney
   b) Liver
   c) Brain
   d) Pancreas
   Answer: b) Liver
   Explanation: The urea cycle, detoxifying ammonia into urea, occurs in the liver's mitochondria and cytosol.

7. **Phenylketonuria (PKU) is caused by a deficiency of which enzyme?**
   a) Tyrosinase
   b) Phenylalanine hydroxylase
   c) Homogentisate oxidase
   d) Branched-chain ketoacid dehydrogenase
   Answer: b) Phenylalanine hydroxylase
   Explanation: PKU results from a deficiency in phenylalanine hydroxylase, leading to phenylalanine accumulation and neurological damage.

8. **Which neurotransmitter is synthesized from tyrosine?**
   a) Serotonin
   b) Dopamine
   c) Melatonin
   d) Histamine
   Answer: b) Dopamine
   Explanation: Dopamine is synthesized from tyrosine via L-DOPA in the catecholamine synthesis pathway.

9. **Hyperbilirubinemia is associated with which condition?**
   a) Jaundice
   b) Atherosclerosis
   c) Phenylketonuria
   d) Alkaptonuria
   Answer: a) Jaundice
   Explanation: Hyperbilirubinemia, elevated bilirubin levels, causes yellowing of skin and eyes in jaundice.

10. **Which reaction removes an amino group as ammonia from amino acids?**
    a) Transamination
    b) Deamination
    b) Decarboxylation

c) Deamination
Answer: b) Deamination
Explanation: Deamination removes an amino group as ammonia, often catalyzed by enzymes like glutamate dehydrogenase.

Long Answer Questions

(Answer 1 out of 3, 10 marks)

1. **Describe the β-oxidation pathway of palmitic acid, its energetics, and its significance.**
   Answer:
   β-Oxidation Pathway: β-Oxidation is the mitochondrial process of breaking down fatty acids into acetyl-CoA for energy production. For palmitic acid (16-carbon saturated fatty acid):
   - **Steps (per cycle)**:
     1. **Activation**: Palmitic acid is activated to palmitoyl-CoA (uses 2 ATP equivalents via acyl-CoA-CoA synthetase).
     2. **Oxidation**: Palmitoyl-CoA → trans-Δ^2-trans-Δ2-enoyl-CoA (by acyl-CoA-CoA dehydrogenase, produces 1 $FADH_2$).
     3. **Hydration**: Trans-Δ^2-trans-Δ2-enoyl-CoA → L-β-L-β-hydroxyacyl → CoA (by enoyl-CoA hydratase).
     4. **Oxidation**: L-β-L-β-hydroxyacyl → CoA → β-ketoacyl-CoA (by β-hydroxyacyl-CoA dehydrogenase, produces produces **1 NADH**).
     5. **Cleavage**: β-ketoacyl-CoA → acetyl-CoA + shortened acyl-CoA (by thiolase).
   - Palmitic acid (16C) undergoes **7 cycles**, producing **8 acetyl-CoA**, **7 $FADH_2$**, and **7 NADH**.
     Energetics:
   - **8 acetyl-CoA**: Each enters the TCA cycle, yielding 8 × 10 = **80 ATP** (via oxidative phosphorylation).
   - **7 NADH**: Each yields ~2.5 ATP → **17.5 ATP**.
   - **7 $FADH_2$**: Each yields ~1.5 ATP → **10.5 ATP**.
   - **Total**: 80 + 17.5 + + 10.5 = **108 ATP**.
   - **Net**: Subtract 2 ATP for activation → **106 ATP**.
     Significance:
   - **Energy Source**: Major energy source during fasting, yielding more ATP per gram than carbohydrates or proteins.
   - **Metabolic Flexibility**: Acetyl-CoA feeds into the TCA cycle or ketogenesis.

- **Regulation**: Activated by glucagon and inhibited by insulin, ensuring fatty acid oxidation during low glucose conditions.

2. **Explain the formation and utilization of ketone bodies, including the causes and consequences of ketoacidosis.**
   Answer:
   Formation (Ketogenesis): Ketone bodies are formed in the liver mitochondria during prolonged fasting, starvation, or uncontrolled diabetes.
   - **Steps**:
     1. Two acetyl-CoA molecules condense to form **acetoacetyl-CoA** (by thiolase).
     2. Acetoacetyl-CoA + acetyl-CoA → **HMG-CoA** (by HMG-CoA synthase, rate-limiting).
     3. HMG-CoA → **acetoacetate** (by HMG-CoA lyase).
     4. Acetoacetate is reduced to **β-hydroxybutyrate** (by β-hydroxybutyrate dehydrogenase) or spontaneously decarboxylated to **acetone**.
   - Primary ketone bodies: **acetoacetate**, **β-hydroxybutyrate** (most abundant), and **acetone** (volatile, exhaled).
     Utilization (Ketolysis):
   - Extrahepatic tissues (e.g., brain, muscle, heart) use ketone bodies during glucose scarcity.
   - **Steps**:
     1. β-Hydroxybutyrate → acetoacetate (by β-hydroxybutyrate dehydrogenase).
     2. Acetoacetate + succinyl-CoA → acetoacetyl-CoA (by succinyl-CoA:acetoacetate CoA transferase).
     3. Acetoacetyl-CoA → 2 acetyl-CoA (by thiolase), entering the TCA cycle for ATP production.
   - The brain adapts to use ketone bodies during prolonged fasting, sparing glucose.
     Ketoacidosis:
   - **Causes**: Excessive ketogenesis in uncontrolled diabetes mellitus (type 1) or prolonged starvation, due to high fatty acid oxidation and low insulin.
   - **Consequences**: Accumulation of acidic ketone bodies (acetoacetate, β-hydroxybutyrate) lowers blood pH (<7.35), causing metabolic acidosis. Symptoms include nausea, confusion, fruity breath (acetone), and, if untreated, coma or death.

- **Management**: Insulin administration and fluid/electrolyte correction to restore glucose metabolism and reduce ketogenesis.

3. **Discuss the urea cycle, its disorders, and the general reactions of amino acid metabolism.**
   Answer:
   General Reactions of Amino Acid Metabolism:

   - **Transamination**: Transfer of an amino group from an amino acid to a keto acid (usually α-ketoglutarate), forming a new amino acid and keto acid. Catalyzed by transaminases (e.g., ALT, AST) using pyridoxal phosphate. Example: Alanine + α-ketoglutarate → pyruvate + glutamate.
   - **Deamination**: Removal of an amino group as ammonia, often from glutamate, by glutamate dehydrogenase, producing α-ketoglutarate and NH_3. Ammonia enters the urea cycle.
   - **Decarboxylation**: Removal of a carboxyl group as CO_2, forming biogenic amines. Example: Histidine → histamine (by histidine decarboxylase).
     Urea Cycle: Converts toxic ammonia into urea in the liver (mitochondria and cytosol).
   - **Steps**:
     1. $NH_3 + CO_2 + 2$ ATP → carbamoyl phosphate (by carbamoyl phosphate synthetase I, in mitochondria).
     2. Carbamoyl phosphate + ornithine → citrulline (by ornithine transcarbamoylase).
     3. Citrulline + aspartate + ATP → argininosuccinate (by argininosuccinate synthetase, in cytosol).
     4. Argininosuccinate → arginine + fumarate (by argininosuccinate lyase).
     5. Arginine → urea + ornithine (by arginase), recycling ornithine.
   - **Energetics**: Requires 4 ATP equivalents per urea molecule (2 ATP in step 1, 2 high-energy bonds in step 3).
     Disorders:
   - **Hyperammonemia**: Deficiency in urea cycle enzymes (e.g., ornithine transcarbamoylase deficiency) leads to ammonia accumulation, causing neurological symptoms, coma, or death.
   - **Citrullinemia**: Deficiency in argininosuccinate synthetase, causing citrulline buildup and ammonia toxicity.
   - **Symptoms**: Lethargy, seizures, developmental delays; treated with low-protein diets and ammonia scavengers (e.g., sodium benzoate).

Significance: The urea cycle detoxifies ammonia, preventing toxicity, and integrates with amino acid catabolism to manage nitrogen waste.

Short Answer Questions

(Answer 2 out of 3, 5 marks each)

1. **Explain the de novo synthesis of palmitic acid and its regulation.**
   Answer:
   De Novo Synthesis of Palmitic Acid: Fatty acid synthesis occurs in the cytoplasm, producing palmitic acid (16:0) from acetyl-CoA.
   - **Steps**:
     1. Acetyl-CoA → malonyl-CoA (by acetyl-CoA carboxylase, rate-limiting, uses biotin and CO_2).
     2. Acetyl-CoA and malonyl-CoA are loaded onto fatty acid synthase (FAS), a multi-enzyme complex.
     3. FAS catalyzes four reactions per cycle: condensation, reduction, dehydration, and reduction, adding 2 carbons from malonyl-CoA.
     4. After 7 cycles, palmitic acid (16C) is released from FAS.
   - **Requirements**: NADPH (from HMP shunt), ATP, and acetyl-CoA (from citrate shuttle).
     Regulation:
   - **Activated by**: Insulin (promotes acetyl-CoA carboxylase activity), citrate (allosteric activator).
   - **Inhibited by**: Glucagon/epinephrine (via phosphorylation of acetyl-CoA carboxylase), palmitoyl-CoA (feedback inhibition), and low NADPH.
   - Occurs in fed states when glucose and energy are abundant, storing excess energy as fat.
2. **Describe the biological significance of cholesterol and its conversion into bile acids, steroid hormones, and vitamin D.**
   Answer:
   Biological Significance of Cholesterol:
   - **Membrane Component**: Cholesterol maintains membrane fluidity and forms lipid rafts for signaling.
   - **Precursor**: Serves as a precursor for bile acids, steroid hormones, and vitamin D.
   - **Signaling**: Modulates cellular processes via lipid-protein interactions.
     Conversions:

- **Bile Acids**: In the liver, cholesterol is hydroxylated to form primary bile acids (e.g., cholic acid, chenodeoxycholic acid) via 7α-hydroxylase. Bile acids emulsify dietary fats for absorption and are stored in the gallbladder.
- **Steroid Hormones**: In endocrine glands, cholesterol is converted to pregnenolone by cytochrome P450scc, then to hormones like cortisol (adrenal cortex), testosterone (testes), and estradiol (ovaries).
- **Vitamin D**: In skin, 7-dehydrocholesterol (cholesterol derivative) is converted to cholecalciferol (vitamin D3) upon UV exposure, regulating calcium and phosphate homeostasis.
  Significance: These derivatives are critical for digestion, endocrine function, and bone health.

3. **Discuss the catabolism of phenylalanine and tyrosine, including two associated metabolic disorders.**
   Answer:
   Catabolism of Phenylalanine and Tyrosine:
   - **Phenylalanine Catabolism**: Phenylalanine is hydroxylated to tyrosine by phenylalanine hydroxylase (uses tetrahydrobiopterin).
   - **Tyrosine Catabolism**:
     1. Tyrosine → p-hydroxyphenylpyruvate (by tyrosine aminotransferase).
     2. p-Hydroxyphenylpyruvate → homogentisate (by p-hydroxyphenylpyruvate dioxygenase).
     3. Homogentisate → maleylacetoacetate (by homogentisate oxidase).
     4. Maleylacetoacetate → fumarylacetoacetate (by maleylacetoacetate isomerase).
     5. Fumarylacetoacetate → fumarate + acetoacetate (by fumarylacetoacetate hydrolase), entering the TCA cycle or ketogenesis.
        Metabolic Disorders:
   - **Phenylketonuria (PKU)**: Deficiency in phenylalanine hydroxylase, causing phenylalanine accumulation, leading to intellectual disability, seizures, and hypopigmentation. Treated with a low-phenylalanine diet.
   - **Alkaptonuria**: Deficiency in homogentisate oxidase, causing homogentisate buildup, which oxidizes to alkapton, leading to dark urine, joint pain, and connective tissue pigmentation.
     Significance: Proper catabolism prevents toxic metabolite accumulation and supports energy production and biosynthesis.