

1. Which of the following is NOT a consequence of ATP depletion in an injured cell?

- A. Failure of energy-dependent membrane transport
- B. Increased intracellular pH due to lactic acid breakdown
- C. Detachment of ribosomes from the endoplasmic reticulum
- D. Reduced protein synthesis and lipogenesis

2. Which of the following enzymes converts hydrogen peroxide into hypochlorite (HOCL) in phagocytic leukocytes?

- A. Superoxide dismutase
- B. Catalase
- C. Myeloperoxidase
- D. Glutathione peroxidase

3. Which statement regarding ischemia-reperfusion injury is INCORRECT?

- A. It refers to additional cellular injury that can occur when blood flow is restored to ischemic tissue
- B. Damaged mitochondria and impaired antioxidant defenses can contribute to increased ROS production
- C. It protects cells by promoting the complete degradation of ROS before they can cause damage
- D. Infiltrating leukocytes can contribute to further tissue injury through inflammation and ROS production

4. Which mechanism best explains the decrease in intracellular pH during ATP depletion?

- A. Increased protein synthesis produces acidic byproducts
- B. Increased anaerobic glycolysis leads to lactic acid accumulation
- C. Increased oxidative phosphorylation causes excessive lactic acid production
- D. Failure of sodium transport directly releases hydrogen ions into the cytoplasm

5. Which of the following is an example of an indirect-acting toxin?

- A. Mercury binding to sulfhydryl groups of membrane proteins and disrupting ATP-dependent transport
- B. Carbon tetrachloride requiring metabolic activation to form reactive metabolites
- C. A chemotherapeutic agent that damages critical cellular components in malignant cells
- D. A substance that immediately disrupts membrane integrity without metabolic conversion

6. Which of the following is NOT an adaptive response to protein misfolding in the endoplasmic reticulum?

- A. Decreased protein translation
- B. Increased production of molecular chaperones
- C. Increased degradation of misfolded proteins
- D. Increased ribosome synthesis

7. Which of the following best describes the consequence of lysosomal membrane damage?

- A. Increased synthesis of membrane phospholipids
- B. Leakage of lysosomal enzymes, contributing to cellular digestion
- C. Release of cytochrome c to activate the mitochondrial apoptotic pathway
- D. Increased sodium pump activity, resulting in cell shrinkage

8. A child develops multiple malignancies due to an inherited defect in the cellular response to DNA damage. The affected cells fail to adequately arrest the cell cycle or eliminate cells with irreparable DNA damage. Which molecular abnormality most likely explains this finding?

- A. Increased activity of superoxide dismutase
- B. Mutation of the p53 tumor suppressor gene
- C. Increased production of endoplasmic reticulum chaperones
- D. Increased activity of glutathione peroxidase

9. A 58-year-old man experiences an acute myocardial infarction. Following successful restoration of coronary blood flow, additional myocardial injury is observed. Investigations reveal increased ROS production and infiltration of inflammatory cells into the affected tissue. Which mechanism best explains this finding?

- A. Reperfusion completely restores mitochondrial function, causing excessive ATP

- B. Reperfusion promotes ROS generation from damaged mitochondria and infiltrating leukocytes, while inflammation contributes to further tissue injury
- C. Reperfusion prevents leukocyte recruitment, allowing lysosomal enzymes to accumulate inside intact cells
- D. Restoration of blood flow prevents oxidative stress by immediately restoring all cellular antioxidant defenses

****10. A patient develops acute liver injury following exposure to carbon tetrachloride (CCl₄). The substance is metabolized by cytochrome P450 enzymes in the smooth endoplasmic reticulum. Subsequent examination reveals fatty changes in hepatocytes and decreased ATP levels. Which of the following is NOT an expected consequence of this injury?**

- A. Increased mitochondrial ATP production due to enhanced oxidative phosphorylation
- B. Formation of reactive metabolites that promote lipid peroxidation of endoplasmic reticulum membranes
- C. Detachment of ribosomes from the endoplasmic reticulum and reduced apoprotein synthesis
- D. Mitochondrial membrane damage, decreased ATP production, and eventual cell death

****11. A researcher investigates diseases associated with abnormal protein folding and accumulation. The findings suggest that misfolded proteins contribute to cellular dysfunction or extracellular deposits. Which of the following conditions is LEAST directly associated with protein misfolding?**

- A. Fatty liver
- B. Alzheimer's disease
- C. Type 2 diabetes mellitus
- D. Amyloidosis

****12. A patient develops severe cellular injury following prolonged hypoxia. Laboratory investigations reveal ATP depletion, loss of mitochondrial membrane potential, and increased ROS production. Which of the following outcomes is most consistent with mitochondrial dysfunction in this setting?**

- A. Increased oxidative phosphorylation to compensate for ATP depletion
- B. Release of cytochrome c from damaged mitochondria, potentially activating the intrinsic apoptotic pathway
- C. Stabilization of mitochondrial membranes and complete prevention of ROS formation
- D. Increased ATP-dependent sodium pump activity, preventing cellular swelling

1 B

2 C

3 C

4 B

5 B

6 D

7 B

8 B

9 B

10 A

11 A

12 B