LEC 5 Q- VESICLE

1. **What is the primary role of clathrin in the formation of transport vesicles?**

    A) To facilitate vesicle fusion

    B) To provide structural integrity and ensure proper targeting

    C) To degrade macromolecules

    D) To transport vesicles to the lysosomes

    Answer: B

2. **Which proteins are responsible for determining the membrane targets of vesicles?**

    A) SNARE proteins

    B) Clathrin proteins

    C) Rab proteins

    D) Motor proteins

    Answer: C

3. **What is the sequence of events in the vesicle fusion process?**

    A) Membrane fusion, SNARE disassembly, Rab binding

    B) Rab binding, SNARE complex formation, membrane fusion

    C) SNARE disassembly, vesicle docking, membrane fusion

    D) Clathrin uncoating, receptor binding, membrane fusion

    Answer: B

4. **What is a consequence of vesicle fusion when the vesicle contains secretory contents?**

    A) They remain in the Golgi apparatus

B) They are integrated into the cell membrane

C) They are released outside the cell

D) They are degraded in lysosomes

Answer: C

5. **Which genetic mutations are associated with Griscelli Syndrome?**

A) MYO5A, RAB27A, MLPH

B) SNARE, clathrin, Rab

C) Glucocerebrosidase, acid hydrolases, mannose

D) Lysosomal enzymes, G proteins, macrophages

Answer: A

6. **What is the primary function of lysosomes?**

A) Synthesize proteins

B) Degrade materials from inside and outside the cell

C) Transport vesicles to the Golgi apparatus

D) Store genetic material

Answer: B

7. **What leads to lysosomal storage diseases?**

A) Excessive production of lysosomal enzymes

B) Defects in lysosomal enzymes

C) Overactivity of Rab proteins

D) Increased membrane fluidity

Answer: B

8. **Which condition is characterized by defective targeting of lysosomal enzymes?**

A) Griscelli Syndrome

B) I-Cell Disease

C) Macropinocytosis

D) Autophagy

Answer: B

9. **What is the process of endocytosis?**

A) The release of materials from the cell

B) The uptake of molecules from outside the cell

C) The degradation of cellular components

D) The synthesis of membrane proteins

Answer: B

10. **In phagocytosis, what is formed when a bacterium is engulfed by a macrophage?**

A) A phagosome

B) An autophagosome

C) A lysosome

D) An early endosome

Answer: A

11. **Which process involves the degradation of cellular components for nutrient recycling during starvation?**

A) Endocytosis

B) Phagocytosis

C) Autophagy

D) Macropinocytosis

Answer: C

12. **What characterizes macropinocytosis?**

    A) Clathrin-dependent uptake of large particles

    B) Clathrin-independent uptake of fluids

    C) Fusion of phagosomes with lysosomes

    D) Transport of secretory vesicles

Answer: B

13. **Which of the following best describes the role of SNARE proteins in vesicle fusion?**

    A) They catalyze the hydrolysis of macromolecules within lysosomes.

    B) They create a clathrin coat around the vesicle.

    C) They mediate the interaction and fusion between vesicular and target membranes.

    D) They facilitate the transport of enzymes to the Golgi apparatus.

Answer: C

14. **In the context of vesicle trafficking, what is the significance of the Rab protein's interaction with its effector proteins?**

    A) It triggers the breakdown of vesicular contents.

    B) It enhances the fusion of vesicles with lysosomes.

    C) It facilitates the correct docking and tethering of vesicles to their target membranes.

    D) It maintains the acidic environment within lysosomes.

Answer: C

15. **What is the primary reason lysosomal enzymes remain inactive when released into the cytoplasm?**

    A) They require a specific substrate for activation.

B) The cytoplasmic pH is too high for their activity.

C) They are degraded by cytoplasmic proteins.

D) They are immediately transported back to the lysosome.

Answer: B

16. **Griscelli Syndrome's symptoms, such as pigmentary dilution and silver-grey hair, are primarily due to what underlying mechanism?**

A) Defective synthesis of melanin

B) Impaired transport of melanosomes

C) Increased degradation of keratinocytes

D) Overproduction of Rab proteins

Answer: B

17. **What type of genetic mutation is responsible for I-Cell Disease?**

A) Mutations in Rab proteins affecting vesicular transport

B) Defective enzyme mutations leading to lysosomal enzyme targeting failure

C) Mutations causing excess production of lysosomal enzymes

D) Changes in the structure of clathrin that prevent vesicle formation

Answer: B

18. **Which of the following best describes the process of clathrin-dependent endocytosis?**

A) It relies solely on passive diffusion of molecules into the cell.

B) It involves the binding of ligands to membrane receptors, leading to vesicle formation.

C) It exclusively transports large particles into the cell.

D) It occurs without any protein involvement.

Answer: B

19. **The formation of autophagosomes during autophagy primarily originates from which cellular structure?**

A) Golgi apparatus

B) Plasma membrane

C) Endoplasmic reticulum

D) Mitochondria

Answer: C

20. **Which statement about lysosomal storage diseases is true?**

A) They only affect the nervous system.

B) They are caused by defects in vesicular transport mechanisms.

C) Severity is consistent regardless of the type of enzyme defect.

D) They lead to the accumulation of undigested macromolecules due to enzyme deficiencies.

Answer: D

21. **What is the role of proton pumps in lysosomes?**

A) They synthesize lysosomal enzymes.

B) They maintain the acidic pH necessary for enzyme activity.

C) They transport substrates into the lysosome.

D) They facilitate vesicle fusion with target membranes.

Answer: B

22. **What happens during the acidic pH shift in early endosomes?**

A) Receptors are degraded to prevent recycling.

B) Ligands are released from their receptors, allowing recycling of receptors.

C) Vesicles are formed for transport to the Golgi apparatus.

D) Fusion with lysosomes is prevented.

Answer: B

23. **In macropinocytosis, which of the following is a key feature?**

A) Specificity in ligand-receptor interactions

B) Formation of small vesicles primarily for nutrient uptake

C) The engulfment of large particles through membrane extensions

D) Dependence on clathrin for vesicle formation

Answer: C

24. **Which condition would most likely result from a mutation affecting the phosphorylation of mannose in lysosomal enzymes?**

A) Griscelli Syndrome

B) I-Cell Disease

C) Gaucher Disease

D) Tay-Sachs Disease

Answer: B

25. **In the vesicle fusion process, which specific interaction initiates the physical proximity required for membrane fusion?**

A) Binding of clathrin to the vesicle membrane

B) Interaction between v-SNAREs and t-SNAREs

C) Docking of Rab proteins to tethering factors

D) Release of GTP from Rab proteins

Answer: B

26. **Given the role of Rab proteins in vesicular transport, which of the following statements is most accurate regarding their diversity?**

A) Each Rab protein can bind to any type of vesicle regardless of its cargo.

B) Over 60 distinct Rab proteins exist, each with unique combinations that determine vesicular identity and targeting.

C) All Rab proteins function independently of other cellular signaling pathways.

D) Rab proteins primarily facilitate lysosomal degradation of substrates.

Answer: B

27. **Which mechanism is primarily responsible for the containment of lysosomal enzymes within lysosomes, preventing cytotoxicity?**

A) The proton pump creating an acidic environment

B) The intrinsic stability of acid hydrolases at higher pH

C) The lipid bilayer of the lysosomal membrane

D) The active transport of substrates out of lysosomes

Answer: C

28. **In the context of I-Cell Disease, what is the specific biochemical defect leading to the failure of lysosomal enzyme targeting?**

A) Absence of the glucocerebrosidase enzyme

B) Mutation in the tagging enzyme responsible for mannose phosphorylation

C) Loss of SNARE protein function

D) Defective Rab protein interactions with effector proteins

Answer: B

29. **During autophagy, what is the fate of the double-membraned autophagosome after it fuses with a lysosome?**

A) It is recycled to the endoplasmic reticulum.

B) It undergoes degradation, releasing the nutrients for cellular use.

C) It is converted back into a vesicle for transport.

D) It remains intact, serving as a storage compartment.

Answer: B

30. **What distinguishes macropinocytosis from traditional receptor-mediated endocytosis?**

A) Macropinocytosis exclusively involves clathrin-coated pits.

B) Macropinocytosis does not rely on specific receptor-ligand interactions for uptake.

C) Macropinocytosis is limited to the uptake of large particles only.

D) Macropinocytosis is dependent on SNARE proteins for membrane fusion.

Answer: B

31. **Which of the following is the most critical step that occurs immediately after the fusion of a transport vesicle with its target membrane?**

A) The vesicle releases its contents into the cytoplasm.

B) The clathrin coat is reassembled for the next cycle of vesicle formation.

C) The SNARE complex undergoes disassembly.

D) The Rab proteins dissociate from the target membrane.

Answer: C

32. **How do lysosomal storage diseases typically impact cellular function beyond the accumulation of undigested macromolecules?**

A) They primarily disrupt mitochondrial function.

B) They result in secondary effects on cell signaling pathways and metabolic processes.

C) They only affect the lysosome without broader implications.

D) They enhance the efficiency of cellular metabolism.

Answer: B

33. **In the context of glucocerebroside metabolism, which pathway is most likely disrupted in Gaucher disease?**

A) The synthesis of ceramide from sphingolipids

B) The degradation of glucocerebroside within lysosomes

C) The recycling of lipid membranes during autophagy

D) The transport of glucocerebroside to the endoplasmic reticulum

Answer: B

34. **Which of the following best explains why defects in lysosomal enzyme targeting lead to systemic symptoms rather than localized effects?**

A) Lysosomal enzymes are only required in specific tissues.

B) Accumulation of substrates affects multiple cell types and organ systems.

C) Lysosomal enzymes are redundant, and their absence can be compensated by other enzymes.

D) The immune system exclusively manages the consequences of enzyme defects.

Answer: B

35. **In the vesicle trafficking process, how do alterations in Rab protein function potentially impact the entire intracellular transport system?**

A) They exclusively affect lysosomal enzyme activity.

B) They can disrupt the entire cargo delivery mechanism, leading to mislocalization of multiple proteins.

C) They enhance the activity of SNARE proteins, improving fusion efficiency.

D) They only affect the degradation pathways without impacting transport.

Answer: B

36. **Which specific structural characteristic of clathrin-coated vesicles is critical for their function during vesicle budding?**

A) Their lipid bilayer composition

B) The geometric arrangement of clathrin triskelions that forms a basket-like structure

C) The presence of specific phospholipids on the cytosolic side of the membrane

D) Their interaction with cytoskeletal elements for movement

Answer: B

37. **What is the molecular mechanism by which lysosomal enzymes are activated once they reach the lysosome?**

A) They undergo proteolytic cleavage at neutral pH.

B) They require phosphorylation of mannose residues to become active.

C) They are activated by the acidic environment and conformational changes.

D) They require co-factors derived from cytosolic proteins.

Answer: C

38. **In Griscelli Syndrome, the impaired transport of melanosomes results in which of the following cellular consequences?**

A) Increased apoptosis of melanocytes

B) Accumulation of melanin within melanocytes, leading to cellular dysfunction

C) Enhanced phagocytosis of surrounding keratinocytes

D) Loss of pigmentation in keratinocytes due to increased transport to the skin

Answer: B

39. **What critical role do tethering factors play in the process of vesicle targeting?**

A) They assist in the docking of vesicles without fusion.

B) They directly catalyze the enzymatic degradation of vesicular contents.

C) They modulate the interaction between Rab proteins and SNARE complexes.

D) They stabilize the clathrin coat during vesicle formation.

Answer: A

40. **How does the mechanism of autophagy contribute to cellular homeostasis under nutrient-deprived conditions?**

A) It prevents apoptosis by blocking lysosomal degradation pathways.

B) It recycles organelles and macromolecules to provide essential nutrients for survival.

C) It increases the synthesis of lysosomal enzymes to enhance degradation.

D) It facilitates the exocytosis of waste materials to the extracellular space.

Answer: B

41. **What specific biochemical changes occur to SNARE proteins following the fusion of a vesicle with its target membrane?**

A) They undergo glycosylation, enhancing their affinity for cargo.

B) They are proteolytically cleaved to prevent subsequent fusion events.

C) They form a stable complex that remains attached to the target membrane.

D) They dissociate to facilitate the recycling of the vesicle components.

Answer: D

42. **In the context of lysosomal storage diseases, why do mutations causing partial enzyme defects generally lead to milder symptoms compared to total enzyme loss?**

A) Partial defects allow for some residual enzymatic activity, which can partially degrade substrates.

B) They primarily affect only one organ system rather than being systemic.

C) Partial defects are often compensated by alternative metabolic pathways.

- D) They trigger compensatory mechanisms in adjacent cells.

Answer: A

43. **What is the significance of the acidic pH maintained in lysosomes with respect to enzymatic activity and cellular homeostasis?**

A) It protects lysosomal contents from proteolytic degradation.

B) It enhances the solubility of hydrophobic substrates.

C) It activates acid hydrolases while deactivating harmful cytoplasmic enzymes.

D) It facilitates the export of lysosomal enzymes to the extracellular space.

Answer: C

44. **How do defects in the tagging enzyme for mannose phosphorylation specifically affect lysosomal enzyme transport?**

A) They prevent the synthesis of lysosomal enzymes altogether.

B) They lead to the mislocalization of enzymes to the extracellular space.

C) They disrupt the interaction between vesicles and lysosomal membranes.

D) They result in an inability to recognize lysosomal enzymes, causing their secretion instead of transport.

Answer: D

45. **In the context of phagocytosis, what distinguishes the formation of the phagolysosome from the formation of autophagosomes?**

A) Phagolysosomes involve extracellular material, while autophagosomes degrade intracellular components.

B) Phagolysosomes are formed only in immune cells, whereas autophagosomes can be formed in any cell type.

C) Phagolysosomes require clathrin-coated vesicles, while autophagosomes do not.

D) Phagolysosomes are not involved in nutrient recycling, unlike autophagosomes.

Answer: A

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1. **Case: A 10-year-old boy presents with developmental delays, distinct facial features, and skeletal abnormalities. Genetic testing reveals a mutation affecting the phosphorylation of mannose residues on lysosomal enzymes. What is the most likely diagnosis?**

A) Griscelli Syndrome

B) I-Cell Disease

C) Gaucher Disease

D) Tay-Sachs Disease

Answer: B

2. **Case: A researcher is investigating a novel compound that inhibits clathrin-mediated endocytosis. Which of the following outcomes is the most direct result of this inhibition?**

A) Increased secretion of lysosomal enzymes

B) Decreased uptake of extracellular ligands

C) Enhanced fusion of vesicles with lysosomes

D) Increased recycling of membrane receptors

Answer: B

3. **Case: A 5-year-old girl with a rare genetic disorder presents with pigmentary dilution of the skin and silver-grey hair. Genetic analysis shows defects in the MYO5A gene. Which of the following best explains her symptoms?**

   A) Defective synthesis of melanin in melanocytes

   B) Impaired transport of melanosomes from melanocytes to keratinocytes

   C) Increased degradation of melanin by lysosomes

   D) Overproduction of melanin in keratinocytes

   Answer: B

4. **Case: A study finds that a mutation in a Rab protein results in misdirected vesicle transport within a cell. Which of the following cellular functions is most likely to be impaired as a direct consequence?**

   A) Protein synthesis in the endoplasmic reticulum

   B) Targeted delivery of lysosomal enzymes to lysosomes

   C) Fusion of vesicles with the plasma membrane

   D) Recycling of membrane receptors

   Answer: B

5. **Case: An adult patient is diagnosed with a lysosomal storage disease and exhibits symptoms of severe psychomotor retardation. Which mechanism is most likely responsible for these symptoms?**

   A) Accumulation of unprocessed substrates due to enzyme deficiencies

   B) Increased activity of lysosomal enzymes in the cytoplasm

   C) Enhanced fusion of lysosomes with other organelles

   D) Overproduction of lysosomal membranes

Answer: A

6. **Case: A 15-year-old boy is diagnosed with a genetic condition characterized by defective transport of melanosomes. He shows symptoms of hypopigmentation and silver-grey hair. Which specific gene mutation is most likely involved in this condition?**

    A) RAB27A

    B) MYO5A

    C) MLPH

    D) GBA

Answer: A

7. **Case: A researcher discovers a drug that enhances the activity of SNARE proteins. What is the most likely effect of this drug on vesicular transport?**

    A) Increased mislocalization of vesicular contents

    B) Enhanced specificity of vesicle docking and fusion

    C) Decreased efficiency of endocytosis

    D) Inhibition of lysosomal degradation

Answer: B

8. **Case: An adult develops an autoimmune disorder with symptoms linked to defective endocytosis of insulin. Which cellular mechanism is likely impaired in this individual?**

    A) SNARE-mediated vesicle fusion

    B) Clathrin-mediated receptor recycling

    C) Autophagosome formation

    D) Lysosomal enzyme activation

Answer: B

9. **Case: A researcher studies a new therapy targeting Rab proteins to improve lysosomal function in a model of storage disease. Which outcome would indicate successful therapy?**

    A) Decreased fusion of lysosomes with autophagosomes

    B) Enhanced delivery of lysosomal enzymes to their correct compartments

    C) Increased levels of unprocessed substrates in lysosomes

    D) Impaired recycling of damaged organelles

    Answer: B

10. **Case: An experiment shows that a mutation in the clathrin coat protein prevents the formation of clathrin-coated vesicles. What is the most immediate cellular consequence of this mutation?**

    A) Decreased membrane recycling

    B) Increased phagocytosis

    C) Enhanced lysosomal degradation

    D) Impaired exocytosis of secretory vesicles

    Answer: A

11. **Case: A patient with a lysosomal storage disease experiences organ dysfunction due to the accumulation of undigested substrates. Which of the following pathways is most directly affected by the underlying enzyme deficiency?**

    A) Glycolipid metabolism

    B) Protein synthesis in the cytosol

    C) Autophagy of damaged organelles

    D) Receptor-mediated endocytosis

    Answer: A

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