LEC 11 Q CYTOKOGY:

1. Which of the following is primarily responsible for the initial interaction between leukocytes and endothelial cells during inflammation?

A) Integrins
B) Cadherins
C) Selectins
D) Claudins

2. Which of the following is a key function of E-cadherins in epithelial cells?

A) Prevents the migration of mesenchymal cells
B) Connects actin filaments between adjacent cells
C) Stabilizes the intermediate filaments of cells
D) Promotes the passage of ions between cells

3. Which of the following cell adhesion molecules recognizes carbohydrates on the surface of other cells?

A) Selectins
B) Integrins
C) Cadherins
D) ICAMs

4. What is the role of integrins in leukocyte-endothelial cell interaction?

A) They mediate the initial weak adhesion between leukocytes and endothelial cells
B) They recognize carbohydrates on endothelial cells
C) They promote stable interactions with ICAMs on endothelial cells
D) They prevent the migration of leukocytes into tissues

5. Which of the following types of cell junctions is primarily involved in maintaining the polarity of cells by separating the apical and basolateral domains?

A) Adherens junctions
B) Desmosomes
C) Tight junctions
D) Gap junctions

6. What happens to cells that lose E-cadherin expression in the context of cancer metastasis?

A) Cells become more adhesive and less migratory
B) Cells adopt a mesenchymal-like phenotype and become more motile
C) Cells form stable junctions and remain in place
D) Cells undergo apoptosis and stop dividing

7. Which cell adhesion molecule family is primarily responsible for the formation of focal adhesions and hemidesmosomes?

A) Cadherins
B) Integrins

C) Selectins
D) Members of the Ig superfamily

8. Which of the following is NOT true about gap junctions?

A) They allow the passage of ions and small molecules between adjacent cells
B) Connexons are composed of six connexin proteins
C) They are involved in the direct transmission of electrical signals in nerve cells
D) They allow the passage of proteins and nucleic acids between cells

9. Desmosomes connect intermediate filaments of adjacent cells. Which of the following is true about desmosomal cadherins?

A) They are involved in homophilic interactions with cadherins
B) They link actin filaments between adjacent cells
C) They interact with proteins that bind to the extracellular matrix
D) They contribute to the formation of tight junctions

10. Which of the following junctions forms a barrier that prevents the passage of molecules, including ions, between epithelial cells?

A) Gap junctions
B) Adherens junctions
C) Desmosomes
D) Tight junctions

11. Which disease is commonly associated with mutations in connexins, leading to defective gap junctions?

A) Marie-Charcot-Tooth disease
B) Cystic fibrosis
C) Duchenne muscular dystrophy
D) Hemophilia

12. The interaction between cadherins in adherens junctions involves binding to which of the following components inside the cell?

A) Microtubules
B) Actin filaments
C) Intermediate filaments
D) Connexons

13. Which of the following adhesion molecules are involved in homophilic interactions?

A) Selectins
B) Integrins
C) Cadherins
D) ICAMs

14. In which junction do desmosomal cadherins link cells via intermediate filaments?

A) Adherens junctions
B) Tight junctions

C) Focal adhesions
D) Desmosomes

15. What is the primary function of tight junctions in epithelial tissues?

A) Provide structural support to cells through intermediate filaments
B) Facilitate the passage of small molecules between adjacent cells
C) Seal spaces between cells and prevent molecule passage
D) Link actin filaments between cells

16. In carcinoma in situ, how do cells behave in relation to cadherins?

A) Cells exhibit reduced cadherin expression, become elongated, and are motile
B) Cells exhibit increased cadherin expression, remain in place, and do not migrate
C) Cells do not express cadherins at all, leading to metastasis
D) Cells only express desmosomal cadherins, forming stable junctions

17. What is the relationship between cadherins and catenins in adherens junctions?

A) Catenins directly bind to intermediate filaments to strengthen adhesion
B) Catenins interact with actin filaments through cadherins, contributing to junction stability
C) Catenins block cadherin interactions to prevent junction formation
D) Catenins mediate the transport of cadherins to the plasma membrane

18. Which of the following is a characteristic feature of claudin-low breast cancer?

A) High expression of E-cadherin
B) A mesenchymal-like phenotype with poor prognosis
C) Increased cell-cell adhesion via tight junctions
D) Low expression of connexins in tumor cells

19. Which cell junction is typically found in the apical domain of epithelial cells and plays a key role in separating external spaces?

A) Desmosomes
B) Focal adhesions
C) Tight junctions
D) Gap junctions

20. Which of the following is true about the formation of gap junctions?

A) They involve the assembly of homomeric connexons from the same type of connexins
B) They allow the passage of nucleic acids between adjacent cells
C) Connexons are composed of four transmembrane proteins
D) Gap junctions form by a single connexon connecting the cytoplasm of two cells

Answers:

1. **C**
2. **B**
3. **A**
4. **C**
5. **C**
6. **B**
7. **B**
8. **D**
9. **A**

10. **D**
11. **A**
12. **B**
13. **C**
14. **D**
15. **C**
16. **B**
17. **B**
18. **B**
19. **C**
20. **A**

21. Which of the following is true about the structure and function of cadherins in adherens junctions?

A) Cadherins form heterophilic interactions with other types of cadherins in neighboring cells
B) Cadherins interact with actin filaments and provide structural support to cell junctions
C) Cadherins are responsible for sealing the spaces between adjacent epithelial cells
D) Cadherins form gap junctions by interacting with connexins

22. In the context of leukocyte-endothelial cell interaction, what happens after leukocytes slow down due to selectin-carbohydrate interactions?

A) Leukocytes extravasate into the tissue immediately
B) Leukocytes increase the expression of ICAMs on their surface
C) Integrins on leukocytes interact with ICAMs on endothelial cells
D) The interaction is reversed, and leukocytes resume fast movement

23. Which of the following statements is correct regarding the role of integrins in cell adhesion?

A) Integrins link cells to each other via cadherins
B) Integrins connect cells to the extracellular matrix through focal adhesions
C) Integrins promote the formation of tight junctions between cells
D) Integrins are involved in homophilic interactions between similar cell types

24. Which type of cadherin is typically found in epithelial cells?

A) E-cadherin
B) N-cadherin
C) P-cadherin
D) Desmosomal cadherins

25. What is the role of desmosomal cadherins in desmosomes?

A) They link actin filaments between adjacent cells
B) They mediate the homophilic interaction between cadherins
C) They connect intermediate filaments of adjacent cells, providing mechanical strength
D) They form channels between cells for direct communication

26. What is the function of gap junctions in tissues?

A) To mediate adhesion between cells by linking actin filaments
B) To allow the passage of ions, small molecules, and signaling molecules between adjacent cells
C) To form a barrier that prevents the movement of ions and small molecules between cells
D) To stabilize the intermediate filaments of cells during cell division

27. How do mutations in connexins affect cellular function?

A) They enhance the formation of gap junctions
B) They may disrupt the assembly of connexons and prevent gap junction formation
C) They lead to an increase in the activity of cadherins
D) They cause cells to migrate and spread, similar to cancer metastasis

28. Which of the following best describes the type of interaction between integrins and ICAMs during leukocyte adhesion?

A) Homophilic interaction
B) Heterophilic interaction
C) Both are involved in the formation of gap junctions
D) Both are responsible for cell migration during metastasis

29. What is the role of catenins in adherens junctions?

A) Catenins form the core structure of gap junctions
B) Catenins link cadherins to actin filaments, contributing to the stability of the junctions
C) Catenins facilitate the translocation of integrins to the plasma membrane
D) Catenins mediate the attachment of desmosomal cadherins to intermediate filaments

30. Which of the following is NOT a feature of tight junctions?

A) Tight junctions are located in the apical domain of epithelial cells
B) Tight junctions help maintain cell polarity by separating apical and basolateral proteins
C) Tight junctions allow the passage of large proteins and nucleic acids between cells
D) Tight junctions prevent the free passage of ions and small molecules between cells

31. What is the primary consequence of defective gap junctions in tissues?

A) Disruption of tissue-specific connexin expression
B) Decreased cell-cell adhesion leading to tissue breakdown
C) Disruption of the passage of ions and small molecules between cells
D) Enhanced communication between cells leading to cancer metastasis

32. Which of the following best describes the role of cadherins in development and differentiation?

A) Cadherins facilitate cell migration and metastasis during development
B) Cadherins mediate the formation of stable junctions between cells in tissues
C) Cadherins prevent the formation of adherens junctions in epithelial tissues
D) Cadherins promote the loss of epithelial properties and enhance mesenchymal behavior

33. What is the significance of E-cadherin loss in carcinoma in situ (a tumor mass)?

A) E-cadherin loss causes cells to become more adhesive and less migratory
B) E-cadherin loss allows tumor cells to migrate and metastasize
C) E-cadherin loss leads to cell death and prevents cancer progression
D) E-cadherin loss causes cells to become more stable and resistant to cancer treatment

34. What do desmosomal cadherins specifically interact with to provide structural support in desmosomes?

A) Actin filaments
B) Intermediate filaments
C) Extracellular matrix proteins
D) Connexons

35. How does the interaction between selectins and carbohydrates impact the behavior of leukocytes during inflammation?

A) It allows leukocytes to exit the blood vessels and enter tissues
B) It enhances the adhesion of leukocytes to the endothelial cells
C) It promotes the migration of leukocytes into tissues
D) It initiates the binding of integrins to ICAMs on the endothelial cells

36. Which of the following junctions is directly responsible for preventing the passage of small molecules and ions between epithelial cells?

A) Tight junctions
B) Gap junctions
C) Desmosomes
D) Focal adhesions

37. What distinguishes gap junctions from other types of cell-cell junctions?

A) They connect cells via cadherin interactions
B) They allow direct communication between adjacent cells through connexons
C) They prevent the movement of molecules between adjacent cells
D) They provide structural support by linking intermediate filaments

38. In what way do desmosomes contribute to the mechanical strength of tissues?

A) By allowing the passage of ions and molecules between cells
B) By forming strong adhesive connections between cells via actin filaments
C) By linking intermediate filaments of adjacent cells, providing structural integrity
D) By forming tight seals between cells to prevent molecule leakage

39. Which of the following is a characteristic of "claudin-low" breast cancer cells?

A) Cells exhibit strong adhesion through tight junctions
B) Cells are characterized by a mesenchymal-like, motile phenotype
C) Cells express high levels of E-cadherin
D) Cells exhibit tight junction formation between tumor cells

40. Which adhesion molecule family is responsible for the formation of hemidesmosomes?

A) Selectins
B) Integrins
C) Cadherins
D) Members of the Ig superfamily

Answers:

21. **B**
22. **C**
23. **B**
24. **A**
25. **C**
26. **B**
27. **B**
28. **B**
29. **B**
30. **C**
31. **C**
32. **B**
33. **B**
34. **B**
35. **A**
36. **A**
37. **B**
38. **C**
39. **B**
40. **B**

41. Which of the following best explains how mutations in cadherins could affect tissue morphogenesis during development?

A) Mutations in cadherins could lead to the disassembly of tight junctions, causing leakage between cells
B) Mutations in cadherins may cause a loss of homophilic interactions, resulting in impaired cell-cell adhesion and improper tissue organization
C) Mutations in cadherins could cause aberrant expression of extracellular matrix proteins, leading to faulty adhesion to the ECM
D) Mutations in cadherins could promote excessive cell proliferation and contribute to tumor formation

42. What is the primary molecular event that allows leukocytes to extravasate during inflammation, based on the interaction with endothelial cells?

A) The activation of integrins, allowing leukocytes to firmly adhere to the endothelial surface
B) The binding of selectins to carbohydrates on endothelial cells, causing leukocytes to slow down
C) The breakdown of tight junctions between endothelial cells, allowing passage of leukocytes
D) The activation of desmosomal cadherins, allowing endothelial cells to loosen and permit leukocyte passage

43. In the context of cancer metastasis, why is the loss of E-cadherin considered a critical step in the epithelial-to-mesenchymal transition (EMT)?

A) Loss of E-cadherin results in decreased cell motility, preventing metastasis
B) Loss of E-cadherin leads to the stabilization of tight junctions, inhibiting cell movement
C) Loss of E-cadherin weakens cell-cell adhesion, facilitating the detachment and migration of tumor cells
D) Loss of E-cadherin prevents the formation of gap junctions, impairing intercellular communication

44. In the leukocyte-endothelial cell interaction, why is the heterophilic interaction between selectins and carbohydrates considered essential for the initiation of inflammation?

A) It allows leukocytes to form stable junctions with endothelial cells, preventing extravasation
B) It initiates the activation of integrins on leukocytes, enabling firm adhesion to the endothelial cells
C) It triggers a cascade of cytokine release from endothelial cells, recruiting more immune cells
D) It slows down the movement of leukocytes, allowing them to be guided by integrins to the site of inflammation

45. How does clustering of cadherins at adherens junctions contribute to tissue stability and integrity?

A) Clustering of cadherins strengthens the physical connection between cells, reducing the chance of mechanical rupture
B) Clustering of cadherins promotes the recruitment of intermediate filaments, enhancing the structural stability of the junctions
C) Clustering of cadherins initiates the formation of gap junctions between adjacent cells, allowing for direct communication
D) Clustering of cadherins induces the phosphorylation of catenins, promoting cell proliferation and tissue growth

46. What would be the effect of overexpression of claudin proteins in epithelial cells in the context of tight junction formation?

A) It would enhance the permeability of the epithelial barrier by promoting the leakage of ions and small molecules
B) It would lead to the loss of epithelial polarity, disrupting the apical-basolateral segregation of proteins
C) It would strengthen the epithelial barrier by increasing the tightness of the junctions and reducing paracellular transport
D) It would weaken the tight junctions, allowing for easier passage of large molecules between cells

47. Given that gap junctions are involved in direct intercellular communication, which of the following diseases could be exacerbated by impaired connexin function?

A) Epithelial cancers, due to reduced cell-cell communication and failure to prevent cellular transformation
B) Cardiomyopathies, because the failure of gap junctions disrupts electrical coupling between cardiac cells, leading to arrhythmias
C) Pulmonary fibrosis, as gap junctions fail to regulate the flow of ions and small molecules, promoting tissue scarring
D) Alzheimer's disease, where reduced gap junction communication in glial cells inhibits neuronal support and plasticity

48. Which statement best describes the role of focal adhesions in tissue mechanics and cellular migration?

A) Focal adhesions mediate homophilic interactions between cells, allowing for stable tissue cohesion
B) Focal adhesions connect integrins in the cell membrane to actin filaments, allowing cells to anchor to the extracellular matrix and migrate
C) Focal adhesions are essential for the formation of tight junctions, preventing the passage of molecules between cells
D) Focal adhesions are primarily involved in linking intermediate filaments to cadherins at desmosomes for structural integrity

49. In the development of clauidin-low breast cancer, which characteristic phenotype is most commonly observed?

A) Increased epithelial cell-cell adhesion, preventing tumor spread
B) A more motile, mesenchymal-like phenotype that allows cells to invade and metastasize
C) Stable epithelial architecture with no loss of cell junctions or polarity
D) Enhanced expression of connexins, promoting gap junction-mediated communication

50. How do integrins contribute to the structural integrity of tissues, particularly in focal adhesions and hemidesmosomes?

A) Integrins link the actin cytoskeleton to the extracellular matrix, providing a stable anchor for cells within the tissue
B) Integrins promote cell-cell adhesion through cadherin interactions, ensuring tissue cohesion
C) Integrins initiate the formation of tight junctions, preventing molecule passage between cells
D) Integrins form gap junctions by interacting with connexins, allowing direct intercellular communication

51. What is the primary molecular mechanism behind desmosomal cadherins providing mechanical support to tissues?

A) They mediate strong homophilic interactions between cells, linking their intermediate filaments
B) They facilitate communication between cells by allowing ions and small molecules to pass through gap junctions
C) They anchor cadherins to the extracellular matrix, contributing to tissue rigidity
D) They recruit catenins to the plasma membrane, stabilizing tight junctions between adjacent cells

52. How would a defect in desmosomal cadherins most likely affect epithelial tissue?

A) It would disrupt the formation of adherens junctions, weakening cell-cell adhesion and promoting tissue instability
B) It would lead to the accumulation of intermediate filaments, causing cell overgrowth and tumor formation
C) It would reduce the mechanical strength of epithelial tissues, leading to increased susceptibility to mechanical stress and tissue rupture
D) It would cause the disintegration of gap junctions, impairing intercellular communication and leading to tissue dysfunction

53. What role do catenins play in the regulation of adherens junctions and cellular signaling?

A) Catenins bind to cadherins to stabilize adherens junctions and provide a link to the cytoskeleton, influencing cell shape and signaling pathways
B) Catenins promote the breakdown of tight junctions, facilitating the passage of molecules between cells
C) Catenins inhibit the activity of gap junctions, preventing communication between adjacent cells
D) Catenins recruit integrins to the plasma membrane, mediating focal adhesion formation

54. Which cellular process is directly facilitated by selectin-carbohydrate interactions in the early stages of inflammation?

A) Leukocyte activation and immune response modulation
B) Leukocyte rolling and slow-down on the endothelial surface, allowing for stable interactions with integrins
C) Endothelial cell migration and tissue repair during injury
D) Integrin clustering and formation of focal adhesions in leukocytes

55. What is the primary function of cadherin-catenin complexes at adherens junctions in terms of cellular behavior during development?

A) They prevent the migration of cells, ensuring that tissues remain in a stable state
B) They promote the formation of gap junctions, facilitating intercellular communication during development
C) They regulate the mechanical strength of tissues by anchoring cells to extracellular matrix proteins
D) They regulate the behavior of cells by transmitting mechanical and chemical signals that govern differentiation and morphogenesis

Answers:

41. **B**
42. **C**
43. **C**
44. **B**
45. **B**
46. **C**
47. **B**
48. **B**
49. **B**
50. **A**
51. **A**
52. **C**
53. **A**
54. **B**
55. **D**

56. In the context of cancer metastasis, what role do cadherins play in preventing the migration of tumor cells?

A) Cadherins promote the formation of tight junctions, which block the migration of cancer cells
B) Cadherins maintain cell-cell adhesion, and their loss enables epithelial cells to transition to a mesenchymal-like, motile phenotype, aiding metastasis
C) Cadherins create mechanical barriers that prevent cancer cells from detaching from the primary tumor site
D) Cadherins suppress the expression of integrins, preventing cell migration and reducing metastasis

57. Why might a deficiency in E-cadherin lead to increased tumor aggressiveness in carcinoma in situ?

A) Loss of E-cadherin destabilizes the cytoskeleton, leading to a more rigid and non-migratory phenotype
B) Loss of E-cadherin allows for the detachment of cancer cells from the primary tumor, facilitating invasion into surrounding tissues
C) E-cadherin promotes the assembly of gap junctions that prevent cancer cells from invading adjacent tissues
D) E-cadherin loss enhances the formation of desmosomes, strengthening cell adhesion and preventing migration

58. Selectins on leukocytes interact with endothelial cell carbohydrates in the early stages of inflammation. What is the significance of this interaction in terms of immune response regulation?

A) Selectins initiate a signaling cascade in endothelial cells, leading to leukocyte adhesion and activation
B) Selectins cause endothelial cells to downregulate integrins, preventing excessive leukocyte adhesion
C) Selectins slow down leukocytes, allowing them to roll along the endothelial surface and eventually bind integrins for stable adhesion
D) Selectins enhance the formation of tight junctions between endothelial cells, preventing leukocyte extravasation

59. In epithelial tissues, how do adherens junctions and desmosomes work together to maintain tissue integrity under mechanical stress?

A) Adherens junctions stabilize epithelial cells by anchoring them to intermediate filaments, while desmosomes anchor cells to the extracellular matrix
B) Adherens junctions link actin filaments between cells, providing flexibility, while desmosomes link intermediate filaments, giving the tissue mechanical strength
C) Adherens junctions and desmosomes both work to seal the paracellular space, preventing leakage between cells under pressure
D) Adherens junctions bind to tight junctions, while desmosomes mediate cell-cell communication

60. What molecular changes are involved when cadherins undergo endocytosis during epithelial-mesenchymal transition (EMT), and how does this process contribute to cancer progression?

A) Cadherins are internalized, leading to the loss of tight junctions and the enhancement of cell-cell adhesion, promoting metastasis
B) The endocytosis of cadherins weakens cell-cell adhesion, allowing cells to become motile and invade surrounding tissues, a key step in metastasis
C) Internalized cadherins strengthen cell-cell adhesion, preventing the loss of epithelial characteristics and reducing the likelihood of cancer metastasis
D) Cadherin endocytosis inhibits cell migration by maintaining tight junctions and promoting a stationary phenotype

61. Gap junctions are responsible for intercellular communication, but what impact would defective connexins have on cardiac function?

A) Defective connexins would enhance electrical coupling between cardiac cells, improving heart contraction
B) Defective connexins would impair the transmission of electrical signals between cardiac cells, leading to arrhythmias and abnormal heart rhythms
C) Defective connexins would have no impact on cardiac function since heart cells do not rely on gap junctions for electrical signaling
D) Defective connexins would disrupt the formation of adherens junctions, compromising the structural integrity of cardiac muscle tissue

62. Why are integrins important in the process of cell migration, particularly in the context of wound healing?

A) Integrins facilitate the formation of tight junctions between migrating cells, stabilizing their position
B) Integrins link the extracellular matrix to the actin cytoskeleton, allowing cells to move through tissues during wound repair
C) Integrins prevent cell migration by promoting adhesion to the basement membrane, halting the healing process
D) Integrins enhance the formation of gap junctions, enabling direct communication between cells to coordinate migration during healing

63. What is the relationship between cadherins and actin filaments in adherens junctions, and how does this interaction contribute to cell shape regulation?

A) Cadherins bind directly to actin filaments, regulating cell shape by anchoring cells to the extracellular matrix
B) Cadherins interact with actin filaments through catenins, helping to maintain the shape and tension of the cell membrane during tissue formation
C) Cadherins and actin filaments are not involved in cell shape regulation; instead, they regulate cell division during development
D) Cadherins bind to intermediate filaments, contributing to cell shape changes in response to mechanical stress

64. How do tight junctions influence the development of Claudin-low breast cancer, and what does this imply for potential therapeutic strategies?

A) Low expression of Claudin proteins in tight junctions leads to enhanced epithelial integrity, preventing metastasis
B) Low expression of Claudin proteins destabilizes tight junctions, allowing cells to become more mesenchymal-like and enabling migration and metastasis
C) Tight junctions enhance tight cell-cell adhesion, preventing Claudin-low breast cancer cells from proliferating or invading tissues
D) Claudin proteins, when overexpressed, strengthen tight junctions, inhibiting cancer cell invasion into the bloodstream

65. In the gap junction communication system, how does the heteromeric composition of connexons influence their channel properties?

A) Homomeric connexons are more efficient at passing large molecules, while heteromeric connexons are specialized for small molecules and ions
B) Heteromeric connexons allow for greater flexibility in channel opening, which helps regulate ion flow and signal transmission between cells
C) Homomeric connexons are more stable, leading to faster communication between cells, while heteromeric connexons slow down signaling
D) The composition of connexons does not affect their function, as all connexons pass the same size molecules regardless of their structure

66. What role do desmosomes play in the mechanical stability of tissues under stress, and how could their dysfunction lead to disease?

A) Desmosomes link actin filaments between cells, providing resistance to mechanical shear forces in tissues such as muscle
B) Desmosomes anchor intermediate filaments to cell membranes, providing strong adhesion and mechanical stability in tissues like skin and heart
C) Desmosomes facilitate the passage of ions between cells, helping tissues withstand physical stress
D) Desmosomes promote cell-cell communication, preventing cell detachment under mechanical stress

67. How do selectin-carbohydrate interactions contribute to leukocyte recruitment during an immune response, and what effect would a mutation in the selectin gene have on this process?

A) Selectin-carbohydrate interactions promote the rolling of leukocytes along the endothelial surface, facilitating their extravasation into the tissue; mutations would reduce leukocyte recruitment and immune response
B) Selectin-carbohydrate interactions initiate a strong, irreversible adhesion between leukocytes and endothelial cells, impairing the immune response during infection
C) Mutations in the selectin gene would lead to excessive leukocyte rolling, causing chronic inflammation and tissue damage
D) Selectin-carbohydrate interactions would enhance the signaling of integrins, promoting leukocyte migration, and mutations would have no effect on immune function

68. How might defective cadherin-mediated adhesion contribute to embryonic development defects?

A) Defective cadherin-mediated adhesion could cause cells to lose their ability to migrate, leading to improper tissue patterning during development
B) Cadherin defects would lead to increased cell-cell adhesion, disrupting the normal separation of tissues during organogenesis
C) Loss of cadherin function would prevent the formation of tight junctions, resulting in abnormal morphogenesis of the epithelial layer
D) Defective cadherin adhesion would cause excessive cell movement, leading to malformed or mislocalized organs

69. What is the significance of gap junctions in neuronal signaling, and how might defective connexin expression affect neurological function?

A) Gap junctions enable direct electrical coupling between neurons, and defective connexins could lead to impaired neurotransmission and cognitive dysfunction
B) Gap junctions inhibit electrical signaling in neurons, and mutations in connexins could enhance neural communication and increase brain activity
C) Gap junctions are involved in synaptic vesicle recycling, and defects in connexins would cause neurotransmitter accumulation and synaptic dysfunction
D) Gap junctions maintain the integrity of myelin sheaths around neurons, and defective connexins could result in neuronal degeneration

70. What might be the consequences of overexpressing integrins in a tissue undergoing repair after injury?

A) Overexpression of integrins would promote excessive cell migration, leading to fibrosis and scarring at the injury site
B) Overexpression of integrins would inhibit tissue repair by preventing the re-establishment of proper cell adhesion
C) Overexpression of integrins would enhance cell adhesion to the extracellular matrix, accelerating tissue regeneration and functional recovery
D) Overexpression of integrins would prevent cell differentiation, impairing the repair process and leading to tissue dysfunction

Answers:

56. **B**
57. **B**
58. **C**
59. **B**
60. **B**
61. **B**
62. **B**
63. **B**
64. **B**
65. **B**
66. **B**
67. **A**
68. **A**
69. **A**
70. **C**

71. What is the potential impact of selectin-mediated interactions in the tumor microenvironment, and how could this be exploited in targeted cancer therapies?

A) Selectins facilitate tumor cell extravasation from the bloodstream, and targeting selectin-carbohydrate interactions could reduce metastasis
B) Selectins promote the adhesion of immune cells to tumor cells, enhancing tumor surveillance and immune destruction
C) Selectins enhance angiogenesis by promoting endothelial cell migration, and inhibiting selectins could prevent tumor vasculature formation
D) Selectins mediate the formation of tight junctions in endothelial cells, and disrupting selectin activity could reduce the permeability of tumor blood vessels

72. How might a loss of E-cadherin and gain of N-cadherin during epithelial-to-mesenchymal transition (EMT) affect tumor progression and metastasis?

A) The loss of E-cadherin and gain of N-cadherin promotes cell-cell adhesion, leading to enhanced tissue integrity and reduced metastatic potential
B) The loss of E-cadherin weakens cell-cell junctions, while the gain of N-cadherin strengthens cell-matrix adhesion, facilitating invasion and migration
C) E-cadherin loss and N-cadherin gain stabilize the extracellular matrix, preventing tumor cells from breaking free and metastasizing
D) The transition from E-cadherin to N-cadherin promotes cell-cell cohesion, limiting invasion but enhancing local tissue spreading

73. In the context of cardiac function, how does the gap junction composition, specifically the types of connexins, impact cardiac conduction and what would happen if connexins were mutated?

A) Different connexin types regulate the permeability of gap junctions to ions, and mutations could result in abnormal cardiac conduction, potentially leading to arrhythmias
B) Connexins primarily control the synthesis of neurotransmitters in cardiac cells, and mutations would have no effect on the electrical properties of the heart
C) Gap junctions formed by connexins in cardiac cells only serve structural purposes, and mutations would result in reduced heart muscle contractility but normal electrical conduction
D) Mutations in connexins would improve electrical coupling between cardiac cells, leading to an overly synchronized heartbeat and potentially causing fibrillation

74. Given that desmosomal cadherins anchor intermediate filaments between adjacent cells, how might defective desmosomal cadherins contribute to skin blistering diseases?

A) Defective desmosomal cadherins would weaken the attachment between cells, leading to mechanical stress-induced tearing and the formation of blisters in the epidermis
B) Defective desmosomal cadherins strengthen the adhesion between skin cells, preventing blister formation but causing other cellular dysfunctions
C) Defective desmosomal cadherins enhance cell migration, which could lead to epithelial overgrowth and abnormal blistering in certain skin conditions
D) Desmosomal cadherins prevent water retention in the skin, and their defects would result in excessive water loss and a dry skin appearance

75. What role does the extracellular matrix (ECM) play in the regulation of integrin-mediated adhesion, and how could disrupting ECM-integrin interactions be used therapeutically in treating fibrosis?

A) The ECM provides a scaffold for integrins to bind, promoting tissue homeostasis; disrupting ECM-integrin interactions could decrease fibrosis by preventing excessive cell adhesion and migration
B) ECM-integrin interactions are irrelevant in fibrosis development, and disrupting them would have no impact on the disease progression
C) Disrupting ECM-integrin binding would promote fibroblast differentiation into myofibroblasts, worsening fibrosis by enhancing collagen deposition
D) The ECM is only involved in providing nutrients to cells, and ECM-integrin disruption would have minimal therapeutic effects in fibrosis treatment

76. How might the dynamic regulation of cadherins and catenins influence cellular differentiation and tissue-specific function during development?

A) Catenins are essential for linking cadherins to the cytoskeleton, and changes in cadherin expression or catenin signaling could alter the polarity and organization of cells during development
B) Cadherins and catenins work independently in different tissues, with no functional overlap during developmental processes
C) The regulation of cadherins and catenins primarily affects cell proliferation, with minimal impact on differentiation or tissue function
D) The disruption of cadherin-catenin binding would promote premature differentiation, leading to abnormal tissue development in organs like the heart and lung

77. In the immune response, how does the interaction between integrins and ICAMs influence the leukocyte transmigration process, and what might be the effect of inhibiting this interaction in autoimmune diseases?

A) Integrins on leukocytes interact with ICAMs on endothelial cells to facilitate firm adhesion and transmigration into tissues; inhibiting this interaction would prevent excessive immune cell infiltration, helping to reduce inflammation in autoimmune diseases
B) Integrins and ICAMs prevent leukocyte attachment to endothelial cells, and inhibiting their interaction would increase immune cell migration, worsening autoimmune symptoms
C) Inhibition of integrin-ICAM interaction would disrupt tight junctions between endothelial cells, leading to increased vascular permeability and further immune cell infiltration in autoimmune diseases
D) ICAM-integrin interactions prevent apoptosis of immune cells, and inhibiting this interaction would enhance the immune response by promoting immune cell survival

78. What might be the consequence of overexpression of cadherins during epithelial repair after an injury, and how could this impact wound healing?

A) Overexpression of cadherins would stabilize epithelial cells and prevent cell migration, potentially impairing wound closure by reducing the ability of cells to move into the wound site
B) Overexpression of cadherins would facilitate cell migration into the wound, enhancing tissue regeneration and accelerating healing
C) Overexpression of cadherins would lead to excessive cell proliferation at the wound site, potentially resulting in hyperplastic tissue formation and scarring
D) Overexpression of cadherins would disrupt cell-matrix adhesion, preventing proper formation of the epithelial barrier and delaying wound healing

79. How do tight junctions influence the development of Claudin-low breast cancer, and what does this imply for potential therapeutic strategies?

A) Claudin proteins regulate tight junction integrity, and low expression of Claudins in breast cancer cells facilitates their mesenchymal transition, suggesting that restoring Claudin expression might reduce invasiveness and improve prognosis
B) Tight junctions promote the expression of mesenchymal markers, and enhancing Claudin expression in Claudin-low breast cancer cells could induce a more epithelial phenotype and reduce metastasis
C) Claudin expression has no impact on breast cancer progression; rather, it only influences the permeability of the tumor vasculature, making it a non-therapeutic target
D) Low Claudin expression enhances tight junction formation, stabilizing cell-cell adhesion and inhibiting cancer cell invasion into surrounding tissues

80. What is the role of gap junctions in the synchronization of tissue activity, and how could defective connexin expression lead to diseases like Marie-Charcot-Tooth disease?

A) Gap junctions facilitate direct electrical coupling between cells, and defective connexins prevent this coupling, leading to the loss of coordinated tissue function and diseases like neuropathy or sensory impairments
B) Gap junctions serve primarily as structural components in tissues, and defective connexin expression results in tissue degeneration but does not affect functional coordination
C) Defective connexins in gap junctions enhance the transmission of electrical signals, leading to overactive tissue responses in diseases like Marie-Charcot-Tooth disease
D) Gap junctions inhibit direct electrical coupling, and defective connexins in gap junctions allow excessive ion flux, contributing to the development of neurological disorders

Answers:

71. **A**
72. **B**
73. **A**
74. **A**
75. **A**
76. **A**
77. **A**
78. **A**
79. **A**
80. **A**

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