LEC 16 CYTOLOGY Q:

1. Which of the following statements about differentiated cells is correct?

A) Most differentiated cells can proliferate indefinitely.
B) Differentiated cells can only proliferate if they are self-renewing stem cells.
C) Differentiated cells lose the ability to proliferate after full maturation, and must be replaced by less differentiated progenitor cells.
D) All differentiated cells can proliferate when needed.

2. Hematopoietic stem cells are responsible for the renewal of which type of cells?

A) Epithelial cells in the gut lining
B) Blood cells, replacing over 100 x 10^9 cells daily
C) Fibroblasts in connective tissue
D) Smooth muscle cells in vascular tissue

3. Which of the following types of cells can temporarily enter the G0 phase but can re-enter the cell cycle when needed?

A) Muscle cells
B) Endothelial cells
C) Neurons
D) Oocytes

4. What is the main difference between apoptosis and necrosis?

A) Necrosis is a programmed process, while apoptosis is not.
B) Necrosis involves cell shrinkage, while apoptosis involves cell swelling.
C) Apoptosis is a regulated and controlled process, while necrosis is caused by acute injury and is uncontrolled.
D) Both apoptosis and necrosis involve inflammation and immune response.

5. Which of the following is true regarding caspases in apoptosis?

A) Caspases inhibit apoptosis by cleaving proapoptotic proteins.
B) Caspases are proteases that cleave over 100 target proteins, triggering apoptosis.
C) Caspases directly activate the cell cycle to repair DNA damage.
D) Caspases prevent chromatin condensation and DNA fragmentation.

6. During apoptosis, the exposure of phosphatidylserine (PS) on the outer leaflet of the cell membrane signals for what?

A) The activation of the apoptosome complex
B) Phagocytosis by immune cells

C) Activation of antiapoptotic proteins
D) Membrane rupture and necrosis

7. Which of the following proteins is directly involved in the mitochondrial pathway of apoptosis?

A) Caspase-8
B) Apaf-1
C) Bcl-2
D) MLKL

8. Which protein complex forms in response to the release of cytochrome c from the mitochondria during apoptosis?

A) The apoptosome complex (cytochrome c + Apaf-1 + caspase-9)
B) The Fas death-inducing complex (Fas + caspase-8)
C) The MLKL complex
D) The autophagosome complex

9. What is the function of the Bcl-2 family of proteins in apoptosis regulation?

A) They inhibit the activation of caspase-9.
B) They prevent DNA fragmentation in apoptotic cells.
C) They control the permeability of the mitochondrial membrane, promoting or inhibiting apoptosis.
D) They directly degrade the plasma membrane during apoptosis.

10. Which of the following best describes necroptosis?

A) A form of programmed cell death that results in inflammation and is regulated like apoptosis.
B) A form of cell death that results from mitochondrial damage and leads to cell shrinkage.
C) A form of death where cells undergo swelling and leakage of their contents, but no immune response is triggered.
D) A caspase-independent cell death mechanism activated by autophagy.

11. Pyroptosis is a type of cell death primarily associated with which of the following?

A) Inflammatory cells such as macrophages
B) Mitochondrial damage and cytochrome c release
C) Depletion of antioxidants and lipid peroxidation
D) Damage to the DNA caused by external radiation

12. Which of the following best describes the molecular mechanism of ferroptosis?

A) Iron accumulation leads to the activation of caspases, resulting in apoptosis.
B) Oxidative damage and lipid peroxidation lead to cell membrane damage and rupture.
C) Caspase inhibitors prevent the activation of proapoptotic proteins.
D) Pyroptotic proteins form pores in the membrane, releasing cytokines like IL-1β.

13. Which cellular event triggers the intrinsic pathway of apoptosis?

A) Activation of death receptors on the cell surface
B) DNA damage or cell stress leading to mitochondrial membrane permeabilization
C) Activation of caspase-8 by Fas receptor signaling
D) Release of gasdermin proteins during infection

14. In the extrinsic pathway of apoptosis, which molecule binds to death receptors like Fas to induce apoptosis?

A) Cytochrome c
B) Tumor necrosis factor (TNF)
C) Growth factors
D) Glutathione peroxidase

15. What is the role of autophagy in cell survival?

A) Autophagy promotes apoptosis by degrading damaged organelles.
B) Autophagy helps cells survive under stress conditions by providing energy from damaged organelles.
C) Autophagy is triggered only during inflammatory cell death.
D) Autophagy leads to excessive accumulation of damaged proteins, ultimately triggering necrosis.

Answers:

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

16. Which of the following is the primary function of caspase-3 in apoptosis?

A) To activate proapoptotic proteins by cleaving antiapoptotic proteins
B) To initiate the apoptotic cascade by activating caspase-9
C) To cleave structural proteins such as nuclear lamins and cytoskeletal proteins
D) To degrade phosphatidylserine and prevent the “eat me” signal

17. The externalization of phosphatidylserine (PS) during apoptosis is a signal for which process?

A) Caspase activation and mitochondrial membrane permeabilization
B) The formation of the apoptosome complex in the cytoplasm
C) Phagocytosis by macrophages or neighboring cells
D) DNA repair and inhibition of cell death

18. In the intrinsic pathway of apoptosis, the activation of which protein is crucial for the release of cytochrome c from mitochondria?

A) Caspase-8
B) Bcl-2
C) Bax or Bak
D) Apaf-1

19. The activation of which protein complex leads to the formation of pores in the mitochondrial outer membrane, a hallmark of the intrinsic apoptotic pathway?

A) Apaf-1 + Cytochrome c + Caspase-9
B) Bax/Bak oligomers
C) Fas + caspase-8 complex
D) MLKL oligomers

20. How does Akt promote cell survival in response to growth factors?

A) By inhibiting proapoptotic proteins like Bax
B) By activating caspase-9, leading to apoptosis
C) By activating the Bcl-2 family of antiapoptotic proteins
D) By deactivating DNA repair mechanisms to allow cell death

21. Which of the following statements best describes the role of p53 in response to DNA damage?

A) p53 activates antiapoptotic genes to protect cells from apoptosis
B) p53 stimulates DNA repair processes and halts the cell cycle if the damage is reparable
C) p53 directly activates caspase-3 to initiate apoptosis
D) p53 promotes mitochondrial dysfunction to induce necrosis

22. Which of the following best characterizes necroptosis compared to necrosis?

A) Necroptosis involves active signaling through death receptors, whereas necrosis is purely caused by external injury
B) Necroptosis results in cell shrinkage, while necrosis results in cell swelling
C) Necroptosis involves caspases and is regulated, while necrosis is unregulated and caused by external factors
D) Necroptosis leads to autophagy-mediated cell death, while necrosis causes mitochondrial rupture

23. In pyroptosis, which of the following cytokines are released due to gasdermin pore formation?

A) IL-6 and TNF-α
B) IL-1β and IL-18
C) IL-10 and TGF-β
D) IFN-γ and IL-12

24. Which of the following is a key molecular event in ferroptosis?

A) Activation of caspase-3 and degradation of nuclear proteins
B) Iron accumulation and lipid peroxidation leading to cell membrane rupture
C) Inhibition of autophagic pathways and clearance of damaged organelles
D) Release of cytochrome c from mitochondria and activation of the apoptosome

25. During apoptosis, the cleavage of which protein leads to nuclear fragmentation and chromatin condensation?

A) ICAD (Inhibitor of Caspase-Activated DNase)
B) Actin
C) Nuclear lamins
D) Vimentin

26. The protein MLKL is associated with which form of regulated cell death?

A) Apoptosis
B) Necroptosis
C) Pyroptosis
D) Ferroptosis

27. Which of the following is true about the role of Bcl-2 family members in the intrinsic apoptotic pathway?

A) Proapoptotic Bcl-2 family members inhibit mitochondrial membrane permeability.
B) Antiapoptotic Bcl-2 family members promote the release of cytochrome c from mitochondria.
C) Proapoptotic Bcl-2 family members form pores in the mitochondrial membrane to

release cytochrome c.
D) Antiapoptotic Bcl-2 family members activate the apoptosome complex.

28. Which of the following mechanisms is responsible for the selective destruction of damaged or dysfunctional mitochondria through autophagy?

A) Lysosomal degradation of damaged mitochondria after fusion with autophagosomes
B) Activation of proapoptotic proteins that disrupt mitochondrial membranes
C) Cytochrome c release leading to caspase activation and mitochondrial fragmentation
D) MLKL formation of membrane pores leading to mitochondrial rupture

29. What is the relationship between autophagy and apoptosis in cellular stress responses?

A) Autophagy prevents apoptosis by degrading proapoptotic proteins
B) Autophagy and apoptosis are mutually exclusive processes that cannot occur simultaneously
C) Autophagy promotes apoptosis by activating caspase-3 and cleaving cellular proteins
D) Autophagy promotes cell survival by providing energy and inhibiting apoptosis under stress conditions

30. Which of the following best describes the role of ICAD in apoptosis?

A) ICAD activates caspases to induce apoptosis
B) ICAD prevents DNA fragmentation by inhibiting DNase activity
C) ICAD cleaves the nuclear lamins to promote chromatin condensation
D) ICAD stabilizes mitochondrial membranes to prevent cytochrome c release

Answers:

16. C
17. C
18. C
19. B
20. C
21. B
22. A
23. B
24. B
25. C
26. B
27. C
28. A

29. D
30. B

31. What is the primary role of the scramblase protein during apoptosis?

A) It cleaves phosphatidylserine (PS) to prevent its externalization on the cell membrane.
B) It translocates phosphatidylserine (PS) from the inner to the outer leaflet of the cell membrane, signaling for phagocytosis.
C) It forms pores in the mitochondrial membrane to release cytochrome c.
D) It activates caspase-9 by forming the apoptosome complex.

32. Which of the following statements correctly describes the relationship between caspases and autophagy?

A) Caspases activate autophagy pathways by cleaving key autophagy-related proteins.
B) Caspases inhibit autophagy, leading to excessive cell death.
C) Autophagy prevents caspase activation by degrading proapoptotic proteins.
D) Caspases and autophagy act independently and are not connected in any cell death pathways.

33. In the extrinsic apoptotic pathway, what is the role of caspase-8?

A) It activates the mitochondrial pathway by cleaving Bcl-2 family proteins.
B) It cleaves MLKL to induce necroptosis.
C) It directly activates effector caspases (caspase-3 and caspase-7) to initiate apoptosis.
D) It activates the apoptosome complex by cleaving Apaf-1.

34. In the context of apoptosis, what function does the protein ICAD serve before being cleaved by caspases?

A) It inhibits the formation of apoptosome complexes by binding cytochrome c.
B) It prevents the activation of caspases by sequestering proapoptotic proteins.
C) It binds and inhibits DNase, preventing DNA fragmentation.
D) It stabilizes the nuclear lamins to prevent nuclear fragmentation.

35. The apoptosome complex involves the binding of cytochrome c to which protein, leading to the activation of caspase-9?

A) Bax
B) Apaf-1
C) Fas
D) MLKL

36. Which of the following molecular events occurs during the activation of the extrinsic apoptotic pathway?

A) The release of cytochrome c from mitochondria into the cytoplasm
B) The formation of the death-inducing signaling complex (DISC) at the cell membrane
C) The activation of Bcl-2 family proteins to form mitochondrial pores
D) The activation of proapoptotic regulatory proteins such as p53

37. How do death receptors like Fas activate the extrinsic apoptotic pathway?

A) By forming pores in the mitochondrial membrane to release proapoptotic proteins.
B) By binding TNF and activating caspase-3 directly.
C) By binding ligands, such as Fas ligand, and recruiting adaptor proteins like FADD to activate caspase-8.
D) By promoting the externalization of phosphatidylserine (PS) to signal phagocytosis.

38. Which of the following proteins is responsible for mitochondrial outer membrane permeabilization (MOMP) in response to proapoptotic signals?

A) Bax/Bak
B) Caspase-8
C) MLKL
D) Apaf-1

39. p53 functions as a tumor suppressor primarily by which of the following mechanisms in response to cellular stress?

A) p53 directly activates the Bcl-2 family of antiapoptotic proteins to inhibit apoptosis.
B) p53 stabilizes the mitochondrial membrane to prevent apoptosis in stressed cells.
C) p53 activates genes that repair DNA damage or induce apoptosis if the damage is irreparable.
D) p53 promotes necrosis by disrupting the plasma membrane integrity.

40. Which protein family regulates the release of cytochrome c from mitochondria by controlling the permeability of the mitochondrial membrane during apoptosis?

A) Bcl-2 family of proteins
B) Caspase family of proteins
C) Fas-associated protein family
D) Gasdermin family

41. Which of the following statements best describes the role of MLKL in necroptosis?

A) MLKL cleaves proapoptotic proteins, leading to mitochondrial fragmentation.
B) MLKL forms pores in the plasma membrane, resulting in ion influx and cell rupture.
C) MLKL activates autophagy by fusing with damaged mitochondria.
D) MLKL recruits caspase-3 to initiate apoptosis through the extrinsic pathway.

42. Autophagy is often activated under conditions of cellular stress. Which of the following is the primary role of autophagy during stress?

A) To induce apoptosis and clear damaged organelles through the caspase pathway.
B) To promote cell survival by recycling damaged organelles and providing energy.
C) To cause necrosis by destabilizing the plasma membrane during nutrient starvation.
D) To initiate pyroptosis by releasing inflammatory cytokines from damaged organelles.

43. Which of the following is a defining feature of ferroptosis compared to other forms of regulated cell death?

A) It is driven by mitochondrial dysfunction and cytochrome c release.
B) It is induced by the accumulation of reactive oxygen species (ROS) and lipid peroxidation.
C) It involves the activation of caspases and DNA fragmentation.
D) It is a pro-inflammatory cell death that triggers the release of IL-1β and IL-18.

44. Which of the following is a common characteristic shared by necroptosis and pyroptosis?

A) Both are caspase-dependent cell death processes.
B) Both involve the activation of MLKL, which forms pores in the plasma membrane.
C) Both are regulated by Bcl-2 family proteins.
D) Both involve chromatin condensation and DNA fragmentation.

45. The release of gasdermin proteins is associated with which type of cell death?

A) Apoptosis
B) Necrosis
C) Pyroptosis
D) Ferroptosis

46. In the context of necroptosis, which of the following is the key event that leads to plasma membrane rupture?

A) Activation of the apoptosome complex.
B) Formation of pores by the protein MLKL in the plasma membrane.
C) Release of cytochrome c into the cytoplasm.
D) Exposure of phosphatidylserine on the outer leaflet of the plasma membrane.

47. Which of the following accurately describes the role of scramblase in apoptosis?

A) Scramblase is involved in the externalization of phosphatidylserine (PS) during the early stages of apoptosis.
B) Scramblase prevents the formation of the apoptosome complex in the cytoplasm.
C) Scramblase helps degrade the Golgi apparatus during apoptosis.
D) Scramblase activates caspase-3 and initiates DNA fragmentation.

48. Which of the following statements is true regarding the cross-talk between autophagy and apoptosis?

A) Autophagy always inhibits apoptosis by degrading proapoptotic proteins.
B) Autophagy and apoptosis are mutually exclusive, meaning one cannot occur if the other is active.
C) Under nutrient starvation, autophagy promotes cell survival by preventing apoptosis.
D) Autophagy promotes apoptosis by releasing cytochrome c from mitochondria.

Answers:

31. B
32. C
33. C
34. C
35. B
36. B
37. C
38. A
39. C
40. A
41. B
42. B
43. B
44. B
45. C
46. B
47. A
48. C

49. Which of the following is the primary mechanism by which autophagy helps cells survive under nutrient deprivation?

A) By activating caspase-3 to remove damaged organelles and proteins.
B) By inducing apoptosis to remove dysfunctional cells.
C) By recycling damaged organelles and proteins for energy production, thereby

maintaining cellular functions.
D) By inhibiting the externalization of phosphatidylserine, preventing phagocytosis.

50. In necroptosis, which protein is primarily responsible for forming oligomeric pores in the plasma membrane, leading to cell rupture?

A) Caspase-9
B) MLKL (Mixed Lineage Kinase Domain-Like Protein)
C) Bax
D) Apaf-1

51. During necrosis, what is the primary factor that triggers the inflammatory response following cell rupture?

A) The leakage of cellular contents like ATP and histones, which activate immune receptors.
B) The exposure of phosphatidylserine on the cell surface, signaling macrophages.
C) The activation of caspases, which mediate the immune response.
D) The release of cytochrome c from the mitochondria into the cytosol.

52. In the context of apoptosis, how does the activation of Bax/Bak proteins lead to mitochondrial outer membrane permeabilization (MOMP)?

A) Bax/Bak proteins cleave caspases that degrade mitochondrial membranes.
B) Bax/Bak oligomerize and form pores in the mitochondrial membrane, allowing the release of pro-apoptotic factors like cytochrome c.
C) Bax/Bak translocate to the nucleus to initiate chromatin condensation and nuclear fragmentation.
D) Bax/Bak inhibit antiapoptotic proteins like Bcl-2, leading to cell survival.

53. Which of the following best describes the role of Bcl-2 family proteins in regulating apoptosis?

A) Bcl-2 family proteins directly cleave caspase-3 and other apoptotic proteases.
B) Antiapoptotic Bcl-2 family members inhibit mitochondrial permeabilization, whereas proapoptotic members promote it.
C) Proapoptotic Bcl-2 family members activate the extrinsic pathway of apoptosis through death receptor signaling.
D) Bcl-2 family proteins block autophagy by inhibiting autophagosome formation.

54. What is the role of cytochrome c in apoptosis?

A) Cytochrome c activates caspase-8, leading to the initiation of the extrinsic apoptotic pathway.
B) Cytochrome c leaks from mitochondria and binds to Apaf-1 and caspase-9 to form the apoptosome complex, triggering apoptosis.
C) Cytochrome c inhibits DNA fragmentation by blocking the activation of DNase.
D) Cytochrome c prevents mitochondrial swelling by inhibiting Bax and Bak proteins.

55. MLKL activation in necroptosis results in which of the following cellular events?

A) DNA fragmentation and chromatin condensation.
B) Cell shrinkage and phagocytosis by neighboring cells.
C) Formation of pores in the plasma membrane that disrupt ionic balance, leading to cell swelling and rupture.
D) Release of proinflammatory cytokines such as IL-1β and IL-18 into the extracellular space.

56. p53 activation in response to DNA damage results in which of the following outcomes?

A) p53 inhibits autophagy and promotes cell death through the extrinsic pathway.
B) p53 induces the expression of proapoptotic proteins like Puma and Noxa to initiate apoptosis when DNA damage is irreparable.
C) p53 directly activates caspase-3 to trigger apoptosis.
D) p53 inhibits Bcl-2 family proteins, leading to mitochondrial integrity and cell survival.

57. Which of the following events is triggered by Fas receptor activation in the extrinsic pathway of apoptosis?

A) Cytochrome c release from mitochondria.
B) Formation of the death-inducing signaling complex (DISC) and activation of caspase-8.
C) Bax/Bak oligomerization and mitochondrial outer membrane permeabilization.
D) Exposure of phosphatidylserine on the outer leaflet of the plasma membrane.

58. Ferroptosis is primarily characterized by which of the following molecular processes?

A) The activation of caspases and DNA fragmentation.
B) The accumulation of iron and lipid peroxidation, leading to oxidative damage of cellular components.
C) The leakage of cytochrome c from mitochondria and activation of the apoptosome.
D) The release of proinflammatory cytokines like IL-1β and IL-18.

59. The apoptosome complex is essential for initiating apoptosis in the intrinsic pathway. Which of the following is the key structural feature of the apoptosome complex?

A) The binding of cytochrome c to Apaf-1 and caspase-9, leading to caspase activation.
B) The activation of Bax/Bak proteins to form pores in the mitochondrial membrane.
C) The phosphorylation of p53 to activate DNA repair proteins.
D) The interaction between Fas receptors and FADD to initiate the extrinsic apoptotic pathway.

60. How does scramblase help in the detection and clearance of apoptotic cells?

A) Scramblase triggers the activation of caspase-3, leading to nuclear fragmentation.
B) Scramblase translocates phosphatidylserine to the outer membrane leaflet, marking cells for phagocytosis.
C) Scramblase enhances mitochondrial membrane permeability, leading to cytochrome c release.
D) Scramblase induces the formation of autophagosomes that degrade damaged cellular components.

61. Which of the following statements correctly describes the role of scramblase in the cell membrane during apoptosis?

A) Scramblase prevents the externalization of phosphatidylserine (PS), thereby blocking phagocytosis.
B) Scramblase promotes the flipping of phosphatidylserine (PS) from the inner to the outer leaflet, marking the cell for clearance.
C) Scramblase induces cell shrinkage by disrupting the cytoskeleton.
D) Scramblase activates autophagy to degrade apoptotic bodies and damaged organelles.

62. In response to DNA damage, ATM kinase activates which of the following proteins to trigger apoptosis?

A) Caspase-9
B) p53
C) MLKL
D) Bax

63. The formation of autophagosomes during autophagy involves the fusion of which two key cellular compartments?

A) Mitochondria and endoplasmic reticulum
B) Lysosomes and mitochondria
C) Lysosomes and autophagic vesicles containing damaged organelles or proteins
D) Golgi apparatus and endosomes

Answers:

49. C
50. B
51. A
52. B
53. B
54. B
55. C

56. B
57. B
58. B
59. A
60. B
61. B
62. B
63. C

64. Which of the following statements best describes the difference between apoptosis and necrosis?

A) Apoptosis is a regulated process that results in cell shrinkage, while necrosis is an uncontrolled process that causes cell swelling and rupture.
B) Necrosis is programmed cell death triggered by internal signals, while apoptosis is caused by external damage.
C) Apoptosis always triggers inflammation, while necrosis does not.
D) Apoptosis results in the formation of an inflammatory response, while necrosis is a silent, non-inflammatory form of cell death.

65. Caspase-3 plays a crucial role in the execution of apoptosis. Which of the following targets is cleaved by caspase-3 during the apoptotic process?

A) Phosphatidylserine (PS) to expose it on the cell membrane for phagocytosis.
B) The inhibitor of DNase (ICAD) to activate DNase and cause DNA fragmentation.
C) Mitochondrial outer membrane proteins like Bax/Bak to release cytochrome c.
D) The Golgi membrane proteins to fragment the Golgi apparatus.

66. The release of cytochrome c from mitochondria into the cytosol is a key step in the intrinsic pathway of apoptosis. Which of the following proteins is responsible for mediating mitochondrial outer membrane permeabilization (MOMP)?

A) Bcl-2
B) Caspase-8
C) Bax/Bak
D) Fas

67. Which of the following is a major function of phosphatidylserine (PS) exposure on the outer leaflet of the plasma membrane during apoptosis?

A) It initiates mitochondrial outer membrane permeabilization.
B) It signals for the activation of caspases in the apoptotic cascade.
C) It serves as a "find-me" signal for phagocytes to clear the dying cell.
D) It causes the release of proinflammatory cytokines such as IL-1β.

68. What is the role of ATM kinase in the response to DNA damage during apoptosis?

A) It activates the extrinsic apoptotic pathway by cleaving caspase-8.
B) It phosphorylates p53, stabilizing it to either repair DNA damage or induce apoptosis if damage is irreparable.
C) It cleaves Bcl-2 proteins to promote mitochondrial permeabilization.
D) It activates autophagy pathways to clear damaged organelles.

69. Necroptosis is a regulated form of cell death that shares some similarities with necrosis. What is the key feature of necroptosis?

A) It is always initiated by DNA damage and leads to apoptosis.
B) It is caspase-independent and involves the activation of MLKL to form pores in the plasma membrane.
C) It involves the release of proinflammatory cytokines such as IL-1β but does not cause plasma membrane rupture.
D) It is a form of autophagy, where cells self-digest damaged organelles for survival.

70. Which of the following is the primary role of scramblase during apoptosis?

A) It activates caspases to trigger DNA fragmentation.
B) It translocates phosphatidylserine (PS) to the outer leaflet of the cell membrane to signal phagocytosis.
C) It forms pores in the mitochondrial membrane to release cytochrome c.
D) It cleaves ICAD to activate DNase for DNA fragmentation.

71. Autophagy serves as a protective mechanism during cellular stress. Which of the following is a key characteristic of autophagy under starvation conditions?

A) It inhibits the intrinsic apoptotic pathway by stabilizing Bcl-2 proteins.
B) It activates the caspase cascade to induce cell death.
C) It degrades damaged organelles and proteins to recycle essential nutrients for

survival.
D) It triggers inflammation by activating the NLRP3 inflammasome.

72. Ferroptosis is a unique form of regulated cell death. Which of the following best describes the mechanism of ferroptosis?

A) It is characterized by DNA fragmentation and chromatin condensation.
B) It involves the accumulation of reactive oxygen species (ROS) and lipid peroxidation, leading to membrane damage.
C) It is triggered by the release of cytochrome c from the mitochondria.
D) It results in a proinflammatory response, with the release of IL-1β and IL-18.

73. Which of the following best describes the role of Bcl-2 family proteins in regulating mitochondrial outer membrane permeabilization (MOMP) during apoptosis?

A) Antiapoptotic Bcl-2 proteins inhibit MOMP by preventing Bax/Bak oligomerization.
B) Proapoptotic Bcl-2 proteins inhibit MOMP, preventing apoptosis.
C) Bcl-2 proteins directly cleave caspase-3 to induce cell death.
D) Bcl-2 family proteins activate autophagy to prevent cell death.

74. What is the function of caspase-9 in the apoptotic signaling pathway?

A) It is activated by Fas receptor signaling and cleaves caspase-8.
B) It forms the apoptosome complex with cytochrome c and Apaf-1 to activate executioner caspases like caspase-3.
C) It degrades mitochondrial DNA to trigger cell death.
D) It activates MLKL to induce necroptosis.

75. Which of the following best describes the key difference between the intrinsic and extrinsic apoptotic pathways?

A) The intrinsic pathway is triggered by external signals such as Fas ligand, while the extrinsic pathway is triggered by internal damage, such as DNA damage.
B) The intrinsic pathway involves mitochondrial dysfunction and the release of cytochrome c, while the extrinsic pathway involves death receptor signaling.
C) The intrinsic pathway always leads to inflammation, while the extrinsic pathway is silent.

D) The extrinsic pathway involves Bcl-2 family proteins, while the intrinsic pathway does not.

76. Pyroptosis is a proinflammatory form of cell death. Which of the following proteins plays a critical role in the induction of pyroptosis?

A) Caspase-3
B) Caspase-8
C) Gasdermin
D) MLKL

77. What is the primary feature that distinguishes necrosis from other forms of regulated cell death, such as apoptosis?

A) Necrosis is characterized by caspase activation and DNA fragmentation.
B) Necrosis is typically caused by external factors, such as trauma or ischemia, leading to cell rupture.
C) Necrosis always leads to the release of proinflammatory cytokines, but apoptosis does not.
D) Necrosis involves the externalization of phosphatidylserine (PS) for phagocytosis.

78. What is the role of p53 in the intrinsic apoptotic pathway following DNA damage?

A) p53 stabilizes antiapoptotic proteins like Bcl-2 to promote cell survival.
B) p53 activates proapoptotic proteins such as Puma and Noxa to promote apoptosis if DNA damage is irreparable.
C) p53 directly cleaves caspase-3 to trigger cell death.
D) p53 prevents mitochondrial membrane permeabilization by blocking Bax/Bak proteins.

79. Which of the following events characterizes apoptosis?

A) DNA fragmentation and chromatin condensation
B) The release of cytochrome c and activation of the inflammasome
C) Swelling of the cell and leakage of cellular contents
D) The activation of MLKL, leading to plasma membrane rupture

80. Tissue renewal in the body involves stem cells. Which of the following stem cells is responsible for replacing blood cells in the human body?

A) Hematopoietic stem cells
B) Intestinal epithelial stem cells
C) Fibroblast stem cells
D) Neural stem cells

Answers:

64. A
65. B
66. C
67. C
68. B
69. B
70. B
71. C
72. B
73. A
74. B
75. B
76. C
77. B
78. B
79. A
80. A

STEP 1 QUSTIONS:

1. A 45-year-old man with a history of chronic alcohol abuse presents with severe liver damage due to alcoholic hepatitis. On histologic examination of his liver tissue, you observe the presence of swollen hepatocytes with disrupted cell membranes, leakage of cellular contents, and an inflammatory response. What type of cell death is most likely occurring in this patient's liver cells?

A) Apoptosis
B) Necrosis
C) Autophagy
D) Necroptosis
E) Ferroptosis

2. During apoptosis, the activation of caspases is a key event. Which of the following caspases is primarily responsible for the cleavage of ICAD (inhibitor of DNase), leading to DNA fragmentation?

A) Caspase-3
B) Caspase-8
C) Caspase-9
D) Caspase-12
E) Caspase-1

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3. A 60-year-old woman is diagnosed with lung cancer. Molecular analysis reveals a mutation in the Bcl-2 family of proteins, which impairs the function of proapoptotic proteins. How would this mutation most likely affect the apoptotic process?

A) Increased mitochondrial membrane permeabilization, leading to cytochrome c release
B) Inhibition of cytochrome c release from mitochondria, preventing apoptosome formation
C) Activation of caspase-8 and subsequent activation of executioner caspases
D) Decreased exposure of phosphatidylserine on the outer membrane
E) Increased activation of proinflammatory cytokines

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4. A 5-year-old child presents with skin and soft tissue infections. A biopsy of the tissue reveals large areas of necrosis with a lack of inflammation. Which of the following best explains the absence of inflammation in this type of necrosis?

A) The cells underwent apoptosis instead of necrosis.
B) The tissue damage was caused by viral infection, which does not induce inflammation.
C) Necrosis in this case is caspase-independent and does not trigger an inflammatory response.
D) The necrosis was due to ischemia, and there was no immune response because of the limited blood flow.
E) The cells released proinflammatory cytokines, but these were cleared too rapidly to initiate inflammation.

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5. In the context of DNA damage, which of the following proteins directly leads to the activation of the mitochondrial apoptotic pathway by forming pores in the mitochondrial outer membrane?

A) p53
B) Bcl-2
C) Bax
D) Fas
E) Apaf-1

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6. Which of the following cellular events during apoptosis is directly triggered by caspase-9?

A) DNA fragmentation through activation of DNase
B) Formation of the apoptosome complex
C) Disruption of the Golgi apparatus
D) Phosphatidylserine exposure on the outer leaflet of the cell membrane
E) Release of interleukins like IL-1β and IL-18

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7. A 35-year-old man presents with an infection caused by Salmonella. On examination, his inflammatory cells show large areas of cell death characterized by the formation of pores in the plasma membrane, leading to the release of proinflammatory cytokines. Which form of regulated cell death is most likely occurring in these cells?

A) Apoptosis
B) Necrosis
C) Pyroptosis
D) Necroptosis
E) Ferroptosis

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8. A researcher is studying the molecular mechanisms of apoptosis in response to cellular stress. She inhibits the activity of the enzyme caspase-3. Which of the following effects would most likely result from this inhibition?

A) Failure of mitochondrial membrane permeabilization
B) Prevention of DNA fragmentation
C) Inhibition of the release of cytochrome c
D) Inability to form the apoptosome
E) Accumulation of phosphatidylserine on the outer membrane

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9. A 45-year-old woman with a history of chronic hepatitis B develops cirrhosis of the liver. She is diagnosed with liver failure. Histological

examination of the liver reveals massive hepatocyte death. The cells appear to be undergoing programmed cell death without causing an inflammatory response. Which of the following best describes this type of cell death?

A) Apoptosis
B) Necrosis
C) Necroptosis
D) Pyroptosis
E) Ferroptosis

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10. A 70-year-old patient presents with severe sepsis. In his laboratory results, there is elevated serum ferritin and evidence of extensive cell death characterized by iron accumulation and lipid peroxidation. Which of the following cell death pathways is most likely responsible for this finding?

A) Apoptosis
B) Necrosis
C) Autophagy
D) Ferroptosis
E) Pyroptosis

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11. A 22-year-old man presents with a cutaneous eruption, and a skin biopsy reveals the presence of epidermal cells undergoing programmed cell death due to a viral infection. The cells exhibit cell shrinkage, chromatin condensation, and formation of apoptotic bodies. Which of the following best characterizes this type of cell death?

A) Apoptosis
B) Necrosis
C) Autophagy
D) Pyroptosis
E) Necroptosis

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12. A 25-year-old woman with a family history of cancer is found to have a mutation in the p53 gene that impairs its ability to activate the mitochondrial apoptotic pathway. Which of the following events would be most likely in this patient's cells?

A) Increased release of cytochrome c into the cytoplasm
B) Decreased activation of Bax and Bak proteins

C) Increased mitochondrial outer membrane permeabilization
D) Increased expression of antiapoptotic Bcl-2 proteins
E) Failure of caspase activation and cell death

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13. A 30-year-old patient develops a mass in his abdomen, which is later confirmed to be a tumor. The tumor cells are found to be undergoing necrosis. Which of the following mechanisms most likely explains the necrosis in these tumor cells?

A) Caspase activation and DNA fragmentation
B) Lack of oxygen and nutrients due to poor blood supply
C) Induction of mitochondrial membrane permeabilization
D) Release of cytokines from macrophages
E) Activation of death receptors on the plasma membrane

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14. A 50-year-old man develops sepsis due to a bacterial infection. His blood work reveals high levels of serum interleukin-1 (IL-1) and interleukin-18 (IL-18). Which of the following mechanisms is responsible for the release of these cytokines in this patient?

A) Apoptosis
B) Necroptosis
C) Pyroptosis
D) Autophagy
E) Ferroptosis

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15. A 65-year-old woman with a history of chronic kidney disease presents with a gradual decline in kidney function. A kidney biopsy reveals significant tubular cell death associated with inflammatory cell infiltration. Which form of cell death is likely occurring in the kidney cells?

A) Apoptosis
B) Necrosis
C) Necroptosis
D) Pyroptosis
E) Autophagy

Answers:

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

1. A 60-year-old patient presents with signs of hepatic failure due to chronic alcohol consumption. Liver biopsy shows hepatocytes with swollen cytoplasm and damaged membranes, with leakage of cellular contents into the surrounding tissue. Which of the following best describes this type of cell death?

A) Apoptosis
B) Necrosis
C) Necroptosis
D) Pyroptosis
E) Ferroptosis

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2. A 45-year-old man with a history of smoking is diagnosed with non-small cell lung cancer. Genetic testing reveals a mutation in Bcl-2 leading to an increased ratio of antiapoptotic to proapoptotic proteins. How would this mutation most likely affect the process of apoptosis?

A) Increased mitochondrial outer membrane permeabilization
B) Inhibition of cytochrome c release from mitochondria
C) Increased cleavage of ICAD, leading to DNA fragmentation
D) Increased formation of the apoptosome complex
E) Increased activation of executioner caspases

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3. During an investigation into cellular responses to injury, a researcher observes the release of cytochrome c from mitochondria, followed by activation of caspase-9. This leads to cleavage of various cellular proteins, including DNA fragmentation. Which form of cell death is being described?

A) Apoptosis
B) Necrosis
C) Necroptosis
D) Pyroptosis
E) Autophagy

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4. A 52-year-old patient with a history of rheumatoid arthritis develops a severe infection. Blood tests reveal elevated levels of IL-1β and IL-18, both of which are released from dying cells. Which form of regulated cell death is most likely responsible for the release of these cytokines?

A) Apoptosis
B) Necroptosis
C) Pyroptosis
D) Autophagy
E) Ferroptosis

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5. A 28-year-old woman with no significant medical history presents with sudden-onset muscle weakness. A muscle biopsy reveals extensive muscle cell death characterized by swollen cells, membrane rupture, and release of intracellular contents. The histological findings suggest an inflammatory response. Which mechanism of cell death is most likely responsible?

A) Apoptosis
B) Necrosis
C) Necroptosis
D) Pyroptosis
E) Ferroptosis

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6. A researcher is studying apoptosis in response to DNA damage. In a specific experiment, they block the release of cytochrome c from mitochondria. Which of the following steps in the apoptotic pathway would be inhibited as a result of this block?

A) Formation of the apoptosome complex
B) Cleavage of ICAD and activation of DNase
C) Release of proinflammatory cytokines from dying cells
D) Activation of caspase-8
E) Formation of pores in the mitochondrial outer membrane

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7. A 40-year-old woman with chronic systemic lupus erythematosus (SLE) develops an infection. Histopathological examination of her immune cells reveals cell death accompanied by the formation of pores in the plasma membrane, causing rapid swelling and leakage of intracellular contents. What type of cell death is most likely occurring?

A) Apoptosis
B) Necrosis
C) Necroptosis
D) Pyroptosis
E) Autophagy

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8. A 60-year-old male smoker presents with severe lung dysfunction, and a biopsy shows cells with an increased accumulation of lipid peroxides and oxidative damage to cellular structures. Which form of cell death is most likely implicated in this scenario?

A) Apoptosis
B) Necrosis
C) Necroptosis
D) Ferroptosis
E) Pyroptosis

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9. A 32-year-old man with an infected wound develops widespread tissue death due to a bacterial infection. The cell death is characterized by the release of intracellular contents, triggering a severe immune response. Which of the following is most likely responsible for the release of proinflammatory cytokines from the dying cells?

A) Apoptosis
B) Necrosis
C) Pyroptosis
D) Necroptosis
E) Autophagy

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10. A genetic disorder in a 40-year-old woman leads to a mutation in the Fas receptor, impairing its ability to bind to its ligand. What is the most likely consequence of this mutation on the extrinsic apoptotic pathway?

A) Reduced activation of caspase-3
B) Increased mitochondrial outer membrane permeabilization
C) Reduced activation of caspase-8

D) Inhibition of cytochrome c release
E) Increased phosphatidylserine exposure on the outer leaflet

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11. A 25-year-old woman is diagnosed with a viral infection that results in cell death in her immune cells. The cell death process is characterized by the formation of large pores in the plasma membrane, release of interleukins, and an inflammatory response. Which form of cell death is being described?

A) Apoptosis
B) Necrosis
C) Pyroptosis
D) Necroptosis
E) Ferroptosis

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12. A patient with a history of chronic kidney disease presents with a progressive decline in kidney function. A biopsy reveals tubular necrosis, which is associated with cell swelling, rupture of the plasma membrane, and an inflammatory response. What mechanism is most likely responsible for this type of cell death?

A) Apoptosis
B) Necrosis
C) Necroptosis
D) Pyroptosis
E) Autophagy

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13. A 60-year-old woman with a history of colon cancer develops resistance to chemotherapy. Analysis of her cancer cells reveals increased levels of antiapoptotic Bcl-2 proteins, leading to decreased apoptosis. What is the most likely mechanism through which these proteins prevent cell death?

A) Inhibition of proapoptotic proteins like Bax
B) Activation of the extrinsic apoptotic pathway
C) Increased cytochrome c release from mitochondria
D) Upregulation of proinflammatory cytokines
E) Increased activation of executioner caspases

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14. A 30-year-old man with iron overload presents with progressive liver damage. His liver biopsy reveals extensive lipid peroxidation and damage to cellular structures. Which form of cell death is most likely responsible for the observed damage?

A) Apoptosis
B) Necrosis
C) Necroptosis
D) Ferroptosis
E) Autophagy

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15. A 50-year-old man presents with an autoimmune disorder and is undergoing treatment with a new drug that inhibits mTOR signaling. Which of the following processes would most likely be inhibited by this drug?

A) Apoptosis
B) Necroptosis
C) Autophagy
D) Pyroptosis
E) Ferroptosis

Answers:

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

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