LEC 14 CYTOLOGY Q:

1. What is the primary function of the cell cycle?

A) To ensure the synthesis of proteins
B) To facilitate DNA replication and chromosome separation
C) To carry out cellular respiration
D) To produce energy for the cell

2. Which phase of the cell cycle is the shortest in duration?

A) G1 Phase
B) S Phase
C) G2 Phase
D) M Phase

3. In which phase of the cell cycle do chromosomes replicate and form sister chromatids?

A) G1 Phase
B) S Phase
C) G2 Phase
D) M Phase

4. What happens during the G0 phase?

A) Cells are preparing for division
B) Cells grow and replicate DNA
C) Cells remain metabolically active but do not divide
D) Cells enter mitosis directly

5. What is the result of the M phase in the cell cycle?

A) DNA replication
B) Chromosomal segregation and two daughter cells
C) Formation of the nuclear membrane
D) Activation of cyclin-dependent kinases

6. Which of the following is true regarding flow cytometry?

A) It measures protein synthesis levels in cells
B) It sorts cells based on their DNA content
C) It is used to monitor ATP production in cells
D) It analyzes the movement of chromosomes during mitosis

7. What is the main role of cyclins in cell cycle regulation?

A) To bind with DNA to start replication
B) To phosphorylate and activate cyclin-dependent kinases
C) To inhibit the activity of cyclin-dependent kinases
D) To prevent chromosome condensation

8. Which of the following cyclin-Cdk complexes regulates the transition from G1 to S phase?

A) Cyclin A/Cdk1
B) Cyclin D/Cdk4,6
C) Cyclin E/Cdk2
D) Cyclin B/Cdk1

9. What is the primary function of the p53 protein in response to DNA damage?

A) To activate cyclin D synthesis
B) To prevent cyclin degradation
C) To promote the activation of p21 and arrest the cell cycle
D) To bind to E2F transcription factors

10. What happens if the Rb protein is phosphorylated by Cdk4/Cyclin D?

A) It binds to E2F and prevents S phase entry
B) It releases E2F, promoting the transition to S phase
C) It activates p53 for cell cycle arrest
D) It blocks DNA replication in the S phase

11. Which checkpoint is responsible for monitoring DNA integrity before entering S phase?

A) G1/S checkpoint
B) G2/M checkpoint
C) Spindle assembly checkpoint
D) S/G2 checkpoint

12. What is a characteristic feature of zygote cell division?

A) Cells divide slowly with long G1 and G2 phases
B) Zygote cells only undergo S and M phases, skipping G1 and G2
C) Zygote cells are unable to divide rapidly
D) Zygote cells differentiate immediately after fertilization

13. Which of the following statements is true about cyclin D in cell cycle regulation?

A) It is continuously present throughout the cell cycle
B) It is synthesized in response to growth factors to drive G1 phase progression
C) It inhibits Cdk4/Cdk6 activity during G1 phase
D) It is involved in the DNA damage checkpoint regulation

14. What is the role of the Ink4 family of Cdk inhibitors?

A) They activate Cdk2/Cyclin E to drive the G1 to S transition
B) They inhibit Cdk4 and Cdk6 during G1 phase
C) They degrade cyclins during M phase
D) They promote the progression from G2 to mitosis

15. Which signaling pathway is activated by growth factor binding to its receptor and regulates cyclin D expression?

A) Ras/Raf/ERK pathway
B) PI3K/Akt pathway
C) Wnt/β-catenin pathway
D) Notch signaling pathway

Answers:

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

1. Which of the following best describes the function of the G2 checkpoint in the cell cycle?

A) It ensures that cyclin D is degraded before entry into S phase.
B) It checks for DNA damage and ensures proper chromosome alignment before mitosis.
C) It activates cyclin E to allow progression through the G1/S transition.
D) It initiates the synthesis of DNA for the next round of cell division.

2. How does the p53 protein prevent the propagation of cells with damaged DNA?

A) It binds to and inactivates cyclin B.
B) It phosphorylates Rb to release E2F, enabling the S phase.
C) It stabilizes and activates p21, leading to cell cycle arrest at the G1/S checkpoint.
D) It degrades cyclin E, preventing entry into S phase.

3. What mechanism ensures the degradation of cyclin B at the end of mitosis?

A) Ubiquitination of cyclin B by the anaphase-promoting complex (APC)
B) Phosphorylation of cyclin B by cyclin A/Cdk1
C) Deactivation of Cdk1 by the Wee1 kinase
D) Inhibition of Cdk1 activity by the Cip/Kip family of inhibitors

4. What role does the Ras/Raf/ERK signaling pathway play in regulating the G1/S transition?

A) It stimulates the production of cyclin A, initiating DNA replication in S phase.
B) It promotes the synthesis of cyclin D, which activates Cdk4/Cdk6 and allows progression through the G1 restriction point.
C) It activates the p53 protein to induce DNA repair before S phase.
D) It inhibits Cdk2 activity to prevent premature S phase entry.

5. Which of the following is a direct consequence of a mutation in the Rb gene that leads to a loss of function?

A) Increased stability of cyclin B, leading to uncontrolled mitosis.
B) A failure to phosphorylate E2F, causing excessive expression of genes required for S phase entry.
C) Activation of p21 to prevent cell cycle progression in the presence of DNA damage.
D) Inhibition of cyclin E/Cdk2 complexes, leading to a delay in G1/S transition.

6. What is the role of the Cip/Kip family of Cdk inhibitors (e.g., p21, p27, p57) in the regulation of the cell cycle?

A) They activate Cdk2/Cyclin A to promote progression from G2 to M phase.
B) They inhibit cyclin D/Cdk4 and cyclin E/Cdk2 complexes, thus controlling G1 to S phase transitions.
C) They prevent the degradation of cyclins during mitosis.
D) They facilitate the phosphorylation of Rb by Cdk4/Cyclin D.

7. In the context of cancer, what is a likely outcome of a mutation that inactivates the Ink4 family of Cdk inhibitors?

A) Hyperactivation of Cdk4/Cdk6, promoting uncontrolled G1 to S phase progression.
B) Inhibition of the Ras/Raf/ERK pathway, preventing cyclin D synthesis.
C) Activation of p53, leading to permanent cell cycle arrest.
D) Enhanced degradation of cyclin E, blocking S phase entry.

8. During which phase of the cell cycle is DNA content measured by flow cytometry at its maximum intensity, corresponding to 4n?

A) G1 Phase
B) S Phase
C) G2 Phase
D) M Phase

9. Which of the following statements accurately describes the behavior of a zygote during early development?

A) Zygote cells enter G1 and G2 phases, with a slower, regulated cell cycle.
B) The zygote undergoes a series of S and M phases only, with no G1 or G2 phases.
C) Zygote cells become differentiated and stop dividing after several cycles.
D) Zygote cells enter a quiescent G0 phase to begin differentiation.

10. How do spindle assembly checkpoints prevent improper chromosome segregation during mitosis?

A) They monitor the alignment of chromosomes at the metaphase plate and halt progression if any chromosomes are not correctly attached to the spindle fibers.
B) They prevent the degradation of cyclin B to allow continued progression through mitosis.
C) They activate the synthesis of cyclin D to ensure continued G1 progression.
D) They induce apoptosis in cells that have incomplete DNA replication.

11. What is the effect of an ATM mutation on the DNA damage response during the cell cycle?

A) It prevents the phosphorylation and activation of p53, leading to failure of the DNA damage checkpoint.
B) It leads to the excessive degradation of cyclin E, inhibiting entry into S phase.
C) It activates cyclin D/Cdk4 complexes, promoting premature entry into S phase.
D) It stabilizes cyclin A, ensuring normal G1/S transition.

12. What key event marks the transition from G2 to M phase in the cell cycle?

A) Activation of Cdk2 by cyclin E
B) Phosphorylation of Rb by Cdk4/Cyclin D
C) Dephosphorylation of Cdk1 by CDC25
D) Activation of the p21 protein to inhibit Cdk2 activity

13. What occurs during the "restriction point" in the G1 phase of the cell cycle?

A) Cells pass through a critical checkpoint in response to external signals such as growth factors, committing to DNA replication and cell division.
B) DNA damage is repaired, and the cell waits for the next signal to enter G1.
C) Cyclin D binds to Cdk2, promoting entry into S phase.
D) The cell enters the G0 phase, where it becomes quiescent and does not divide.

14. In what way does the action of p53 relate to tumor suppression?

A) It activates cyclin D, promoting progression from G1 to S phase.
B) It inhibits the Ras/Raf/ERK pathway, preventing cell cycle progression.
C) It stabilizes p21, which inhibits Cdk2/Cyclin E complexes, thereby causing cell cycle arrest to prevent the propagation of damaged DNA.
D) It degrades cyclin B to prevent mitosis.

15. How does the degradation of cyclin B at the end of mitosis affect the cell cycle?

A) It triggers the activation of Cdk2, leading to DNA replication in the next cycle.
B) It prevents the transition from G2 to M phase.
C) It inactivates Cdk1, thereby halting mitosis and allowing the cell to exit mitosis.
D) It stimulates cyclin D synthesis to promote G1 progression.

Answers:

1. B
2. C
3. A

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

1. Which of the following statements about the regulation of the cell cycle by Cdk inhibitors (CKIs) is correct?

A) CKIs of the Cip/Kip family (e.g., p21, p27) are involved in inhibiting G1/S and G2/M transitions by binding to cyclins.
B) CKIs of the Ink4 family (e.g., p15, p16) inhibit Cyclin D/Cdk4/6 complexes, promoting G1/S phase progression.
C) CKIs only function in S phase to prevent DNA replication.
D) Both families of CKIs inhibit Cdk activity by preventing phosphorylation of cyclins.

2. What is the primary role of the proteasome during cell cycle regulation, particularly concerning cyclins and Cdk inhibitors?

A) The proteasome stabilizes cyclins to maintain consistent cell cycle progression.
B) The proteasome degrades cyclins and Cdk inhibitors to allow transition through key checkpoints, such as G1/S and G2/M.
C) The proteasome helps activate Cdk inhibitors by phosphorylating them.
D) The proteasome prevents the activation of cyclin-dependent kinases by removing DNA-binding proteins.

3. How does the phosphorylation of Cdk1 by CDC25 specifically facilitate the transition from G2 to M phase?

A) Phosphorylation of Cdk1 by CDC25 inactivates it, ensuring that the cell does not prematurely enter mitosis.
B) Phosphorylation of Cdk1 by CDC25 leads to the activation of the anaphase-promoting complex (APC), promoting mitotic exit.
C) CDC25 activates Cdk1 by removing inhibitory phosphates, thereby driving the cell into mitosis.
D) CDC25 phosphorylates Cdk1 to increase the stability of cyclin B, promoting mitotic entry.

4. Which of the following best explains why the zygote skips G1 and G2 phases during early division cycles?

A) The zygote has a reduced need for metabolic activities and DNA repair mechanisms in early development.
B) The zygote lacks the necessary resources to enter G1 and G2, so it only proceeds through S and M phases.
C) The zygote undergoes rapid cell division without needing to grow or replicate DNA.
D) The zygote maintains a G0 phase to prevent DNA replication until differentiation begins.

5. What role does the spindle assembly checkpoint play in preventing aneuploidy?

A) It ensures that DNA replication occurs without errors during S phase.
B) It prevents mitosis if the chromosomes are not correctly attached to spindle fibers, ensuring accurate segregation.
C) It arrests the cell cycle during G1 to repair DNA damage before entering S phase.
D) It activates Cdk1 to trigger chromosome condensation during early mitosis.

6. Which of the following is a result of the activation of the Ras/Raf/ERK signaling pathway in response to growth factors?

A) It promotes the degradation of Cdk2 and Cyclin A, preventing the G2/M transition.
B) It stimulates the synthesis of cyclin D, which binds to Cdk4/Cdk6, allowing the cell to pass through the G1 restriction point and enter S phase.
C) It prevents the binding of Cdk inhibitors, ensuring the activation of Cdk2/Cyclin E.
D) It activates p53 to arrest the cell cycle in response to DNA damage.

7. What is the effect of an ATM (Ataxia-telangiectasia mutated) protein mutation on the DNA damage checkpoint in the cell cycle?

A) It leads to a hyperactive p53 response, causing premature cell cycle arrest.
B) It prevents the phosphorylation of p53, blocking its stabilization and thus the activation of DNA repair mechanisms.
C) It allows for the unregulated degradation of cyclins, resulting in premature mitotic entry.
D) It enhances the activity of the anaphase-promoting complex (APC), facilitating mitotic exit.

8. How do quiescent cells (G0 phase) maintain metabolic activity without dividing?

A) They cease all protein synthesis and metabolic activities until re-entry into the cell cycle.
B) They continue to transcribe genes required for DNA replication but do not replicate their DNA.
C) They remain metabolically active, carrying out normal cellular functions while remaining out of the cell cycle.
D) They enter a state of permanent differentiation and cease all metabolic functions.

9. Which of the following statements is true about the role of cyclin E in the transition from G1 to S phase?

A) Cyclin E activates Cdk2, which inhibits DNA replication and prevents entry into S phase.
B) Cyclin E promotes the degradation of cyclin D, thus preventing cell cycle progression.
C) Cyclin E binds to Cdk2 to activate DNA replication and facilitate the entry into S phase.
D) Cyclin E inactivates Cdk4/Cdk6, which are necessary for the transition into G1.

10. What happens to the DNA content during the transition from G1 to S phase in terms of flow cytometry measurements?

A) The DNA content doubles from 2n to 4n, and flow cytometry shows a shift to high-intensity staining.
B) The DNA content remains constant at 2n, and flow cytometry shows low-intensity

staining.
C) The DNA content increases from 2n to an intermediate value, showing a gradual increase in flow cytometry staining intensity.
D) The DNA content decreases from 4n to 2n, with a decrease in flow cytometry staining intensity.

11. In cancer cells, what is the consequence of mutations in the p16INK4 gene?

A) p16INK4 overexpression leads to excessive inhibition of Cdk4/Cdk6, arresting the cell cycle in G1.
B) Loss of p16INK4 leads to uncontrolled activation of Cdk4/Cdk6, allowing continuous progression through the G1 restriction point.
C) p16INK4 mutations prevent cyclin E from binding to Cdk2, inhibiting progression into S phase.
D) p16INK4 mutations lead to increased expression of p21, blocking Cdk2/Cyclin E activity.

12. Why does the anaphase-promoting complex (APC) regulate cyclin B degradation during mitosis?

A) To allow for proper chromosome segregation and to signal the end of mitosis.
B) To prevent DNA replication by inactivating Cdk2/Cyclin A.
C) To prevent the formation of the mitotic spindle, inhibiting mitosis.
D) To activate cyclin D, ensuring proper entry into G1 phase.

13. How does the degradation of cyclin B at the end of mitosis contribute to the exit from mitosis and entry into G1?

A) It directly activates Cdk4/Cdk6, leading to the initiation of the next cell cycle.
B) It causes the dephosphorylation of Cdk1, inactivating it and terminating mitosis.
C) It promotes DNA repair processes before entering G1.
D) It prevents the activation of the spindle assembly checkpoint.

14. How does the activity of p21 affect the cell cycle when p53 is activated in response to DNA damage?

A) p21 binds to and inhibits Cdk4/Cdk6, preventing entry into S phase.
B) p21 binds to and inhibits Cdk2/Cyclin E, halting progression from G1 to S phase.
C) p21 activates Cdk1/Cyclin B, allowing the cell to proceed into mitosis.
D) p21 binds to Rb, releasing E2F and promoting S phase entry.

15. What is the clinical significance of targeting cyclin D in cancer therapy?

A) Cyclin D inhibition prevents DNA replication, which halts tumor growth by blocking S phase.
B) Cyclin D inhibition promotes uncontrolled cell growth by preventing G1/S checkpoint activation.
C) Cyclin D inhibition reduces the levels of p53, allowing damaged cells to continue dividing.
D) Cyclin D inhibition blocks progression through G1, thereby halting the growth of cancer cells that are dependent on cyclin D for progression into S phase.

Answers:

1. A

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

1. How does the action of p53 contribute to preventing the accumulation of mutations during the cell cycle?

A) By binding to and inactivating cyclin D, preventing the cell from entering the G1 phase.
B) By activating cyclin A, which promotes DNA replication in S phase.
C) By inhibiting the transition from G1 to S phase by activating p21, leading to cell cycle arrest.
D) By degrading cyclin B, ensuring proper progression from G2 to M phase.

2. What would be the likely consequence of a mutation that prevents the phosphorylation of Rb protein by Cyclin D/Cdk4?

A) Rb would remain bound to E2F transcription factors, preventing transcription of genes necessary for S phase entry, thus blocking the cell cycle progression from G1 to S.
B) Rb would be continuously phosphorylated, leading to excessive gene expression and uncontrolled entry into S phase.
C) The E2F transcription factors would remain inactive, preventing both DNA replication and G1/S transition.
D) Cyclin D/Cdk4 would be unable to initiate the cell cycle, causing a permanent G0 phase arrest.

3. Which of the following is the primary mechanism through which cyclin E/Cdk2 regulates the G1 to S phase transition?

A) By binding to and inactivating p53, preventing DNA damage-induced cell cycle arrest.
B) By promoting the degradation of Rb, allowing the release of E2F transcription factors to drive gene expression for DNA replication.
C) By deactivating the anaphase-promoting complex (APC), preventing mitosis.
D) By inhibiting the function of cyclin D, preventing progression through the G1 phase.

4. In the event of DNA damage during the S phase, what mechanism ensures that cells do not progress to G2/M before repair is completed?

A) The activation of p21, which inhibits cyclin A/Cdk2 complexes, halting progression through S phase and preventing entry into G2.
B) The activation of p16INK4, which inhibits Cdk2 activity and prevents DNA replication.
C) The dephosphorylation of Cdk1 by CDC25, ensuring that mitosis cannot begin

prematurely.
D) The binding of Rb to E2F, preventing the transcription of genes required for S phase.

5. What role does the anaphase-promoting complex (APC) play in mitotic progression and ensuring accurate chromosome segregation?

A) APC degrades cyclin B at the end of mitosis, allowing the cell to exit mitosis and enter G1.
B) APC activates Cdk2, driving the transition from G1 to S phase.
C) APC phosphorylates Rb, releasing E2F and promoting the entry into S phase.
D) APC binds to and inhibits the function of p21, preventing cell cycle arrest during DNA damage.

6. What is the effect of a mutation that prevents CDC25 from dephosphorylating Cdk1 during the G2 phase?

A) Cdk1 would remain inactive, delaying or preventing the onset of mitosis and the G2 to M phase transition.
B) Cdk1 would become constitutively active, leading to premature entry into M phase even before DNA replication is complete.
C) The cell would arrest in G1 due to excessive cyclin B degradation.
D) Cdk1 would be inactivated by p21, preventing the progression from G1 to S phase.

7. In the context of cancer, how might a mutation in the Ink4 family of Cdk inhibitors (such as p16INK4) contribute to tumorigenesis?

A) The loss of p16INK4 function would lead to continuous activation of Cyclin D/Cdk4, driving uncontrolled cell cycle progression from G1 to S phase and promoting tumor growth.
B) The loss of p16INK4 function would prevent the degradation of cyclin B, causing cell cycle arrest in G1.
C) The loss of p16INK4 function would activate the Ras/Raf/ERK signaling pathway, inhibiting cyclin E synthesis and preventing cell proliferation.
D) The loss of p16INK4 function would increase the activity of p21, causing a permanent G0 phase arrest.

8. What is the significance of G0 phase in cells like neurons and muscle cells, and how does it relate to the cell cycle?

A) G0 is a phase of complete cell cycle arrest where cells stop metabolizing and remain dormant.
B) G0 is a reversible phase in which cells remain metabolically active but do not replicate DNA or divide unless stimulated by external signals.
C) G0 cells can re-enter the cycle after receiving signals that promote progression from G1 to S phase.
D) G0 is a phase where cells continue to divide but do not undergo DNA replication.

9. How does flow cytometry distinguish cells in G1, S, and G2 phases based on their DNA content?

A) Cells in G1 have 4n DNA content, cells in S phase show intermediate staining intensity, and cells in G2 have 2n DNA content.
B) Cells in G1 have 2n DNA content and low staining intensity, cells in S phase have between 2n and 4n DNA content, and cells in G2 have 4n DNA content and high staining

intensity.
C) Cells in G1 have 2n DNA content, cells in S phase have 4n DNA content, and cells in G2 show intermediate intensity.
D) Cells in G1, S, and G2 all have identical DNA content but vary in staining intensity.

10. What could be the outcome if the activity of the Cdk inhibitors from the Cip/Kip family (e.g., p21) is reduced in a cancer cell?

A) Reduced Cip/Kip activity would prevent activation of cyclin E/Cdk2 complexes, thus halting the G1 to S phase transition.
B) Reduced Cip/Kip activity would enhance the activity of cyclin D/Cdk4, pushing the cell through the G1 restriction point without proper regulation.
C) Reduced Cip/Kip activity would increase the expression of cyclin B, promoting premature mitosis.
D) Reduced Cip/Kip activity would block DNA replication and prevent the transition from S to G2 phase.

11. What is the effect of the activation of the Ras/Raf/ERK pathway in the context of growth factor signaling during the G1 phase?

A) It activates cyclin E synthesis, leading to the progression through the G1/S transition by stimulating Cdk2 activity.
B) It inhibits the synthesis of cyclin D, preventing entry into G1 and slowing down cell proliferation.
C) It promotes the synthesis of cyclin D, which binds to and activates Cdk4/Cdk6, allowing progression through the G1 restriction point.
D) It directly activates p53, causing cell cycle arrest in G1 due to DNA damage.

12. In cells undergoing the mitotic spindle checkpoint, what ensures that chromosome segregation occurs correctly during metaphase?

A) The degradation of cyclin E, which prevents the transition from G1 to S phase.
B) The phosphorylation of p53, which stabilizes the cell cycle to ensure DNA replication is complete.
C) The activation of the anaphase-promoting complex (APC), which ensures proper chromosome attachment and segregation.
D) The inhibition of cyclin B, preventing mitotic entry.

13. Why do zygotes undergo rapid divisions without entering G1 or G2 phases, and what is the functional consequence of this behavior?

A) Zygotes skip G1 and G2 to accelerate cell division for early development, producing smaller cells without growth.
B) Zygotes divide rapidly but enter G1 and G2 phases after the third division, promoting differentiation.
C) Zygotes remain arrested in G0, preventing any form of cell division until later stages of embryonic development.
D) Zygotes require long G1 and G2 phases to repair DNA before entering mitosis, making early divisions slower.

14. How does the phosphorylation of Cdk2 during S phase regulate DNA replication?

A) Cdk2 phosphorylates cyclin A, preventing DNA replication from occurring.
B) Cdk2 activity is inhibited by the Cip/Kip family of Cdk inhibitors, ensuring DNA replication does not occur prematurely.
C) Cdk2 phosphorylates key proteins required for DNA replication, ensuring that the process proceeds smoothly during S phase.
D) Cdk2 inhibits p53, allowing the cell to bypass DNA damage checkpoints during replication.

15. How does the regulation of cyclin D/Cdk4 activity at the G1 restriction point impact cell cycle progression in response to growth factors?

A) The activation of cyclin D/Cdk4 at the G1 restriction point enables cells to pass through the checkpoint and proceed into S phase when growth factors are present.
B) The activation of cyclin D/Cdk4 inhibits the activity of cyclin E, preventing the transition from G1 to S phase.
C) Cyclin D/Cdk4 activation leads to the degradation of Rb, preventing the cell from entering G0.
D) Cyclin D/Cdk4 activity is blocked by growth factors, preventing any progression through the G1 restriction point.

Answers:

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

1. A 56-year-old patient is diagnosed with a mutation in the p16INK4 gene, a Cdk inhibitor. Which of the following is the most likely consequence of this mutation in the patient's cells?

A) Inhibition of cyclin D/Cdk4 complexes, resulting in cell cycle arrest at the G1 phase.
B) Unregulated activation of cyclin D/Cdk4 complexes, promoting cell cycle progression from G1 to S phase and potentially leading to uncontrolled cell division.
C) Increased expression of p21, leading to the inhibition of cyclin E/Cdk2 and G1/S transition arrest.
D) Premature degradation of cyclin B, resulting in a delay in entering mitosis.

2. A 33-year-old woman has a mutation in the ATM gene, leading to a loss of function of the ATM protein. This prevents proper activation of p53 in response

to DNA damage. Which of the following is the most likely outcome in this patient?

A) Increased activation of the spindle checkpoint during mitosis, preventing chromosomal missegregation.
B) Failure to induce p21 expression in response to DNA damage, allowing the cell to continue cycling despite the presence of DNA damage.
C) Enhanced repair of DNA damage due to a hyperactive G1/S checkpoint.
D) Premature entry into mitosis due to lack of cyclin D degradation in response to DNA damage.

3. In a clinical trial, a new drug is shown to inhibit cyclin E/Cdk2 activity. Which of the following cell cycle transitions would be most directly affected by this drug?

A) The transition from G0 to G1.
B) The transition from G1 to S phase.
C) The transition from S to G2 phase.
D) The transition from G2 to M phase.

4. A researcher is studying the anaphase-promoting complex (APC) in a cell culture model. Which of the following is the primary role of the APC in regulating the mitotic phase?

A) It promotes the degradation of cyclin B, allowing the cell to exit mitosis and enter G1.
B) It activates cyclin D, which drives the G1 to S phase transition.
C) It inhibits p53, allowing progression from G1 to S phase.
D) It binds to and degrades p21, preventing G1/S checkpoint arrest.

5. A 5-year-old child presents with retinoblastoma, which is associated with a mutation in the RB gene. Which of the following molecular events is most likely to be disrupted in this child's cells?

A) Release of E2F from Rb, allowing transcription of genes necessary for S phase entry.
B) Activation of cyclin E/Cdk2, driving the G1 to S phase transition.
C) Inhibition of p53, preventing DNA damage-induced cell cycle arrest.
D) Degradation of cyclin D, preventing progression through the G1 phase.

6. A 24-year-old patient is diagnosed with a form of cancer that involves mutations in the ras gene. How would this mutation most likely affect the cell cycle?

A) Activation of the Ras/Raf/ERK pathway, leading to increased cyclin D synthesis and continuous progression through the G1/S checkpoint.
B) Inactivation of cyclin D, leading to cell cycle arrest at the G1 phase.
C) Increased activity of p53, leading to the activation of p21 and cell cycle arrest.
D) Inhibition of cyclin E/Cdk2 complexes, preventing progression from G1 to S phase.

7. A 40-year-old woman undergoes chemotherapy for breast cancer. A flow cytometry analysis is performed to monitor the effects of the drug on her cell cycle. Which of the following would you expect to observe in cells treated with a drug that inhibits Cdk1 activity?

A) Accumulation of cells in G1 due to inhibition of G1/S progression.
B) Increased DNA content in cells during S phase due to unchecked DNA replication.
C) Arrest of cells in G2/M phase due to inability to progress through mitosis.
D) Premature progression from G2 to S phase despite incomplete DNA replication.

8. A patient has a mutation that prevents the activation of CDC25, the phosphatase responsible for dephosphorylating Cdk1. Which of the following is the most likely effect of this mutation on the cell cycle?

A) Premature activation of Cdk1, causing the cell to enter mitosis without completing DNA replication.
B) Delayed activation of Cdk1, preventing progression from G2 to M phase.
C) Enhanced progression through the G1 phase, accelerating entry into S phase.
D) Impaired degradation of cyclin B, resulting in prolonged mitosis.

9. A researcher is studying the role of p53 in a cultured cell line. Which of the following events is most directly regulated by p53 in response to DNA damage?

A) Activation of Cdk4/Cdk6 to promote progression from G1 to S phase.
B) Inhibition of Cdk2/Cyclin E complexes to arrest the cell cycle at G1/S.
C) Degradation of cyclin B to facilitate the exit from mitosis.
D) Phosphorylation of Rb to release E2F and allow progression into S phase.

10. A 60-year-old male patient presents with symptoms of prostate cancer. He is found to have mutations in cyclin D that lead to its overexpression. Which of the following is most likely to occur in this patient's cells?

A) Arrest at the G1 restriction point due to inhibition of cyclin D/Cdk4 activity.
B) Uncontrolled progression through the G1/S checkpoint, leading to increased cell proliferation.
C) Inhibition of p21 activity, leading to enhanced repair of DNA damage.
D) Failure to pass the G2/M checkpoint due to dysregulation of cyclin B.

11. A researcher is studying a novel drug that inhibits p21 activity. Which of the following effects would be expected in cells treated with this drug?

A) Increased Cdk2/Cyclin E activity, leading to accelerated G1 to S phase progression.
B) Increased activation of p53, resulting in cell cycle arrest at G1/S.
C) Decreased Cyclin D/Cdk4 activity, delaying the G1 to S transition.
D) Inhibition of Cdk1, causing the cell to arrest in G2 phase.

12. A 50-year-old male patient presents with DNA repair deficiencies and a history of multiple cancers. A genetic analysis reveals a defect in the ATM gene, which plays a key role in the DNA damage response. What is the most likely consequence of this mutation in the patient's cells?

A) Impaired ability to arrest the cell cycle at the G1/S checkpoint in response to DNA damage.
B) Loss of function of Cdk2, preventing progression through S phase.
C) Enhanced DNA replication, leading to chromosomal instability.
D) Unregulated progression through the G2/M checkpoint, resulting in premature mitotic entry.

13. A 28-year-old woman is diagnosed with a rare form of cancer that disrupts the regulation of Rb protein. Which of the following is most likely to be affected in her cells?

A) Unregulated release of E2F, driving excessive transcription of genes required for S phase entry.
B) Persistent inhibition of E2F, preventing entry into S phase.
C) Accumulation of cyclin E, preventing entry into G1.
D) Deactivation of p21, preventing cell cycle arrest in response to DNA damage.

14. A 45-year-old male is diagnosed with a tumor containing cells that are arrested in the G0 phase of the cell cycle. Which of the following is the most likely characteristic of these G0 cells?

A) They are metabolically inactive but incapable of entering the cell cycle.
B) They are quiescent but can re-enter the cell cycle in response to external signals such as growth factors or tissue damage.
C) They are permanently differentiated and unable to proliferate.
D) They undergo rapid DNA replication and mitosis despite being in G0.

15. A 32-year-old patient is diagnosed with a cancer that involves loss of p53 function. Which of the following cellular consequences would you most likely observe in this patient's tumor cells?

A) Increased activation of p21, causing a halt in the cell cycle at G1/S.
B) Unregulated progression through the cell cycle without DNA repair in response to damage.
C) Premature degradation of cyclin B, preventing entry into mitosis.
D) Enhanced repair of DNA damage, leading to stable chromosome segregation during mitosis.

Answers:

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

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