

Cell Division and Cancer Practice Exam (Sample)

Study Guide



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Questions

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- 1. What are cancer stem cells?**
 - A. A group of cells that can differentiate into various cell types**
 - B. A subpopulation of cancer cells that can self-renew and drive tumor growth**
 - C. The first cells to transform into cancerous cells**
 - D. Normal cells that assist in tumor suppression**
- 2. What is formed during the S phase regarding chromosomes?**
 - A. Each chromosome is broken into smaller fragments**
 - B. Each chromosome now has two sister chromatids**
 - C. Homologous chromosomes are aligned**
 - D. Cellular respiration processes are initiated**
- 3. How many mutated alleles of a proto-oncogene are necessary to potentially cause cancer?**
 - A. One allele**
 - B. Two alleles**
 - C. Three alleles**
 - D. Four alleles**
- 4. What occurs in the S phase of the cell cycle?**
 - A. Cell differentiation**
 - B. Synthesis of proteins**
 - C. DNA synthesis**
 - D. Cellular respiration**
- 5. Which of the following is considered a common hallmark of cancer?**
 - A. Increased angiogenesis**
 - B. Cellular quiescence**
 - C. Sustained proliferative signaling**
 - D. Decreased metabolism**

- 6. How does cell division contribute to tissue repair?**
- A. It enhances cell flexibility**
 - B. It generates energy for repairs**
 - C. It replaces damaged or dead cells**
 - D. It increases blood supply to tissues**
- 7. What occurs when Ras is malfunctioning in relation to cell division?**
- A. It remains inactive, preventing cell division**
 - B. It continually stimulates division leading to cancer**
 - C. It promotes apoptosis in cells**
 - D. It enhances the signaling pathways for cell repair**
- 8. What happens to chromatin during cell division?**
- A. It remains unchanged**
 - B. It condenses into chromosomes**
 - C. It transforms into RNA**
 - D. It becomes free-floating DNA**
- 9. What does apoptosis refer to?**
- A. Uncontrolled cell division**
 - B. Programmed cell death**
 - C. DNA damage repair**
 - D. Cellular differentiation process**
- 10. What defines the impact of p53 mutations in the context of cancer treatment?**
- A. They make cancer cells more sensitive to treatment**
 - B. They usually lead to better prognosis**
 - C. They contribute to treatment resistance**
 - D. They always decrease the rate of metastasis**

Answers

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

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Explanations

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1. What are cancer stem cells?

- A. A group of cells that can differentiate into various cell types
- B. A subpopulation of cancer cells that can self-renew and drive tumor growth**
- C. The first cells to transform into cancerous cells
- D. Normal cells that assist in tumor suppression

Cancer stem cells are a subpopulation of cancer cells that have the unique ability to self-renew and drive tumor growth. This concept is rooted in the understanding that not all cells within a tumor are the same; rather, some cells possess the characteristics of stem cells, which include the capacity to proliferate indefinitely and the ability to produce diverse cell types that make up the tumor. This self-renewal ability is critical because it allows cancer stem cells to perpetuate the tumor even after conventional therapies may have targeted the bulk of the cancer cells. The presence of these cells contributes to issues like tumor recurrence and metastasis, as they can survive treatments that eliminate other cancer cells. Understanding the role of cancer stem cells is vital for developing targeted therapies that can more effectively eliminate tumors by addressing the root of tumor growth rather than merely shrinking the overall mass of cancer cells. Recognizing this distinct population helps researchers and clinicians in designing strategies that will specifically target cancer stem cells for better treatment outcomes.

2. What is formed during the S phase regarding chromosomes?

- A. Each chromosome is broken into smaller fragments
- B. Each chromosome now has two sister chromatids**
- C. Homologous chromosomes are aligned
- D. Cellular respiration processes are initiated

During the S phase of the cell cycle, DNA synthesis takes place. This phase is crucial for ensuring that when the cell divides, each daughter cell receives an identical set of chromosomes. Specifically, during the S phase, each chromosome is replicated, resulting in two identical copies known as sister chromatids. These sister chromatids are joined together at a region called the centromere. The formation of sister chromatids is essential for the accurate distribution of chromosomes during cell division, specifically in mitosis and meiosis. The other options do not accurately represent what occurs during the S phase. Chromosomes are not broken into smaller fragments, homologous chromosomes align during a different stage of cell division (metaphase), and cellular respiration processes are separate from the events of the S phase. Thus, the formation of sister chromatids is a key defining feature of this phase, making it the correct answer.

3. How many mutated alleles of a proto-oncogene are necessary to potentially cause cancer?

- A. One allele**
- B. Two alleles**
- C. Three alleles**
- D. Four alleles**

The correct answer is that one mutated allele of a proto-oncogene is sufficient to potentially cause cancer. Proto-oncogenes are normal genes that promote cell growth and division. When these genes are mutated, they can become oncogenes, which can drive excessive cell proliferation and contribute to tumor formation. In general, only a single mutation in one of the alleles of a proto-oncogene is necessary for the gain of function that characterizes oncogenes. This is because most proto-oncogenes act in a dominant manner, meaning that the presence of just one mutant allele is enough to trigger the oncogenic behavior. This contrasts with tumor suppressor genes, where mutations in both alleles are typically required to eliminate their growth-inhibitory effects. In summary, a single mutated allele of a proto-oncogene can initiate a cascade of events that may ultimately lead to cancer, making the understanding of these mutations crucial in cancer biology and treatment strategies.

4. What occurs in the S phase of the cell cycle?

- A. Cell differentiation**
- B. Synthesis of proteins**
- C. DNA synthesis**
- D. Cellular respiration**

During the S phase of the cell cycle, DNA synthesis occurs as the cell prepares for division. This phase is crucial for the replication of the cell's genetic material, ensuring that each daughter cell will receive an identical set of chromosomes. In this phase, the entire genome is duplicated to produce two complete sets of DNA. This process is meticulously regulated and involves several key proteins and enzymes that help unwind the DNA strands, create new complementary strands, and ensure the accuracy of the replication to prevent mutations. Successful completion of the S phase is essential for the subsequent phases of the cell cycle, particularly mitosis, where the duplicated chromosomes are evenly distributed into the daughter cells. Understanding that DNA synthesis is a highly orchestrated process that prepares the cell for division highlights its fundamental role in growth, development, and maintenance of cellular function.

5. Which of the following is considered a common hallmark of cancer?

- A. Increased angiogenesis**
- B. Cellular quiescence**
- C. Sustained proliferative signaling**
- D. Decreased metabolism**

Sustained proliferative signaling is recognized as a hallmark of cancer because cancer cells develop the ability to continuously signal for growth and division, ignoring the normal regulatory mechanisms that limit cellular proliferation. In healthy tissues, cells receive growth signals that promote division in a controlled manner, while also having mechanisms to halt growth when necessary. In contrast, cancer cells can produce their own growth signals or hijack existing signaling pathways to maintain incessant proliferation, leading to tumor growth. This uncontrolled signaling is a key characteristic of cancer, allowing tumors to grow without regard for the normal constraints of cell division. Hence, this ability to sustain proliferative signals is fundamentally linked to the progression of many cancer types, making it a central feature in the development and maintenance of malignancies.

6. How does cell division contribute to tissue repair?

- A. It enhances cell flexibility**
- B. It generates energy for repairs**
- C. It replaces damaged or dead cells**
- D. It increases blood supply to tissues**

Cell division plays a fundamental role in tissue repair primarily by replacing damaged or dead cells. When tissues are injured, there is often a loss of cells that must be replenished to restore the structure and function of the tissue. Cell division allows for new cells to be generated, which can migrate into the damaged area and help to rebuild the tissue architecture. This process is crucial for recovery, as it enables the body to heal wounds, recover from injuries, and maintain overall tissue integrity. While enhancing cell flexibility, generating energy for repairs, and increasing blood supply to tissues can all contribute to the healing process, they do not directly address the need for replacing cells that have been lost or compromised. The process of mitosis, where one cell divides to form two identical daughter cells, is essential for increasing the cell population in the affected area, thus facilitating effective tissue repair and regeneration.

7. What occurs when Ras is malfunctioning in relation to cell division?

- A. It remains inactive, preventing cell division**
- B. It continually stimulates division leading to cancer**
- C. It promotes apoptosis in cells**
- D. It enhances the signaling pathways for cell repair**

When Ras is malfunctioning, it typically leads to a continuous stimulation of cell division, which can result in cancer. Ras is a type of oncogene that plays a critical role in transmitting signals within cells that promote growth and division. Under normal conditions, Ras functions as a switch that is activated by growth factors. When activated, it sends signals that stimulate the proliferation of the cell. If Ras becomes mutated or dysfunctional, it can remain in an active state without the appropriate external signals. This persistent activation leads to uncontrolled cellular proliferation, contributing to the development of tumors and cancer. In essence, the malfunctioning of Ras disrupts the tightly regulated process of cell division, promoting unchecked growth instead, which is a hallmark of many cancers. The other responses do not accurately reflect the role of Ras in cell division and its relationship to cancer progression. For instance, remaining inactive would prevent cell division rather than contribute to cancer, while promoting apoptosis would lead to cell death rather than division, and enhancing repair signaling does not directly tie to the oncogenic function of Ras in the context of cancer.

8. What happens to chromatin during cell division?

- A. It remains unchanged**
- B. It condenses into chromosomes**
- C. It transforms into RNA**
- D. It becomes free-floating DNA**

During cell division, chromatin undergoes a significant transformation in which it condenses into distinct structures known as chromosomes. This process is essential for ensuring proper segregation of genetic material into daughter cells. Chromatin, which is composed of DNA and proteins, exists in a more relaxed and elongated form during interphase, allowing for processes such as transcription and DNA replication to occur. As the cell enters mitosis, chromatin condensation occurs to facilitate the orderly distribution of genetic material. The condensation process involves the coiling and folding of chromatin fibers, making it compact enough to be moved without becoming tangled or damaged. Each chromosome consists of two sister chromatids joined at a region called the centromere, which ensures that each daughter cell receives an identical set of chromosomes during cell division. This condensation is critical because it allows chromosomes to be easily separated and accurately distributed to the resulting daughter cells. If chromatin did not condense, it would be far more challenging to segregate the genetic material during cell division, potentially leading to errors such as aneuploidy, where daughter cells end up with the wrong number of chromosomes.

9. What does apoptosis refer to?

- A. Uncontrolled cell division
- B. Programmed cell death**
- C. DNA damage repair
- D. Cellular differentiation process

Apoptosis refers to programmed cell death, which is a highly regulated and organized process that occurs in multicellular organisms. This process is crucial for maintaining homeostasis within the body by removing damaged, dysfunctional, or unnecessary cells in a controlled manner. Unlike necrosis, which is uncontrolled cell death resulting from external factors and can lead to inflammation, apoptosis is a deliberate process activated by various biochemical signals that ensure the safe elimination of cells without harming adjacent tissues. The mechanisms involved in apoptosis include characteristic cellular changes such as cell shrinkage, chromatin condensation, and the activation of specific enzymes called caspases that facilitate the breakdown of cellular components. This process plays a pivotal role in development, immune response, and prevention of cancer by eliminating cells that might potentially become cancerous due to irreparable DNA damage or abnormal growth signals. Thus, the term best captures the essence of how the body regulates cell population and health at a cellular level.

10. What defines the impact of p53 mutations in the context of cancer treatment?

- A. They make cancer cells more sensitive to treatment
- B. They usually lead to better prognosis
- C. They contribute to treatment resistance**
- D. They always decrease the rate of metastasis

The impact of p53 mutations in the context of cancer treatment is primarily characterized by their role in contributing to treatment resistance. The p53 protein is a crucial tumor suppressor that regulates the cell cycle, promotes DNA repair, and induces apoptosis in the presence of genetic damage. When mutations occur in the TP53 gene, the resulting p53 protein often loses its tumor-suppressing functions. This can lead to an impaired response to conventional therapies, such as chemotherapy and radiation, which rely on the activation of pathways that p53 helps regulate. In cancers with p53 mutations, cells may evade programmed cell death and continue to proliferate despite treatment. This not only makes these tumors more challenging to treat but also often correlates with a more aggressive disease course, leading to reduced overall survival rates. Thus, the presence of p53 mutations significantly complicates cancer treatment strategies and underscores the importance of developing therapies that can overcome this resistance.