

CELL DIVISION AND CANCER PRACTICE EXAM (SAMPLE)

STUDY GUIDE



SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

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THE FULL VERSION STUDY GUIDE

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Introduction

Preparing for a certification exam can feel overwhelming, but with the right tools, it becomes an opportunity to build confidence, sharpen your skills, and move one step closer to your goals. At Examzify, we believe that effective exam preparation isn't just about memorization, it's about understanding the material, identifying knowledge gaps, and building the test-taking strategies that lead to success.

This guide was designed to help you do exactly that.

Whether you're preparing for a licensing exam, professional certification, or entry-level qualification, this book offers structured practice to reinforce key concepts. You'll find a wide range of multiple-choice questions, each followed by clear explanations to help you understand not just the right answer, but why it's correct.

The content in this guide is based on real-world exam objectives and aligned with the types of questions and topics commonly found on official tests. It's ideal for learners who want to:

- Practice answering questions under realistic conditions,
- Improve accuracy and speed,
- Review explanations to strengthen weak areas, and
- Approach the exam with greater confidence.

We recommend using this book not as a stand-alone study tool, but alongside other resources like flashcards, textbooks, or hands-on training. For best results, we recommend working through each question, reflecting on the explanation provided, and revisiting the topics that challenge you most.

Remember: successful test preparation isn't about getting every question right the first time, it's about learning from your mistakes and improving over time. Stay focused, trust the process, and know that every page you turn brings you closer to success.

Let's begin.

How to Use This Guide

This guide is designed to help you study more effectively and approach your exam with confidence. Whether you're reviewing for the first time or doing a final refresh, here's how to get the most out of your Examzify study guide:

1. Start with a Diagnostic Review

Skim through the questions to get a sense of what you know and what you need to focus on. Your goal is to identify knowledge gaps early.

2. Study in Short, Focused Sessions

Break your study time into manageable blocks (e.g. 30 – 45 minutes). Review a handful of questions, reflect on the explanations.

3. Learn from the Explanations

After answering a question, always read the explanation, even if you got it right. It reinforces key points, corrects misunderstandings, and teaches subtle distinctions between similar answers.

4. Track Your Progress

Use bookmarks or notes (if reading digitally) to mark difficult questions. Revisit these regularly and track improvements over time.

5. Simulate the Real Exam

Once you're comfortable, try taking a full set of questions without pausing. Set a timer and simulate test-day conditions to build confidence and time management skills.

6. Repeat and Review

Don't just study once, repetition builds retention. Re-attempt questions after a few days and revisit explanations to reinforce learning. Pair this guide with other Examzify tools like flashcards, and digital practice tests to strengthen your preparation across formats.

There's no single right way to study, but consistent, thoughtful effort always wins. Use this guide flexibly, adapt the tips above to fit your pace and learning style. You've got this!

Questions

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1. How do mutations in the BRCA1 and BRCA2 genes affect cancer risk?

- A. They primarily lead to lung cancer
- B. They increase the risk of breast and ovarian cancers
- C. They decrease overall cancer risk
- D. They have no effect on cancer risk levels

2. What is chromatin?

- A. The tightly coiled form of DNA
- B. The coiled structure of RNA
- C. The uncoiled, less condensed form of DNA
- D. The structural unit of chromosomes

3. What happens to telomeres as cells divide?

- A. They remain constant in length
- B. They lengthen with each division
- C. They shorten, limiting cell divisions
- D. They are replaced by new genetic material

4. What is mitosis?

- A. The division of the cell's cytoplasm
- B. The process of nuclear division
- C. The formation of chromatin from chromosomes
- D. The synthesis of RNA for protein production

5. What is the main purpose of the cell cycle?

- A. To enable cells to grow and perform photosynthesis
- B. To allow cells to grow and divide
- C. To transport nutrients across the cell membrane
- D. To synthesize proteins required for cell function

6. What could happen if cell cycle checkpoints are not functioning properly?

- A. Normal cell growth
- B. Controlled cell division
- C. Uncontrolled cell division leading to cancer
- D. Increased cell apoptosis

7. What is an example of an anti-cancer strategy involving actin disruption?

- A. Agents that promote actin filament growth
- B. Experimental agents that stabilize actin filaments
- C. Drugs that depolymerize actin filaments to prevent cytokinesis
- D. Compounds that enhance actin interactions with microtubules

8. Give an example of a common tumor suppressor gene.

- A. BRCA1
- B. TP53 (p53)
- C. MYC
- D. EGFR

9. What is the primary role of checkpoints in the cell cycle?

- A. To ensure proper replication of DNA
- B. To allow cells to grow uncontrollably
- C. To prevent mutations and uncontrolled growth
- D. To promote rapid cell division

10. What is the main function of taxanes in cancer treatment?

- A. Inhibit DNA synthesis
- B. Prevent depolymerization of microtubules
- C. Cause cross-linking of DNA
- D. Block nucleotide synthesis

Answers

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

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Explanations

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1. How do mutations in the BRCA1 and BRCA2 genes affect cancer risk?

- A. They primarily lead to lung cancer
- B. They increase the risk of breast and ovarian cancers**
- C. They decrease overall cancer risk
- D. They have no effect on cancer risk levels

Mutations in the BRCA1 and BRCA2 genes are specifically linked to an increased risk of developing breast and ovarian cancers. These genes are crucial for the repair of DNA breaks through a process known as homologous recombination, which is essential for maintaining genomic stability. When these genes are mutated, the ability of cells to repair DNA damage is compromised, leading to the accumulation of genetic alterations that can result in cancer. It is particularly well-established that women with BRCA1 mutations have a significantly higher risk of breast and ovarian cancers, and BRCA2 mutations also confer a substantial risk for these cancers, as well as for other types of cancer such as pancreatic and prostate cancers. This connection between BRCA mutations and specific cancer risks is backed by extensive research, making it clear that these mutations are critical factors in assessing cancer risk for individuals.

2. What is chromatin?

- A. The tightly coiled form of DNA
- B. The coiled structure of RNA
- C. The uncoiled, less condensed form of DNA**
- D. The structural unit of chromosomes

Chromatin refers to the uncoiled, less condensed form of DNA that exists within the nucleus of a cell during interphase, when the cell is not dividing. In this relaxed state, DNA is more accessible for processes such as transcription and replication, allowing the genetic material to be expressed and utilized by the cell. During cell division, chromatin will condense to form distinct chromosomes, making it easier to segregate the genetic material into daughter cells. The characterization of chromatin as the uncoiled form of DNA differentiates it from tightly coiled structures, which are referred to as chromosomes. Understanding this distinction is key in the study of DNA organization and gene expression regulation. Additionally, chromatin consists of DNA wrapped around histone proteins, which plays a crucial role in the regulation of gene activity and the structural organization of the genomic DNA in the nucleus.

3. What happens to telomeres as cells divide?

- A. They remain constant in length
- B. They lengthen with each division
- C. They shorten, limiting cell divisions**
- D. They are replaced by new genetic material

Telomeres are repetitive DNA sequences located at the ends of chromosomes, and their primary function is to protect chromosome ends from degradation and prevent them from fusing with one another. As cells undergo division, particularly during mitosis, a phenomenon known as the "end replication problem" occurs. This leads to the progressive shortening of telomeres with each round of cell division. Once a threshold length is reached, the cell may enter a state known as senescence, where it stops dividing, or it may undergo apoptosis (programmed cell death). This mechanism serves as a natural limit on the number of times a cell can divide, playing a significant role in aging and the potential development of cancer. Cells with severely shortened telomeres can also be prone to genomic instability, which is a characteristic feature of cancerous cells. In summary, the correct answer reflects the biological reality that telomeres shorten with consecutive cell divisions, thereby limiting the number of times a cell can successfully replicate and maintain its genomic integrity.

4. What is mitosis?

- A. The division of the cell's cytoplasm
- B. The process of nuclear division**
- C. The formation of chromatin from chromosomes
- D. The synthesis of RNA for protein production

Mitosis is specifically defined as the process of nuclear division, which occurs during the cell cycle when replicated chromosomes are separated into two new nuclei. This fundamental process ensures that each daughter cell receives an identical set of chromosomes, maintaining genetic consistency across cellular generations. During mitosis, the cell goes through several distinct phases, including prophase, metaphase, anaphase, and telophase. Each stage is crucial for the orderly and accurate division of the genetic material. This nuclear division is essential for growth, repair, and asexual reproduction in multicellular organisms. While other processes mentioned in the choices involve critical cellular functions, they do not pertain to the definition of mitosis. For instance, the division of the cytoplasm is a separate process called cytokinesis, which occurs after mitosis. The formation of chromatin involves the packaging of DNA and typically occurs before mitosis begins. Furthermore, the synthesis of RNA for protein production pertains to gene expression and occurs during transcription, not during the mitotic division of the nucleus. These distinctions highlight the importance of understanding mitosis as a unique and specific phase within the broader context of the cell cycle.

5. What is the main purpose of the cell cycle?

- A. To enable cells to grow and perform photosynthesis
- B. To allow cells to grow and divide**
- C. To transport nutrients across the cell membrane
- D. To synthesize proteins required for cell function

The primary purpose of the cell cycle is to facilitate the growth and division of cells. The cell cycle consists of phases that allow a cell to replicate its DNA, grow in size, and ultimately divide to produce two daughter cells. This process is essential for growth, tissue repair, and reproduction in multicellular organisms. During the cell cycle, cells also undergo critical checks to ensure that division occurs properly, minimizing the risk of errors that could lead to cancer or other diseases. While growth is certainly involved in other cellular processes, the cell cycle uniquely encompasses the entire process from one division to the next, ensuring that both the genetic material is accurately replicated and that the cell divides in an organized fashion. This focus on growth and division sets it apart from other cellular functions like photosynthesis or transport of nutrients, which are specific to certain cell types and functions.

6. What could happen if cell cycle checkpoints are not functioning properly?

- A. Normal cell growth
- B. Controlled cell division
- C. Uncontrolled cell division leading to cancer**
- D. Increased cell apoptosis

When cell cycle checkpoints are not functioning properly, the regulation of the cell cycle is compromised. Checkpoints are critical control mechanisms that monitor and regulate the progression of a cell through the various phases of the cell cycle, ensuring that any potential errors in DNA replication or damage are addressed before the cell divides. If these checkpoints fail, cells can continue to divide unchecked, which can lead to uncontrolled cell division. This uncontrolled growth is a hallmark of cancer, as it allows for the accumulation of mutations and abnormalities that can further drive cancerous transformations. In contrast, scenarios such as normal cell growth, controlled cell division, or increased cell apoptosis are less likely to occur when cell cycle checkpoints are impaired. Normal cell growth and controlled division are dependent on the proper functioning of these checkpoints to prevent errors during division. Increased apoptosis may occur in some cases where cells are damaged, but when checkpoints malfunction, it is instead the lack of apoptotic signals that contributes to the survival and proliferation of cells that should not be dividing. Hence, the primary concern with dysfunctional checkpoints is the potential onset of cancerous growth due to unchecked cell division.

7. What is an example of an anti-cancer strategy involving actin disruption?

- A. Agents that promote actin filament growth
- B. Experimental agents that stabilize actin filaments
- C. Drugs that depolymerize actin filaments to prevent cytokinesis**
- D. Compounds that enhance actin interactions with microtubules

The strategy involving actin disruption as an anti-cancer approach primarily focuses on the manipulation of the cytoskeletal components that are critical for cell division. During cytokinesis, the process by which a cell divides its cytoplasm to produce two daughter cells, actin filaments play a vital role in forming the contractile ring that facilitates this separation. Drugs that depolymerize actin filaments can effectively interfere with this process. By disrupting the formation of actin filaments, these agents prevent the contractile machinery from organizing properly, thereby hindering cytokinesis. This interruption can lead to failed cell division, giving rise to cells that may be unable to proliferate—an important aspect in the context of cancer, where uncontrolled cell division is a hallmark of the disease. In contrast to the correct answer, other strategies such as promoting actin filament growth, stabilizing actin filaments, or enhancing actin-microtubule interactions do not hinder cell division through actin disruption, and they could potentially support tumor cell survival and proliferation. Thus, the use of drugs that depolymerize actin filaments is a targeted strategy to disrupt cancer cell division, making it an effective anti-cancer approach.

8. Give an example of a common tumor suppressor gene.

- A. BRCA1
- B. TP53 (p53)**
- C. MYC
- D. EGFR

TP53, commonly referred to as p53, serves as a quintessential example of a tumor suppressor gene. Tumor suppressor genes are critical for regulating cell division and maintaining genomic stability. They produce proteins that help prevent uncontrolled cell growth by promoting DNA repair, inducing apoptosis (programmed cell death), and halting the cell cycle in response to DNA damage. In the case of p53, it is often termed the "guardian of the genome" due to its role in monitoring cellular stress and genomic integrity. When DNA is damaged, p53 can activate genes that stop the cell cycle to allow for repair, or it can initiate apoptosis if the damage is irreparable. Mutations in the TP53 gene can lead to the loss of these essential functions, contributing to the development of various cancers since the cell can then proliferate uncontrollably. The other options provided represent different categories of genes: BRCA1 is also a tumor suppressor but not as universally recognized as p53; MYC is an oncogene that promotes cell proliferation; and EGFR is a receptor that, when mutated or overexpressed, can lead to tumorigenesis but does not function as a tumor suppressor. Therefore, TP53 stands out as a prime example of

9. What is the primary role of checkpoints in the cell cycle?

- A. To ensure proper replication of DNA
- B. To allow cells to grow uncontrollably
- C. To prevent mutations and uncontrolled growth**
- D. To promote rapid cell division

The primary role of checkpoints in the cell cycle is to prevent mutations and uncontrolled growth. Checkpoints serve as critical control mechanisms that monitor the integrity of the cell's genetic material and the cell's overall readiness to progress through different stages of the cell cycle. This includes verifying that DNA has been accurately replicated and ensuring that any damage is repaired before the cell divides. During these checkpoints, if any issues are detected—such as incomplete DNA replication or DNA damage—the cell cycle is halted, allowing the cell time to repair the damage or, if irreparable, to initiate programmed cell death (apoptosis). This regulatory function is crucial for maintaining genomic stability and preventing the development of cancer, as uncontrolled cell growth often arises from cellular abnormalities that checkpoints are designed to catch and rectify. By effectively managing these critical transitions, checkpoints help preserve the health of tissues and organisms as a whole. In contrast, allowing cells to grow uncontrollably would lead to tumorigenesis, which is contrary to the purpose of checkpoints. Promoting rapid cell division and ensuring proper replication of DNA are less comprehensive in capturing the full critical function of checkpoints compared to their role in preventing mutations and unchecked cellular proliferation.

10. What is the main function of taxanes in cancer treatment?

- A. Inhibit DNA synthesis
- B. Prevent depolymerization of microtubules**
- C. Cause cross-linking of DNA
- D. Block nucleotide synthesis

Taxanes are a class of chemotherapeutic agents that primarily function by preventing the depolymerization of microtubules, which are essential components of the cell's cytoskeleton. By stabilizing microtubules, taxanes effectively interfere with the mitotic spindle's ability to function correctly during cell division. This stabilization makes it impossible for cancer cells to complete mitosis, leading to cell cycle arrest and ultimately triggering apoptosis, or programmed cell death. This mechanism is particularly effective against rapidly dividing cancer cells, making taxanes a valuable tool in the treatment of various cancers, including breast and ovarian cancers. The other functions listed do not accurately describe the action of taxanes. Inhibiting DNA synthesis, causing cross-linking of DNA, and blocking nucleotide synthesis are mechanisms associated with different classes of chemotherapeutic agents rather than taxanes specifically.

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Next Steps

Congratulations on reaching the final section of this guide. You've taken a meaningful step toward passing your certification exam and advancing your career.

As you continue preparing, remember that consistent practice, review, and self-reflection are key to success. Make time to revisit difficult topics, simulate exam conditions, and track your progress along the way.

If you need help, have suggestions, or want to share feedback, we'd love to hear from you. Reach out to our team at hello@examzify.com.

Or visit your dedicated course page for more study tools and resources:

<https://celldivisioncancer.examzify.com>

We wish you the very best on your exam journey. You've got this!

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