DNA AND GENE EXPRESSION PRACTICE TEST (SAMPLE)

STUDY GUIDE

SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

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Table of Contents

Copyright 1

Table of Contents 2

Introduction 3

How to Use This Guide 4

Questions 5

Answers 8

Explanations 10

Next Steps 15

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

1. How does CRISPR-Cas9 enable targeted genome editing, and what are common repair outcomes after Cas9-induced double-strand breaks?

A. Cas9 creates a double-strand break at a guide RNA specified locus; repair by non-homologous end joining yields indels; or homology-directed repair uses a template for precise changes.

B. Cas9 integrates a plasmid randomly; no repair.

C. Cas9 stops transcription by binding to promoter.

D. Cas9 replicates the genome.

2. Which double-strand break repair pathway uses a homologous template for repair?

A. Non-homologous end joining

B. Homologous recombination

C. Base excision repair

D. Nucleotide excision repair

3. Semiconservative replication means that each daughter DNA molecule contains one parental strand and one newly synthesized strand. Which experimental evidence supported this model?

A. DNA replication results in two parental strands.

B. DNA replication is conservative.

C. Each daughter molecule contains one parental and one newly synthesized strand.

D. DNA replication results in two newly synthesized strands.

4. What term refers to the accumulation of mutations in a species over time?

A. Epigenetics

B. Activators

C. Hox Genes

D. Genetic Load

5. What is the role of a promoter in a cloning vector?

A. To terminate transcription

B. To replicate the plasmid

C. To regulate transcription levels of the inserted gene by controlling RNA polymerase binding

D. To drive transcription of the inserted gene by recruiting RNA polymerase

6. What is nonsense-mediated decay and when does it occur?

A. A quality-control mechanism that degrades mRNAs with premature stop codons to prevent truncated, potentially harmful proteins.

B. A process that enhances translation efficiency of all mRNAs.

C. A pathway that repairs DNA double-strand breaks.

D. A mechanism that degrades rRNA.

7. What is chromatin remodeling and how does it affect gene expression?

A. Replicating DNA during the S phase.

B. Transcribing DNA to RNA by RNA polymerase.

C. Exposing DNA by repositioning nucleosomes to allow transcription.

D. Stabilizing chromatin to prevent any transcription.

8. In translation initiation, what does the initiator tRNA do?

A. It brings the first amino acid to the ribosome.

B. It catalyzes peptide bond formation.

C. It recognizes stop codons.

D. It degrades mRNA.

9. Which enzyme links together the growing chain of RNA nucleotides during transcription using a DNA template?

A. DNA polymerase

B. Ligase

C. Helicase

D. RNA polymerase

10. What are the main types of mutation that alter a protein's amino acid sequence?

A. Missense, nonsense, silent, and frameshift mutations.

B. Silent mutations only.

C. Only frameshift mutations.

D. Splice-site mutations only.

Answers

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

Explanations

1. How does CRISPR-Cas9 enable targeted genome editing, and what are common repair outcomes after Cas9-induced double-strand breaks?

A. Cas9 creates a double-strand break at a guide RNA specified locus; repair by non-homologous end joining yields indels; or homology-directed repair uses a template for precise changes.

B. Cas9 integrates a plasmid randomly; no repair.

C. Cas9 stops transcription by binding to promoter.

D. Cas9 replicates the genome.

CRISPR-Cas9 edits targeted DNA by using a guide RNA to locate a specific sequence, and Cas9 then makes a clean double-strand break at that site. After the cut, the cell fixes the break mainly through two pathways. Non-homologous end joining quickly rejoin the ends and is error-prone, which often creates small insertions or deletions called indels that can disrupt gene function. If a donor DNA template is provided, the cell can use homology-directed repair to copy the template into the genome, allowing precise changes such as specific base substitutions or insertions. These outcomes reflect how editing works in practice: gene disruption via indels from NHEJ or precise edits via HDR with a template. Other statements miss the mechanism—for example, Cas9 does not randomly integrate plasmids, does not repress transcription merely by binding promoters, and does not replicate the genome.

2. Which double-strand break repair pathway uses a homologous template for repair?

A. Non-homologous end joining

B. Homologous recombination

C. Base excision repair

D. Nucleotide excision repair

Double-strand break repair that uses a homologous template relies on a nearly identical copy of the sequence to guide restoration, ensuring high accuracy. This process, homologous recombination, first processes the broken ends and then uses a sister chromatid or homologous chromosome as a template. The DNA strand from the template is copied into the break site, and the resulting structures are resolved to restore the original sequence. Because it requires a homologous template, this repair pathway is most active when a sister chromatid is available (during S and G2 phases) and tends to produce error-free repair. In contrast, non-homologous end joining simply ligates the broken ends together without using a template, which can introduce small insertions or deletions. Base excision repair and nucleotide excision repair address different types of DNA damage and do not repair a double-strand break by copying from a homologous template.

3. Semiconservative replication means that each daughter DNA molecule contains one parental strand and one newly synthesized strand. Which experimental evidence supported this model?

A. DNA replication results in two parental strands.

B. DNA replication is conservative.

C. Each daughter molecule contains one parental and one newly synthesized strand.

D. DNA replication results in two newly synthesized strands.

Semiconservative replication means each daughter DNA molecule has one parental (old) strand and one newly synthesized strand. The key evidence comes from the Meselson-Stahl experiment, which used density labeling of DNA. Bacteria were grown in heavy nitrogen to label the old strands, then switched to light nitrogen. After one round of replication, the DNA appeared with intermediate density, indicating each molecule contained one old and one new strand. After a second round, the distribution showed both intermediate and light DNA, still consistent with one old and one new strand per molecule. This pattern rules out purely conservative replication (which would give two old or two new molecules after the first round) and dispersive replication (which would keep all molecules at an intermediate density). Therefore, the observed results support the idea that each daughter molecule contains one parental and one newly synthesized strand.

4. What term refers to the accumulation of mutations in a species over time?

A. Epigenetics

B. Activators

C. Hox Genes

D. Genetic Load

Genetic load describes the decrease in a population's average fitness caused by the buildup of deleterious mutations over generations. Mutations continually arise, and while natural selection removes many harmful changes, some persist—especially if they're recessive or kept in check by drift in small populations. Over time, this accumulation lowers the overall vitality or reproductive success of the species, which is what the term genetic load captures. Epigenetics refers to heritable changes in gene expression that don't alter the DNA sequence, so it's not about accumulating mutations. Activators are proteins that boost transcription, not a concept about mutation buildup. Hox genes are developmental regulators that shape body plans, again unrelated to the accumulation of mutations over time.

5. What is the role of a promoter in a cloning vector?

A. To terminate transcription

B. To replicate the plasmid

C. To regulate transcription levels of the inserted gene by controlling RNA polymerase binding

D. To drive transcription of the inserted gene by recruiting RNA polymerase

Promoters are DNA sequences where RNA polymerase binds to begin transcription. In a cloning vector, placing a promoter upstream of the inserted gene provides the signal for the host cell's RNA polymerase to start transcribing that gene, effectively driving its expression. This is why the key role is to recruit RNA polymerase to initiate transcription of the inserted gene. Terminators stop transcription, and the origin of replication handles plasmid copying, so those functions lie outside the promoter's job. While promoter strength and regulatory elements can influence how much transcription occurs, the essential action is recruitment and initiation of transcription by RNA polymerase.

6. What is nonsense-mediated decay and when does it occur?

A. A quality-control mechanism that degrades mRNAs with premature stop codons to prevent truncated, potentially harmful proteins.

B. A process that enhances translation efficiency of all mRNAs.

C. A pathway that repairs DNA double-strand breaks.

D. A mechanism that degrades rRNA.

Nonsense-mediated decay is a cellular quality-control mechanism that degrades messenger RNAs containing premature stop codons to prevent production of truncated, potentially harmful proteins. It typically detects when a stop codon occurs earlier than intended, especially when there are exon junction complexes left behind after RNA splicing. When the ribosome encounters this premature termination, NMD factors recruit decay pathways that remove the message from the cell, often by decapping, deadenylation, or targeted cleavage. This disposal stops the faulty mRNA from being translated into a shortened, possibly harmful protein. It isn't about boosting translation, repairing DNA, or degrading ribosomal RNA.

7. What is chromatin remodeling and how does it affect gene expression?

A. Replicating DNA during the S phase.

B. Transcribing DNA to RNA by RNA polymerase.

C. Exposing DNA by repositioning nucleosomes to allow transcription.

D. Stabilizing chromatin to prevent any transcription.

Chromatin remodeling is the process that changes how DNA is wrapped around histones, altering access to the genetic code. It affects gene expression by repositioning, ejecting, or restructuring nucleosomes so promoter and regulatory regions become exposed or hidden from transcription machinery. When nucleosomes are moved away from a gene's promoter, RNA polymerase II and transcription factors can bind and initiate transcription, increasing gene expression. Conversely, tightening or blocking access reduces transcription. This remodeling is typically driven by ATP-dependent chromatin-remodeling complexes and can be reinforced by histone modifications that recruit these factors, shifting chromatin between open (euchromatin) and closed (heterochromatin) states. The other options describe DNA replication or transcription itself, or stabilization that prevents transcription, none of which capture how altering nucleosome positioning directly regulates gene activity.

8. In translation initiation, what does the initiator tRNA do?

A. It brings the first amino acid to the ribosome.

B. It catalyzes peptide bond formation.

C. It recognizes stop codons.

D. It degrades mRNA.

During translation initiation, the initiator tRNA delivers the first amino acid to the ribosome and helps establish the reading frame for the upcoming protein. In bacteria, this tRNA carries formylmethionine and pairs with the start codon AUG in the P site of the small subunit, guided by initiation factors. This placement sets the N-terminus and marks the true start of synthesis, so the ribosome knows where to begin adding amino acids. The actual formation of peptide bonds happens later when the ribosome catalyzes the bond between the growing chain and the next aminoacyl-tRNA. Stopping translation involves release factors recognizing stop codons, not the initiator tRNA, and mRNA degradation is a separate process. In eukaryotes, the initiator tRNA delivers methionine (not formylated) but serves the same role of starting the polypeptide and setting the frame.

9. Which enzyme links together the growing chain of RNA nucleotides during transcription using a DNA template?

A. DNA polymerase

B. Ligase

C. Helicase

D. RNA polymerase

RNA polymerase is the enzyme that links together the growing RNA chain during transcription using a DNA template. It reads the DNA template and catalyzes the formation of phosphodiester bonds between incoming ribonucleotides, building the RNA strand in the 5′ to 3′ direction and adding each nucleotide to the 3′ end. It does not require a primer, unlike DNA replication. The other enzymes have different roles: DNA polymerase makes DNA from a DNA template and typically needs a primer; ligase seals breaks in DNA; helicase unwinds the DNA helix to allow access.

10. What are the main types of mutation that alter a protein's amino acid sequence?

A. Missense, nonsense, silent, and frameshift mutations.

B. Silent mutations only.

C. Only frameshift mutations.

D. Splice-site mutations only.

Mutations that change the protein's amino acid sequence fall into three classic forms. A missense mutation changes one amino acid to another, altering the protein's building blocks. A nonsense mutation creates a premature stop codon, truncating the protein and usually damaging its function. A frameshift mutation results from insertions or deletions that shift the reading frame, so the entire downstream sequence is altered and most amino acids are wrong. Silent mutations, by contrast, do not change the amino acid sequence because the genetic code is redundant—different codons can encode the same amino acid—so they typically don't alter the protein's sequence. Splice-site mutations can affect which parts of the gene are included in the final mRNA, potentially changing the protein produced, but not by directly changing the amino acids in the coding sequence itself. So, the main types that directly alter the protein's amino acid sequence are missense, nonsense, and frameshift mutations.

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://dnageneexpression.examzify.com

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