

# ASCP Molecular Biology (MB) Technologist Practice Exam (Sample)

## Study Guide



**Everything you need from our exam experts!**

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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 many loci are considered 'core loci' by the FBI for the Combined DNA Indexing System (CODIS)?**
  - A. 10**
  - B. 13**
  - C. 15**
  - D. 20**
  
- 2. What is a consequence of qualitative defects in the hemoglobin molecule?**
  - A. Inability of hemoglobin to bind oxygen**
  - B. Increased oxygen saturation levels**
  - C. Enhanced red blood cell lifespan**
  - D. Formation of abnormal platelet aggregates**
  
- 3. What enzyme is primarily associated with QB replicase technology for amplification?**
  - A. DNA-dependent RNA polymerase**
  - B. RNA-dependent DNA polymerase**
  - C. RNA-dependent RNA polymerase**
  - D. DNA-dependent DNA polymerase**
  
- 4. What is the inheritance pattern of sickle cell anemia?**
  - A. Dominant**
  - B. X-linked**
  - C. Autosomal recessive**
  - D. Autosomal dominant**
  
- 5. What can minor histocompatibility antigens (mHags) cause after a transplant?**
  - A. Increased graft vs. tumor effects**
  - B. Enhanced immune tolerance**
  - C. Graft-vs-host disease and graft failure**
  - D. Successful long-term graft acceptance**

- 6. How do patients with metastatic colorectal carcinoma that have the KRAS mutation respond to anti-EGFR therapy?**
- A. They respond well and should receive this treatment**
  - B. They do not respond well and should not receive this treatment**
  - C. They may have mixed results with this treatment**
  - D. They become resistant after the first dose**
- 7. In the study by Avery, MacLeod, and McCarty, what treatment was used to destroy the transforming factor?**
- A. Protease**
  - B. Ribonuclease**
  - C. Deoxyribonuclease**
  - D. Heating**
- 8. What characterizes an oncogene?**
- A. A gene that inhibits cell growth**
  - B. A normal gene that can become mutated**
  - C. A gene that only promotes apoptosis**
  - D. A gene that leads to normal cell function**
- 9. In a qPCR Amplification plot, where is the threshold set?**
- A. Below background and in the exponential phase**
  - B. At the peak of the amplification curve**
  - C. Above background and in the exponential phase**
  - D. At the baseline of the data**
- 10. What is the rate of DNA translation?**
- A. 30 nucleotides per second**
  - B. 60 nucleotides per second**
  - C. 90 nucleotides per second**
  - D. 120 nucleotides per second**

## Answers

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

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## **Explanations**

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**1. How many loci are considered 'core loci' by the FBI for the Combined DNA Indexing System (CODIS)?**

- A. 10
- B. 13**
- C. 15
- D. 20

The FBI has designated 13 specific loci as 'core loci' for the Combined DNA Indexing System (CODIS). These core loci are essential for forensic DNA testing because they provide a standardized set of markers that allow for reliable comparisons of DNA profiles across different crime scenes and datasets. The selection of these loci is critical for ensuring that the DNA profiles generated are consistent and have sufficient discriminatory power to distinguish between individuals. The 13 loci include a combination of short tandem repeats (STRs), which are known to vary greatly among individuals, making them suitable for use in criminal investigations and paternity testing. This established standard facilitates the sharing of DNA information across jurisdictions, enhances the database's utility, and improves the chances of solving crimes through DNA evidence.

**2. What is a consequence of qualitative defects in the hemoglobin molecule?**

- A. Inability of hemoglobin to bind oxygen**
- B. Increased oxygen saturation levels
- C. Enhanced red blood cell lifespan
- D. Formation of abnormal platelet aggregates

Qualitative defects in the hemoglobin molecule typically refer to mutations or alterations in the structure of hemoglobin that impair its normal function. The most significant consequence of these defects is the inability of hemoglobin to effectively bind oxygen. Hemoglobin's primary role is to transport oxygen from the lungs to tissues and carry carbon dioxide from tissues back to the lungs. When qualitative defects occur, such as in conditions like sickle cell disease or various forms of thalassemia, the altered hemoglobin may have a diminished affinity for oxygen or it might not release oxygen efficiently to the tissues, resulting in inadequate oxygen delivery throughout the body. The other options do not accurately reflect the consequences of qualitative hemoglobin defects. Increased oxygen saturation levels would imply improved oxygen binding and transport, which typically does not occur with qualitative defects. Enhanced red blood cell lifespan is usually not associated with qualitative defects; in fact, some conditions can lead to a reduced lifespan of red blood cells. Finally, the formation of abnormal platelet aggregates relates to platelet function rather than hemoglobin function and is not a direct consequence of qualitative defects in hemoglobin.

**3. What enzyme is primarily associated with QB replicase technology for amplification?**

- A. DNA-dependent RNA polymerase**
- B. RNA-dependent DNA polymerase**
- C. RNA-dependent RNA polymerase**
- D. DNA-dependent DNA polymerase**

In QB replicase technology, the enzyme primarily associated with the amplification process is RNA-dependent RNA polymerase. This enzyme is capable of synthesizing RNA from an RNA template, playing a crucial role in amplifying the RNA sequences necessary for various applications, including those in molecular biology and virology. The significance of using RNA-dependent RNA polymerase lies in its ability to replicate the viral RNA genomes, which is essential for the QB phage system utilized in amplification techniques. This enzyme allows for the efficient production of multiple copies of RNA, thus facilitating further study or manipulation of the RNA molecules involved in the process. Other types of polymerases have distinct functions. DNA-dependent RNA polymerase synthesizes RNA from a DNA template and is not applicable in this context. RNA-dependent DNA polymerase, commonly associated with reverse transcription, converts RNA templates into DNA, which is also not the primary function in QB replicase technology. Lastly, DNA-dependent DNA polymerase focuses on synthesizing DNA from a DNA template, which is irrelevant to RNA amplification processes.

**4. What is the inheritance pattern of sickle cell anemia?**

- A. Dominant**
- B. X-linked**
- C. Autosomal recessive**
- D. Autosomal dominant**

Sickle cell anemia follows an autosomal recessive inheritance pattern. This means that an individual must inherit two copies of the mutated gene, one from each parent, to express the disease. In the case of sickle cell anemia, the mutation affects the HBB gene, which encodes the beta-globin subunit of hemoglobin. When both alleles are mutated, the hemoglobin produced is abnormal, leading to the characteristic sickle-shaped red blood cells, which can cause various complications. In autosomal recessive conditions, carriers (those with only one copy of the mutated gene) typically do not exhibit symptoms of the disease, as their normal allele compensates for the defective one. However, they can pass the mutated gene to their offspring, making it important to understand family history and genetic counseling for those who carry the sickle cell trait. This inheritance pattern highlights key aspects such as the roles of both parents in contributing to the trait and the potential for passing it on to the next generation.

**5. What can minor histocompatibility antigens (mHags) cause after a transplant?**

- A. Increased graft vs. tumor effects**
- B. Enhanced immune tolerance**
- C. Graft-vs-host disease and graft failure**
- D. Successful long-term graft acceptance**

Minor histocompatibility antigens (mHags) play a significant role in transplant immunology, particularly in the context of mismatched grafts. These antigens can trigger immune responses because they are recognized as foreign by the recipient's immune system. When mHags are present and not matched between the donor and recipient, the recipient's immune cells can mount an attack against the transplanted tissue. This immune response can lead to graft-versus-host disease (GVHD), where the donor immune cells recognize the recipient's tissues as foreign, causing inflammation and damage. Furthermore, the reaction to mHags can also contribute to the potential failure of the graft due to rejection processes initiated by the recipient's immune system. In contrast to other options, the presence of mHags typically does not enhance immune tolerance or contribute positively to graft acceptance. Instead, they are more associated with adverse outcomes such as graft-versus-host disease and the risk of graft failure. Understanding the influence of minor histocompatibility antigens is crucial for improving transplant outcomes and managing patient care post-transplantation.

**6. How do patients with metastatic colorectal carcinoma that have the KRAS mutation respond to anti-EGFR therapy?**

- A. They respond well and should receive this treatment**
- B. They do not respond well and should not receive this treatment**
- C. They may have mixed results with this treatment**
- D. They become resistant after the first dose**

Patients with metastatic colorectal carcinoma who possess a KRAS mutation generally do not respond well to anti-EGFR (epidermal growth factor receptor) therapy. The presence of a KRAS mutation indicates a downstream alteration in the signaling pathway that leads to cell growth and proliferation. This mutation results in a constitutively active signaling pathway, meaning that tumor growth can continue independently of EGFR signaling. As a consequence, anti-EGFR therapies, which are designed to block the effects of EGFR and inhibit cell growth, prove ineffective in this group of patients. The KRAS mutation effectively bypasses the blockade that these therapies attempt to impose, leading to poor treatment outcomes. Therefore, patients identified with this mutation are typically advised against receiving anti-EGFR therapy because it is unlikely to provide any meaningful clinical benefit. This biomarker testing for KRAS status before initiating treatment is crucial in the management of metastatic colorectal carcinoma to guide therapy choices appropriately.

**7. In the study by Avery, MacLeod, and McCarty, what treatment was used to destroy the transforming factor?**

- A. Protease**
- B. Ribonuclease**
- C. Deoxyribonuclease**
- D. Heating**

In the study conducted by Avery, MacLeod, and McCarty, the transforming factor was identified as DNA. To demonstrate this, they performed a series of experiments where various enzymes were used to treat different samples. The use of deoxyribonuclease (DNase) was crucial because this enzyme specifically hydrolyzes DNA, effectively destroying the transforming ability of the extract. When the DNA was degraded by DNase, the transformation of non-virulent bacteria into virulent bacteria could not occur, confirming that DNA was indeed the genetic material responsible for the transformation. The other treatments addressed different components of the cell and their potential roles in transformation. For instance, protease would degrade proteins, ribonuclease would target RNA, and heating would denature proteins but would not selectively eliminate DNA. The success of deoxyribonuclease in abrogating the transforming factor highlights the critical role of DNA in heredity and transformation, which was pivotal in advancing the understanding of molecular genetics.

**8. What characterizes an oncogene?**

- A. A gene that inhibits cell growth**
- B. A normal gene that can become mutated**
- C. A gene that only promotes apoptosis**
- D. A gene that leads to normal cell function**

An oncogene is characterized by its potential to promote uncontrolled cell growth and division, which can lead to cancer. In its normal state, an oncogene is referred to as a proto-oncogene, which is involved in regular cellular functions such as growth and division. However, when this gene undergoes mutations or is expressed at higher levels than normal, it can lead to a gain of function that contributes to the transformation of a normal cell into a cancerous one. The choice that identifies an oncogene as a normal gene that can become mutated accurately captures this critical aspect of its function. Mutations in proto-oncogenes can result in proteins that are hyperactive or constitutively active, leading to the promotion of malignancy. This is why understanding the transformation of proto-oncogenes to oncogenes is essential in the study of cancer biology and therapy. Other choices do not encapsulate the essence of what an oncogene represents: inhibiting growth is contrary to the defining characteristics of oncogenes, while the promotion of apoptosis does not align with the fundamental role of oncogenes in driving cellular proliferation rather than cell death. Finally, a gene that leads to normal cell function does not typically classify as an oncogene since oncogenes

**9. In a qPCR Amplification plot, where is the threshold set?**

- A. Below background and in the exponential phase**
- B. At the peak of the amplification curve**
- C. Above background and in the exponential phase**
- D. At the baseline of the data**

The threshold in a qPCR amplification plot is set above the background signal and within the exponential phase of the amplification curve. This is done to ensure that the threshold exceeds the noise levels that may occur at the starting phase of the reaction while capturing the true exponential growth of the PCR products. Setting the threshold in this manner allows for accurate quantification of the target DNA. During the exponential phase, the amount of DNA doubles with each cycle, making it the most reliable point for quantification, as the increase in signal is proportional to the starting template concentration. Thus, placing the threshold above the background ensures that only the specific amplification signal, which indicates the presence of the target, is measured, avoiding potential interference from non-specific signals or fluctuations in baseline readings. A threshold set below background would not effectively differentiate between the signal generated from the amplification and the noise inherent in the dataset, leading to unreliable results. Similarly, placing it at the peak of the curve would not account for the exponential nature of the reaction, and setting it at the baseline would not capture the amplification signal at all. Hence, the correct choice emphasizes the importance of establishing the threshold in a range that supports precise quantification of the amplifying target.

**10. What is the rate of DNA translation?**

- A. 30 nucleotides per second**
- B. 60 nucleotides per second**
- C. 90 nucleotides per second**
- D. 120 nucleotides per second**

The rate of DNA translation refers specifically to the speed at which ribosomes synthesize proteins from messenger RNA (mRNA). In eukaryotic cells, this process generally occurs at an approximate rate of 2 to 6 amino acids incorporated into a growing polypeptide chain per second, which translates to around 60 codons (or nucleotides) per second, given that each codon consists of three nucleotides. This rate can vary depending on several factors such as the specific organisms, cellular conditions, and the presence of various initiation factors and elongation factors that influence translation speed. However, the widely accepted and commonly cited rate of translation in eukaryotes is around 60 nucleotides per second, making it the correct choice for this question. Understanding this rate is crucial for molecular biology as it provides insight into the efficiency and regulation of protein synthesis within cells.

## 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](mailto:hello@examzify.com).**

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

**<https://ascpmolecularbiology.examzify.com>**

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

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