

COMAT Foundational Biomedical Science (FBS) 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. What factor in hyperkalemia is associated with metabolic acidosis?**
 - A. Medications**
 - B. Opportunistic infections**
 - C. Renal disease**
 - D. Cellular destruction**

- 2. What is the common symptom associated with peptic ulcers due to H. pylori infection?**
 - A. Pain reduced after meals**
 - B. Pain worse between meals when the stomach is empty**
 - C. Consistent abdominal pain regardless of eating**
 - D. Severe back pain**

- 3. What is a preferred treatment for hyperlipidemia?**
 - A. Atorvastatin**
 - B. Niacin**
 - C. Ezetimibe**
 - D. Gemfibrozil**

- 4. What is the function of the Anti-HBs marker in hepatitis B diagnostics?**
 - A. Indicates active infection**
 - B. Indicates past infection or vaccination**
 - C. Indicates high infectivity**
 - D. Indicates window period**

- 5. Which of the following electrolytes is often found to be low in conjunction with elevated IgM in hyper IgM syndrome?**
 - A. Calcium**
 - B. Potassium**
 - C. Sodium**
 - D. Magnesium**

- 6. What does "polished ivory appearance of bone" indicate?**
- A. Eburnation in end stage osteoarthritis**
 - B. Bone marrow edema in osteomyelitis**
 - C. Subchondral sclerosis in rheumatoid arthritis**
 - D. Osteopenia in early osteoporosis**
- 7. What is the clinical significance of HBeAg in hepatitis?**
- A. Indicates liver inflammation**
 - B. Sign of liver cell necrosis**
 - C. Key marker for infectivity**
 - D. Indicates acute liver failure**
- 8. Which essential enzyme complex is thiamine vital for?**
- A. Fatty acid synthase**
 - B. Pyruvate dehydrogenase complex**
 - C. Glycogen synthase**
 - D. Ribonucleotide reductase**
- 9. What is a common side effect of thiazide diuretics?**
- A. Hypokalemia**
 - B. Hyperkalemia**
 - C. Hypertension**
 - D. Dehydration**
- 10. What does carotid artery atherosclerosis result in?**
- A. Increased blood flow to the brain**
 - B. Narrowing and hardening of the arteries**
 - C. Formation of new blood vessels**
 - D. Increased oxygen delivery to tissues**

Answers

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

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Explanations

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1. What factor in hyperkalemia is associated with metabolic acidosis?

- A. Medications**
- B. Opportunistic infections**
- C. Renal disease**
- D. Cellular destruction**

In hyperkalemia, metabolic acidosis often arises due to cellular destruction. This process releases potassium into the extracellular space, leading to elevated serum potassium levels, or hyperkalemia. When cells break down, they not only release potassium but also free hydrogen ions, which contribute to a decrease in blood pH, hence inducing metabolic acidosis. While certain medications may have effects on potassium levels, they do not directly cause the metabolic acidosis in the context of hyperkalemia.

Opportunistic infections are also not primarily responsible for the interplay between hyperkalemia and metabolic acidosis. Renal disease can lead to hyperkalemia due to the impaired excretion of potassium, but the direct relationship between cellular destruction and metabolic acidosis is more significant. Therefore, cellular destruction is a key factor linking hyperkalemia with metabolic acidosis, highlighting the intricate balance between cellular integrity, electrolyte levels, and acid-base homeostasis in the body.

2. What is the common symptom associated with peptic ulcers due to H. pylori infection?

- A. Pain reduced after meals**
- B. Pain worse between meals when the stomach is empty**
- C. Consistent abdominal pain regardless of eating**
- D. Severe back pain**

The common symptom associated with peptic ulcers caused by H. pylori infection is characterized by pain that worsens between meals when the stomach is empty. This occurs because the acidic environment of an empty stomach can further irritate the ulcerated area. When individuals with peptic ulcers do not eat, they do not provide food in the stomach to buffer the acid, leading to increased discomfort. In contrast, eating can temporarily alleviate the pain as food can serve to neutralize stomach acid. This is why many patients find that their symptoms may improve after meals and then return as their stomach empties. Recognizing this pattern is important for diagnosing and managing peptic ulcers associated with H. pylori infection.

3. What is a preferred treatment for hyperlipidemia?

- A. Atorvastatin
- B. Niacin**
- C. Ezetimibe
- D. Gemfibrozil

Atorvastatin is widely recognized as a first-line treatment for hyperlipidemia, particularly for individuals with elevated low-density lipoprotein (LDL) cholesterol levels. This medication belongs to the statin class, which works by inhibiting the enzyme HMG-CoA reductase. This enzyme plays a crucial role in the cholesterol biosynthesis pathway in the liver. By blocking this enzyme, atorvastatin effectively lowers LDL cholesterol levels, reduces cardiovascular risk, and may also have beneficial effects on triglycerides and high-density lipoprotein (HDL) cholesterol levels. While niacin, ezetimibe, and gemfibrozil also have roles in managing lipid levels, they are generally not considered first-line treatments. Niacin primarily serves to raise HDL cholesterol levels and lower triglycerides, but it can have side effects that limit its use in some patients. Ezetimibe works by reducing intestinal absorption of cholesterol, often used in conjunction with statins, but is typically not effective as a standalone therapy. Gemfibrozil is used mainly for lowering triglycerides and increasing HDL levels, but it is less effective than statins in lowering LDL cholesterol. Due to the efficacy of atorvastatin in managing hyperlipidemia and its established benefits

4. What is the function of the Anti-HBs marker in hepatitis B diagnostics?

- A. Indicates active infection
- B. Indicates past infection or vaccination**
- C. Indicates high infectivity
- D. Indicates window period

The Anti-HBs marker, or antibody to hepatitis B surface antigen, is critical in the diagnosis and management of hepatitis B infection. Its presence in a patient's serum signifies that the individual has either cleared a past infection with the hepatitis B virus (HBV) or has been successfully vaccinated against the virus. This is important as it reflects the immune response to the HBV, showing that the body has produced antibodies specifically in response to past exposure to the virus or as a result of vaccination. Testing for Anti-HBs is a key component in determining an individual's immune status regarding hepatitis B. A positive result is typically interpreted as immunity, while a negative result might suggest that there has been no prior infection or vaccination. Therefore, understanding the presence of Anti-HBs helps healthcare providers formulate appropriate medical decisions regarding future vaccinations, screening for liver disease, and potential risks of transmission. The other options are linked to different markers or situations in the context of hepatitis B diagnostics. For instance, the presence of HBsAg typically indicates an active infection, while HBeAg would be associated with high infectivity. The window period in hepatitis B refers to a phase when neither HBsAg nor Anti-HBs are detectable, which can complicate the diagnosis. Thus, the

5. Which of the following electrolytes is often found to be low in conjunction with elevated IgM in hyper IgM syndrome?

- A. Calcium**
- B. Potassium**
- C. Sodium**
- D. Magnesium**

In hyper IgM syndrome, an immunological condition characterized by elevated levels of immunoglobulin M (IgM) and deficient immunoglobulin G (IgG) and immunoglobulin A (IgA), the common finding of low calcium levels can be attributed to the underlying dysfunction related to the immune system and possibly effects on the parathyroid hormone or vitamin D metabolism. Calcium plays a critical role in various physiological processes, including cell signaling and the function of immune cells. In the context of hyper IgM syndrome, the impaired immune response can lead to alterations in calcium metabolism, resulting in hypocalcemia. This may be exacerbated by deficiencies in vitamin D absorption due to gastrointestinal complications or decreased liver function, which can also accompany immune dysfunction. While low levels of potassium, sodium, or magnesium can occur in different clinical contexts, they are not prominently associated with hyper IgM syndrome in the same way that low calcium levels are. Thus, the connection between elevated IgM levels and concurrent low calcium is well established in the clinical understanding of this syndrome, making it the most appropriate choice in this context.

6. What does "polished ivory appearance of bone" indicate?

- A. Eburnation in end stage osteoarthritis**
- B. Bone marrow edema in osteomyelitis**
- C. Subchondral sclerosis in rheumatoid arthritis**
- D. Osteopenia in early osteoporosis**

The term "polished ivory appearance of bone" refers specifically to eburnation, which is a condition often seen in the end stages of osteoarthritis. Eburnation occurs when the cartilage in a joint is worn away, leading to exposure of the underlying bone. This exposed bone undergoes changes characterized by smoothness and density, giving it a polished, ivory-like appearance. In osteoarthritis, as the disease progresses, the continuous stress and friction in the joint cause the bone to remodel and become denser. This can result in the thickening and smoothing of the bone surface, resulting in the distinct appearance that is noted during radiographic evaluations. Other conditions listed do not present with this specific morphology. For example, bone marrow edema associated with osteomyelitis typically presents with signals of inflammation on imaging rather than a polished surface. Subchondral sclerosis in rheumatoid arthritis may indeed involve changes in the bone, but the appearance is more related to increased density rather than the smooth, polished nature seen with eburnation. Osteopenia in early osteoporosis indicates a reduction in bone density rather than a polished appearance, and it would not produce the smooth surface characteristic of eburnation. Thus, the polished ivory appearance of bone is a clinical radi

7. What is the clinical significance of HBeAg in hepatitis?

- A. Indicates liver inflammation
- B. Sign of liver cell necrosis
- C. Key marker for infectivity**
- D. Indicates acute liver failure

HBeAg, or Hepatitis B e-antigen, serves as a crucial marker in the clinical assessment of hepatitis B virus (HBV) infection. It indicates active viral replication and high levels of infectious virus in the patient's blood. When HBeAg is present, it signifies that the virus is actively replicating, which correlates with a higher risk of transmission and greater infectivity. This is particularly important for guiding treatment decisions and infection control measures, as patients with detectable HBeAg are more likely to spread the virus to others. In contrast, the other options do not accurately capture the specific role of HBeAg in the context of hepatitis. While liver inflammation can occur during HBV infection, it is not directly indicated by the presence of HBeAg. Similarly, while liver cell necrosis can happen in chronic hepatitis, this is not what HBeAg specifically reveals. Lastly, HBeAg is not indicative of acute liver failure, which is a separate clinical phenomenon that relates to the severity of liver damage rather than the active state of viral replication. Understanding the significance of HBeAg can help in monitoring disease progression and treatment response, making it an essential aspect of managing patients with hepatitis B infection.

8. Which essential enzyme complex is thiamine vital for?

- A. Fatty acid synthase
- B. Pyruvate dehydrogenase complex**
- C. Glycogen synthase
- D. Ribonucleotide reductase

Thiamine, also known as vitamin B1, is a crucial coenzyme for several important enzyme complexes in metabolism, particularly in carbohydrate metabolism. The pyruvate dehydrogenase complex is one such vital enzyme complex that requires thiamine to function. This complex plays a critical role in converting pyruvate, derived from glycolysis, into acetyl-CoA, which then enters the citric acid cycle for energy production. In the pyruvate dehydrogenase complex, thiamine is incorporated as thiamine pyrophosphate (TPP), which is essential for the decarboxylation reaction of pyruvate. The process involves the removal of a carbon dioxide molecule, ultimately facilitating the transition from anaerobic to aerobic metabolism and linking glycolysis with the citric acid cycle. Deficiencies in thiamine can lead to impaired energy metabolism and conditions such as Wernicke-Korsakoff syndrome and beriberi. The other enzyme complexes listed do not rely on thiamine as a cofactor. Fatty acid synthase is involved in fatty acid synthesis and utilizes different cofactors. Glycogen synthase is responsible for glycogen synthesis and operates independently of thiamine. Ribonucleotide reductase is

9. What is a common side effect of thiazide diuretics?

- A. Hypokalemia**
- B. Hyperkalemia**
- C. Hypertension**
- D. Dehydration**

Thiazide diuretics are well-known for their ability to promote diuresis, which involves the excretion of excess fluid through urine. One of the common side effects of thiazide diuretics is hypokalemia, which refers to a lower than normal concentration of potassium in the blood. This occurs because thiazide diuretics increase the reabsorption of sodium in the distal convoluted tubule of the nephron, while simultaneously leading to increased potassium excretion. As sodium is reabsorbed, potassium is lost in the urine, resulting in a significant reduction in serum potassium levels. Monitoring potassium levels is crucial for patients on thiazide diuretics because severe hypokalemia can lead to serious complications such as arrhythmias and muscle weakness. Educating patients about the importance of maintaining appropriate potassium intake through diet or supplementation may also be recommended to mitigate this side effect.

10. What does carotid artery atherosclerosis result in?

- A. Increased blood flow to the brain**
- B. Narrowing and hardening of the arteries**
- C. Formation of new blood vessels**
- D. Increased oxygen delivery to tissues**

Carotid artery atherosclerosis is characterized by the buildup of plaque within the carotid arteries, which supply blood to the brain. This condition leads to narrowing (stenosis) of the arteries and hardening of the arterial walls. As plaque accumulates, the lumen of the carotid arteries becomes constricted, reducing blood flow to the brain. The hardening is due to the loss of elasticity in the arterial walls, which can also increase the risk of other complications such as thrombus formation or embolic events. In contrast, the other options describe physiological occurrences that do not directly relate to the effects of carotid artery atherosclerosis. For example, atherosclerosis does not increase blood flow to the brain nor does it facilitate the formation of new blood vessels in the affected areas; rather, it can lead to diminished perfusion. Likewise, this condition does not increase oxygen delivery to tissues due to the reduced blood flow caused by the narrowed arteries. Thus, the predominant result of carotid artery atherosclerosis is indeed the narrowing and hardening of the arteries, which is accurately captured by the correct response.

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://comatfbs.examzify.com>

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

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