# NBME Immunology Practice Test (Sample)

## Study Guide


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

# Table of Contents

# 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. What is the main difference between hyperacute and acute rejection after transplantation?**

**A. Hyperacute is antibody-mediated and occurs within minutes to hours; acute involves T-cell and antibody mechanisms occurring days to weeks after transplant**

**B. Hyperacute is T-cell mediated and occurs within days; acute is antibody-mediated within minutes**

**C. Hyperacute occurs weeks after transplant; acute occurs within hours**

**D. Hyperacute and acute are both antibody-mediated within minutes**

**2. Which component is central to all three complement pathways (classical, lectin, and alternative)?**

**A. C3**

**B. C5**

**C. C1q**

**D. Factor B**

**3. Which of the following is NOT a step in MHC class I antigen processing?**

**A. Proteasomal degradation of cytosolic proteins**

**B. TAP transport of peptides into the endoplasmic reticulum**

**C. Loading onto MHC II in endosomes**

**D. Surface expression of peptide-MHC I complex**

**4. Which immunoglobulin class predominantly exists as a pentamer in the serum?**

**A. IgG**

**B. IgA**

**C. IgM**

**D. IgE**

**5. HIV infection primarily leads to immunodeficiency by which mechanism?**

**A. Depletion of B cells due to direct infection of B lymphocytes**

**B. Loss of innate immune signaling due to macrophage dysfunction**

**C. Depletion of CD4+ T helper cells via infection of CD4-expressing cells, leading to impaired adaptive immunity**

**D. Deficiency of complement components leading to reduced opsonization**

**6. What is the role of maternal IgG in immunity?**

**A. Provides passive immunity to the fetus/neonate by crossing the placenta.**

**B. Provides active immunity to the fetus through in utero immune priming.**

**C. Stimulates development of the fetal immune system via thymic education.**

**D. Induces autoimmunity in the newborn.**

**7. Hyperacute graft rejection is best explained by which immunologic mechanism?**

**A. Type I hypersensitivity with IgE-mediated mast cell degranulation**

**B. Type IV hypersensitivity T cell-mediated**

**C. Type III immune complex deposition**

**D. Type II hypersensitivity with antibody-mediated vascular injury and complement activation**

**8. Common variable immunodeficiency is best described as which of the following?**

**A. Hyper-IgM syndrome**

**B. Severe combined immunodeficiency**

**C. IgA deficiency**

**D. Common variable immunodeficiency**

**9. Autoantibodies in myasthenia gravis target which molecule?**

**A. Voltage-gated calcium channels**

**B. Acetylcholine receptors**

**C. Myelin basic protein**

**D. Acetylcholinesterase**

**10. Which autoantibody is most specific for the diagnosis of systemic lupus erythematosus?**

**A. Anti-Smith (anti-Sm) antibodies**

**B. Anti-dsDNA antibodies**

**C. Anti-Ro (SSA) antibodies**

**D. Antiphospholipid antibodies**

# Answers

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

# Explanations

## 1. What is the main difference between hyperacute and acute rejection after transplantation?

**A. Hyperacute is antibody-mediated and occurs within minutes to hours; acute involves T-cell and antibody mechanisms occurring days to weeks after transplant**

**B. Hyperacute is T-cell mediated and occurs within days; acute is antibody-mediated within minutes**

**C. Hyperacute occurs weeks after transplant; acute occurs within hours**

**D. Hyperacute and acute are both antibody-mediated within minutes**

**The timing and the immune mechanism are what differentiate hyperacute from acute rejection. Hyperacute rejection happens almost immediately to a few hours after transplant and is driven by preformed recipient antibodies against donor antigens (such as ABO or HLA), with complement activation leading to rapid vascular injury and graft loss. Acute rejection occurs days to weeks after transplant and is primarily T-cell–mediated, as host T cells react against donor MHC antigens; there can be antibody-mediated components as well, but the cellular response is the key feature and occurs later. This matches the idea that hyperacute is antibody-driven and occurs within minutes to hours, while acute involves T-cell and possibly antibody mechanisms over days to weeks.**

## 2. Which component is central to all three complement pathways (classical, lectin, and alternative)?

**A. C3**

**B. C5**

**C. C1q**

**D. Factor B**

**The central idea is that all three complement pathways converge at activation of C3. In the classical and lectin pathways, activation begins with C1 complex or MASP binding to target surfaces, leading to formation of the C3 convertase C4b2a. In the alternative pathway, C3 undergoes spontaneous hydrolysis and, with factor B and D, forms the C3 convertase C3bBb. In all routes, C3 is cleaved into C3a and C3b. C3b deposition drives opsonization and, together with other components, forms C5 convertases to cleave C5 and generate the membrane attack complex, while C3a acts as an inflammatory mediator. Because this activation step is required for all three pathways to propagate the cascade, C3 is the component central to the system. The other options are more pathway-specific or act downstream (for example, C5 appears later; C1q initiates only the classical pathway; Factor B is specific to the alternative pathway).**

## 3. Which of the following is NOT a step in MHC class I antigen processing?

**A. Proteasomal degradation of cytosolic proteins**

**B. TAP transport of peptides into the endoplasmic reticulum**

**C. Loading onto MHC II in endosomes**

**D. Surface expression of peptide-MHC I complex**

**MHC class I antigen processing relies on endogenous proteins being degraded by the proteasome, with the resulting peptides transported into the endoplasmic reticulum by TAP. In the ER, these peptides are loaded onto newly formed MHC class I molecules with the help of chaperones and editing proteins, and the peptide-MHC I complex is then trafficked to the cell surface for presentation to CD8+ T cells. This pathway is focused in the cytosol and the ER, not in endosomes. Loading peptides onto MHC II in endosomes is not part of this process. MHC class II molecules present exogenous antigens that are taken up by endocytosis and processed in endosomal/lysosomal compartments, with peptide loading occurring there, often involving invariant chain and HLA-DM. The step described—loading onto MHC II in endosomes—belongs to the MHC class II pathway, not to MHC class I.**

## 4. Which immunoglobulin class predominantly exists as a pentamer in the serum?

**A. IgG**

**B. IgA**

**C. IgM**

**D. IgE**

**IgM is the immunoglobulin class that predominantly exists as a pentamer in the serum. This pentameric form arises when five IgM units come together with a joining chain, creating ten antigen-binding sites. The high valency gives IgM very strong overall binding (high avidity) to multivalent antigens, which makes it especially effective at agglutination and at activating the classical complement pathway. This is why IgM is produced first in a primary immune response. Other isotypes are monomeric (IgG and IgE) or, for IgA, typically dimeric in secretions (though serum IgA is usually monomeric). Thus, the pentameric arrangement in serum points to IgM as the predominant form.**

**5. HIV infection primarily leads to immunodeficiency by which mechanism?**

**A. Depletion of B cells due to direct infection of B lymphocytes**

**B. Loss of innate immune signaling due to macrophage dysfunction**

**C. Depletion of CD4+ T helper cells via infection of CD4-expressing cells, leading to impaired adaptive immunity**

**D. Deficiency of complement components leading to reduced opsonization**

**HIV immunodeficiency primarily comes from loss of CD4+ T helper cells, the cells that coordinate the entire adaptive immune response. The virus infects cells that express CD4, using coreceptors to enter them; this leads to death of these helper T cells and a progressive drop in their numbers. When CD4+ T cells are depleted, B cells receive less help to produce high-affinity antibodies and class-switched responses, and cytotoxic T cell responses are less effectively primed. The result is a broad failure of adaptive immunity, paving the way for opportunistic infections and malignancies associated with AIDS. Other ideas—such as direct depletion of B cells, major loss of innate signaling from macrophages, or a primary deficiency in complement—don't account for the central, coordinated role of CD4+ T cells in orchestrating immune responses, which is why this mechanism best explains HIV-related immunodeficiency.**

**6. What is the role of maternal IgG in immunity?**

**A. Provides passive immunity to the fetus/neonate by crossing the placenta.**

**B. Provides active immunity to the fetus through in utero immune priming.**

**C. Stimulates development of the fetal immune system via thymic education.**

**D. Induces autoimmunity in the newborn.**

**Maternal IgG provides passive immunity to the fetus by crossing the placenta and entering the fetal circulation. This transfer happens mainly in the third trimester via Fc receptors (FcRn) in the placenta, so the newborn benefits from antibodies the mother already has without having to produce its own response. This is different from active immunity, where the individual generates its own antibodies after exposure to an antigen and can develop memory. The fetus's immune system isn't being primed in utero by maternal antibodies to create its own protective memory, and maternal IgG does not induce thymic education or autoimmunity in the newborn. After birth, maternal IgG wanes, and the infant must rely on its own immune responses.**

## 7. Hyperacute graft rejection is best explained by which immunologic mechanism?

**A. Type I hypersensitivity with IgE-mediated mast cell degranulation**

**B. Type IV hypersensitivity T cell-mediated**

**C. Type III immune complex deposition**

**D. Type II hypersensitivity with antibody-mediated vascular injury and complement activation**

**Hyperacute graft rejection is driven by preformed antibodies against donor antigens, such as ABO or HLA, that bind to the donor endothelium and fix complement. This antibody binding sets off a cascade that injures the vascular lining, activates platelets, and causes rapid thrombosis of graft vessels, leading to immediate graft failure. This is the classic Type II hypersensitivity mechanism: antibody-mediated destruction of target cells or tissues with complement involvement. In contrast, a Type I response involves IgE and mast cell degranulation (allergic reactions), a Type IV response is a delayed T cell–mediated reaction, and a Type III response involves immune complex deposition. The rapid vascular injury and clotting seen in hyperacute rejection point to the antibody-mediated, complement-activating process described here.**

## 8. Common variable immunodeficiency is best described as which of the following?

**A. Hyper-IgM syndrome**

**B. Severe combined immunodeficiency**

**C. IgA deficiency**

**D. Common variable immunodeficiency**

**Common variable immunodeficiency represents a defect in B cell maturation and antibody production, leading to low levels of immunoglobulins—especially IgG—with poor antibody responses to vaccines. This results in recurrent bacterial infections, particularly of the sinopulmonary tract, often beginning in adolescence or adulthood. The hallmark is hypogammaglobulinemia with impaired specific antibody production, while T-cell function is typically preserved. This differs from hyper-IgM syndrome, where class-switching from IgM to other isotypes is defective, leading to normal or high IgM with low IgG/IgA/IgM; from severe combined immunodeficiency, which involves profound defects in both T and B cell immunity presenting early in infancy; and from IgA deficiency, which is mainly a selective loss of IgA with generally normal IgG and fewer systemic antibody-production issues.**

## 9. Autoantibodies in myasthenia gravis target which molecule?

**A. Voltage-gated calcium channels**

**B. Acetylcholine receptors**

**C. Myelin basic protein**

**D. Acetylcholinesterase**

**In myasthenia gravis the immune system makes antibodies against the postsynaptic nicotinic acetylcholine receptors at the neuromuscular junction. When these antibodies bind the receptors, they cause cross-linking and internalization of the receptors and activate complement, damaging the postsynaptic membrane and reducing the number of functional ACh receptors. With fewer receptors, the normally released acetylcholine fails to produce a sufficient end-plate potential to trigger a muscle action potential, leading to fatigable weakness that worsens with use. That's why therapies that boost acetylcholine in the synapse, like acetylcholinesterase inhibitors, can improve transmission. The other options point to different disorders or targets: antibodies against presynaptic voltage-gated calcium channels occur in Lambert-Eaton, myelin basic protein relates to demyelinating diseases like MS, and acetylcholinesterase is the enzyme that breaks down acetylcholine rather than a typical autoimmune target.**

## 10. Which autoantibody is most specific for the diagnosis of systemic lupus erythematosus?

**A. Anti-Smith (anti-Sm) antibodies**

**B. Anti-dsDNA antibodies**

**C. Anti-Ro (SSA) antibodies**

**D. Antiphospholipid antibodies**

**Anti-Smith antibodies are the most specific marker for systemic lupus erythematosus. These antibodies target small nuclear ribonucleoprotein complexes that are essentially unique to SLE, so their presence strongly supports the diagnosis. They're not very sensitive—many people with SLE don't have anti-Sm—but when you do detect anti-Sm, it's highly indicative of SLE. Anti-dsDNA antibodies are also associated with SLE and relate to disease activity, especially lupus nephritis, but they can appear in a small subset of other conditions, so they're less specific than anti-Sm. Anti-Ro/SSA antibodies are more commonly linked to Sjögren's syndrome (and can occur in a subset of SLE patients), so they're not as specific to SLE. Antiphospholipid antibodies can be found in several diseases and even in healthy individuals, so they're not diagnostic on their own for SLE. In short, a positive anti-Smith antibody test is the most specific serologic finding for confirming SLE.**

# 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://nbmeimmunology.examzify.com**

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