NBME IMMUNOLOGY PRACTICE TEST (SAMPLE)

STUDY GUIDE



SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

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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.

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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!

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Questions

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1. The J chain in immunoglobulins is primarily responsible for which function?

A. Initiating isotype switching

B. Stabilizing IgG structure

C. Linking monomers to form polymeric IgA and IgM

D. Enhancing antigen affinity

2. Which antibody mediates immunity to parasitic worms?

A. IgG

B. IgA

C. IgE

D. IgM

3. Which antibody class crosses the placenta to provide passive immunity to the fetus/neonate?

A. IgA

B. IgM

C. IgG

D. IgE

4. Which immunodeficiency is characterized by severe combined immunodeficiency with defective thymic T-cell development, often requiring stem cell transplantation for survival?

A. Wiskott-Aldrich syndrome

B. SCID (severe combined immunodeficiency)

C. X-linked agammaglobulinemia

D. Hyper-IgM syndrome

5. What is the end product of the complement cascade that forms pores in microbial membranes?

A. Membrane attack complex (MAC; C5b-9)

B. Opsonization by C3b

C. Anaphylatoxin C5a

D. C1q initiator complex

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6. How do natural killer cells detect virally infected cells and mediate cytotoxicity?

A. By recognizing increased MHC I expression

B. By recognizing decreased MHC I expression and inducing apoptosis via perforin/granzyme

C. By phagocytosing infected cells

D. By producing antibodies against infected cells

7. Hyperacute transplant rejection is characterized by which mechanism and timing?

A. Acute rejection: CD8+ T cells reacting against foreign MHCs

B. Graft-versus-host disease: donor T cells attack host

C. Chronic rejection: fibrosis months later

D. Hyperacute rejection: preformed antibodies (type II) - occurs in minutes

8. X-linked agammaglobulinemia is due to a defect in which protein?

A. Bruton tyrosine kinase

B. CD40L

C. AID

D. RAG1

9. Which statement about central tolerance and peripheral tolerance is correct?

A. Central tolerance occurs during lymphocyte development in the thymus or bone marrow; peripheral tolerance occurs in mature lymphocytes to prevent autoimmunity.

B. Central tolerance occurs in mature lymphocytes; peripheral occurs during development in thymus and bone marrow.

C. Both central and peripheral tolerance occur only in the thymus.

D. Tolerance is not essential for preventing autoimmunity.

10. Which immunotherapy uses monoclonal antibodies to block PD-1/PD-L1 or CTLA-4 to enhance anti-tumor immunity?

A. Cytokine therapy with IL-2

B. Adoptive transfer of NK cells

C. CAR-T cell therapy

D. Immune checkpoint inhibitors

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Answers

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

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Explanations

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1. The J chain in immunoglobulins is primarily responsible for which function?

A. Initiating isotype switching

B. Stabilizing IgG structure

C. Linking monomers to form polymeric IgA and IgM

D. Enhancing antigen affinity

The joining (J) chain's main role is to link antibody subunits to form polymeric immunoglobulins, specifically dimers of IgA and pentamers of IgM. This polymerization enables these antibodies to be secreted across mucosal surfaces and to function effectively in mucosal immunity. It's not involved in triggering isotype switching (that's driven by cytokines and activation-induced cytidine deaminase), nor does it stabilize IgG (IgG is a monomer), and it doesn't directly enhance antigen affinity (affinity maturation comes from somatic hypermutation and selection).

2. Which antibody mediates immunity to parasitic worms?

A. IgG

B. IgA

C. IgE

D. IgM

IgE is the antibody specialized for defense against large extracellular parasites like helminths. When parasite antigens are bound by IgE, the Fc region engages Fc receptors on eosinophils, as well as on mast cells and basophils. This sets up eosinophil-mediated cytotoxicity: eosinophils attach to IgE-coated parasites and release granule proteins such as major basic protein that damage the worm's surface. At the same time, mast cell and basophil degranulation releases mediators that increase mucus production, smooth muscle contraction, and vascular permeability, helping to expel the parasite. The Th2 response drives IgE production and eosinophil activation, reinforcing this defense. Other antibodies like IgG, IgM, or IgA have their roles (IgG in opsonization, IgM in early responses, IgA at mucosal surfaces), but they're not as specialized for targeting large helminths as IgE is.

3. Which antibody class crosses the placenta to provide passive immunity to the fetus/neonate?

A. IgA

B. IgM

C. IgG

D. IgE

The key idea is that only one antibody class is actively transported from mother to fetus to provide passive immunity: IgG. This transfer happens through the placental Fc receptor (FcRn), which binds the Fc portion of IgG in maternal blood, shuttles it across placental cells, and releases it into fetal circulation. This gives the newborn maternal antibodies that help protect against infections for the first months of life. Other antibody classes don't cross the placenta effectively. IgM is a large pentamer and cannot pass the placental barrier. IgA mainly protects mucosal surfaces and is supplied to the newborn mainly through breast milk in secretory form, not by placental transfer. IgE is not transferred across the placenta either and is involved in allergy and parasite defense at mucosal surfaces. So, IgG is the antibody class responsible for passive immunity acquired in utero and continued after birth, making it the correct choice.

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4. Which immunodeficiency is characterized by severe combined immunodeficiency with defective thymic T-cell development, often requiring stem cell transplantation for survival?

A. Wiskott-Aldrich syndrome

B. SCID (severe combined immunodeficiency)

C. X-linked agammaglobulinemia

D. Hyper-IgM syndrome

Severe combined immunodeficiency is defined by a profound defect in T-cell development in the thymus, which cripples cellular immunity and, as a consequence, impairs humoral responses as well. When thymic T-cell maturation is blocked, you lose mature CD4+ and CD8+ T cells, and without functional T-helper activity, B cells don't receive the signals they need to generate proper antibody responses. Infants with this condition typically present early with recurrent, severe infections, failure to thrive, chronic diarrhea, and thrush. Because the immune system is so severely compromised, definitive treatment often requires hematopoietic stem cell transplantation to reconstitute immune function; gene therapies are being used for some genetic forms (like ADA-SCID or IL-2R gamma chain defects). Other immunodeficiencies differ in which cells are affected: Wiskott-Aldrich has eczema, thrombocytopenia, and immunodeficiency; X-linked agammaglobulinemia features absent or very few B cells and low immunoglobulins with relatively normal T cells; Hyper-IgM syndrome involves defective class switching with high or normal IgM but low IgG/IgA, and normal or near-normal T-cell numbers. The hallmark of this scenario is the defective thymic T-cell development leading to a life-threatening, early-onset combined immunodeficiency that often needs stem cell transplantation for survival.

5. What is the end product of the complement cascade that forms pores in microbial membranes?

A. Membrane attack complex (MAC; C5b-9)

B. Opsonization by C3b

C. Anaphylatoxin C5a

D. C1q initiator complex

The key concept is how the complement system ends by creating a pore-forming structure that lyses microbes. After activation, C5 is split to C5b, which then sequentially binds C6, C7, C8, and multiple C9 molecules to form the membrane attack complex. This MAC inserts into the microbial membrane and creates a transmembrane pore, disrupting the membrane and causing lysis. Other outcomes of complement, like C3b-mediated opsonization or C5a-driven inflammation, do not produce pores, and C1q is involved in initiating the pathway rather than forming the pore-forming complex.

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6. How do natural killer cells detect virally infected cells and mediate cytotoxicity?

A. By recognizing increased MHC I expression

B. By recognizing decreased MHC I expression and inducing apoptosis via perforin/granzyme

C. By phagocytosing infected cells

D. By producing antibodies against infected cells

Natural killer cells patrol for cells with signs of stress or infection by sensing the absence of normal self markers, particularly MHC class I molecules. When a virally infected cell downregulates MHC I to evade CD8+ T cells, inhibitory signals on NK cells decline and activating signals prevail, tipping the balance toward activation. Once activated, NK cells unleash cytotoxic granules containing perforin and granzymes. Perforin forms pores in the target cell membrane, allowing granzymes to enter and activate caspases, driving the target cell to apoptosis. This allows rapid elimination of infected cells without requiring antigen presentation on MHC I. NK cells can also kill via antibody-dependent mechanisms when antibodies coat targets, but the described action is the recognition of decreased MHC I with perforin/granzyme–mediated apoptosis.

7. Hyperacute transplant rejection is characterized by which mechanism and timing?

A. Acute rejection: CD8+ T cells reacting against foreign MHCs

B. Graft-versus-host disease: donor T cells attack host

C. Chronic rejection: fibrosis months later

D. Hyperacute rejection: preformed antibodies (type II) - occurs in minutes

Hyperacute rejection hinges on preformed antibodies against donor antigens, a type II hypersensitivity reaction. In the recipient, circulating antibodies (often against donor HLA or ABO antigens) bind the graft endothelium and activate complement, triggering rapid thrombosis of graft vessels and ischemic graft failure. Because these antibodies are already present before transplantation, the reaction happens within minutes to hours after the transplant. This immediate, antibody-mediated process distinguishes it from other forms: acute rejection is mainly T cell–mediated against donor MHCs and occurs days to weeks later; graft-versus-host disease involves donor T cells attacking the recipient, typically after hematopoietic stem cell transplantation; chronic rejection develops months later with fibrotic changes.

8. X-linked agammaglobulinemia is due to a defect in which protein?

A. Bruton tyrosine kinase

B. CD40L

C. AID

D. RAG1

Bruton's tyrosine kinase is essential for signaling through the B cell receptor and for B cell maturation. When BTK is defective, B cells cannot progress from the pro-B/pre-B stage to mature B cells, so there are very few circulating B cells and immunoglobulins of all isotypes are markedly reduced. This humoral immunodeficiency presents with recurrent bacterial infections in affected males, reflecting the X-linked inheritance of BTK. In contrast, issues with CD40L impair class switching (Hyper-IgM due to CD40-CD40L interaction), AID affects class switching and somatic hypermutation, and RAG1 defects disrupt V(D)J recombination, leading to more severe combined immunodeficiencies. Therefore, the defect described fits Bruton's tyrosine kinase.

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9. Which statement about central tolerance and peripheral tolerance is correct?

A. Central tolerance occurs during lymphocyte development in the thymus or bone marrow; peripheral tolerance occurs in mature lymphocytes to prevent autoimmunity.

B. Central tolerance occurs in mature lymphocytes; peripheral occurs during development in thymus and bone marrow.

C. Both central and peripheral tolerance occur only in the thymus.

D. Tolerance is not essential for preventing autoimmunity.

Central tolerance is established during lymphocyte development in primary lymphoid organs—thymus for T cells and bone marrow for B cells—where self-reactive clones are deleted or inactivated through negative selection. Peripheral tolerance then acts on mature lymphocytes outside these organs to prevent autoimmunity, using mechanisms such as anergy, regulatory T cell suppression, clonal deletion in the periphery, or ignorance of sequestered antigens. This combination fits the statement: central tolerance during development in thymus or bone marrow, and peripheral tolerance in the periphery to prevent autoimmunity. The other options misstate where tolerance occurs or its necessity—central tolerance does not occur in mature lymphocytes, peripheral tolerance is not limited to the thymus, and tolerance is essential to prevent autoimmunity.

10. Which immunotherapy uses monoclonal antibodies to block PD-1/PD-L1 or CTLA-4 to enhance anti-tumor immunity?

A. Cytokine therapy with IL-2

B. Adoptive transfer of NK cells

C. CAR-T cell therapy

D. Immune checkpoint inhibitors

Immune checkpoint inhibitors use monoclonal antibodies to block inhibitory signals that restrain T cells, boosting anti-tumor immunity. CTLA-4 and PD-1 are brakes on T-cell activation: CTLA-4 competes with CD28 for B7, so blocking CTLA-4 enhances T cell priming and activation; PD-1 on T cells binds PD-L1 in the tumor environment to dampen activity, and blocking PD-1 or PD-L1 reactivates exhausted T cells to attack cancer cells. This mechanism is distinct from cytokine therapy, NK cell transfer, or CAR-T cell therapy, which rely on different approaches rather than blocking these checkpoints with monoclonal antibodies.

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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://nbmeimmunology.examzify.com

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

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