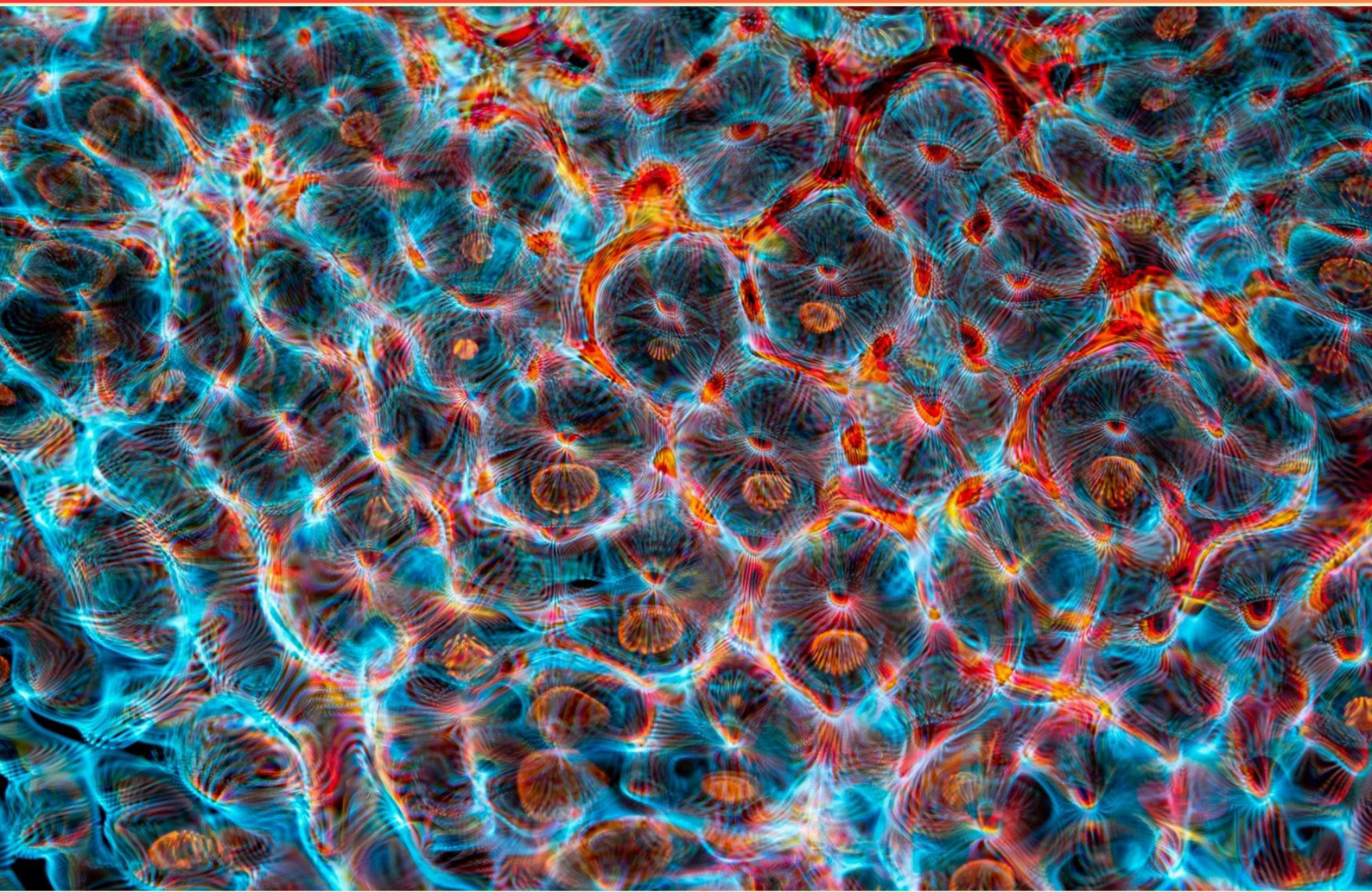


UNIVERSITY OF CENTRAL FLORIDA (UCF) PCB3233 IMMUNOLOGY EXAM 2 PRACTICE TEST (SAMPLE)

STUDY GUIDE



SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

**VISIT US ONLINE FOR
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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. Which statement about Type IV hypersensitivity is correct?

- A. Delayed-type hypersensitivity is mediated by T cells and typically occurs 24-72 hours after exposure.
- B. It is mediated primarily by IgE antibodies.
- C. It results from immune complex deposition.
- D. It requires cytotoxic T cells to directly lyse target cells.

2. Which statement about T cell costimulation is correct?

- A. Provides a necessary costimulatory signal that enhances TCR signaling via CD28-B7 interactions.
- B. Inhibits TCR signaling to prevent autoimmunity.
- C. Acts as an antigen receptor on B cells.
- D. Is a cytokine produced by T cells to recruit neutrophils.

3. Which cytokine profile best defines a Th1 response?

- A. IL-4, IL-5, IL-13 production
- B. IFN- γ production and macrophage activation
- C. IL-10 production
- D. IL-17 production

4. Which statement correctly describes the four types of hypersensitivity reactions?

- A. Type I: immediate IgE-mediated allergy; Type II: antibody-mediated cytotoxic against cells/tissues (IgG/IgM); Type III: immune complex-mediated; Type IV: delayed-type T cell-mediated.
- B. Type I: delayed-type T cell-mediated; Type II: antibody-mediated cytotoxic against cells/tissues (IgG/IgM); Type III: immune complex-mediated; Type IV: immediate IgE-mediated allergy.
- C. Type I: immune complex-mediated; Type II: immediate IgE-mediated; Type III: antibody-mediated cytotoxic; Type IV: delayed-type T cell-mediated.
- D. Type I: immediate IgG-mediated; Type II: antibody-mediated cytotoxic against pathogens; Type III: immune complex-mediated; Type IV: cytotoxic T cell-mediated.

5. IgG subclass concept: Which statement is true?

- A. There are three IgG subclasses
- B. There is only one IgG subclass
- C. There are four IgG subclasses: IgG1, IgG2, IgG3, IgG4
- D. IgG subclasses do not differ functionally

6. Which statement correctly maps TLRs to their ligands?

- A. TLR3 and TLR7 recognize RNA viruses; TLR4 recognizes Gram-negative bacteria; TLR5 recognizes bacteria.
- B. TLR3 recognizes DNA viruses; TLR4 recognizes Gram-positive bacteria; TLR5 recognizes viruses.
- C. TLR3 and TLR7 recognize RNA viruses; TLR4 recognizes viruses; TLR5 recognizes bacteria.
- D. TLR3 recognizes RNA viruses; TLR4 recognizes Gram-negative bacteria; TLR5 recognizes bacteria.

7. Which cells are primarily responsible for cross-presentation of extracellular antigens to CD8+ T cells?

- A. B cells
- B. Macrophages
- C. Neutrophils
- D. Dendritic cells

8. Which adhesion molecule binds to ICAM-1?

- A. L-selectin
- B. CD2 (LFA-2)
- C. LFA-3
- D. LFA-1

9. Which molecule presents endogenous peptides to CD8+ T cells?

- A. MHC II
- B. CD1
- C. MHC I
- D. TCR

10. In MHC class II antigen processing, what is the role of CLIP?

- A. A placeholder peptide that blocks loading of other peptides until HLA-DM replaces it
- B. The peptide that binds MHC I
- C. A chaperone that transports MHC II to the cell surface
- D. A transcription factor regulating MHC II expression

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Answers

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

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Explanations

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1. Which statement about Type IV hypersensitivity is correct?

- A. Delayed-type hypersensitivity is mediated by T cells and typically occurs 24-72 hours after exposure.
- B. It is mediated primarily by IgE antibodies.
- C. It results from immune complex deposition.
- D. It requires cytotoxic T cells to directly lyse target cells.

Type IV hypersensitivity is driven by T lymphocytes, not antibodies. After exposure, sensitized T cells release cytokines that recruit and activate macrophages, causing tissue damage that typically peaks 24–72 hours later. This delay and reliance on T cells distinguish it from antibody-mediated types. The other statements describe mechanisms of other hypersensitivity types: IgE drives immediate Type I reactions; immune complex deposition underlies Type III; and direct cytotoxic lysis by T cells is more characteristic of antibody-independent cytotoxic processes seen in Type II, not the classic delayed, T cell–mediated response.

2. Which statement about T cell costimulation is correct?

- A. Provides a necessary costimulatory signal that enhances TCR signaling via CD28-B7 interactions.
- B. Inhibits TCR signaling to prevent autoimmunity.
- C. Acts as an antigen receptor on B cells.
- D. Is a cytokine produced by T cells to recruit neutrophils.

T cell activation relies on two signals: recognition of antigen by the TCR (signal one) and a costimulatory signal (signal two) that heightens the response and prevents anergy. The classic costimulatory interaction is CD28 on T cells binding to B7 molecules (CD80/CD86) on antigen-presenting cells. This engagement amplifies TCR signaling, promotes IL-2 production, supports T cell proliferation and survival, and drives differentiation into effective effector cells. Without this second signal, T cells may become anergic or undergo apoptosis despite TCR engagement, which helps keep autoimmunity in check but can blunt immune responses. The other statements don't fit because costimulation is not an inhibitory brake on TCR signaling (inhibition is mediated by molecules like CTLA-4 later in activation, not by the normal costimulatory interaction). It isn't an antigen receptor on B cells, and it isn't a cytokine produced by T cells. The accurate description is that costimulation provides a necessary signal that enhances TCR signaling via CD28-B7 interactions.

3. Which cytokine profile best defines a Th1 response?

- A. IL-4, IL-5, IL-13 production
- B. IFN- γ production and macrophage activation
- C. IL-10 production
- D. IL-17 production

Th1 responses are defined by producing IFN- γ , which activates macrophages and drives cell-mediated immunity against intracellular pathogens. This IFN- γ signaling enhances macrophage killing, boosts antigen presentation, and supports effective clearance of intracellular microbes. The option describing IFN- γ production and macrophage activation captures this profile most accurately. By contrast, IL-4, IL-5, and IL-13 signal a Th2 response geared toward humoral immunity and allergy; IL-10 is an anti-inflammatory/regulatory cytokine; IL-17 characterizes Th17 responses involved in neutrophil recruitment and mucosal defenses. Hence, IFN- γ –driven macrophage activation best defines the Th1 pattern.

4. Which statement correctly describes the four types of hypersensitivity reactions?

- A. Type I: immediate IgE-mediated allergy; Type II: antibody-mediated cytotoxic against cells/tissues (IgG/IgM); Type III: immune complex-mediated; Type IV: delayed-type T cell-mediated.**
- B. Type I: delayed-type T cell-mediated; Type II: antibody-mediated cytotoxic against cells/tissues (IgG/IgM); Type III: immune complex-mediated; Type IV: immediate IgE-mediated allergy.
- C. Type I: immune complex-mediated; Type II: immediate IgE-mediated; Type III: antibody-mediated cytotoxic; Type IV: delayed-type T cell-mediated.
- D. Type I: immediate IgG-mediated; Type II: antibody-mediated cytotoxic against pathogens; Type III: immune complex-mediated; Type IV: cytotoxic T cell-mediated.

Understanding hypersensitivity types by mechanism helps you see why this pairing is correct. Type I is immediate and IgE-mediated, where antibodies bound to mast cells and basophils crosslink when an allergen is encountered, causing rapid release of histamine and other mediators. Type II is antibody-mediated cytotoxic, with IgG or IgM targeting antigens on cell surfaces or extracellular matrix, leading to complement activation and cell lysis or opsonization. Type III is immune complex-mediated, in which circulating antigen-antibody complexes deposit in tissues, activate complement, and recruit inflammatory cells to cause tissue injury. Type IV is delayed-type hypersensitivity, driven by T cells (especially Th1 cells and macrophages) rather than antibodies, producing local inflammation after a delay. The statement that assigns immediate IgE-mediated allergy to Type I, antibody-mediated cytotoxicity with IgG/IgM to Type II, immune complex-mediated to Type III, and delayed-type T cell-mediated to Type IV corresponds with these mechanisms. Other options mix up the roles (for example, calling Type I delayed or Type IV antibody-mediated) which contradicts the established classifications.

5. IgG subclass concept: Which statement is true?

- A. There are three IgG subclasses
- B. There is only one IgG subclass
- C. There are four IgG subclasses: IgG1, IgG2, IgG3, IgG4**
- D. IgG subclasses do not differ functionally

Humans have four IgG subclasses: IgG1, IgG2, IgG3, and IgG4. These arise from different constant regions of the heavy chain, giving each subclass distinct effector capabilities. This diversity allows the antibody response to be fine-tuned for different types of threat. For example, IgG1 and IgG3 are strong at activating complement and engaging Fc gamma receptors, which promotes phagocytosis and antibody-dependent cellular cytotoxicity. IgG3, in particular, is very effective at complement activation. IgG2 is more specialized for responses to polysaccharide antigens and has comparatively weaker Fc receptor interactions. IgG4 tends to be less inflammatory and can undergo Fab-arm exchange, reducing crosslinking and complement activation. So the statement that there are four IgG subclasses named IgG1 through IgG4 is correct. The idea that there are only three or only one IgG subclass is inaccurate, and the notion that IgG subclasses do not differ functionally is incorrect because they have distinct receptor binding and effector profiles.

6. Which statement correctly maps TLRs to their ligands?

- A. TLR3 and TLR7 recognize RNA viruses; TLR4 recognizes Gram-negative bacteria; TLR5 recognizes bacteria.
- B. TLR3 recognizes DNA viruses; TLR4 recognizes Gram-positive bacteria; TLR5 recognizes viruses.
- C. TLR3 and TLR7 recognize RNA viruses; TLR4 recognizes viruses; TLR5 recognizes bacteria.
- D. TLR3 recognizes RNA viruses; TLR4 recognizes Gram-negative bacteria; TLR5 recognizes bacteria.

Pattern recognition receptors like Toll-like receptors have specific microbial components they recognize. TLR3 and TLR7 sit in endosomes and specialize in sensing viral RNA: TLR3 detects double-stranded RNA, a common replication intermediate for many viruses, while TLR7 detects single-stranded RNA. TLR4 sits on the cell surface and recognizes lipopolysaccharide, a hallmark of Gram-negative bacteria, activating a typical inflammatory response. TLR5 recognizes bacterial flagellin, the structural protein of flagella. Therefore, saying that TLR3 and TLR7 recognize RNA viruses, TLR4 recognizes Gram-negative bacteria, and TLR5 recognizes bacteria is the correct mapping. The other statements misassign these ligands—for example, DNA viruses, Gram-positive bacteria, or viruses being recognized by TLR4 or TLR5 are not correct in the standard ligand mappings.

7. Which cells are primarily responsible for cross-presentation of extracellular antigens to CD8+ T cells?

- A. B cells
- B. Macrophages
- C. Neutrophils
- D. Dendritic cells

Cross-presentation is the process by which an extracellular (exogenous) antigen is shown on MHC class I to activate CD8+ T cells. Dendritic cells are best suited for this because they can take up external proteins, process them, and route peptides into the MHC I pathway to prime naive CD8+ T cells in lymph nodes. They possess specialized mechanisms to load exogenous antigens onto MHC I, enabling robust cross-priming of cytotoxic T cells against pathogens that do not directly infect antigen-presenting cells. B cells mainly present to CD4+ T cells via MHC II and support helper responses; macrophages present primarily to CD4+ T cells and are less efficient at initiating naive CD8+ T cell responses through cross-presentation. Neutrophils have limited antigen-presenting capabilities. So, the primary cells responsible for cross-presentation to CD8+ T cells are dendritic cells.

8. Which adhesion molecule binds to ICAM-1?

- A. L-selectin
- B. CD2 (LFA-2)
- C. LFA-3
- D. LFA-1

ICAM-1 serves as a ligand for LFA-1, which is an integrin on leukocytes (CD11a/CD18, also known as $\alpha L\beta 2$). When leukocytes become activated, inside-out signaling increases LFA-1's affinity so it can snugly bind ICAM-1 on the endothelial surface. This strong adhesion is what stops rolling and allows the cells to crawl through the vessel wall during an inflammatory response (diapedesis). Other adhesion pairs include L-selectin binding to HEV addressins to mediate rolling, and CD2 binding to LFA-3 on other cells; those interactions are about initial tethering or costimulation, not the firm ICAM-1–LFA-1 adhesion.

9. Which molecule presents endogenous peptides to CD8+ T cells?

- A. MHC II
- B. CD1
- C. MHC I**
- D. TCR

Endogenous peptides are presented to CD8+ T cells by MHC class I. Proteins made inside a cell are chopped into short peptides by the cytosolic proteasome, then transported into the endoplasmic reticulum by TAP transporters. There, peptides bind to MHC class I molecules with the help of chaperones, and the peptide–MHC I complexes move to the cell surface. CD8+ T cells recognize these complexes via their T cell receptors and can become activated to kill the presenting cell if the peptide is foreign or abnormal. In contrast, MHC class II presents exogenous peptides to CD4+ helper T cells, CD1 presents lipid antigens, and the T cell receptor is the molecule on T cells that recognizes the peptide–MHC complex rather than the presenting molecule itself.

10. In MHC class II antigen processing, what is the role of CLIP?

- A. A placeholder peptide that blocks loading of other peptides until HLA-DM replaces it**
- B. The peptide that binds MHC I
- C. A chaperone that transports MHC II to the cell surface
- D. A transcription factor regulating MHC II expression

CLIP serves as a placeholder peptide in MHC class II antigen processing. It sits in the peptide-binding groove after the invariant chain has guided the MHC II molecule to the endosome and been degraded. This CLIP peptide blocks other peptides from binding too early, preventing premature or inappropriate loading. HLA-DM acts as a catalyst that facilitates the removal of CLIP and promotes binding of the appropriate antigenic peptide with the groove, allowing the MHC II-peptide complex to travel to the cell surface for presentation to CD4+ T cells. The other options don't fit because CLIP is not the peptide for MHC I, not a trafficking chaperone, and not a transcription factor.

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:

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

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