

PATHOPHYSIOLOGY EXAM 1: IMMUNODEFICIENCY AND AUTOIMMUNE DISEASE PRACTICE EXAM (SAMPLE)

STUDY GUIDE



SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

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THE FULL VERSION STUDY GUIDE**

<https://pathophys1immunodeficiency.examzify.com>



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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 set of defects are immunologic defects seen in HIV-1 infection?**
 - A. complete loss of cellular immunity
 - B. opportunistic infections
 - C. B cell lymphomas and Kaposi sarcoma
 - D. all of the above
- 2. What is IVIG and what is its therapeutic use?**
 - A. Intracellular immunoglobulin; used to block T cells
 - B. Isovolem immunoglobulin; used to promote growth of immune cells
 - C. Intravenous immunoglobulin; used to replace antibodies in immunodeficiency and modulate immune responses in autoimmune conditions
 - D. Intravenous immunoglobulin; used only in lab testing
- 3. Which complement components are typically measured to assess SLE activity?**
 - A. C3 and C4
 - B. C4 alone
 - C. C1q and C3
 - D. C5 and C9
- 4. What overall description best summarizes diffuse systemic sclerosis pathophysiology?**
 - A. Isolated skin involvement with no internal organ fibrosis
 - B. Widespread skin thickening and fibrosis of internal organs due to excessive collagen deposition and microvascular injury
 - C. Predominantly lymph node enlargement without tissue fibrosis
 - D. Primary autoimmune destruction of neurons
- 5. During seroconversion in early HIV infection, what change occurs in CD4+ T cell count during weeks 1-6?**
 - A. Temporary decline in CD4+ T cells during weeks 1-6
 - B. Sustained rise in CD4+ T cells
 - C. No change
 - D. Immediate depletion with no fluctuations

- 6. Which infection is associated with CVID due to gastrointestinal involvement?**
- A. Streptococcus pneumoniae bacteremia
 - B. Candida esophagitis
 - C. Giardia lamblia causing persistent diarrhea
 - D. Mucocutaneous candidiasis
- 7. In SLE, deposition of immune complexes in tissues leads to which combination of tissue changes?**
- A. Vasculitis, synovitis, and glomerulonephritis.
 - B. Necrotizing myopathy.
 - C. Pulmonary fibrosis.
 - D. Pancreatic acinar destruction.
- 8. Central tolerance is primarily established during lymphocyte development in which locations?**
- A. Peripheral tissues.
 - B. Circulation.
 - C. Central tolerance occurs during lymphocyte development in primary organs; peripheral tolerance occurs in mature lymphocytes in the periphery.
 - D. Only occurs in B cells.
- 9. What treatment may decrease morbidity/mortality in patients with HIV/AIDS?**
- A. antiretroviral therapy and chemoprophylaxis
 - B. vaccines alone
 - C. antibiotics alone
 - D. hydration only
- 10. Anti-thyroid peroxidase antibodies are most commonly associated with which condition?**
- A. Hashimoto thyroiditis
 - B. Graves disease
 - C. Type 1 diabetes mellitus
 - D. Addison disease

Answers

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

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Explanations

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1. Which set of defects are immunologic defects seen in HIV-1 infection?

- A. complete loss of cellular immunity
- B. opportunistic infections
- C. B cell lymphomas and Kaposi sarcoma
- D. all of the above**

HIV-1 infection causes progressive loss of CD4+ T cells, leading to a profound reduction in cell-mediated (cellular) immunity. As this cellular defense wanes, the body becomes vulnerable to intracellular pathogens, giving rise to opportunistic infections. With advancing immune suppression, surveillance against malignancies also falters, increasing the risk of cancers like B cell lymphomas and Kaposi sarcoma (the latter linked to HHV-8). Because HIV's impact spans loss of cellular immunity, susceptibility to opportunistic infections, and AIDS-related malignancies, all of these immunologic defects arise in HIV infection, making the broad answer the best one. In advanced disease the decline in CD4+ T cells is substantial, representing severe impairment of cell-mediated immunity even though "complete" depletion is not always literal.

2. What is IVIG and what is its therapeutic use?

- A. Intracellular immunoglobulin; used to block T cells
- B. Isovolemic immunoglobulin; used to promote growth of immune cells
- C. Intravenous immunoglobulin; used to replace antibodies in immunodeficiency and modulate immune responses in autoimmune conditions**
- D. Intravenous immunoglobulin; used only in lab testing

IVIG stands for intravenous immunoglobulin, a product made from pooled IgG antibodies collected from many donors. In immunodeficiency, it provides the antibodies that patients can't make well on their own, delivering passive immunity to help prevent infections. In autoimmune and inflammatory conditions, IVIG helps modulate the immune system through several overlapping mechanisms: it can block Fc receptors on immune cells to dampen inflammatory responses, interfere with complement activation, neutralize circulating autoantibodies, and alter cytokine signaling and regulatory T cell activity. This combination of replacement and immunomodulation is why IVIG is used in a range of conditions—from antibody deficiencies to diseases like Guillain-Barré syndrome, Kawasaki disease, immune thrombocytopenia, and certain neuropathies. It is not an intracellular immunoglobulin, not used to promote growth of immune cells, and not limited to laboratory testing.

3. Which complement components are typically measured to assess SLE activity?

- A. C3 and C4**
- B. C4 alone
- C. C1q and C3
- D. C5 and C9

Active SLE often drives immune complex-mediated activation of the classical complement pathway, which uses and depletes complement proteins. Because C3 and C4 are consumed during flares, their serum levels drop when the disease is active and tend to rise again with treatment and remission. Checking both C3 and C4 gives a more reliable picture of disease activity than any single component, since each can vary and together they reflect overall classical pathway activation. C3 is often more sensitive to activity, including nephritis, while C4 adds specificity for classical pathway involvement. C4 alone can miss activity in some patients who have normal C4 but low C3, so it's less informative on its own. C1q and C3 might be helpful in specific contexts (for example, C1q deficiency is associated with lupus-like disease), but they are not routinely used to monitor ongoing activity. C5 and C9 are terminal components and don't correlate as consistently with disease activity, so they're not standard measures for monitoring SLE flares.

4. What overall description best summarizes diffuse systemic sclerosis pathophysiology?

- A. Isolated skin involvement with no internal organ fibrosis
- B. Widespread skin thickening and fibrosis of internal organs due to excessive collagen deposition and microvascular injury**
- C. Predominantly lymph node enlargement without tissue fibrosis
- D. Primary autoimmune destruction of neurons

Diffuse systemic sclerosis is driven by autoimmune-mediated damage to small blood vessels (microvascular injury) with a subsequent overactive fibrotic response. Endothelial injury and vascular dysfunction set off a cascade of profibrotic signals, leading to activation of fibroblasts that become myofibroblasts and lay down large amounts of collagen. This results not only in thickened, stiff skin but also in progressive fibrosis of internal organs such as the lungs, kidneys, heart, and gastrointestinal tract. The combination of widespread skin involvement and internal organ fibrosis, all rooted in excessive collagen deposition and vascular injury, is the hallmark of diffuse systemic sclerosis. Other options don't fit because they describe non-fibrotic processes or localized disease (isolated skin involvement), lack of fibrosis, lymph node-dominated features, or a primary neuronal autoimmune process, none of which capture the vascular-driven fibrotic pathology of this condition.

5. During seroconversion in early HIV infection, what change occurs in CD4+ T cell count during weeks 1-6?

- A. Temporary decline in CD4+ T cells during weeks 1-6**
- B. Sustained rise in CD4+ T cells
- C. No change
- D. Immediate depletion with no fluctuations

In acute HIV infection, seroconversion brings a surge of viral replication that directly targets CD4+ T cells and triggers widespread immune activation. This causes a rapid loss of circulating CD4+ T cells, so their count in the blood falls during the early weeks, typically around weeks 2–4 and can be observed through week 6. As the immune response ramps up and viral replication is partly controlled, CD4+ T cell numbers in the blood often partially rebound, though they may not return to baseline. This pattern explains why the correct answer is a temporary decline in CD4+ T cells during weeks 1–6—the drop is real and transient rather than a sustained rise, no change, or immediate permanent depletion.

6. Which infection is associated with CVID due to gastrointestinal involvement?

- A. Streptococcus pneumoniae bacteremia
- B. Candida esophagitis
- C. Giardia lamblia causing persistent diarrhea**
- D. Mucocutaneous candidiasis

CVID involves deficient antibody production, especially impaired mucosal immunity in the gut. Giardia lamblia is a protozoan that causes persistent diarrhea when secretory IgA in the intestinal lumen is limited. In CVID, the lack of IgA and other humoral responses prevents efficient clearance of Giardia, leading to chronic, troublesome diarrhea and malabsorption. The other options fit infections seen with immune compromise in general, but they don't highlight the GI-specific vulnerability seen with CVID: Streptococcus pneumoniae bacteremia reflects susceptibility to encapsulated bacteria rather than a gut-focused issue; Candida esophagitis and mucocutaneous candidiasis are more typical of T-cell-related immunodeficiencies.

7. In SLE, deposition of immune complexes in tissues leads to which combination of tissue changes?

- A. Vasculitis, synovitis, and glomerulonephritis.**
- B. Necrotizing myopathy.
- C. Pulmonary fibrosis.
- D. Pancreatic acinar destruction.

In SLE, immune complexes form and deposit in tissues, triggering a type III hypersensitivity reaction. This activates complement and attracts inflammatory cells, causing inflammation in small vessels, the joint lining, and the kidneys. That leads to vasculitis in vessels, synovitis in joints, and glomerulonephritis in the kidneys—exactly the combination described. Other options describe tissue damage more typical of different processes (necrotizing muscle disease, fibrotic lung disease, or pancreatic destruction), which aren't the hallmark immune-complex-driven changes seen in lupus.

8. Central tolerance is primarily established during lymphocyte development in which locations?

- A. Peripheral tissues.
- B. Circulation.
- C. Central tolerance occurs during lymphocyte development in primary organs; peripheral tolerance occurs in mature lymphocytes in the periphery.**
- D. Only occurs in B cells.

Central tolerance is the process by which self-reactive lymphocytes are eliminated or inactivated during their development in primary lymphoid organs. For B cells this happens in the bone marrow, and for T cells in the thymus, ensuring that the mature repertoire is largely self-tolerant. The best answer expresses that central tolerance occurs during development in primary organs and that peripheral tolerance—happening in mature lymphocytes in the body's periphery—addresses self-reactivity outside those primary sites. It's also worth noting that this process isn't limited to B cells; T cells undergo central tolerance in the thymus as well. Peripheral tissues are the arena for peripheral tolerance mechanisms, such as anergy or regulatory T cell-mediated suppression, after lymphocytes have already matured and circulated.

9. What treatment may decrease morbidity/mortality in patients with HIV/AIDS?

- A. antiretroviral therapy and chemoprophylaxis**
- B. vaccines alone
- C. antibiotics alone
- D. hydration only

*The key idea is that lowering HIV replication while preventing opportunistic infections best reduces illness and death in HIV/AIDS. Antiretroviral therapy suppresses HIV viral load, preserves CD4 T cells, and strengthens immune function, which slows disease progression and cuts mortality. When you add chemoprophylaxis, you proactively guard against common opportunistic pathogens (like *Pneumocystis jirovecii* pneumonia and other infections), further lowering morbidity and mortality. Vaccines alone aren't sufficient in advanced HIV because vaccine responses can be weak, and they don't address the underlying immunodeficiency or the broad spectrum of opportunistic infections. Antibiotics alone treat infections as they occur but don't control HIV replication or prevent new infections. Hydration alone has no impact on the immune failure or infection risk. So, combining antiretroviral therapy with chemoprophylaxis provides the most effective strategy to decrease disease burden and death in HIV/AIDS.*

10. Anti-thyroid peroxidase antibodies are most commonly associated with which condition?

- A. Hashimoto thyroiditis**
- B. Graves disease
- C. Type 1 diabetes mellitus
- D. Addison disease

Anti-thyroid peroxidase antibodies reflect autoimmune attack on the thyroid gland, targeting the enzyme thyroid peroxidase essential for thyroid hormone synthesis. They are most strongly linked to Hashimoto thyroiditis, where autoimmune destruction of thyroid follicles leads to hypothyroidism and often a non-tender goiter; TPO antibodies are commonly present and support the diagnosis of autoimmune thyroiditis. In Graves disease, the primary autoimmune issue is antibodies stimulating the TSH receptor, causing hyperthyroidism, and while TPO antibodies can be seen, they are not the defining feature. Other conditions like Type 1 diabetes mellitus and Addison disease involve different autoantibodies (beta-cell-specific or adrenal cortex-specific) and are not characteristically associated with thyroid peroxidase 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://pathophys1immunodeficiency.examzify.com>

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

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