

The Immune System: Innate and Adaptive Body Defenses

Objectives

Surface Barriers: Skin and Mucosae

1. Explain the roles of the skin and mucous membranes in the innate defense.
2. List and discuss the secretions of the skin and mucous membranes.

Internal Defenses: Cells and Chemicals

3. Describe the different types of phagocytes.
4. Identify the role of natural killer cells.
5. Explain the inflammatory response.
6. Discuss complement, interferon, and fever.

Antigens

7. Define antigen and differentiate between self and nonself.
8. Compare a complete and incomplete antigen.

Humoral Immune Response

9. Examine the clonal selection and differentiation of B cells.
10. Compare the primary immune response and the secondary immune response.
11. Discuss active and passive immunities, both naturally acquired and artificially acquired.
12. Explain the structure of an antibody.
13. List the five classes of antibodies and their functions.
14. Identify the ways antibodies function.

Cell-Mediated Immune Response

15. Discuss clonal selection and differentiation of T cells.
16. Explain how T cells are activated.
17. List the types of T cells and their roles.
18. Describe the different types of tissue grafts involved in organ transplants.

Homeostatic Imbalances of Immunity

19. Examine immunodeficiencies, autoimmune diseases, and hypersensitivities.

Developmental Aspects of the Immune System

20. Identify the events of embryonic development and the effects of aging on the immune system.

Suggested Chapter Outline

1. Innate Defenses (pp. 789–798; Figs. 21.1–21.6; Tables 21.1–21.2)

- A. Surface Barriers: Skin and Mucosae

1. Skin, a highly keratinized epithelial membrane, represents a physical barrier to most microorganisms and their enzymes and toxins.
 2. Mucous membranes line all body cavities open to the exterior and function as an additional physical barrier.
 3. Secretions of the epithelial tissues include acidic secretions, sebum, hydrochloric acid, saliva, and mucus.
- B. Internal Defenses: Cells and Chemicals
1. Phagocytes confront microorganisms that breach the external barriers.
 - a. Macrophages are the main phagocytes of the body.
 - b. Neutrophils are the first responders and become phagocytic when they encounter infectious material.
 - c. Eosinophils are weakly phagocytic but are important in defending the body against parasitic worms.
 - d. Mast cells have the ability to bind with, ingest, and kill a wide range of bacteria.
 2. Natural killer cells are able to lyse and kill cancer cells and virally infected cells before the adaptive immune system has been activated.
 3. Inflammation occurs any time the body tissues are injured by physical trauma, intense heat, irritating chemicals, or infection by viruses, fungi, or bacteria.
 - a. The four cardinal signs of acute inflammation are redness, heat, swelling, and pain.
 - b. Chemicals cause dilation of surrounding blood vessels to increase blood flow to the area and increase permeability, which allows fluid containing clotting factors and antibodies to enter the tissues.
 - c. Soon after inflammation the damaged site is invaded by neutrophils and macrophages.
 4. Antimicrobial proteins enhance the innate defenses by attacking microorganisms directly or by hindering their ability to reproduce.
 - a. Interferons are small proteins produced by virally infected cells that help protect surrounding healthy cells.
 - b. Complement refers to a group of about 20 plasma proteins that provide a major mechanism for destroying foreign pathogens in the body.
 5. Fever, or an abnormally high body temperature, is a systemic response to microorganisms.

II. Adaptive Defenses (pp. 798–803; Figs. 21.7–21.9)

- A. Aspects of the Adaptive Immune Response
1. The adaptive defenses recognize and destroy the specific antigen that initiated the response.
 2. The immune response is a systemic response; it is not limited to the initial infection site.
 3. After an initial exposure the immune response is able to recognize the same antigen and mount a faster and stronger defensive attack.
 4. Humoral immunity is provided by antibodies produced by B lymphocytes present in the body's "humors" or fluids.
 5. Cellular immunity is associated with T lymphocytes and has living cells as its protective factor.
- B. Antigens are substances that can mobilize the immune system and provoke an immune response.
1. Complete antigens are able to stimulate the proliferation of specific lymphocytes and antibodies, and to react with the activated lymphocytes and produced antibodies.

2. Haptens are incomplete antigens that are not capable of stimulating the immune response, but if they interact with proteins of the body they may be recognized as potentially harmful.
 3. Antigenic determinates are a specific part of an antigen that are immunogenic and bind to free antibodies or activated lymphocytes.
- C. Cells of the Adaptive Immune System: An Overview
1. Lymphocytes originate in the bone marrow and when released become immunocompetent in either the thymus (T cells) or the bone marrow (B cells).
 2. Antigen-presenting cells engulf antigens and present fragments of these antigens on their surfaces where they can be recognized by T cells.

III. Humoral Immune Response (pp. 804–810; Figs. 21.10–21.14; Table 21.3)

- A. The immunocompetent but naive B lymphocyte is activated when antigens bind to its surface receptors.
1. Clonal selection is the process of the B cell growing and multiplying to form an army of cells that are capable of recognizing the same antigen.
 2. Plasma cells are the antibody-secreting cells of the humoral response; most clones develop into plasma cells.
 3. The clones that do not become plasma cells develop into memory cells.
- B. Immunological Memory
1. The primary immune response occurs on first exposure to a particular antigen with a lag time of about 3–6 days.
 2. The secondary immune response occurs when someone is reexposed to the same antigen. It is faster, more prolonged, and more effective.
- C. Active and Passive Humoral Immunity
1. Active immunity occurs when the body mounts an immune response to an antigen.
 - a. Naturally acquired active immunity occurs when a person suffers through the symptoms of an infection.
 - b. Artificially acquired active immunity occurs when a person is given a vaccine.
 2. Passive immunity occurs when a person is given preformed antibodies.
 - a. Naturally acquired passive immunity occurs when a mother's antibodies enter fetal circulation.
 - b. Artificially acquired passive immunity occurs when a person is given preformed antibodies that have been harvested from another person.
- D. Antibodies or immunoglobulins are proteins secreted by plasma cells in response to an antigen that are capable of binding to that antigen.
1. The basic antibody structure consists of four looping polypeptide chains linked together by disulfide bonds.
 2. Antibodies are divided into five classes based on their structure: IgM, IgG, IgA, IgD, and IgE.
 3. Embryonic cells contain a few hundred gene segments that are shuffled and combined to form all of the different B cells that are found in the body.
 4. Antibody Targets and Functions
 - a. Complement fixation and activation occurs when complement binds to antibodies attached to antigens, and leads to lysis of the cell.

- b. Neutralization occurs when antibodies block specific sites on viruses or bacterial exotoxins, causing them to lose their toxic effects.
 - c. Agglutination occurs when antibodies cross-link to antigens on cells, causing clumping.
 - d. Precipitation occurs when soluble molecules are cross-linked into large complexes that settle out of solution.
5. Monoclonal antibodies are commercially prepared antibodies specific for a single antigenic determinant.

IV. Cell-Mediated Immune Response (pp. 810–820; Figs. 21.15–21.20; Table 21.4)

- A. The stimulus for clonal selection and differentiation of T cells is binding of antigen, although their recognition mechanism is different from B cells.
 - 1. T cells must accomplish a double recognition process: they must recognize both self (an MHC protein of a body cell) and nonself (antigen) at the same time.
 - 2. T Cell Activation
 - a. Step 1: T cell antigen receptors (TCRs) bind to antigen-MHC complex on the surface of a body cell.
 - b. Step 2: A T cell must recognize one or more co-stimulatory signals.
 - c. Once activated, a T cell enlarges and proliferates to form a clone of cells that differentiate and perform functions according to their T cell class.
 - 3. Cytokines include hormonelike glycoproteins released by activated T cells and macrophages.
- B. Specific T Cell Roles
 - 1. Helper T cells stimulate proliferation of other T cells and B cells that have already become bound to antigen.
 - 2. Cytotoxic T cells are the only T cells that can directly attack and kill other cells displaying antigen to which they have been sensitized.
 - 3. Regulatory T cells release cytokines that suppress the activity of both B cells and other types of T cells.
 - 4. Gamma delta T cells are found in the intestine and are more similar to NK cells than other T cells.
 - 5. Without helper T cells there is no adaptive immune response because the helper T cells direct or help complete the activation of all other immune cells.
- C. Organ Transplants and Prevention of Rejection
 - 1. Grafts
 - a. Autografts are tissue grafts transplanted from one body site to another in the same person.
 - b. Isografts are grafts donated to a patient by a genetically identical individual such as an identical twin.
 - c. Allografts are grafts transplanted from individuals that are not genetically identical but belong to the same species.
 - d. Xenografts are grafts taken from another animal species.
 - 2. Transplant success depends on the similarity of the tissues because cytotoxic T cells, NK cells, and antibodies work to destroy foreign tissues.

V. Homeostatic Balances of Immunity (pp. 820–825; Fig. 21.21)

- A. Immunodeficiencies are any congenital or acquired conditions that cause immune cells, phagocytes, or complement to behave abnormally.

1. Severe combined immunodeficiency (SCID) is a congenital condition that produces a deficit of B and T cells.
 2. Acquired immune deficiency syndrome (AIDS) cripples the immune system by interfering with helper T cells.
- B. Autoimmune diseases occur when the immune system loses its ability to differentiate between self and nonself and ultimately destroys itself.
- C. Hypersensitivities, or allergies, are the result of the immune system causing tissue damage as it fights off a perceived threat that would otherwise be harmless.
1. Immediate hypersensitivities begin within seconds after contact and last about half an hour.
 2. Subacute hypersensitivities take 1–3 hours to occur and last 10–15 hours.
 3. Delayed hypersensitivity reactions take 1–3 days to occur and may take weeks to go away.

VI. Developmental Aspects of the Immune System (pp. 825–826)

- A. Embryologic Development
1. Stem cells of the immune system originate in the liver and spleen during weeks 1–9 of embryonic development; later the bone marrow takes over this role.
 2. In late fetal life and shortly after birth the young lymphocytes develop self-tolerance and immunocompetence.
- B. Later in life the ability and efficiency of our immune system declines.

Cross References

Additional information on topics covered in Chapter 21 can be found in the chapters listed below.

1. Chapter 2: Protein structure
2. Chapter 3: Cilia; lysosomes
3. Chapter 5: Mechanical and chemical protection of the skin; Langerhans' cells
4. Chapter 15: Lysozyme
5. Chapter 16: Thymus
6. Chapter 17: Granulocytes; agranulocytes; chemotaxis; diapedesis
7. Chapter 22: Inflammatory processes involving respiratory tissues
8. Chapter 23: Protection of the mucous barrier in the stomach; role of saliva in protection of mucous barriers; Kupffer cells
9. Chapter 24: Body temperature regulation
10. Chapter 28: Antibody protection of the fetus due to maternal antibodies

Laboratory Correlations

1. Marieb, E. N. *Human Anatomy & Physiology Laboratory Manual: Cat and Fetal Pig Versions*. Eighth Edition Updates. Benjamin Cummings, 2006.
Exercise 35: The Lymphatic System and Immune Response
2. Marieb, E. N. *Human Anatomy & Physiology Laboratory Manual: Main Version*. Seventh Edition Update. Benjamin Cummings, 2006.
Exercise 35: The Lymphatic System and Immune Response

Lecture Hints

1. Although specific and nonspecific defense mechanisms are treated as separate entities, emphasize that there is much overlap of function. For example, in antibody-mediated complement lysis, formation of the antibody that labels a cell is due to specific processes, but the actual lysis of that cell is accomplished nonspecifically by complement fixation.
2. Point out that the body has several lines of defense—mechanical, chemical, and cellular. It is helpful to orient students toward the idea that even with all of its complexity, the immune system has one underlying theme: rid the body of unwanted substances/life forms.
3. Students often have problems deciding what mechanisms are specific and which are nonspecific. Stress that the immune system tailors its response to each individual antigen, while the nonspecific mechanisms respond to cues that are much more broad.
4. Emphasize the logic behind the four cardinal signs of inflammation. For example, to bring the large quantities of oxygen and nutrients for repair processes, blood supply to an area must be increased. Redness results as vasodilation increases; heat, as warm blood is delivered; swelling as capillary walls become more permeable; pain as pressure due to swelling is transmitted to nerve endings.
5. Point out that neutrophils are seen early in an infection, but that macrophages are characteristic of chronic infection.
6. To illustrate the action of pyrogens on the hypothalamus, use the example of resetting the thermostat to a higher temperature in a home. As always, relating a physiological concept to something familiar to students will help reinforce the idea.
7. Mention that cytotoxic T cells must come in contact with the invader, but that B cells send out antibodies from sometimes remote locations to target specific antigens.
8. Emphasize the difference between antigens and haptens, and that size is the cause of the distinction between the two. Essentially, one can think of a hapten as an antigenic determinant if it is bound to a carrier molecule; alternatively, if an antigenic determinant were not part of a large molecule, it would be a hapten.
9. To reinforce the idea of clonal selection, point out that a single B cell could not possibly produce enough antibody to neutralize a large quantity of antigen.
10. Stress the difference between active and passive immunity, and that the body does not care where antibodies come from. If the students understand the concept, the question should arise: “Why don’t the foreign antibodies generate a response in the recipient?” If no one asks, ask this of the class to generate discussion.
11. In the discussion of complement, ask the class, “What would happen if an antibody bound to a completely normal body cell?” Use this as a lead-in to the topic of complement recognition of the constant region of the antibody.
12. It is often difficult for students to grasp the concept of somatic recombination in the generation of antibody diversity. Ask plenty of questions during lecture to reinforce concepts.
13. Clearly distinguish between different types of allergies. As the material is being covered, ask students which arm of the immune system is responsible for what type of hypersensitivity—immediate, subacute, or delayed. Students should be able to make the connection that a cell-mediated response will take time (delayed hypersensitivity).

Activities/Demonstrations

1. Audio-visual materials listed under Multimedia in the Classroom and Lab.

2. To open a discussion of the inflammatory process, ask if anyone has a cut or injury that is in the process of healing. If so, have the class members observe it, describe all obvious signs, and provide the underlying reason for the signs seen.
3. Ask students to come prepared to discuss the following questions during a subsequent lecture:
 - a. Explain why vaccination provides long-term protection against a particular disease while passive immunization provides only temporary protection.
 - b. What is the important difference between natural killer cells and cytotoxic T cells?
 - c. Why can T helper cells be called the “managers” of the immune system?
4. Use a lock and several keys (with only one that fits the lock) to demonstrate the specificity of antigens and antibodies.

Critical Thinking/Discussion Topics

1. Discuss the pros and cons surrounding the use of immunizations for mumps, measles, etc.
2. Explore the autoimmune diseases, how they occur, symptoms, prognosis, and treatment.
3. Discuss why some individuals are sensitive (allergic) to drugs from one source, but are not so sensitive to drugs from another source.
4. Discuss the social implications of immunity disorders such as AIDS, ARC, SCID, etc.
5. Identify the role of the Epstein-Barr virus in immunity and immunity disorders.
6. Discuss the effects of AIDS both immunologically and socially.
7. Explain why we need specific resistance mechanisms even though nonspecific resistance mechanisms attack all foreign substances (i.e., why is specific resistance necessary at all?).
8. Explain what the body’s immune response is to an antitoxin or other passive immunization.
9. Discuss why chemotherapeutics attached to monoclonal antibodies are an advantage over injection of the chemical agent alone. Could there be any drawbacks to this therapy?

Library Research Topics

1. Research some of the opportunistic diseases that often accompany AIDS.
2. Study the difficulties involved in transplant surgeries.
3. Explore the causes of several known autoimmune diseases.
4. Examine the signs, symptoms, and treatment of anaphylactic shock.
5. Investigate the possible side effects of vaccines.

Multimedia in the Classroom and Lab

Online Resources for Students

www.anatomyandphysiology.com

www.myaandp.com

The following shows the organization of the Chapter Guide page in both the Anatomy & Physiology Place and MyA&P™. The Chapter Guide organizes all the chapter-specific online media resources for Chapter 21 in one convenient location, with e-book links to each section of the textbook. Please note that both sites also give you access to other

general A&P resources, like InterActive Physiology[®], PhysioEx 6.0[™], Anatomy 360°, Flashcards, a Glossary, a Histology Tutorial, and much more.

Objectives

PART ONE: INNATE DEFENSES

Section 21.1 Surface Barriers: Skin and Mucosae (pp. 789–790)

Section 21.2 Internal Defenses: Cells and Chemicals (pp. 790–798)

Art Labeling Activity: Phagocytosis (Fig. 21.2, p. 791)

Art Labeling Activity: Phagocyte Mobilization (Fig. 21.4, p. 794)

PART TWO: ADAPTIVE DEFENSES

Section 21.3 Antigens (pp. 799–800)

Section 21.4 Cells of the Adaptive Immune System: An Overview (pp. 800–803)

Section 21.5 Humoral Immune Response (pp. 804–810)

Art Labeling Activity: Mechanisms of Antibody Actions (Fig. 21.14, p. 809)

PhysioEx: Serological Testing

Section 21.6 Cell-Mediated Immune Response (pp. 810–820)

Memory: The Immune Response, Part 1

Memory: The Immune Response, Part 2

Section 21.7 Homeostatic Imbalances of Immunity (pp. 820–825)

Case Study: Genetic Immunodeficiency

Section 21.8 Developmental Aspects of the Immune System (pp. 825–826)

Chapter Summary

Self-Study Quizzes

Art Labeling Quiz

Matching Quiz

Multiple-Choice Quiz (Level I)

Multiple-Choice Quiz (Level II)

True-False Quiz

Crossword Puzzles

Crossword Puzzle 21.1

Crossword Puzzle 21.2

Media

See *Guide to Audio-Visual Resources in Appendix A* for key to AV distributors.

Video

1. *AIDS: A Biological Perspective* (FHS; 30 min., 1995). Award-winning video that explores many of the difficult questions surrounding AIDS, including why a vaccine has been so difficult to find.
2. *Basic Immunology* (IM; 37 min., 1994). A highly recommended video that examines the anatomy and physiology of the immune system. It illustrates the system's tissues, organs, and cellular and soluble components.
3. *The Body Against Disease Video* (WNS; 48 min.). Presents a detailed picture of how the body defends itself against disease. Students are shown an in-depth analysis of the immune system, stressing the many ways in which it protects the body against disease.

4. *Cell Wars* (FHS; 22 min.). This program explains the role of antibodies in vaccinations and allergies, and shows the uses of monoclonal antibodies in the diagnosis and treatment of a variety of different types of tumors, as well as the immune system deficiency syndrome AIDS.
5. *Human Immune System* (IM; 20 min., 2002). Explains how the immune system defends the body against foreign invaders.
6. *Immunizations* (FHS; 20 min., 1994). Explains the need for vaccinations against disease and identifies the recommended pediatric immunization schedule.
7. *Your Immune System* (FHS; 20 min., 2001). Maps out the human immune system and what it does to keep the body healthy.

Software

1. *Blood and Immunity* (see p. 199 of this guide for full listing).
2. *Immunology* (IM; Win/Mac). Provides in-depth coverage of the principles of immunology.
3. *InterActive Physiology[®] 9-System Suite CD-ROM* (BC; Win/Mac). Interactive software that explores the physiology of the immune system.

Lecture Enhancement Material

To view thumbnails of all of the illustrations for Chapter 21, see Appendix B.

Transparencies Index/Media Manager

Figure 21.1	Overview of innate and adaptive defenses.
Figure 21.2	Phagocytosis.
Figure 21.3	Flowchart of events in inflammation.
Figure 21.4	Phagocyte mobilization.
Figure 21.5	The interferon mechanism against viruses.
Figure 21.6	Complement activation.
Figure 21.7	Antigenic determinants.
Figure 21.8	Lymphocyte traffic.
Figure 21.9	T cell selection in the thymus.
Figure 21.10	Clonal selection of a B cell.
Figure 21.11	Primary and secondary humoral responses.
Figure 21.12	Types of acquired immunity.
Figure 21.13	Antibody structure.
Figure 21.14	Mechanisms of antibody action.
Figure 21.15	Major types of T cells based on displayed cell differentiation glycoproteins (CD4, CD8).
Figure 21.16	MHC proteins, and antigen processing and display.
Figure 21.17	Clonal selection of T _H and T _C cells involves simultaneous recognition of self and nonself.
Figure 21.18	The central role of T _H cells.
Figure 21.19	Cytotoxic T cells attack infected and cancerous cells.
Figure 21.20	The primary immune response.
Figure 21.21	Mechanism of an acute allergic (immediate hypersensitivity) response.
Table 21.1	Inflammatory Chemicals
Table 21.2	Summary of Nonspecific Body Defenses
Table 21.3	Immunoglobulin Classes

*Indicates images that are on the Media Manager only.

Answers to End-of-Chapter Questions

Multiple Choice and Matching Question answers appear in Appendix G in the main text.

Short Answer Essay Questions

13. Mucosae are found on the outer surface of the eye and in the linings of all body cavities open to the exterior, i.e., the digestive, respiratory, urinary, and reproductive tracts. The epidermis is the outermost covering of the body surface. Mucus provides a sticky mechanical barrier that traps pathogens.
Lysosyme, an enzyme that destroys bacteria, is found in saliva and lacrimal fluid.
Keratin, a tough waterproofing protein in epithelial membranes, presents a physical barrier to microorganisms on the skin. It is resistant to most weak acids and bases and to bacterial enzymes and toxins.
The acid pH of skin secretions inhibits bacterial growth. Vaginal secretions and urine (as a rule) are also very acidic. Hydrochloric acid is secreted by the stomach mucosa and acts to kill pathogens.
Cilia of the upper respiratory tract mucosae sweep dust and bacteria-laden mucus superiorly toward the mouth, restraining it from entering the lower respiratory passages. (pp. 789–790)
14. Attempts at phagocytosis are not always successful because to accomplish ingestion, the phagocyte must first adhere to the particle. Complement proteins and antibodies coat foreign particles, providing binding sites to which phagocytes can attach, making phagocytosis more efficient. (p. 790)
15. The term *complement* refers to a heterogenous group of at least 20 plasma proteins that normally circulate in an inactive state. Complement is activated by one of two pathways (classical or alternative) involving the plasma proteins. Each pathway involves a cascade in which complement proteins are activated in an orderly sequence leading to the cleavage of C3. Once C3b is bound to the target cell's surface, it enzymatically initiates the remaining steps of complement activation, which incorporates C5 through C9 (MAC) into the target cell membrane, ensuring lysis of the target cell.
Other roles of complement include opsonization, inflammatory actions such as stimulating mast cells and basophils to release histamine (which increases vascular permeability), and attracting neutrophils and other inflammatory cells to the area. (pp. 796–798)
16. Interferons are secreted by virus-infected cells. They diffuse to nearby cells where they interfere with the ability of viruses to multiply within these cells. Cells that form interferon include macrophages, lymphocytes, and other leukocytes. (pp. 795–796)
17. Humoral immunity is provided by the antibodies in the body's fluids. Cell-mediated immunity is provided by non-antibody-producing lymphocytes, i.e., T cells. (p. 799)
18. Cytokines released by Helper T cells help to amplify and regulate both the humoral and cellular immune response as well as the nonspecific defense responses. (pp. 814–815)
19. Immunocompetence is the ability of the immune system's cells to recognize foreign substances (antigens) in the body by binding to them. Acquisition is signaled by the appearance of a single, unique type of cell surface receptor protein on each T or B cell that enables the lymphocyte to recognize and bind to a specific antigen. (p. 800)
20. Helper T cell activation involves a double recognition: a simultaneous recognition of the antigen and an MHC II membrane glycoprotein of an antigen-presenting cell (macrophage). One or more costimulators also appear necessary. (pp. 813–814)

21. A primary immune response results in cellular proliferation, differentiation of mature effector and memory lymphocytes, and the synthesis and release of antibodies—a series of events that takes 3 to 6 days. The secondary immune response results in huge numbers of antibodies flooding into the bloodstream within hours after recognition of the antigen, as well as an amplified cellular attack. Secondary responses are faster because the immune system has been primed to the antigen and sizable numbers of sensitized memory cells are already in place. (p. 805)
22. An antibody is a soluble protein secreted by sensitized B cells and plasma cell offspring of B cells in response to an antigen. (pp. 806–807; Fig. 21.13)
23. The variable regions of an antibody combine to form an antigen-binding site that is shaped to “fit” a specific antigenic determinant or an antigen. The constant regions of an antibody determine what class of antibody will be formed, how the antibody class will carry out its immune roles in the body, and with which cell types or chemicals the antibody will bind. (pp. 807–808)
24. The antibody classes and their probable locations in the body include the following:
 - Class IgD—virtually always attached to B cells; B cell receptor
 - Class IgM—monomer attached to B cells; pentamer free in plasma (during primary response)
 - Class IgG—in plasma
 - Class IgA—some in plasma, most in secretions such as saliva, tears, intestinal juice, and milk
 - Class IgE—secreted by plasma cells in skin, mucosae of gastrointestinal and respiratory tracts and tonsils (pp. 807–808; Table 21.3)
25. Antibodies help defend the body by complement fixation, neutralization, agglutination, and precipitation. Complement fixation and neutralization are most important in body protection. (pp. 808–810)
26. Vaccines produce active humoral immunity because most contain dead or extremely weakened pathogens which have the antigenic determinants necessary to stimulate the immune response but are generally unable to cause disease. Passive immunity is less than satisfactory because neither active antibody production nor immunological memory is established. (pp. 805–806)
27. Helper T cells function to chemically or directly stimulate the proliferation of other T cells and of B cells that have already become bound to antigen. Suppressor T cells function to temper the normal immune response by dampening the activity of both T cells and B cells by releasing cytokines that suppress their activity. Cytotoxic T cells function to kill virus-invaded body cells and cancer cells and are involved in rejection of foreign tissue grafts. (pp. 814–817)
28. Cytokines are soluble glycoproteins released by activated T cells. They enhance the defensive activity of T cells, B cells, and macrophages. Specific cytokines and their role in the immune response are summarized in Table 21.4.
29. Hypersensitivity is an antigen-induced state that results in abnormally intense immune responses to an innocuous antigen. Immediate hypersensitivities include anaphylaxis and atopy. Subacute hypersensitivities include cytotoxic and immune complex hypersensitivities. All of these involve antibodies. Delayed hypersensitivities include allergic contact dermatitis and graft rejection. These hypersensitivities involve T cells. (pp. 822–825)
30. Autoimmune disease results from changes in the structure of self-antigens, ineffective or inefficient lymphocyte programming, and by cross-reaction of antibodies produced against foreign antigens with self-antigens. (pp. 821–822)
31. Declining efficiency of the immune system with age probably reflects genetic aging. (p. 826)

Critical Thinking and Clinical Application Questions

1. a. Jenny has severe combined immunodeficiency disease (SCID) in which T cells and B cells fail to develop. At best there are only a few detectable lymphocytes. Bone marrow transplant is the treatment of choice; however, this is unsuccessful in some cases. The transplanted cells may not survive, or may mount an immune response against the recipient's tissues (graft versus host response).
- b. Jenny's brother has the closest antigenic match since both children are from the same parents.
- c. Bone marrow transplant using umbilical cord stem cells is the next best chance for survival. It is hoped that by replacing marrow stem cells, the populations of T cells and B cells would approach normal.
- d. Epstein-Barr virus is the etiologic agent of infectious mononucleosis, usually a self-limiting problem with recovery in a few weeks. Rarely, the virus causes the formation of cancerous B cells—Burkitt lymphoma.
- e. SCID is a congenital defect in which there is a lack of the common stem cell that develops into T cells and B cells. AIDS is the result of an infectious process by a virus that selectively incapacitates the CD4 (helper) T cells. Both result in a severe immunodeficiency that leaves the individual open to opportunistic pathogens and body cells that have lost normal control functions (cancerous). (p. 820)
2. IgA is found primarily in mucus and other secretions that bathe body surfaces. It plays an important role in preventing pathogens from entering the body. Lack of IgA would result in frequent major/minor infections of the sinuses or respiratory tract infections. (p. 808)
3. The mechanisms for the cardinal signs of acute inflammation involve the entire inflammatory process. The inflammatory process begins as a host of inflammatory chemicals are released into the extracellular fluid. They promote local vasodilation, allowing more blood to flow into the area, causing a local hyperemia that accounts for the redness and heat of an inflamed area. The liberated chemicals also increase the permeability to local capillaries and large amounts of exudate seep from the bloodstream into the tissue space, causing local edema or swelling. The excessive fluid in the extracellular space presses on adjacent nerve endings, contributing to a sensation of pain. (pp. 791–795)
4. Costanza was exhibiting the typical signs of anaphylaxis, an immediate hypersensitivity response. This typical inflammatory response (redness, edema, etc.) at the site of exposure to the allergen (in this case, the sting) is triggered any time the body tissues are injured. He would benefit from a topical cream containing an antihistamine drug. (p. 822–823)
5. The HIV virus is transferred from the mother to the baby through the placenta. Caroline's Helper T cells are infected. This is so devastating to the immune response because of the role of the Helper T cells in activating both the humoral immune response of the B cells and the activation of the T cytotoxic cells. Caroline is taking medications to control the infection and slow the progression of the disease to full-blown AIDS. She is taking a combination of drugs from three categories of action: reverse transcriptase inhibitors, protease inhibitors, and fusion inhibitors. (p. 821)

Suggested Readings

- Brown, Phyllida. "Cinderella Goes to the Ball." *Nature* 410 (6832) (April 2001): 1018–1020.
- Casellas, Rafael, et al. "Contribution of Receptor Editing to the Antibody Repertoire." *Science* 291 (5508) (Feb. 2001): 1541–1544.
- Check, Erika. "The Virtue of Tolerance." *Nature* 418 (6896) (July 2002): 364–366.
- Dove, Alan. "New Class of HIV Drugs Shows Promise." *Nature Medicine* 7 (12) (Dec. 2001): 1265.

- DuClos, Terry. K. "C-Reactive Protein and the Immune Response." *Science and Medicine* 8 (2) (March/April 2002): 108–117.
- Gura, Trisha. "Innate Immunity: Ancient System Gets New Respect." *Science* 291 (5511) (March 2002): 2068–2071.
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