chapter

# 21 Immune System

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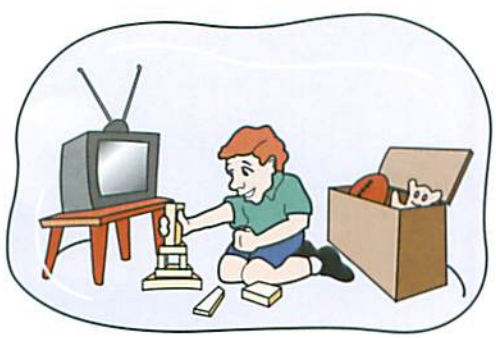
## Key Terms

**active immunity** (p. 391)
**allergens** (p. 393)
**antibodies** (p. 389)
**antibody-mediated immunity (AMI)** (p. 387)
**antigen** (p. 386)
**artificially acquired immunity** (p. 391)
**autoimmunity** (p. 393)
**B lymphocytes (B cells)** (p. 387)
**cell-mediated immunity (CMI)** (p. 387)
**clone** (p. 388)
**complement proteins** (p. 385)
**immunity** (p. 382)
**immunoglobulins** (p. 389)
**inflammation** (p. 384)
**interferons** (p. 385)
**macrophages** (p. 384)
**naturally acquired immunity** (p. 391)
**passive immunity** (p. 391)
**phagocytosis** (p. 384)
**T lymphocytes (T cells)** (p. 387)
**vaccine** (p. 391)

## Objective

1. Discuss nonspecific immunity, including:
   - Describe the process of phagocytosis.
   - Explain the causes of the signs of inflammation.
   - Explain the role of fever in fighting infection.
2. Discuss specific immunity, including:
   - Differentiate between specific and nonspecific immunity.
   - Explain the role of T cells in cell-mediated immunity.
   - Explain the role of B cells in antibody-mediated immunity.
3. Differentiate between genetic immunity and acquired immunity.
4. Describe naturally and artificially acquired active and passive immunity.
5. Describe other immune responses, including:
   - Identify the steps in the development of anaphylaxis.
   - Define *autoimmunity*.
   - Explain ways to prevent organ rejection.

Joey was born with an immunodeficiency disease; his immune system was not functioning well. This condition put Joey at high risk for life-threatening infections. As a result of this constant danger of infection, Joey spent most of his life in the sterile environment of a plastic bubble. The bubble protected him from a world of microorganisms. For persons with healthy immune systems, most microorganisms are harmless, but for Joey, the same microorganisms became dangerous pathogens. The use of bone marrow transplants has now eliminated the lifelong use of bubbles and has offered new hope for children with immunodeficiency diseases. Today we are more apt to encounter persons who are immunosuppressed because of human immunodeficiency virus (HIV) infection or treatment for cancer in the form of chemotherapy or radiation.



The study of the immune system is called *immunology*. The human body has an elaborate defense system called **immunity**. In addition to protecting the body from pathogens, the immune system protects the body from other foreign agents, including pollens such as ragweed, toxins such as bee stings, and our own cells that have gone astray (cancer cells).

## CLASSIFICATION OF THE IMMUNE SYSTEM

The defense mechanisms of the immune system are classified as nonspecific and specific immunity.

## NONSPECIFIC IMMUNITY

Nonspecific immunity protects the body against many different types of foreign agents. With nonspecific immunity, the body need not recognize the specific foreign agent. A number of defense mechanisms are included in the category of nonspecific immunity (Figure 21-1). Nonspecific immunity can be divided

7. The term MALT
   a. refers to lymphoid tissue in the mucosal membrane of many organs.
   b. is the medical term for lymph.
   c. is restricted to the spleen.
   d. is located exclusively in the mediastinum.
8. Which of the following is not true of lymph?
   a. It is formed from tissue or interstitial fluid.
   b. It is pumped into the main lymphatic ducts by the heart.
   c. It drains from the right lymphatic and thoracic ducts into the subclavian veins.
   d. It is cleansed by phagocytes as it flows through lymphoid organs.

## Go Figure

1. **According to Figures 20-2, 20-3, and 20-4**
   a. The lymphatic vessels accompany the distribution of veins.
   b. Lymph nodes act as pumps to move the lymph from node to node.
   c. Most lymph drains into the right lymphatic duct.
   d. All lymph eventually drains into the thoracic duct.
2. **According to Figures 20-1 and 20-2**
   a. Water and protein are normally filtered by the lymphatic capillaries into the interstitium.
   b. Lymphatic capillaries normally reabsorb water from the interstitium.
   c. Lymphatic vessels surround and accompany the aorta.
   d. Lymphatic vessels are found exclusively in the upper torso.
3. **According to Figures 20-5 and 20-6**
   a. Lymphoid organs are restricted to the abdominopelvic cavity.
   b. Tonsils are considered cervical lymph nodes.
   c. *Adenoids* is another name for the spleen.
   d. The lymphatic system is widely dispersed throughout the body.

into lines of defense. The first line of defense includes mechanical barriers, chemical barriers, and reflexes. The second line of defense includes phagocytosis, inflammation, fever, protective proteins (interferons and complement proteins), and natural killer (NK) cells. Remember that the nonspecific defense mechanisms work against all foreign agents; no recognition of a specific agent is necessary. Table 21-1 (later in this chapter) lists the many types of cells involved in the immune response.

## FIRST LINE OF DEFENSE

The first line of defense includes mechanical barriers, chemical barriers, and reflexes. Intact skin and mucous membranes serve as mechanical barriers; pathogens cannot cross these structures and enter the body. Destruction of mechanical barriers is an invitation to microbial invasion and subsequent infection (see Figure 21-1). Assisting the skin and mucous membranes with their defensive functions are their secretions, the chemical barriers.

For example, tears, saliva, and perspiration provide chemical barriers that wash away microorganisms. They also establish a hostile environment, thereby killing the potential pathogens. The acid and digestive enzymes secreted by the cells of the stomach kill most of the microorganisms that are swallowed. Tears secrete a substance called *lysozyme*, which discourages the growth of pathogens on the surface of the eye.

Other secretions make the environment sticky and so provide another type of chemical barrier. The mucus secreted by the mucous membranes of the respiratory tract traps inhaled foreign material. Then the cilia, which line most of the respiratory structures, sweep the entrapped material toward the throat so that the material can eventually be coughed up or swallowed. In addition to the mechanical and chemical barriers, reflexes assist in the removal of pathogens. Sneezing and coughing help remove pathogens from the respiratory tract, whereas vomiting and diarrhea help remove pathogens from the digestive tract.

Mechanical barriers, chemical barriers, and reflexes are not an adequate defense against all pathogens, however. If a pathogen penetrates this first line of defense, it encounters processes that make up the second line of defense.

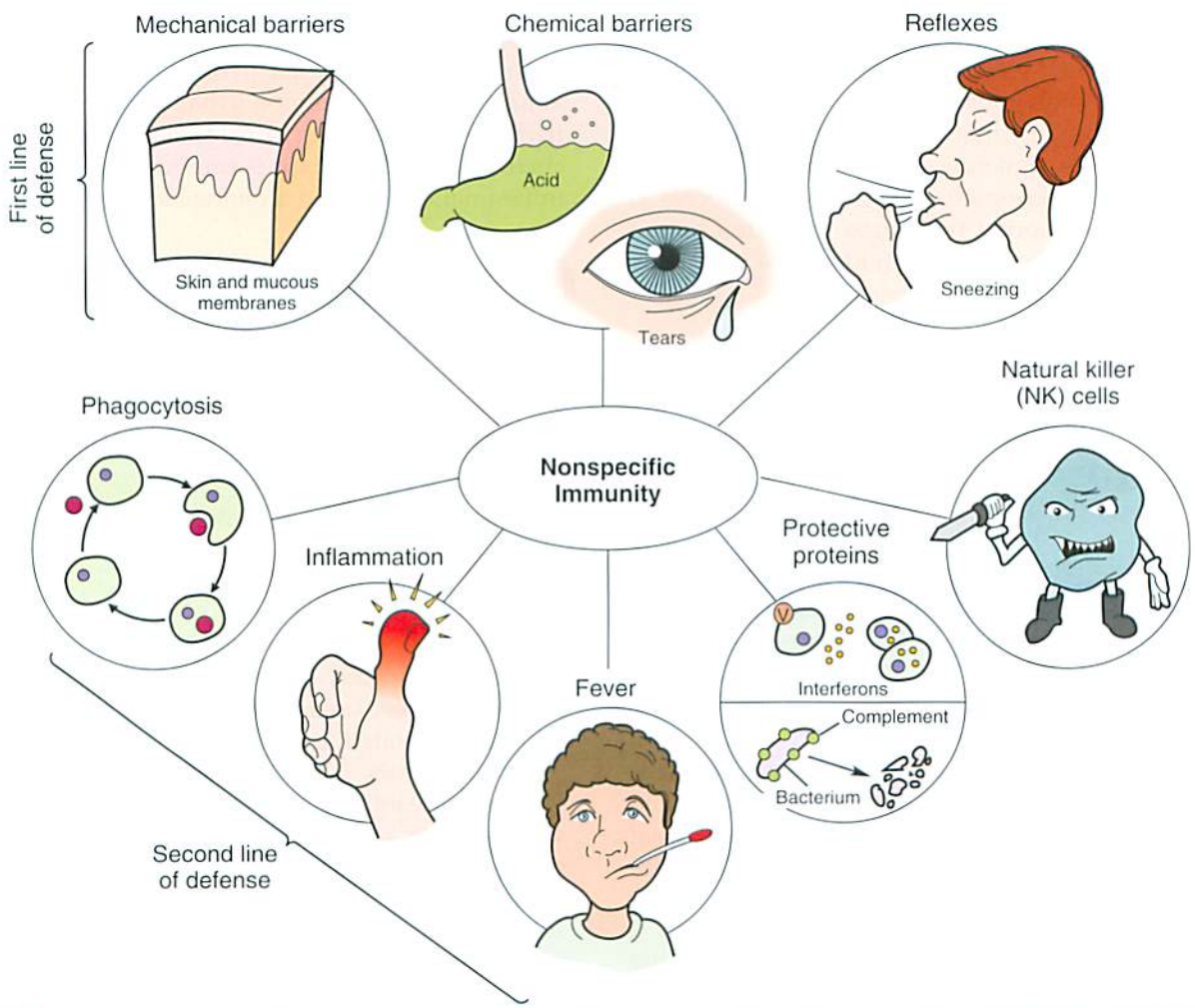



FIGURE 21-1 Nonspecific immunity. First line of defense: mechanical barriers, chemical barriers, and reflexes. Second line of defense: phagocytosis, inflammation, fever, protective proteins, and natural killer (NK) cells.

## Re-Think

1. Describe the three mechanisms that make up the first line of defense.
2. Why do health care workers wear gloves while handling the body secretions of patients?

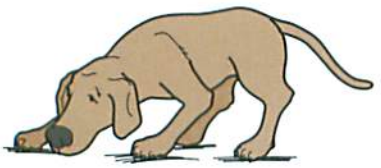
## SECOND LINE OF DEFENSE

The second line of defense includes phagocytosis, inflammation, fever, protective proteins, and natural killer (NK) cells.

### Phagocytosis

Some of the white blood cells (leukocytes) can ingest and destroy pathogens and other foreign substances by **phagocytosis**. Some phagocytes—the neutrophils and monocytes—are *motile;* they wander around the body through the blood and tissue fluid, doing their job. Other phagocytes are confined within a particular tissue and are *fixed*.

Traveling through the blood to the site of infection, the neutrophils and monocytes can squeeze through the tiny gaps between the endothelial cells of the capillary walls and enter the tissue spaces at the site of infection. The process of squeezing through the tiny gaps is called *diapedesis* (dye-ah-peh-DEE-sis). How do the neutrophils and monocytes know where to go? Chemicals released by injured cells attract them to the injured site. This signaling to attract phagocytes is called *chemotaxis* (kee-moh-TAK-sis). This process is similar to a bloodhound tracking a scent; the hound picks up the signal (odor), which identifies its source.

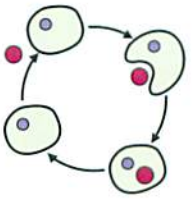


## Re-Think

What point is the bloodhound making about white blood cells?

### What Does a Phagocyte Do?

A phagocyte engulfs, or eats, particles or pathogens, much like an ameba does. The phagocyte's plasma membrane sends out pseudopods ("false feet") that surround the pathogen. The surfaces of the pseudopods then fuse, thereby enclosing the pathogen within the phagocyte. The trapped pathogen encounters a lysosome; the lysosomal membrane fuses with the pathogen, releasing potent enzymes that destroy the pathogen. The process of phagocytosis can be summarized as "ingested (eaten) and digested."



One group of phagocytic cells, the monocytes, deposit themselves in various organs and give rise to **macrophages**. As the name implies, the macrophages are big eaters. They become fixed within a particular organ and are thus nonmotile. They can, however, divide and produce new macrophages at their fixed site. The Kupffer (KOOP-fer) cells in the liver, for example, are fixed to the walls of the large capillaries called *sinusoids*. As blood flows through the sinusoids, pathogens and other foreign substances are removed from the blood and phagocytosed. The liver, spleen, lungs, and lymph nodes have a particularly rich supply of fixed phagocytes. Some fixed macrophages in the lungs are called "dust" cells because they phagocytose inhaled solid particles (better known as dust).

### Inflammation

**Inflammation** refers to the responses the body makes when confronted by an irritant. The irritant can be almost anything; common irritants include pathogens, friction, excessive heat or cold, radiation, injuries, and chemicals. If the irritant is caused by a pathogen, the inflammation is called an *infection*.

## Do You Know...

**Where You Have Seen *Rubor, Calor, Tumor,* and *Dolor?***

Enough with the Latin! These Latin words refer to the classic signs of inflammation: redness (rubor), heat (calor), swelling (tumor), and pain (dolor). Note that the Latin word for swelling is *tumor*. When the ancients used the term *tumor,* it referred to any type of swelling, even the swelling of edema. When we use the word today, we generally mean a solid mass, as in cancer.

Inflammation is characterized by redness, heat, swelling, and pain (see Figure 21-1). What are the causes of these symptoms? When the tissues are injured or irritated, injured cells release histamine and other chemicals. These chemicals cause the blood vessels in the injured tissue to dilate. The dilated blood vessels bring more blood to the area; the increased blood flow causes redness and heat. The histamine causes the blood vessel walls to leak fluid and dissolved substances into the tissue spaces, causing swelling. Fluid and irritating chemicals accumulating at the injured site also stimulate pain receptors; therefore, the person experiences pain. Redness, heat, swelling, and pain are the classic signs of inflammation.

The increased blood flow also carries an increased number of phagocytes (neutrophils and monocytes) to the injured site. As the phagocytes do their job, many are killed in the process. In a severe infection, the area becomes filled with dead leukocytes, pathogens, injured cells, and tissue fluid. This thick, yellowish accumulation of dead material is called *pus*. The presence of pus indicates that the phagocytes are doing their job.

Because of the leaky blood vessels, fluid collects in the tissue spaces. This tissue fluid contains some blood-clotting factors, such as fibrinogen, a protein present in plasma. Fibrinogen creates fibrin threads within the tissue spaces. Later, fibroblasts, the cells that form connective tissue, may also invade the injured area. The connective tissue helps contain, or restrict, the area of inflammation and thereby prevents the infection from spreading throughout the body. Fibroblastic activity is also involved in tissue repair.

### Do You Know...

**How and Why the Body "Walls Off the Pus"?**

When an area becomes infected, the cells involved in the inflammatory response do two things. First, they kill the pathogens. As the war continues, dead cells (including phagocytes, injured cells, and pathogens) and secretions accumulate in the area as pus. Second, the cells build a wall of tissue around the infected debris. This walled-off area is an abscess. An abscess performs a beneficial role in that it restricts the spread of the infection throughout the body. A large abscess may require a surgical procedure in which the abscess is lanced and drained.

### Re-Think

1. In what sense does a phagocyte act like an ameba?
2. List the four signs of inflammation and explain the physiological basis of each.

## Fever

Fever, also known as *pyrexia* (pye-REK-see-ah), is an abnormal elevation in body temperature. As phagocytes perform their duty, they release fever-producing substances called *pyrogens* (from the Latin word for "fire"). The pyrogens stimulate the hypothalamus in the brain to reset the body's temperature, producing a fever. The elevation in temperature is thought to be beneficial in two ways: a fever stimulates phagocytosis and decreases the ability of certain pathogens to multiply. In fact, the elimination of mild fevers may do more harm than good.

What happens when the hypothalamus resets the body temperature? First, the person shivers in an attempt to generate heat; the heat is conserved as the blood vessels of the skin constrict. The person may have chills and feel cold and clammy, even though the body temperature is rising. The elevated temperature hovers around the new set point while the pathogen is active, but when the infection is contained and the secretion of pyrogens diminishes, the hypothalamus resets its thermostat back to normal. Heat-losing mechanisms are activated and the blood vessels of the skin dilate, thereby losing heat as the person sweats.

Evidence suggests that the reduction of fever prolongs an infection. Note, however, that a very high fever must be reduced because high body temperature may cause severe and irreversible brain damage. High fever, especially in children, is frequently accompanied by seizures. Seizures resulting from an elevated body temperature are called *febrile (fever) seizures*.

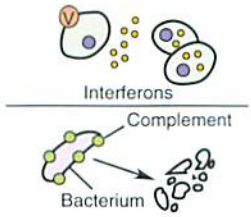
### Re-Think

Explain why pyrexia, but not hyperthermia, is considered a second line of defense. (Refer back to Chapter 7, if needed.)

## Protective Proteins

Two groups of protective proteins, the interferons and complement proteins, act nonspecifically to protect the body (see Figure 21-1). **Interferons** (in-ter-FEER-ons) are a group of proteins secreted by cells infected by a virus. The interferons diffuse to surrounding cells where they prevent viral replication. Researchers first found interferons in cells infected by the influenza virus and named them accordingly because they interfered with viral replication. Interferons also activate NK cells and macrophages, thus boosting the immune system.

A second group of proteins that protect the body are the complement proteins. **Complement proteins** circulate in the blood in their inactive form. When the complement proteins are activated against a bacterium, they swarm over it. The complement attaches to



the bacterium's outer membrane and punches holes in it. The holes in the membrane allow fluid and electrolytes to flow into the bacterium, causing it to burst and die. The activated complements perform other functions that enhance phagocytosis and the inflammatory response.

### Natural Killer Cells

Natural killer (NK) cells are a special type of lymphocyte that acts nonspecifically to kill a variety of cells. NK cells are effective against many microbes and certain cancer cells. NK cells cooperate with the specific defense mechanisms to mount the most effective defense possible.

### 2+2 Sum It Up!

Figure 21-2 summarizes the functions of the nonspecific defense mechanisms. The wall of the fortress is the first line of defense. It protects the body from invaders such as bacteria, fungi, and viruses. The first line of defense includes mechanical barriers, chemical barriers, and reflexes. Behind the wall of the fortress is the second line of defense: phagocytosis, inflammation, fever, protective proteins, and NK cells. The third line of defense includes lymphocytes that are concerned with specific immunity, the topic of the next section.

## SPECIFIC IMMUNITY: THIRD LINE OF DEFENSE

Specific immunity homes in on a foreign substance and provides protection against one specific substance but not others. It protects against a specific foreign agent such as the measles virus (a specific pathogen) or ragweed pollen. The two cells that play key roles in specific immunity are the lymphocytes (B lymphocytes and T lymphocytes) and the macrophages. Understanding the function of lymphocytes requires an understanding of antigens.

### ANTIGENS

An **antigen** is a substance that stimulates the formation of antibodies. Antigens are generally large molecules; most are proteins, but a few are polysaccharides and lipids. Antigens are found on the surface of many substances, such as pathogens, red blood cells, pollens, foods, toxins, and cancer cells. Foreign substances that display antigens are described as antigenic. Antigenic substances are attacked by lymphocytes.

### SELF AND NONSELF: IS THAT ME?

Before birth, your lymphocytes somehow get to know who belongs and who does not. In effect, your

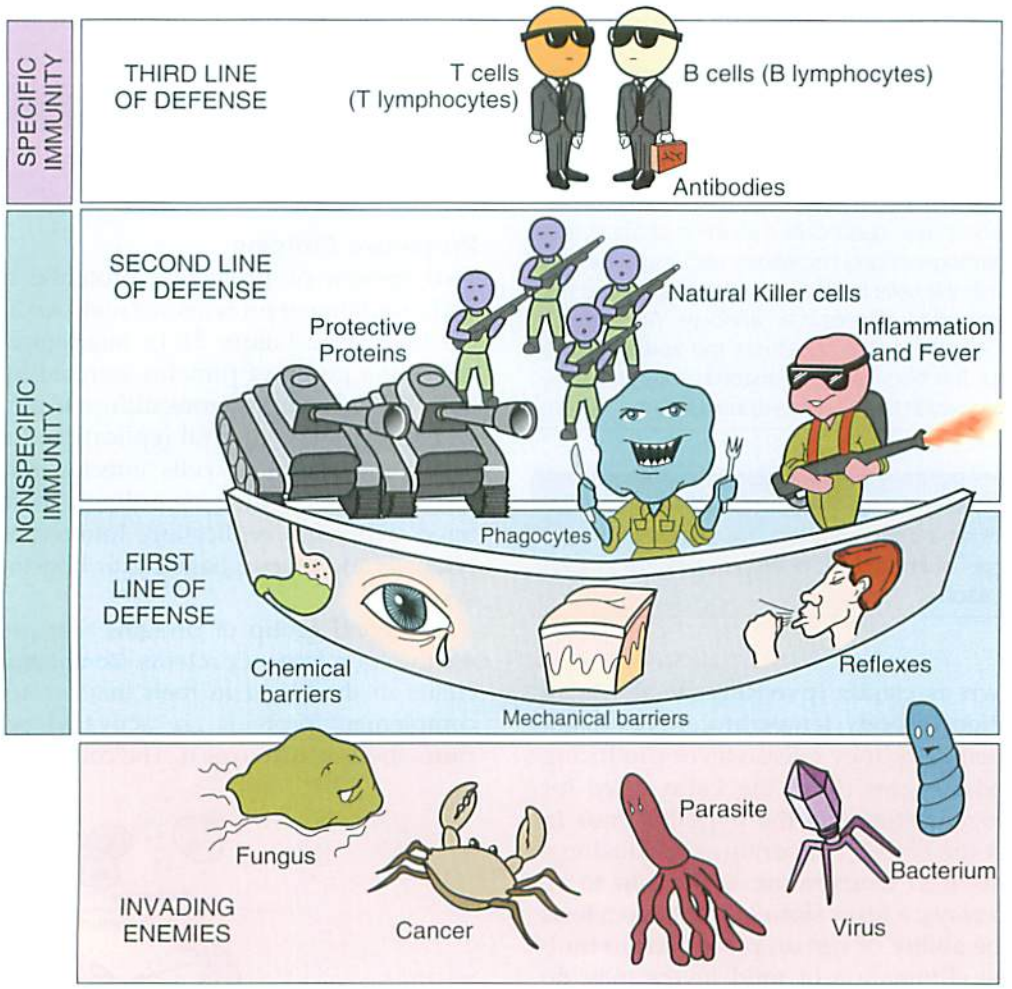



FIGURE 21-2 The immune system wages its battle with three lines of defense. (Read from bottom to top.)

**Table 21-1** Cells Involved in Immunity

| CELL TYPE | PRODUCTION SITE | FUNCTION |
|---|---|---|
| **Granular Leukocytes** | | |
| Neutrophils | Bone marrow | Phagocytosis |
| Basophils | Bone marrow | Secrete histamine and heparin |
| Eosinophils | Bone marrow | Destroy parasites |
| **Nongranular Leukocytes** | | |
| Monocytes | Bone marrow | Phagocytosis; they enter tissues and are transformed into macrophages |
| Lymphocytes | | |
| • B cells | Bone marrow | Antibody-mediated immunity; accounts for 20% to 30% of blood lymphocytes |
| Plasma cells | | Secrete antibodies |
| Memory B cells | | Remember the antigens |
| • T cells | Bone marrow and lymphoid tissue | Cell-mediated immunity; accounts for 70% to 80% of blood lymphocytes |
| Killer T cells | | Kill cells |
| Helper T cells | | Secrete lymphokines, which activate B cells and other cells |
| Suppressor T cells | | Inhibit B-cell and T-cell activity (help control immune response) |
| Memory T cells | | Remember the antigens |
| • Natural killer (NK) cells | Lymphoid tissue | Kill cells |
| **Other Cells** | | |
| Macrophages | Almost all organs and tissues | Phagocytosis; present antigens to lymphocytes |
| Mast cells | Almost all organs and tissues, especially liver and lungs | Release histamine and other chemicals involved in inflammation |

lymphocytes learn to recognize "you" (self) and take steps to eliminate "not you" (nonself, or foreign agent). Your body perceives your own cells and secretions as nonantigenic and other cells as antigenic. The antigenic cells are subsequently eliminated. Recognition of self is called *immunotolerance*. Sometimes, a person's immune system fails to identify self and mounts an immune attack against its own cells. This attack is the basis of autoimmune diseases, such as rheumatoid arthritis.

## LYMPHOCYTES

The two types of lymphocytes are **T lymphocytes (T cells)** and **B lymphocytes (B cells)**. Although both come from the stem cells in the bone marrow, they differ in their development and functions (Table 21-1).

### Why the Names "T" and "B" Cells?

During fetal development, stem cells in bone marrow produce lymphocytes. The blood carries lymphocytes throughout the body. About half of the lymphocytes travel to the thymus gland, where they mature and differentiate into T cells (the "T" is for *t*hymus-derived lymphocytes). Eventually, the blood carries T cells away from the thymus gland to various lymphoid tissues, particularly the lymph nodes and spleen. T cells live, work, and reproduce in the lymphoid tissue as well as circulate in the blood, making up 70% to 80% of the blood's lymphocytes.

What about the B lymphocytes? B cells differentiate in the fetal liver and bone marrow (the "B" is for *b*one marrow). Like the T cells, the B cells take up residence in lymphoid tissue. B cells make up 20% to 30% of the circulating lymphocytes.

Both T cells and B cells attack antigens, but they do so in different ways. T cells attack antigens directly, through cell-to-cell contact. This immune response is called **cell-mediated immunity (CMI)**. B cells, on the other hand, interact with the antigen indirectly, through the secretion of antibodies. This response is called **antibody-mediated immunity (AMI)**. Because the antibodies are carried by the blood and other tissue fluid (the body "humors"), this type of immunity is also called *humoral immunity*.

### Cell-Mediated Immunity: T-Cell Function

CMI is effective against many pathogens, tumor cells, and foreign tissue such as organ transplants. Refer to Figure 21-3 as you read about the following steps in CMI:

- *Step 1.* The antigen, on the surface of the pathogen, is phagocytosed by a macrophage. The macrophage digests the antigen and pushes the antigen to its surface. The macrophage's ability to push the antigen to its surface is called *antigen presentation*.

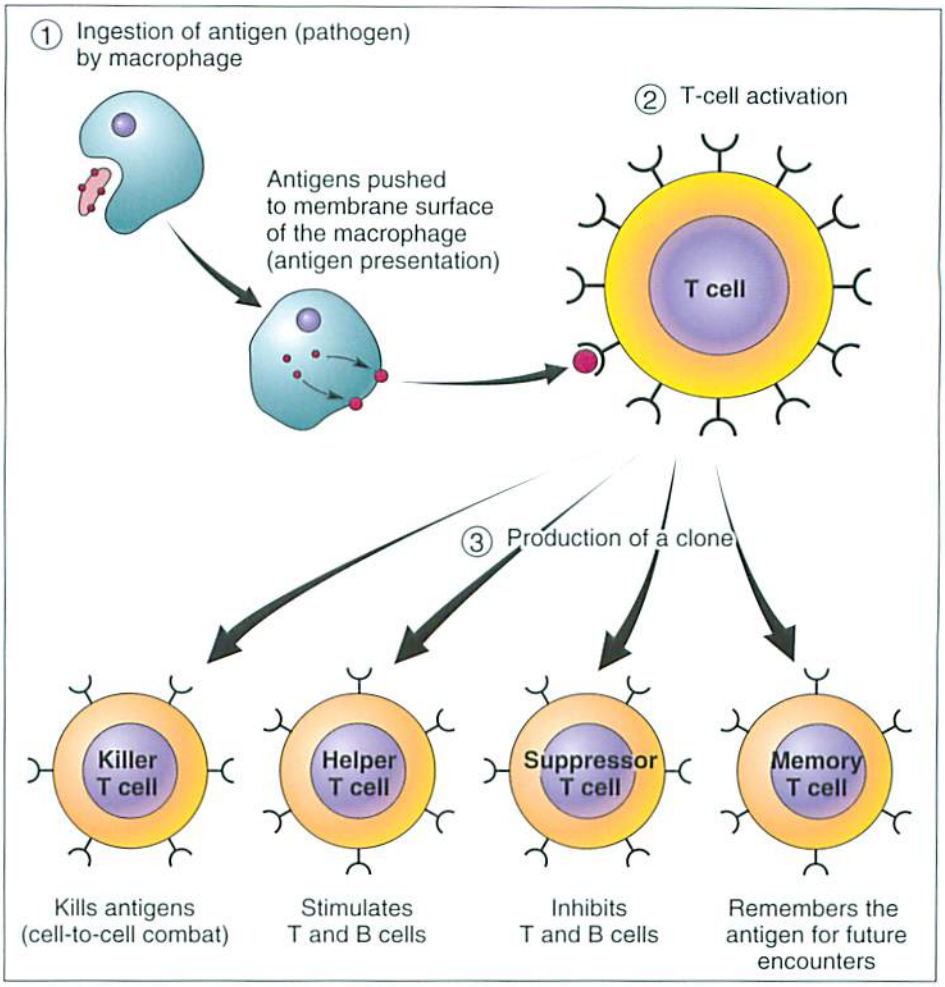



FIGURE 21-3 Cell-mediated immunity.

- *Step 2.* T cells that have receptor sites bind to the antigen and become activated. This process is called *T-cell activation*. Activation of the T cell always requires an antigen-presenting cell, such as a macrophage.
- *Step 3.* The activated T cell divides repeatedly, resulting in large numbers of T cells. This group of T cells is called a **clone**, a group of identical cells formed from the same parent cell. Four subgroups are within the clone: killer T cells, helper T cells, suppressor T cells, and memory T cells.

The killer T cells destroy the antigen (pathogen) through the use of two mechanisms, punching holes in the pathogen's cell membrane and secreting substances called *lymphokines*, which enhance phagocytic activity. The killer T cells engage in cell-to-cell combat. The helper T cells also secrete a lymphokine that stimulates T cells and B cells and, in general, enhances the immune response. The suppressor T cells inhibit the immune response when the antigen has been destroyed. The suppressor T cells control B- and T-cell activity.

The memory T cells do not participate in the destruction of the antigen. These cells "remember" the initial encounter with the antigen. If the antigen is presented at some future time, the memory cells quickly reproduce and thus allow a faster immune response to occur.

### Antibody-Mediated Immunity: B-Cell Function

B cells engage in AMI. Activated B cells produce a clone of cells that secrete antibodies. The antibodies are carried by the blood and body fluids to the antigen-bearing pathogens. Individual B cells can produce over 10 million different antibodies, each of which reacts against a specific antigen. The large numbers of antibodies allow the body to develop immunity against many different diseases. Follow Figure 21-4 as you read about the following steps in AMI:

- *Step 1.* A macrophage engulfs and processes an antigen. The antigen is pushed to the surface of the macrophage and presented to the B cell and helper T cell.
- *Step 2.* The presented antigens bind to the B cell and helper T cell, activating both cells. Only those B cells and helper T cells with the proper receptors are activated.

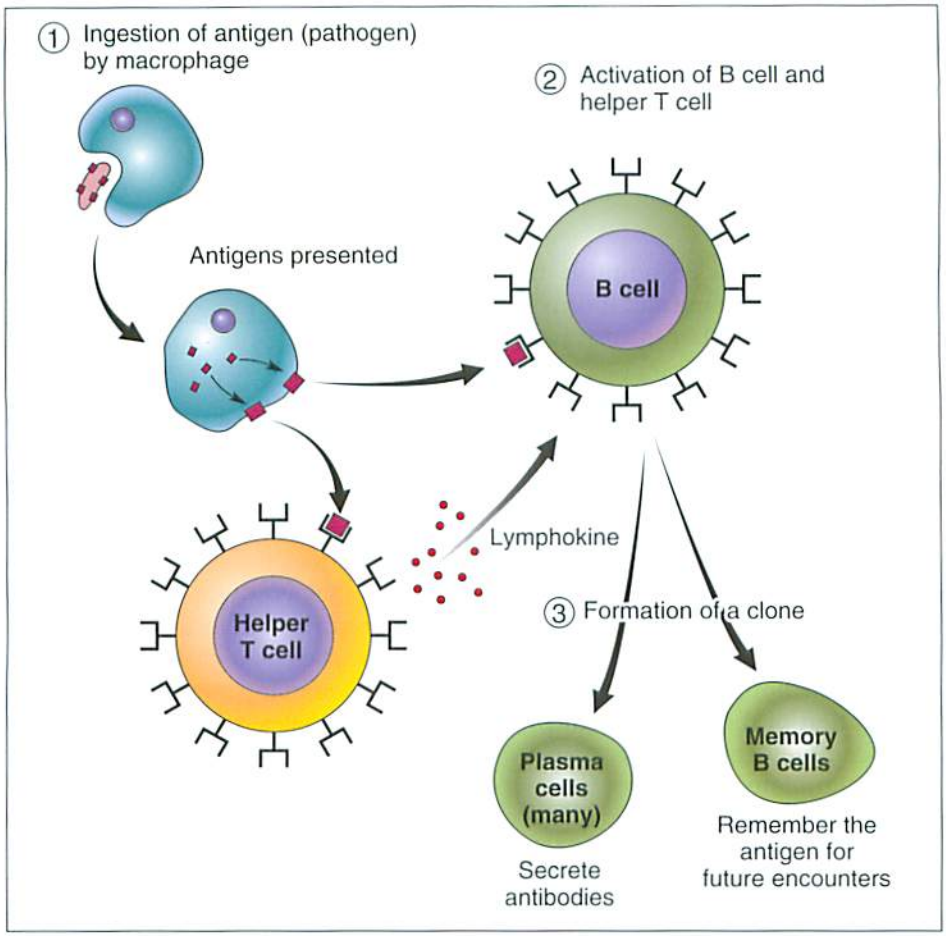



FIGURE 21-4 Antibody-mediated immunity.

- *Step 3.* The activated helper T cells secrete a lymphokine that stimulates the B cells to reproduce, producing a clone. The two subgroups of the clone include plasma cells and memory B cells. Plasma cells produce large quantities of antibodies that travel through the blood to the antigens (pathogens). The memory B cells do not participate in the attack; they remember the specific antigen during future encounters and allow a quicker response to the invading antigen.

Note that B- and T-cell activation depends on helper T-cell activity. HIV attacks the helper T cells, thereby producing severe impairment of the immune system. This syndrome is called *acquired immunodeficiency syndrome* (AIDS). Because of the impairment of their immune system, persons with HIV infection and AIDS experience numerous bouts of infection, some of which may be lethal. The immune response of an AIDs patient may be so inadequate that even the common cold presents a life-threatening challenge.

The helper T cell is also called the $CD4^+$ T-cell (because of a surface protein called CD4). The $CD4^+$ T cell is a marker for immune function and the progression of HIV infection is monitored by the $CD4^+$ T-cell count. The $CD4^+$ T-cell count usually decreases as the infection progresses.

## Re-Think

1. List two lymphocytes that engage in specific immunity.
2. What is the consequence of the failure of the immune system to "recognize self"?
3. How does the action of the killer T cell differ from that of the plasma cell?

## ANTIBODIES

### What Antibodies Are

The **antibodies** secreted by the B cells are proteins called **immunoglobulins**. There are five major types of immunoglobulins. The three most abundant immunoglobulins are immunoglobulin G, immunoglobulin A, and immunoglobulin M. A fourth immunoglobulin, immunoglobulin D, is present in small amounts. A fifth immunoglobulin, immunoglobulin E, is involved in hypersensitivity reactions and is described later in this chapter.

- Immunoglobulin G (IgG) is an antibody found in plasma and body fluids. It is particularly effective against certain bacteria, viruses, and toxins.
- Immunoglobulin A (IgA) is an antibody found primarily in the secretions of exocrine glands. IgA in milk, tears, and gastric juice helps protect against

infection. Breast milk contains IgA antibodies and helps the infant ward off infection.

- Immunoglobulin M (IgM) is an antibody found in blood plasma. The anti-A and anti-B antibodies associated with red blood cells are a type of IgM antibody.

### What Antibodies Do

Antibodies destroy antigens. They accomplish this task directly by attacking the membrane and indirectly by activating complement proteins that in turn facilitate the attack on the antigens.

When antibodies react with antigens directly, the antibodies bind to antigens in a process called an *antigen–antibody reaction*. By engaging in an antigen–antibody reaction, the antigen–antibody components clump together, or agglutinate (ah-GLOO-tin-ate). Agglutination makes it easier for the phagocytic cells to destroy the antigen. Under normal conditions, direct attack by the antibodies is not very helpful in protecting the body against invasion by pathogens.

A more effective way for antibodies to attack an antigen is through activation of the complement proteins. These activated complement proteins cause a variety of effects: they stimulate chemotaxis (attract more phagocytes), promote agglutination, make pathogens more susceptible to phagocytosis, and encourage lysis, or rupture of the pathogen's cell membrane. Direct and indirect attacks by antibodies provide an effective defense against foreign agents.

### ? Re-Think

1. CMI achieves cell death by cell-to-cell combat. How does AMI achieve the death of a pathogen?
2. How does agglutination help out the phagocytes?

### Remember Me? Primary and Secondary Responses

Activated when exposed to an antigen, B cells produce many plasma cells and memory cells. The plasma cells secrete antibodies. This initial response to an antigen is called the *primary response* (Figure 21-5). The primary response develops slowly and produces a small number of antibodies. Note what happens when the immune system is challenged for a second time by the same antigen. The immune system responds quickly and produces a larger number of antibodies. This second challenge is called the *secondary response*. Compare the plasma levels of antibodies in the primary and secondary responses.

Why is the secondary response so much greater? The initial exposure to the antigen has stimulated the formation of antibody-secreting plasma cells and memory cells. The memory cells, which live for a long time in the plasma, are activated very quickly on the second exposure. The activated memory cells, in turn, induce the formation of many antibody-secreting plasma cells.

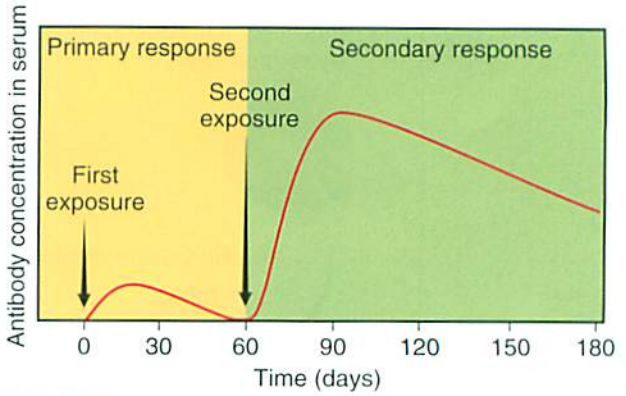



FIGURE 21-5 Primary and secondary responses to an antigen.

What does the secondary response mean for you? It means that you won't get the disease a second time; you are immune to that disease. For example, if you had measles as a child, you developed measles antibodies and many memory cells. If you are then exposed to the measles virus later in life, the memory cells "remember" the first exposure and produce antibody-secreting plasma cells very quickly. The measles antibodies, in turn, attack the measles virus and prevent you from becoming ill.

The level of antibodies in your blood is called an *antibody titer*. If you have had measles, for example, your measles antibody titer is higher than the titer of someone who has never had measles.

### ? Re-Think

List two ways that the secondary response differs from the primary response.

### 2+2 Sum It Up!

Specific immunity forms the third line of defense of the immune response. It allows the immune system to recognize and destroy specific substances called *antigens*. The B and T lymphocytes and the macrophages are the most important cells associated with specific immunity. T cells engage in cell-to-cell combat (CMI), whereas B cells fight at a distance through the mediation of antibodies (AMI). Macrophages not only engage in phagocytosis, but also present the antigen to the lymphocytes. Antibodies are called *immunoglobulins* (IgG, IgA, IgM, IgD, and IgE); they engage antigens causing agglutination. Agglutination, in turn, facilitates phagocytosis. The primary and secondary responses refer to the secretion of antibodies by plasma cells in response to antigen stimulation.

## TYPES OF IMMUNITY

The two main categories of immunity are genetic immunity and acquired immunity (Figure 21-6).

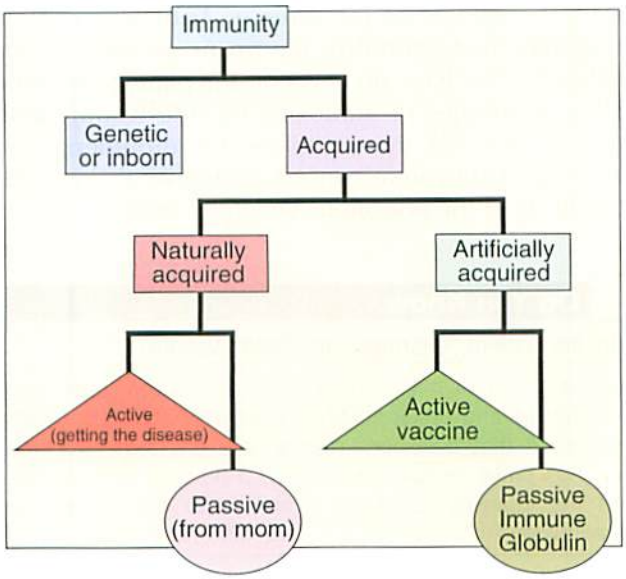



FIGURE 21-6 Types of immunity.

## GENETIC IMMUNITY

Do you ever wonder why you have never gotten heartworms from your dog or why your dog did not pick up chickenpox from you? As a human, you have inherited immunity to certain diseases such as canine heartworm; you are immunologically protected from your pet. Similarly, your dog will never contract chickenpox; Rover is immunologically protected from you. Another comforting thought is that neither you nor your dog is in danger of contracting Dutch elm disease from your tree. Each of you was born with genetic information that provides immunity to certain diseases. Genetic immunity is also called *inborn*, *innate*, or *species immunity*. As you can see, your species protects you from many diseases that afflict other species.

## ACQUIRED IMMUNITY

Unlike genetic immunity, acquired immunity is received during a person's lifetime. Acquired immunity comes either naturally or artificially.

### NATURALLY ACQUIRED IMMUNITY

You can acquire immunity naturally in two ways. The first is by getting the disease. As a child, you probably had one of the childhood diseases such as chickenpox. Your body responded to the specific pathogen by developing antibodies. After that first exposure, you never became ill with chickenpox again because your immune system had a ready supply of antibodies and memory cells with which to respond quickly to the second invasion of the chickenpox virus. Because your own body produced the antibodies, this type of **naturally acquired immunity** is called **active immunity**. Active immunity is generally long lasting.

The second way to acquire immunity naturally is by receiving antibodies from your mother. Some antibodies (IgG) crossed the placenta from your mother into you as a fetus. Your mother developed these antibodies in response to the pathogens that she encountered throughout her lifetime. Because your immune system did not produce these antibodies (you received them as a gift from your mother), this type of immunity is called **passive immunity**. Antibodies can also be transferred passively from mother to infant through breast milk. Breast milk contains IgA antibodies.

Unlike active immunity, which often lasts a lifetime, passive immunity is short lived. The antibodies that are acquired passively are broken down and eliminated from the baby's body. The mother's antibodies afford protection to the infant for about the first 6 months after birth. Breast-feeding may extend the length of immunoprotection.

### ARTIFICIALLY ACQUIRED IMMUNITY

You can also acquire immunity artificially in two ways. The first is by a vaccine; the second is by injection of immune globulin. Both provide **artificially acquired immunity**.

A **vaccine** is an antigen-bearing substance, such as a pathogen, injected into a person in an attempt to stimulate antibody production. For example, the measles virus is first killed or weakened or attenuated (ay-TEN-yoo-ayt-ed). The attenuated virus cannot cause the disease (measles) when injected into the person, but it can still act as an antigen and stimulate the person's immune system to produce antibodies. The use of a dead or attenuated pathogen to stimulate antibody production is vaccination, or immunization. The solution of dead or attenuated pathogens is the vaccine. Because the use of a vaccine stimulates the body to produce its own antibodies, vaccines induce active immunity.

A vaccine can also be made from the toxin secreted by the pathogen. The toxin is altered to reduce its harmfulness, but it can still act as an antigen to induce immunity. The altered toxin is called a *toxoid*. Because a toxoid stimulates the production of antibodies, it causes active immunity.

The purpose of vaccination is to provide an initial exposure and stimulate the formation of memory cells (the primary response). The purpose of a booster shot is to stimulate the secondary response by administering another dose of the vaccine.

Vaccines have almost eradicated certain diseases. For example, infants routinely receive a series of DTP injections. DTP injections stimulate active immunity for *d*iphtheria (diphtheria toxoid), *t*etanus (tetanus toxoid), and *p*ertussis, or whooping cough (pertussis vaccine). MMR vaccine (*m*easles-rubeola, *m*umps, and *r*ubella) is also used preventively during early childhood.

Immune globulin differs from a vaccine. Immune globulin is obtained from a donor (human or animal) and contains antibodies (immune globulins). The antibodies are formed in the donor in response to a specific antigen. These preformed antibodies are taken from the donor and injected into a recipient, thereby conveying passive immunity.

### Do You Know...

**Why Joey is Mooing?**

Here's the story. Vaccination against smallpox was originally accomplished by injecting the cowpox virus, a cousin to the smallpox virus. Although Edward Jenner (circa 1850) had demonstrated some success with vaccination, many doctors opposed the procedure. His fellow doctors therefore spread a nasty rumor designed to scare the peasant population. The rumor claimed that the injection of the cowpox virus makes the child take on the characteristics of a cow...Moooooo! Pictures were widely distributed of Joey mooing, swatting flies with his tail, and clanging a cow bell hung around his neck.

Fortunately, the peasants weren't duped because they knew that milkmaids rarely came down with smallpox. Most attributed the immunity of the milkmaids to the cowpox lesions that developed when the milkmaid milked the cows infected with the cowpox virus. (Holy cow—this sounds like bull!) Just remember, the Latin word for cow is *vacca*, the root word for *vaccination*.

Why might this procedure be done? Assume for the moment that you are not immune to hepatitis B and so do not have antibodies against the hepatitis B virus. You are then exposed to the virus. Because you have no immunity to the virus, you may receive immune globulin (antibodies) in an attempt to provide immediate protection against the virus. Because this is a form of passive immunity, the immunity is short-lived. Immune globulins are available for rubella (German measles), hepatitis A and B, rabies, and tetanus. A comparison of the different types of acquired immunity appears in Table 21-2.

Other forms of passive immunity are commonly used to prevent the disease or the development of severe symptoms of the disease. Antitoxins contain antibodies that neutralize the toxins secreted by the pathogens but have no effect on the pathogens themselves. Examples of antitoxins include tetanus antitoxin (TAT) and the antitoxins for diphtheria and botulism. Antivenoms contain antibodies that combat the effects of the poisonous venom of snakes.

### Do You Know...

**Why the Ancient "Charmers" Ate Snake Venom?**

Long ago, Indian snake charmers squeezed the venom from their cobras and drank it. Why? They realized that by eating the venom, they developed resistance to the bite of the family pet. This practice was observed by a number of physicians, and the concept eventually evolved into our modern-day immunology.

### Re-Think

1. Differentiate between active and passive immunity. Give an example of each.
2. Differentiate between naturally and artificially acquired immunity. Give an example of each.

## OTHER IMMUNE RESPONSES

Normally the immune system protects the body from nonself; foreign agents are recognized and eliminated. Sometimes, however, the immune system goes awry: it attacks the self, causing autoimmune disease, or it overreacts, causing allergies.

## ALLERGIC REACTIONS

The immune system sometimes forms antibodies to substances not usually recognized as foreign. This response forms the basis of allergic reactions. The two common allergic reactions are the delayed-reaction allergy and the immediate-reaction allergy.

**Table 21-2** Types of Acquired Immunity

| TYPE | STIMULUS | RESULT |
|---|---|---|
| **Naturally Acquired** | | |
| Active immunity | Exposed to live pathogens (e.g., get the disease) | Long-term immunity; makes antibodies |
| Passive immunity | Antibodies are passed from mother to infant (across placenta and/or by breast-feeding) | Short-term immunity (lasts approximately for the first 6 months and for duration of breast-feeding); does not stimulate the production of antibodies |
| **Artificially Acquired** | | |
| Active immunity | Vaccination | Long-term immunity; makes antibodies |
| Passive immunity | Injection with gamma globulin (antibodies) | Short-term immunity; does not stimulate the production of antibodies |

The *delayed-reaction allergy* is so named because it usually takes about 48 hours to occur; its onset is delayed. This type of allergic response can occur in anyone. It usually results from the repeated exposure of the skin to chemicals such as household detergents. Repeated exposure to the chemical activates T cells, which eventually accumulate in the skin. Local tissue response to T-cell activity causes skin eruptions and other signs of inflammation. This skin response is called *contact dermatitis*. Other forms of contact dermatitis are associated with poison ivy, poison oak, certain cosmetics, and soaps.

The *immediate-reaction allergy*, as its name implies, occurs rapidly in response to its stimulus. It is more commonly called *immediate hypersensitivity reaction* and involves immunoglobulin E, the IgE antibodies. **Allergens** (antigens) are substances capable of inducing allergy. Allergens that are apt to be involved in this type of allergic response include pollens, such as ragweed, insect venom, drugs such as penicillin, and foods such as peanuts. See Figure 21-7, the steps involved in the development of an immediate hypersensitivity reaction.

- *Step 1.* An allergen activates a B cell.
- *Step 2.* The activated B cell forms a clone of antibody-secreting plasma cells.
- *Step 3.* The plasma cells secrete large amounts of IgE antibodies against the specific allergen.
- *Step 4.* The IgE antibodies bind to the mast cells in body tissues.
- *Step 5.* More of the allergen invades the body.

The allergen binds with the IgE antibodies on the mast cells. The mast cells release large amounts of histamine, leukotrienes, and other chemicals that cause systemic effects. The systemic effects can be severe; they include a massive vasodilation, which causes a sharp drop in blood pressure and severe constriction of the respiratory passages (bronchoconstriction), making breathing extremely difficult and, in some cases, impossible. This severe form of the immediate hypersensitivity reaction is called *anaphylaxis* or *anaphylactic shock*. Persons allergic to penicillin are at particular risk for anaphylaxis. As a result, always ask a person about allergies to medications before administering any type of drug, particularly antibiotics. (Some persons are even allergic to aspirin.)

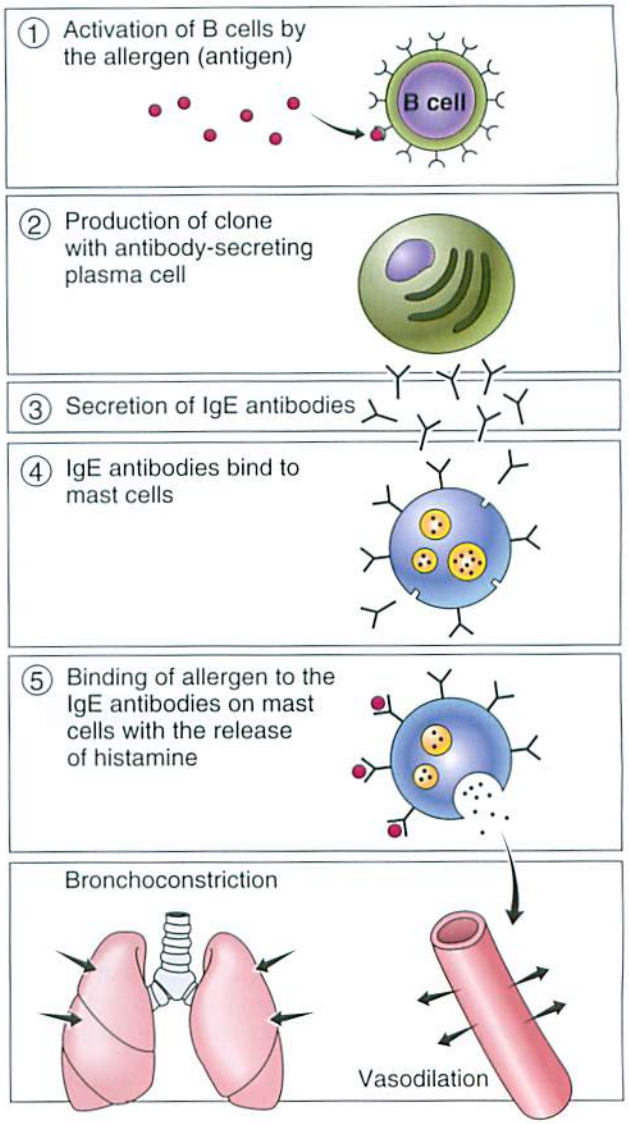



FIGURE 21-7 Immediate hypersensitivity reaction.

**? Re-Think**

Describe the steps involved in the development of anaphylaxis.

## AUTOIMMUNE DISEASE

Sometimes, a person's T cells attack his or her own body, causing extensive tissue damage and organ dysfunction. This process is **autoimmunity**. Diseases that develop in response to self-attack are called *autoimmune diseases*. A surprisingly large number of diseases are considered autoimmune.

Consider this. The immune system is affected by your endocrine system and your nervous system. Your endocrine and nervous systems are both affected by your emotional state (happy, sad, angry). What are the chances that your emotional state can affect your immune system and hence your physical well-being? In other words, does happiness promote health, whereas anger and depression cause disease? A branch of science called *psychoneuroimmunology* seeks to explore this relationship. In the meantime, be happy!

## ORGAN REJECTION

Organ transplantation has become a common means of dealing with organ failure. A patient in kidney failure, for example, may receive a kidney transplant.

One of the greatest problems associated with this surgical technology is organ rejection. The recipient's immune system recognizes the donated kidney as *foreign* and mounts an immune attack against it. When the immune attack is successful, the organ is destroyed and is said to be rejected.

There are several ways to prevent organ rejection. The physician first selects a donor organ that is immunologically similar to the recipient's tissues. The physician then administers drugs that suppress the recipient's immune system. Cyclosporine is a commonly used immunosuppressant that inhibits the secretion of certain lymphokines, which, in turn, diminishes the immune attack against the donated organ. Unfortunately, these measures are not always successful, and the recipient may ultimately reject the organ.

## Do You Know...

### Why This Bee Might Kill Auntie Bea?

Auntie Bea has become sensitized to bee venom (antigen). IgE antibodies to the bee venom attached to the surface of her mast cells. If Auntie is stung again, the bee venom antigen will bind to the IgE antibodies on the mast cells, causing a massive release of histamine. The histamine causes an anaphylactic reaction. Unless treated immediately, she is apt to die of anaphylactic shock. Treatment is usually an injection of epinephrine (Adrenalin), which opens breathing passages and elevates blood pressure. Steroids (cortisol) are also given to suppress the immune response.

## Do You Know...

### About the Ultimate Rejection?

When dealing with transplant patients, we watch closely for signs of organ rejection by the host (recipient). However, some patients experience the opposite—the transplanted organ (or blood) rejects the host. This response is called *graft-versus-host disease* (GVHD); GVHD usually occurs in immunodeficient patients and is caused in part by transplanted T cells. The target organs for rejection are the skin, digestive tract, and liver. The biggest clinical problem is a variety of infections that gradually overwhelm the granulocytopenic patient. Imagine being rejected by an organ!

## 2+2 Sum It Up!

Immunity is classified as genetic or acquired. Immunity may be acquired naturally or artificially. Immunity is also classified as active or passive. If a person makes antibodies in his or her own body, the immunity is active. If the person merely receives antibodies that were made by another person or animal, the immunity is passive. Active immunity is generally long lasting, whereas passive immunity is short lasting. Although the immune system normally works to protect the body, it can go awry, causing allergic reactions and autoimmune disease. Anaphylaxis is a serious IgE hypersensitivity reaction that is life threatening.

## As You Age

1. T-cell and B-cell function are somewhat deficient in older adults. Depressed lymphocyte function is accompanied by a decrease in macrophage activity. Consequently, older adults are more prone to develop infections and they recover more slowly. Depressed lymphocyte function might also explain the higher incidence of cancer in older adults.
2. Older adults often have a reduced fever response to infection and may therefore have difficulty in combating infection.
3. Older adults have increased levels of circulating autoantibodies (antibodies directed against self). This increase explains, in part, why they are more prone to the development of autoimmune disease.
4. Older adults often take drugs or undergo treatment that depresses the immune system. For example, the use of steroids in the treatment of arthritis and the use of drugs and radiation in the treatment of cancer all cause immunosuppression.

## MEDICAL TERMINOLOGY AND DISORDERS Disorders of the Immune System

| Medical Term | Word Parts | Word Part Meaning or Derivation | Description |
|---|---|---|---|
| **Words** | | | |
| anaphylaxis | ana- | up | **Anaphylaxis** is *a life-threatening hypersensitivity reaction mediated by IgE antibodies.* |
| | -phylaxis | From a Greek word meaning "watching or guarding" | |
| immune | immune/o- | From a Latin word meaning "exempt from" | To be **immune** is *to be exempt or protected from a particular disease.* |
| lymphocyte | lymph/o- | lymph | A **lymphocyte** is *a nongranular white blood cell.* There are T lymphocytes (T cells) and B lymphocytes (B cells). |
| | -cyte | cell | |
| macrophage | macro- | large | A **macrophage** (fixed or wandering) is *a type of white blood cell that digests foreign materials, including pathogens.* |
| | -phage | eat | |
| wheal and flare reaction | | | A **wheal and flare reaction** is *a skin reaction to an allergen or antigen.* The response is a circular (wheal) blanched area surrounded by an area of redness. |
| **Disorders** | | | |
| hypersensitivity reactions | | | A **hypersensitivity reaction** is *the immune system response to a foreign substance, or what is perceived as "foreign" in the case of autoimmune disease. Hypersensitivity reactions include allergies and autoimmune diseases.* |
| allergies | | | An **allergy** occurs when the immune system reacts to a foreign substance. Allergies are described in many ways: atopic, bacterial, contact, cold, drug, immediate, delayed, etc. The symptoms vary in intensity from mild to life threatening. An **atopic allergy** is genetic and refers to the development of a hypersensitivity reaction to an environmental allergen (e.g., pollen, bee venom, dander, food, environmental chemicals). Atopic reactions generally include eczema (atopic dermatitis), allergic rhinitis (hay fever), allergic conjunctivitis, or allergic asthma. A **latex allergy** is an allergic reaction to proteins found in natural rubber latex. Latex is found in many articles, such as shoes, balloons, condoms, medical equipment, drugs, paints, and adhesives, and for some is considered a major occupational hazard. About 50% of persons with latex allergy have allergic reactions to certain foods: avocado, banana, plum, strawberry, and tomato. **Anaphylaxis** is a severe and life-threatening hypersensitivity reaction, mediated by IgE antibodies. |
| autoimmune diseases | auto- | self | **Autoimmune system disorders** occur *when a person's immune system produces antibodies against its own cells; the immune system no longer recognizes self from nonself.* There are many **autoimmune diseases,** including rheumatoid arthritis, Hashimoto thyroiditis, diabetes mellitus (type 1), and rheumatic fever. **Rheumatic fever** is an immune disorder in which the antibodies produced in response to a streptococcal infection attack the heart muscle and its valves. Many of the autoimmune diseases are described in other chapters. |
| | -immun/o- | immunity | |

*Continued*

**MEDICAL TERMINOLOGY AND DISORDERS** Disorders of the Immune System—cont'd

| Medical Term | Word Parts | Word Part Meaning or Derivation | Description |
|---|---|---|---|
| immunodeficiency disorders | immun/o-<br>-deficiency | | *Disorders caused by an impaired immune response.* Immunodeficiency disorders are classified as primary or secondary. **Primary immunodeficiency disorders** are rare, an example being **severe combined immunodeficiency disease (SCID). Secondary immunodeficiency disorders** are common, the most common cause being a drug-induced immune deficiency such as the granulocytopenia resulting from cancer chemotherapy. Other causes include radiation therapy, steroid therapy, stress, malignancies, aging, and many diseases, especially infection with the human immunodeficiency virus (HIV). **HIV infection** is usually transmitted sexually. Because it is also transmitted through accidental or intended sharing of injection equipment, HIV is a serious threat to hospital workers and others who routinely handle blood or blood-related products or equipment. Initial infection with HIV causes **viremia** (virus in the blood), which persists for many years and which targets the immune system, particularly the $CD4^+$ T cells (helper T cells). The late and severe manifestation of HIV infection is called **acquired immunodeficiency syndrome (AIDS).** At this stage all organ systems are adversely affected and death is most often caused by infection. |
| scleroderma | scler/o-<br>-derm/o | hard<br>skin | *A chronic autoimmune disease characterized by a hardening of the skin or other organs.* The systemic form of the disease may be fatal because of heart, kidney, lung, and intestinal involvement. The limited form of the disease is called **CREST,** an acronym for the five main signs: *C*alcinosis, *R*aynaud's syndrome, *E*sophageal dysfunction, *S*clerodactyly (hardness of fingers or toes), *T*elangiectasia. CREST usually spares the lungs and kidneys. |
| systemic lupus erythematosus (SLE) | | From the Latin word meaning "wolf" (lupus) | **SLE** is a chronic autoimmune disorder. Symptoms are due to inflammation and are highly variable, but all persons experience joint pain (especially fingers, hands, wrist, and knees). A "butterfly" rash over the bridge of the nose and patchy skin color are characteristic of SLE. Persons who experience only the skin symptoms have **discoid erythematosus.** A **drug-induced lupus erythematosus (DIL)** can develop in response to certain medications; symptoms gradually disappear when the medication is stopped. |

## Get Ready for Exams!

### Summary Outline

The immune system is a defense system that protects the body from foreign agents such as pathogens, pollens, toxins, and cancer cells.

**I. Nonspecific Immunity**

A. Nonspecific immune mechanisms protect the body against many different types of foreign agents and do not require recognition of the specific agent.

B. Lines of defense

1. The first line of defense includes mechanical barriers, chemical barriers, and reflexes.
2. The second line of defense includes phagocytosis, inflammation, fever, protective proteins, and natural killer (NK) cells.

**II. Immunity**

A. Specific immunity protects the body against specific foreign agents and requires recognition of the specific agent involved.

B. T cells, or T lymphocytes

1. The T cells make up 70% to 80% of the blood's lymphocytes.
2. T cells engage in cell-mediated immunity.
3. Activated T cells produce a clone (killer T cells, helper T cells, suppressor T cells, and memory T cells).

C. B cells, or B lymphocytes

1. B cells make up 20% to 30% of the blood's lymphocytes.
2. B cells engage in antibody-mediated immunity.

3. Activated B cells produce a clone (memory cells and plasma cells). The plasma cells secrete antibodies that travel through the blood to the antigens.
4. The antibodies are called *immunoglobulins*.

**III. Types of Immunity**

A. Genetic or acquired immunity
1. With genetic immunity, a person is genetically immune to an antigen.
2. A person can acquire immunity naturally or artificially.
3. A person can acquire immunity naturally in two ways: by getting the disease or by receiving antibodies from the mother across the placenta and/or through breast milk.
4. Immunity can be acquired artificially in two ways: by the use of a vaccine or by injection of immune globulin made by another person or animal.

B. Active or passive immunity
1. Active immunity means that a person's body makes the antibodies (usually long-lasting).
2. Passive immunity means that the antibodies are made by another animal and then injected into a patient's body (usually short-lasting).

**IV. Other Immune Responses**

A. Allergic reactions
1. Allergic reactions are caused by the formation of antibodies to substances usually not recognized as foreign.
2. There are two types of allergic reactions: delayed-onset allergy and immediate-reaction allergy.
3. A delayed-onset allergy takes about 48 hours to develop. Contact dermatitis to a household chemical is a common example.
4. An immediate-reaction allergy (also called an *immediate hypersensitivity reaction*) is often caused by exposure to pollens and drugs such as penicillin. The most severe form is anaphylaxis.

B. Autoimmunity and tissue or organ rejection
1. Autoimmune disease develops when the immune system mounts an attack against self.
2. Tissue or organ rejection occurs when the immune system recognizes the transplanted organ as foreign and attacks it.

## Review Your Knowledge

### Matching: Nonspecific Immunity

Directions: Match the following words with their descriptions below. Some words may be used more than once.

**a.** protective proteins
**b.** phagocytosis
**c.** inflammation
**d.** fever

1. ___ Caused by pyrogens
2. ___ Eats debris and pathogens
3. ___ Redness, heat, swelling, and pain
4. ___ Neutrophils and monocytes
5. ___ Complement and interferons

### Matching: Specific Immunity

Directions: Match the following words with their descriptions below.

**a.** immunotolerance
**b.** cell-mediated immunity
**c.** antibody-mediated immunity
**d.** macrophage
**e.** plasma cells

1. ___ Subgroup of the B-cell clone that secretes antibodies
2. ___ Recognition of self
3. ___ The cell responsible for antigen presentation
4. ___ Also called *humoral immunity*
5. ___ T-cell immunity

### Matching: Active and Passive Immunity

Directions: Indicate whether the following conveys active immunity (a) or passive immunity (b).

1. ___ Vaccine
2. ___ Antivenom
3. ___ Antitoxin
4. ___ Toxoid
5. ___ Gamma globulin
6. ___ Getting the disease

### Multiple Choice

1. Complement and interferons are
   **a.** considered to be specific immunity.
   **b.** protective proteins engaged in nonspecific immunity.
   **c.** secreted by B cells and T cells.
   **d.** vaccines.
2. T and B cells
   **a.** engage in nonspecific immunity.
   **b.** refer to neutrophils.
   **c.** both engage in antibody-mediated immunity.
   **d.** are lymphocytes that are responsible for specific immunity.
3. Which of the following is not related to cell-mediated immunity?
   **a.** Cell-to-cell combat
   **b.** Killer T cells
   **c.** Immunoglobulins
   **d.** Antigen presentation by a macrophage
4. Which of the following best describes the killer T cell, helper T cell, memory T cell, and suppressor T cell?
   **a.** Immunoglobulins
   **b.** Macrophages
   **c.** Clone
   **d.** Agglutination
5. Pyrexia, pyrogens, and febrile seizures are most related to this nonspecific form of immunity?
   **a.** Inflammation
   **b.** Fever
   **c.** Anaphylaxis
   **d.** Diapedesis, chemotaxis, and phagocytosis
6. Plasma cells
   **a.** refer to T cells.
   **b.** are the same as NK cells.
   **c.** secrete antibodies.
   **d.** secrete interferons.

7. With which of the following is anaphylaxis most associated?
   a. Interferons
   b. Phagocytosis
   c. IgE
   d. Contact dermatitis
8. What is the primary concern regarding the care of a person experiencing an anaphylactic reaction?
   a. Inability to breathe
   b. Development of hives
   c. Development of febrile seizures
   d. Intense itching and discomfort
9. Which of the following is least characteristic of a vaccine?
   a. Artificially acquired immunity
   b. Active immunity
   c. Conveys long-lasting immunity
   d. Passive, immediate-onset, and short-lived immunity
10. Inflammation is
    a. specific immunity.
    b. characterized by redness, heat, swelling, and pain.
    c. known as *cell-mediated immunity*.
    d. synonymous with infection.

## Go Figure

1. **According to Figures 21-1 and 21-2**
   a. The natural killer cell is a lymphocyte and is therefore considered a type of specific immunity.
   b. Phagocytosis, inflammation, and fever occur only in response to invasion by a pathogen.
   c. Interferons are lymphocytes.
   d. T and B cells are lymphocytes and are considered a form of specific immunity.

2. **According to Figures 21-3 and 21-4**
   a. Antigens are presented by macrophages in both cell-mediated immunity (CMI) and antibody-mediated immunity (AMI).
   b. Antibodies are secreted by plasma cells in AMI.
   c. Helper T cells enhance B-cell response.
   d. All of the above are true.

3. **According to Figures 21-3 and 21-4**
   a. Only T cells have memory cells in their clone.
   b. A deficiency of helper T cells, as in HIV infection, depresses B-cell activity.
   c. Suppressor T cells engage in cell-to-cell combat.
   d. Helper T cells secrete lymphokines, which in turn suppress B-cell activity.

4. **According to Figures 21-5 and 21-6**
   a. Antibody concentration is higher with the first exposure to a pathogen than the second.
   b. "Getting the disease" is a form of artificially acquired active immunity.
   c. Naturally acquired passive immunity describes the immunity of a breast-fed infant.
   d. A vaccine conveys long-lasting passive immunity.

5. **According to Figure 21-7**
   a. IgE antibodies are released by mast cells.
   b. IgE antibodies bind to mast cells, causing the release of histamine.
   c. Histamine causes peripheral vasoconstriction and anemia.
   d. Histamine binds to mast cells, causing the release of IgE antibodies.

6. **According to Figures 21-4 and 21-7**
   a. T cell activation produces plasma cells.
   b. Plasma cells secrete lymphokines which, in turn, activate helper T cells.
   c. Plasma cells secrete antibodies.
   d. Histamine binds to mast cells.