Chapter 43

The Immune System

Key Concepts

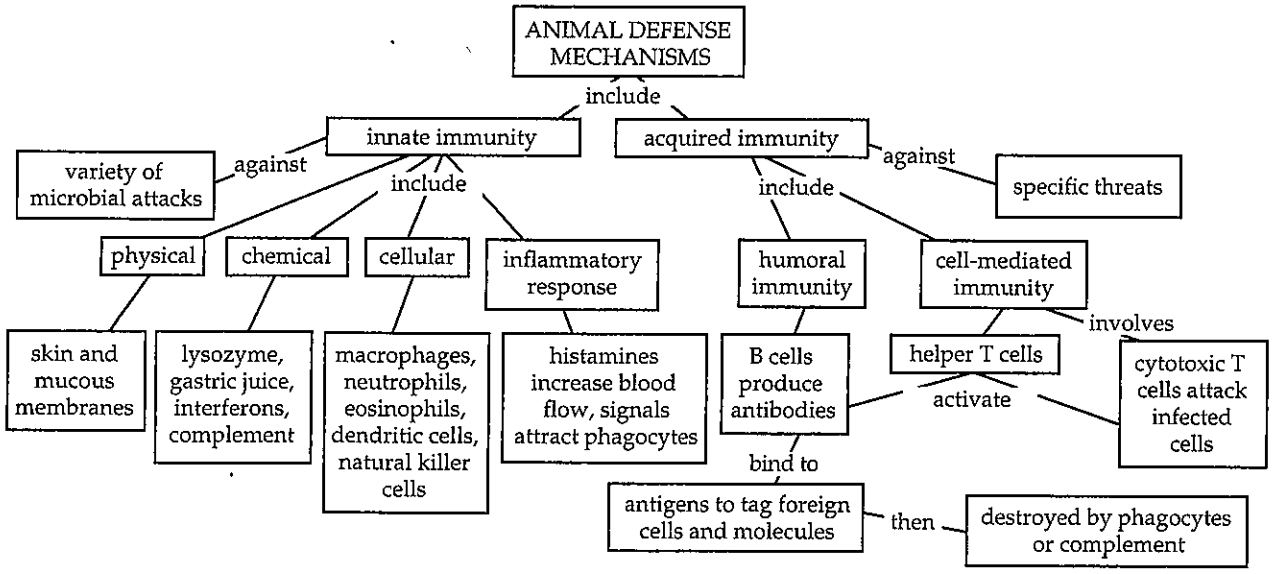
43.1 In innate immunity, recognition and response rely on shared traits of pathogens

43.2 In acquired immunity, lymphocyte receptors provide pathogen-specific recognition

43.3 Acquired immunity defends against infection of body cells and fluids

43.4 Disruptions in immune system function can elicit or exacerbate disease

Framework



Chapter Review

The **immune system** defends against **pathogens,** infectious agents such as bacteria, viruses, protists, and fungi that cause disease. **Innate immunity** includes external physical barriers and internal defenses of immune cells that have a small group of receptor proteins that recognize a broad range of pathogens. **Acquired immunity,** also called *adaptive immunity,* is a line of defense in vertebrates in which immune cells react specifically to pathogens. A vast array of acquired immune receptors allow recognition and response to specific pathogens.

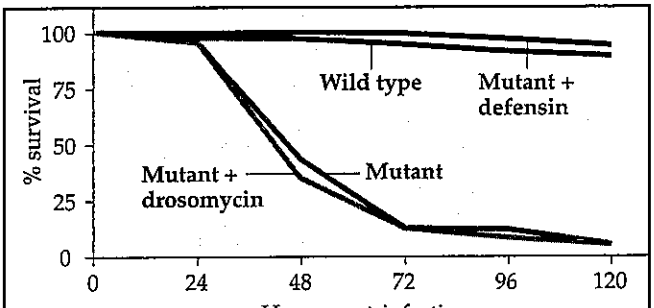
43.1 In innate immunity, recognition and response rely on shared traits of pathogens

Innate Immunity of Invertebrates Insect defenses begin with their protective exoskeleton. **Lysozyme,** an

enzyme that attacks microbial cell walls, a low pH, and the chitin lining of the intestine all protect the digestive system. *Hemocytes* are circulating cells that can engulf and destroy bacteria by **phagocytosis,** trigger production of chemicals that entrap multicellular parasites, and secrete *antimicrobial peptides* that kill fungi and bacteria. Different classes of pathogens bind to distinct Toll receptors that activate pathways for the production of antimicrobial peptides effective against that group of pathogens.

INTERACTIVE QUESTION 43.1

Explain why the data presented in this graph indicate that defensin and not drosomycin is the antimicrobial peptide in fruit flies effective against infection by the bacteria *M. luteus.*



Survival after infection by *M. luteus* bacteria

Innate Immunity of Vertebrates In mammals, skin and the mucous membranes lining the digestive, respiratory, and genitourinary tracts are physical barriers to microbes. The ciliated, *mucus*-coated epithelial lining of the respiratory tract traps and removes microbes. Skin secretions maintain a low pH, which discourages colonization by microbes. The acidity of gastric juice kills most microorganisms that reach the stomach. Lysozyme present in tears, saliva, and mucus kills bacteria.

Cellular innate defenses involve specific **Toll-like receptors (TLRs)** that recognize molecules that are common to a set of pathogens. Recognition by a TLR triggers phagocytosis of microbes by phagocytic white blood cells (leukocytes). Microbes are engulfed and the resulting vacuoles fuse with lysosomes, which contain toxic gases (such as nitric oxide) and digestive enzymes.

Neutrophils are the most numerous phagocytic white blood cells. **Macrophages** may migrate through the body or become permanently attached in various organs of the lymphatic system. These macrophages attack microbes filtered from blood in the spleen and from interstitial fluid that flows as lymph through lymph nodes.

Eosinophils are leukocytes that attack multicellular parasitic invaders with destructive enzymes. The primary role of **dendritic cells,** located in tissues in contact with the environment, is to stimulate acquired immunity.

Various antimicrobial peptides and proteins are released in response to pathogen recognition. Virus-infected cells produce **interferons,** which stimulate neighboring cells to produce substances that inhibit viral reproduction in those cells. Another type of interferon activates macrophages. Interferons produced by recombinant DNA technology are used in treating certain viral infections.

The **complement system** is a group of about 30 proteins in the blood plasma that, when activated by contact with microbes, may lyse cells, trigger inflammation, or assist in acquired defenses.

Chemicals released in response to physical injury or pathogen entry can trigger an **inflammatory response,** characterized by redness, swelling, and heat. Damaged **mast cells** in connective tissue release **histamine,** which triggers dilation and leakiness of blood vessels; activated macrophages and other cells release signals that promote blood flow to the damaged area. Vasodilation as well as signaling proteins result in the congregation of phagocytic cells and the production of antimicrobial compounds. *Pus* is an accumulation of fluid, white blood cells, and dead microbes.

A systemic response to an infection may include an increase in the number of circulating white blood cells and a fever. Fever may be triggered by toxins produced by pathogens or by *pyrogens* released by macrophages. Fevers may stimulate phagocytosis and speed tissue repair. *Septic shock* is a dangerous condition resulting from an overwhelming systemic inflammatory response.

Natural killer (NK) cells recognize an absence of class I MHC molecule on virus-infected or cancer cells, attaching to them and triggering cell death.

Innate Immune System Evasion by Pathogens Certain pathogens may evade the innate immune system with an outer capsule that covers their surface molecules or by resisting breakdown in lysosomes and growing within the host's cells.

INTERACTIVE QUESTION 43.2

Complete the following table that summarizes the functions of the cells and chemicals of the innate defense mechanisms.

Cells or Compounds	Functions
Neutrophils	a.
Macrophages	b.
Eosinophils	c.
Dendritic cells	d.
Natural killer (NK) cells	e.
Mast cells	f.
Histamine	g.
Interferons	h.
Complement system	i.
Lysozyme	j.

43.2 In acquired immunity, lymphocyte receptors provide pathogen-specific recognition

Lymphocytes are the key cells of acquired immunity. **T cells** migrate to the **thymus** to mature, while **B cells** remain and mature in the bone marrow. B cells and T cells are activated by contact with foreign molecules and by **cytokines,** proteins secreted by macrophages and dendritic cells after they engulf microbes. Both B and T cells are involved in *immunological memory,* an enhanced response to a previously encountered foreign molecule.

Acquired Immunity: An Overview Each B cell and T cell has many surface receptors that can bind to a particular foreign molecule. Such binding activates the cells, stimulating cell division and the production of some cells that will fight future infections by the same microbe. Some T cells help to activate other lymphocytes. Other T cells kill infected host cells. B cells secrete proteins that attach to foreign molecules and cells in body fluids.

Antigen Recognition by Lymphocytes Most **antigens** are proteins or polysaccharides, often protruding from the surfaces of microbes. B cells and T cells have membrane-bound **antigen receptors** that allow them to recognize a specific antigen. Each B or T lymphocyte carries about 100,000 identical receptors. B cells may give rise to *plasma cells* that secrete **antibodies,** or **immunoglobulins (Ig),** which are soluble antigen receptors. The small region of an antigen to which a lymphocyte or secreted antibody binds is called an **epitope,** or *antigenic determinant.*

Each Y-shaped **B cell receptor** consists of four polypeptide chains: two identical **light chains** and two identical **heavy chains,** linked together by disulfide bridges. Both heavy and light chains have *variable (V) regions* at the ends of the two arms of the Y, which form two identical antigen-binding sites. The *constant (C) regions* of the molecule vary little from cell to cell. Secreted antibodies lack an anchoring transmembrane region and cytoplasmic tail but are otherwise structurally similar to B cell receptors.

A **T cell receptor,** also anchored by a transmembrane region, consists of one α *chain* and one β *chain,* linked by a disulfide bridge. The variable regions at the tip of the molecule form a single antigen-binding site. T cells recognize small fragments of antigens complexed with MHC molecules. A group of genes called the **major histocompatibility complex (MHC)** codes for these cell-surface proteins.

In a process called **antigen presentation,** MHC molecules bind with antigen fragments within the cell and then display them on the cell's surface, where a T cell receptor can recognize the antigen and MHC molecule complex.

Class I MHC molecules are found on almost all nucleated cells. They bind foreign antigens that an infected cell has produced. **Cytotoxic T cells** recognize class I MHC molecules displaying peptide antigens and kill the infected cell.

Class II MHC molecules are made by dendritic cells, macrophages, and B cells. These cells, called **antigen-presenting cells,** engulf and fragment microbes, then display their antigens in class II MHC molecules.

Cytotoxic T cells and **helper T cells,** which assist B cells and cytotoxic T cells, recognize class II MHC molecule-peptide antigen complexes.

INTERACTIVE QUESTION 43.3

Describe the differences between the antigens that B cell receptors and antibodies recognize and the antigens that T cell receptors on cytotoxic T cells and helper T cells recognize.

Lymphocyte Development Each person may have as many as a million different B cells and 10 million different T cells. The genes coding for this diversity have numerous coding segments that are randomly and permanently rearranged.

For example, *immunoglobulin* (*Ig*) genes code for secreted antibodies. In the Ig light-chain gene, 40 variable (*V*) gene segments and five joining (*J*) gene segments are separated by a long stretch of DNA. The *J* segments are followed by an intron and a C exon that codes for the constant region. Early in B cell development, one *V* gene segment is randomly linked to one *J* segment by a set of enzymes called recombinase, producing one of 200 possible light chains. After transcription, the pre-mRNA is processed, and the mRNA is translated into a light chain with a variable and a constant region. These light chains combine with heavy chains that were produced the same way.

In the bone marrow and thymus, respectively, the antigen receptors of maturing B and T cells are tested for self-reactivity. Lymphocytes with receptors specific for the body's own molecules are either inactivated or destroyed by apoptosis. This critical *self-tolerance* means that normally there are no mature lymphocytes that react against self components.

When an antigen interacts with receptors on B cells or T cells that are specific for epitopes of that antigen, those particular lymphocytes are activated to divide repeatedly and differentiate into two clones—a large number of short-lived **effector cells,** which combat the antigen, and a clone of long-lived **memory cells,** all of which carry receptors for that antigen. By this **clonal selection,** a small number of cells is selected by their interaction with a specific antigen to produce thousands of cells keyed to that particular antigen.

The body mounts a **primary immune response** upon first exposure to an antigen. About 10 to 17 days are required for selected lymphocytes to proliferate and differentiate to yield the maximum response produced by effector T cells and the antibody-producing effector B cells, called **plasma cells.** Should the body reencounter the same antigen, the **secondary immune response** is more rapid, effective, and prolonged. The long-lived T and B memory cells are responsible for this immunological memory. This secondary immune response provides long-term protection against a previously encountered pathogen.

INTERACTIVE QUESTION 43.4

Answer the following questions concerning the three major stages in the development of lymphocytes.

a. How is the great diversity of B and T cells produced?

b. What prevents B and T cells from reacting against the body's own molecules?

c. Describe clonal selection.

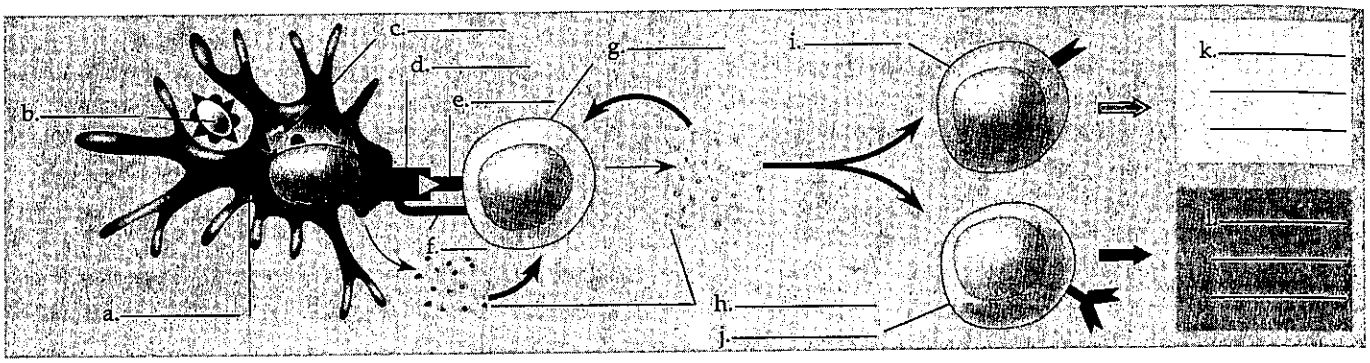
43.3 Acquired immunity defends against infection of body cells and fluids

The **humoral immune response,** also called the antibody-mediated response, involves B cell activation and production of antibodies that circulate in the blood and lymph. The **cell-mediated immune response** involves cytotoxic T cells that destroy target cells. Helper T cells activate both B cells and cytotoxic T cells.

Helper T Cells: A Response to Nearly All Antigens Helper T cells recognize specific class II MHC molecule-antigen complexes on antigen-presenting cells. A T cell surface protein called **CD4** enhances the binding by attaching to the class II MHC molecule. Signaling between the two cells results in the proliferation and differentiation of a clone of activated helper T cells and memory cells. Activated helper T cells secrete cytokines, which stimulate both the cell-mediated and humoral responses.

INTERACTIVE QUESTION 43.5

Label the components in this diagram that show a helper T cell being activated by interaction with a dendritic cell and the central role of the helper T cell in activating both humoral and cell-mediated immunity.



Cytotoxic T Cells: A Response to Infected Cells Cytotoxic T cells recognize nonself proteins synthesized in infected cells and displayed with class I MHC molecules. CD8 surface proteins on cytotoxic T cells bind to the side of class I MHC molecules and enhance the interaction between the cells, while the cytotoxic T cells, also stimulated by cytokines from nearby helper T cells, differentiate into active killers. The activated cell secretes proteins that kill the target cell. Pathogens released from the destroyed cell are marked by circulating antibodies for destruction.

INTERACTIVE QUESTION 43.6

a. What surface molecule of a helper T cell facilitates the interaction with a class II MHC molecule of an antigen-presenting cell and the helper T cell?

b. What surface molecule on a cytotoxic T cell assists in the interaction with a class I MHC molecule displayed on infected cells?

c. What does an activated helper T cell release?

d. What does a cytotoxic T cell attached to an infected body cell release?

B Cells: A Response to Extracellular Pathogens B cells are selectively activated by antigens on the surface of bacteria. The activation is aided by cytokines released from helper T cells also activated by that antigen. Upon first binding antigen, the B cell takes in a few foreign molecules by receptor-mediated endocytosis and presents antigen fragments in its class II MHC molecules to helper T cells. Most protein antigens require the aid of helper T cells to stimulate antibody production. The activated B cell then proliferates into a clone of plasma cells and a clone of memory B cells. When certain antigens bind with multiple receptors on a single cell, a B cell response may not involve cytokines or helper T cells.

The antigen-binding sites on the arms of an antibody allow it to identify a specific antigen. The heavy-chain constant regions in the tail determine the antibody's distribution in the body and its function. There are five major types of constant regions, creating five classes of antibodies: IgM, IgG, IgA, IgD, and IgE. IgG is the most abundant antibody in blood and confers passive immunity on a fetus; IgA is present in tears, saliva, mucus, and breast milk.

Researchers can use the specificity of antigen-antibody binding for biological research, clinical testing, and medical applications. Some antibody tools are *polyclonal* because they were formed by several different B cell clones, each specific for a different epitope. A technique for making **monoclonal antibodies** can supply quantities of identical antibodies.

Antibodies label antigens for disposal by one of several mechanisms. In *neutralization,* antibodies may block the ability of a virus or bacterium to infect a host cell by binding to its surface. In *opsonization,* antibodies coat microbes and enhance phagocytosis by macrophages. Because each antibody molecule has at least two antigen-binding sites, the formation of antigen-antibody

complexes produces clumps, which are then engulfed by phagocytes.

Antigen-antibody complexes on microbes may activate the complement system by binding with complement proteins and triggering the generation of a *membrane attack complex (MAC)*, which produces a pore in the membrane and causes the cell to lyse. Complement proteins can be activated as part of the innate or acquired defenses. In addition to lysing microbes, activated complement proteins promote inflammation and phagocytosis.

INTERACTIVE QUESTION 43.7

List three ways in which antibodies mediate the disposal of antigens.

a.

b.

c.

Active and Passive Immunization **Active immunity** can be acquired when the body produces antibodies and develops immunological memory from either exposure to an infectious agent or from **immunization,** also called **vaccination.** A vaccine may be an inactivated toxin, a killed or weakened microbe, a portion of a microbe, or even genes for microbial proteins. In **passive immunity,** temporary immunity is provided by antibodies supplied through the placenta to a fetus, through milk to a nursing infant, or by an antibody injection.

Immune Rejection The immune response to the chemical markers that determine ABO blood groups must be considered in blood transfusions. Antibodies to other blood group antigens (other than the individual's own antigens) arise in response to normal bacterial flora and circulate in the blood plasma, where they will induce a devastating transfusion reaction to transfused blood cells with matching antigens.

INTERACTIVE QUESTION 43.8

Fill in the following table to review your understanding of the antigens and antibodies of the ABO blood groups. Remember to compare the antigens on the donor cells with the antibodies in the recipient's plasma. (Assume these are packed cell transfusions in which the antibodies in the donor's plasma are not a risk factor.)

Blood Type	Antigens on RBCs	Antibodies in Plasma	Can Receive Blood from	Can Donate Blood to

Transplanted tissues and organs are rejected because the foreign MHC molecules are antigenic and trigger immune responses. The use of closely related donors, as well as drugs that suppress immune responses, helps to minimize rejection.

In bone marrow transplants, used to treat leukemia and blood cell diseases, the graft itself may be the source of immune rejection. The recipient's bone marrow cells are destroyed by irradiation, eliminating the recipient's immune system. The lymphocytes in the bone marrow transplant, however, may produce a *graft versus host reaction* if the MHC molecules of the donor and recipient are not well matched.

43.4 Disruptions in immune system function can elicit or exacerbate disease

Exaggerated, Self-Directed, and Diminished Immune Responses Allergies are hypersensitivities to certain environmental antigens, or **allergens.** IgE antibodies created in response to initial exposure to an allergen may bind to mast cells in connective tissue. When allergens then bind to these cell surface antibodies, the mast cells, in a process called *degranulation,* release histamines. The resulting inflammatory response may include sneezing, a runny nose, and difficulty in breathing due to smooth muscle contractions. Antihistamines are drugs that combat these symptoms by blocking receptors for histamine. *Anaphylactic shock* is a severe allergic response in which the abrupt dilation of peripheral blood vessels caused by widespread mast cell degranulation leads to a life-threatening drop in blood pressure.

Sometimes the immune system turns against self, leading to **autoimmune diseases,** such as *lupus, rheumatoid arthritis, insulin-dependent diabetes mellitus,*

and *multiple sclerosis*. These diseases may be caused by a failure in the regulation of self-reactive lymphocytes.

Exercising to exhaustion and psychological stress have been shown to impair immune system function.

An **immunodeficiency** is the inability of the immune system to protect against pathogens. A genetic or developmental defect in the immune system is called an *inborn immunodeficiency;* a defect that arises later in response to chemical or biological agents is called an *acquired immunodeficiency*. An inborn immunodeficiency may occur in any of the components of the immune system. In the rare congenital disease known as severe combined immunodeficiency (SCID), lymphocytes and the acquired immune response are lacking. Acquired immunodeficiency may be caused by drugs used against autoimmune diseases or to suppress transplant rejection. Certain cancers, such as Hodgkin's disease, and **acquired immunodeficiency syndrome,** or **AIDS,** damage the immune system.

Acquired Immune System Evasion by Pathogens Some viruses and parasites evade the body's defenses through *antigenic variation,* changing their surface epitopes so that the immune system cannot develop an immunological memory of the pathogen. Examples include the trypanosome that causes sleeping sickness and the influenza virus.

Some viruses enter an inactive state called *latency,* in which viral DNA remains in a host cell's nucleus. Should conditions change, the virus can replicate and spread to neighboring cells or to new hosts.

The infectious agent responsible for AIDS is HIV (human immunodeficiency virus). HIV infects cells with surface CD4 molecules, including helper T cells, macrophages, and brain cells. HIV RNA is reverse-transcribed into DNA, which is integrated into the host cell genome, from where it directs production of new viral particles. HIV escapes the immune system through both antigenic variation, when it mutates rapidly during replication, and latency, when it integrates into host cell DNA. HIV infection kills helper T cells through the effects of virus reproduction or by undergoing apoptosis, triggered by the virus. Individuals with AIDS are highly susceptible to opportunistic infections and cancers that take advantage of a suppressed immune system.

While not able to cure HIV, new drug combinations are slowing the progression to AIDS. The frequent mutational changes during replication generate drug-resistant strains of HIV. And frequent mutational changes in surface antigens have made development of an effective vaccine difficult. HIV is transmitted by transfer of body fluids containing infected cells, such as blood or semen.

INTERACTIVE QUESTION 43.9

a. Why is AIDS such a deadly disease?

b. Why has it proved so difficult to prevent and cure this disease?

Cancer and Immunity The incidence of certain cancers increases when the immune system is impaired. A common hypothesis is that a robust immune system destroys cancer cells. An alternative hypothesis is that the infections and inflammatory responses resulting from an impaired immune system contribute to the development of many cancers.

Word Roots

an- = without; **-aphy** = suck (*anaphylactic shock:* an acute, life-threatening, allergic response)

anti- = against; **-gen** = produce (*antigen:* a foreign macromolecule that does not belong to the host organism and that elicits an immune response)

cyto- = cell (*cytokines:* in the vertebrate immune system, protein factors secreted by macrophages and helper T cells as regulators of neighboring cells)

epi- = over; **-topo** = place (*epitope:* a localized region on the surface of an antigen that is chemically recognized by antibodies)

immuno- = safe, free; **-glob** = globe, sphere (*immunoglobulin:* one of the class of proteins comprising the antibodies)

macro- = large; **-phage** = eat (*macrophage:* an amoeboid cell that moves through tissue fibers, engulfing bacteria and dead cells by phagocytosis)

neutro- = neutral; **-phil** = loving (*neutrophil:* the most abundant type of leukocyte; neutrophils tend to self-destruct as they destroy foreign invaders, limiting their life span to but a few days)

perfora- = bore through (*perforin:* a protein that forms pores in a target cell's membrane)

Structure Your Knowledge

This chapter contains a wealth of information that is probably fairly new to you. If you take a little time and pull out the key players of the immune system and organize them first into very basic concept clusters and then develop more interrelated concept maps, you will find that this information is both understandable and fascinating.

1. Create a concept map outlining acquired immunity, showing the cells involved in humoral and cell-mediated responses and their functions.
2. Describe the structure of an antibody molecule and relate this structure to its function.
3. While we presented innate and acquired immunity separately, these defense mechanisms interact in several ways. Describe a few of the chemical and cellular players they share.

Test Your Knowledge

MULTIPLE CHOICE: *Choose the one best answer.*

1. Which of the following is *incorrectly* paired with its effect?
   a. gastric juice—kills bacteria in the stomach
   b. fever—may stimulate phagocytosis
   c. histamine—causes blood vessels to dilate
   d. vaccination—creates passive immunity
   e. lysozyme—attacks cell walls of bacteria
2. Which of the following would release interferon?
   a. a macrophage that has become an antigen-presenting cell
   b. an injured epithelial cell of a blood vessel
   c. a cell infected by a virus
   d. a mast cell that has bound an antigen
   e. a helper T cell bound to an antigen-presenting cell
3. Antibodies are
   a. proteins or polysaccharides usually found on the cell surface of invading bacteria or viruses.
   b. proteins embedded in T cell membranes.
   c. proteins circulating in the blood that tag foreign cells for complement destruction.
   d. proteins that consist of two light and two heavy polypeptide chains.
   e. c and d are both correct.
4. A secondary immune response is more rapid and greater in effect than a primary immune response because
   a. histamines cause rapid vasodilation.
   b. the second response is an active immunity, whereas the primary one was a passive immunity.
   c. helper T cells are available to activate other blood cells.
   d. chemical signals cause the rapid accumulation of phagocytic cells.
   e. memory cells respond to the pathogen and rapidly clone more effector cells.
5. Which of the following is *not* true of Toll-like receptors?
   a. They are found on phagocytic white blood cells.
   b. They recognize molecules specific to individual pathogens.
   c. They resemble the Toll receptor on immune response cells of insects, which initiate signal transduction pathways that result in synthesis of antimicrobial peptides.
   d. TLR proteins may be located on the plasma membrane or inside vesicles of leukocytes.
   e. They trigger innate immune defenses upon binding to molecules common to a set of pathogens.
6. Major histocompatibility complex molecules
   a. are involved in the ability to distinguish self from nonself.
   b. are a collection of cell surface proteins.
   c. may trigger T cell responses after transplant operations.
   d. present antigen fragments on infected cells.
   e. All of the above are correct.
7. In opsonization,
   a. antibodies coat microorganisms and help phagocytes bind to and engulf the foreign cell.
   b. a set of complement proteins lyses a hole in a foreign cell's membrane.
   c. antibodies coat binding sites of viruses.
   d. a flood of histamines is released that may result in anaphylactic shock.
   e. *V* gene segments and *J* gene segments are joined by recombinase.

8. Severe combined immunodeficiency
   a. is an inborn autoimmune disease.
   b. is a form of cancer in which MHC proteins are absent on transformed cells.
   c. is a disease in which both T and B cells are absent or inactive.
   d. is an immune disorder in which the number of helper T cells is greatly reduced.
   e. results from a few types of cancers, such as Hodgkin's disease.

9. A transfusion of type B blood given to a person who has type A blood would result in
   a. the recipient's anti-B antibodies reacting with the donated red blood cells.
   b. the recipient's B antigens reacting with the donated anti-B antibodies.
   c. the recipient forming both anti-A and anti-B antibodies.
   d. no reaction, because B is a universal donor type of blood.
   e. the introduced blood cells being destroyed by innate defense mechanisms.

10. Which of the following are *incorrectly* paired?
    a. variable region—determines antibody specificity for an epitope
    b. immunoglobulins—proteins that form epitopes
    c. constant region—determines class and function of antibody
    d. IgG—most abundant circulating antibodies, confer passive immunity to fetus
    e. IgE—antibody molecules attached to mast cells

11. Which of the following destroys a target cell by phagocytosis?
    a. neutrophil
    b. cytotoxic T cell
    c. natural killer cell
    d. complement proteins
    e. plasma cell

12. Which of the following best describes an insect's immune system?
    a. They rely on the barrier defense of an exoskeleton.
    b. Lysozyme and a chitin-lined intestine with a low pH protect their digestive system.
    c. Hemocytes can carry out phagocytosis of bacteria and foreign substances.
    d. They produce different antimicrobial peptides in response to binding of molecules from a particular type of pathogen to recognition proteins, which activate the protein Toll and a signal transduction pathway.
    e. All of the above are part of an insect's innate immunity.

13. What do IgE antibodies, T cell receptors, and MHC molecules have in common?
    a. They are found exclusively in cells of the immune system.
    b. They are all part of the complement system.
    c. They are antigen-presenting molecules.
    d. They are or can be membrane-bound proteins.
    e. They are involved in the cell-mediated portion of the immune system.

14. Which of the following is an effective defense against bacteria but does *not* work against viral particles?
    a. secretion of interferon by an infected cell
    b. neutralization by antibodies
    c. the enzyme lysozyme
    d. a secondary immune response
    e. humoral immunity

15. How are antibodies and complement related?
    a. They are both coded for by genes that have hundreds of alleles.
    b. They are both involved in innate defenses.
    c. They are both produced by plasma cells.
    d. Antibodies bound to antigens on a pathogen's membrane may activate complement proteins to form a membrane attack complex.
    e. Complement proteins tag foreign cells for destruction; antibodies destroy cells by opsonization.

16. Which of the following describes the main difference between an innate immune response and an acquired immune response?
    a. An innate immune response is only to free pathogens in a localized area; an acquired immune response is only to pathogens that have entered body cells.
    b. An innate immune response involves only leukocytes, whereas an acquired immune response involves only lymphocytes.
    c. An innate immune response relies on phagocytes to destroy pathogens, whereas an acquired immune response does not involve phagocytes.
    d. An innate immune response involves recognition of molecules common to a set of pathogens, whereas an acquired immune response reacts to specific microbes on the basis of their different antigens.
    e. Complement proteins participate in an acquired immune response but not in an innate immune response.

17. Clonal selection is responsible for the
    a. proliferation of clones of effector and memory cells specific for an encountered antigen.
    b. recognition of class I MHC molecules by cytotoxic T cells.
    c. rearrangement of antibody genes for the light and heavy chains.
    d. formation of cell cultures in the commercial production of monoclonal antibodies.
    e. transformation of a clone of helper T cells into cytotoxic T cells keyed to a specific antigen.

18. What role does a dendritic cell play in an acquired immune response?
    a. activates complement proteins to form a membrane attack complex
    b. binds to the CD8 receptors on cytotoxic T cells to activate their production of perforin
    c. releases cytokines to activate B cells to produce clones of plasma cells
    d. activates both humoral and cell-mediated immunity by releasing interferons after it has ingested a virus
    e. presents peptide antigens of an engulfed pathogen in its class II MHC molecules to helper T cells, and releases cytokines

19. All of the following are involved with innate immunity *except*
    a. the inflammatory response.
    b. the complement system.
    c. antimicrobial proteins such as lysozyme.
    d. chemicals that attract phagocytes.
    e. plasma cells.

20. Place the following steps in the helper T cell activation of cell-mediated and humoral immunity in the correct order:
    1. Helper T cell secretes cytokines.
    2. Macrophage engulfs pathogen and presents antigen in class II MHC.
    3. Plasma cells secrete antibodies and cytotoxic T cells attack cells with class I MHC molecule-antigen complex.
    4. T cell receptor recognizes class II MHC molecule-antigen complex.
    5. Macrophage secretes cytokines.
    6. Activated B cells form plasma and memory cells, activated T cells form cytotoxic T cells and memory cells.

    a. 1, 3, 5, 6, 2, 4
    b. 5, 1, 2, 6, 4, 3
    c. 2, 4, 5, 1, 6, 3
    d. 5, 2, 4, 1, 3, 6
    e. 2, 1, 4, 5, 6, 3

21. Helper T cells play which of the following roles in an acquired immune response?
    a. bind to class I MHC molecules and activate complement proteins to attack and lyse cancer cells.
    b. bind to the antigens presented in the CD8 receptors on cytotoxic T cells and release perforin.
    c. produce interferons and histamines that help initiate a specialized inflammatory response.
    d. present antigens of an engulfed pathogen in its class II MHC molecules to B cells, which are then stimulated to develop into a clone of plasma cells.
    e. activate both the humoral and cell-mediated immunities by releasing cytokines after recognizing class II MHC molecule-antigen complexes on an antigen-presenting cell.

22. Which of the following statements about humoral immunity is *correct*?
    a. It is a form of passive immunity produced by vaccination.
    b. It defends against free pathogens with effector mechanisms such as neutralization, opsonization, or complement activation.
    c. It protects the body against pathogens that have invaded body cells as well as against abnormal body cells.
    d. It is mounted by lymphocytes that have matured in the thymus.
    e. It depends on the recognition of class I MHC molecules that are bound to a specific antigen to activate its effector mechanism.

23. What accounts for the huge diversity of antigens to which B cells can respond?
    a. The antibody genes have millions of alleles.
    b. The rearrangement of the antibody genes during development results in millions of possible combinations of randomly combined light and heavy polypeptide chains.
    c. The antigen-binding sites at the arms of the molecule can assume a huge diversity of shapes in response to the specific antigen encountered.
    d. B cells have thousands of copies of antibodies bound to their plasma membranes.
    e. B cells can be antigen-presenting cells when they take in antigens by endocytosis and display fragments in their class II MHC molecules.

24. What is the function of CD4?
    a. a surface molecule on a cytotoxic T cell that enhances its binding to a class I MHC molecule displaying a foreign antigen
    b. a membrane protein on an antigen-presenting cell that helps a helper T cell recognize the MHC molecule-antigen complex
    c. a receptor that normally functions for cytokines, but which HIV uses as its receptor
    d. a surface molecule on a helper T cell that enhances its binding to a class II MHC molecule displaying a foreign antigen
    e. a portion of the class I MHC molecule found on all nucleated cells that identify cells as "self"

25. In which circumstance would B cells display antigens to T cells?
    a. They take in a few antigen molecules by endocytosis and display them in class II MHC molecules to helper T cells.
    b. They phagocytose bacteria and display bacterial peptide antigens in class II MHC molecules to helper T cells.
    c. After being infected by a virus, they display viral peptides they have synthesized in class I MHC molecules to cytotoxic T cells.
    d. They bind free antigens and display them to helper T cells attached to their B cell receptors.
    e. Both a and c are possible ways that B cells can display antigens.

26. All of the following are considered diseases or malfunctions of the immune system *except*
    a. MHC-induced transplant rejection.
    b. SCID (severe combined immunodeficiency).
    c. lupus, multiple sclerosis, and insulin-dependent diabetes.
    d. AIDS.
    e. allergic anaphylactic shock.

27. Which of the following may induce a graft-versus-host reaction?
    a. organ transplant
    b. blood transfusion
    c. bone marrow transplant
    d. skin transplant
    e. gene therapy

28. From which of the following would an AIDS patient be *least* likely to suffer?
    a. Kaposi's sarcoma or other cancers
    b. tuberculosis
    c. pneumonia
    d. rheumatoid arthritis
    e. yeast infections of mucous membranes