Chapter 21

IMMUNITY

STUDENTS'LEARNING OUTCOMES

After studying this chapter, the students will be able to:

- List the structural features of human skin that make it an impenetrable barrier against invasion by microbes (1st line of defense).
- Explain how oil and sweat glands within the epidermis inhibit the growth and also kill microorganisms. (1st line of defense)
- Recognize the role of the acids of the digestive tract as killing bacteria present in food.
- State the role of the ciliated epithelium of the nasal cavity and the mucous of the bronchi and bronchioles in trapping airborne microorganisms.
- Describe the role of macrophages and neutrophils in killing bacteria.
- Explain how Natural Killer (NK) cells kill cells infected by microbes and cancer cells.
- State the way proteins of the complement system kill bacteria and that interferons inhibit viruses from infecting cells.
- State the events of the inflammatory response as a generalized, nonspecific defense.
- Outline the release of pyrogens by microbes and their effect on the hypothalamus to boost the body's temperature.
- List the ways that fever affects microbes.
- Define the specific immune system as providing specific defense and acting as the most powerful means of resisting infection.
- Identify monocytes, T- cells, and B-cells as components of the immune system.
- State inborn and acquired immunity as the two basic types of immunity.
- Differentiate between active and passive immunity as the two types of acquired immunity.
- Describe the role of T-cells in cell-mediated immunity.
- Describe the role of B-cells in antibody-mediated immunity.
- Discuss the role of T-cells and B-cells in transplant rejections.
- Evaluate the discovery of monoclonal antibodies and justify how this accomplishment revolutionized many aspects of biological research.
- Identify the process of vaccination as a means to develop active acquired immunity.
- Draw the structural model of an antibody molecule.
- Explain the role of memory cells in long- term immunity.
- Define allergies and correlate the symptoms of allergies with the release of histamines.
- Describe the autoimmune diseases with examples.

Most human diseases result from microbial infections – invasions of the body by viruses, pathogenic bacteria, fungi, or protists. To defend against infections and against cancers, vertebrates possess a defence system called the **immune system**. It continually checks the bloodstream for the presence of any foreign cells and molecules.

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For example, when the immune system detects an infection, it attacks and destroys the invading microbes. That is why, the diseases that weaken the immune system, such as AIDS, are very dangerous.

Tidbit

Our body is defended from infection the same way that a city is defended against foreign invasion. There are walls to make invaders entry difficult, roaming policemen check strangers, security guards challenge anyone wandering and call policeman if a proper ID is not presented. Our body also has different lines of defence.

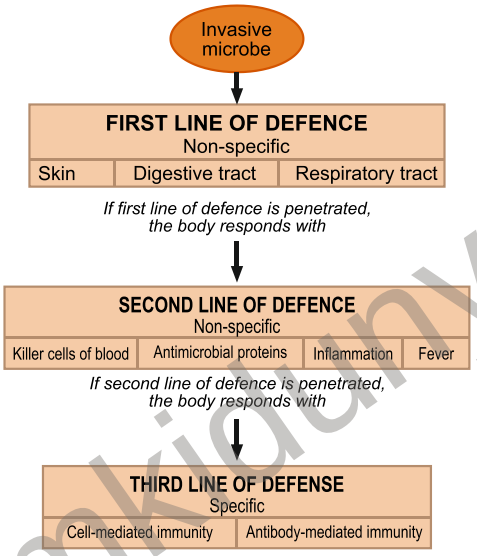

Figure 21.1: Lines of defence against infection

21.1- First Line Of Defence

The first line of defence against infections consists of skin, the digestive tract, and the respiratory tract.

21.1.1- Defence Provided by Skin

Skin is the outermost layer of our body. It provides the first defence against invasion of microbes. The epidermis of skin is 10 to 30 cells thick. The outer layer of epidermis is called **stratum corneum**. Its cells are continuously shed, because they are continuously damaged. New cells are formed in the deep epidermis from where they migrate upward and replace the worn-out cells. These cells form keratin protein which makes the skin tough and impenetrable for microbes.

For Information

Skin is the largest organ of the vertebrate body. In an adult human, 15% of the total weight is skin. One square centimetre of human skin contains 200 nerve endings, 10 hairs and muscles, 100 sweat glands, 15 oil glands, 3 blood vessels, 12 heat receptors, 2 cold receptors, and 25 pressure receptors.

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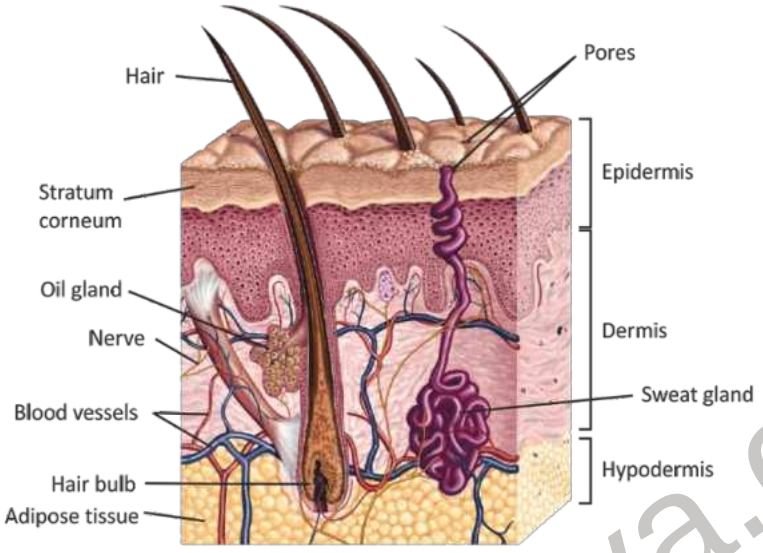

Figure 21.2: Layers of skin

Skin further reinforces the defence by secreting chemicals. The oil and sweat glands present in skin lower the pH at the skin's surface to 3-5. This is enough acid level that inhibits the growth of many microbes. Sweat also contains the enzyme lysozyme, which attacks and digests the cell walls of many bacteria.

21.1.2- Defence Provided by Digestive and Respiratory Tracts

Many bacteria are present in the food that humans eat. Most of these are killed by **saliva in the oral cavity**, which also contains lysozyme. The strong digestive acids present in the stomach and the protein-digesting enzymes in the intestine also kill bacteria.

Many microbes are present in the air that we breathe. Ciliated epithelial cells in the nasal cavity trap many bacteria before they can enter the air passageway. The cells lining the smaller bronchi and bronchioles secrete a layer of sticky **mucous** that traps microbes before they can reach the warm, moist lungs. Cilia on the cells lining these passages continually sweep the mucous upward, where it can be swallowed.

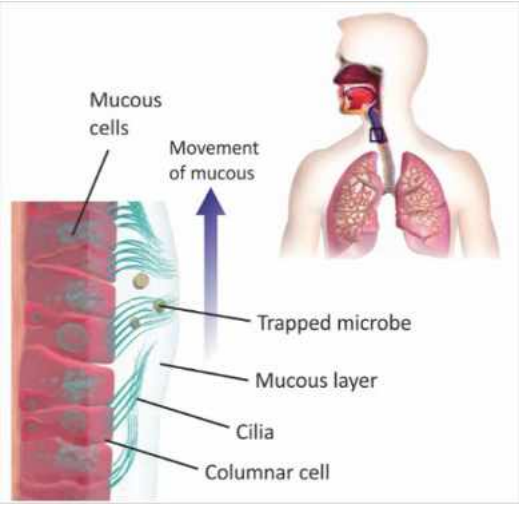

Figure 21.3: Epithelium of trachea, with trapped microbes

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21.2- Second Line of Defence

The first line of defence of the body is very effective. However, it may occasionally be overcome by invaders. When such invaders reach deep tissues, a second line of defence comes into play. Along with the first line of defence, the second line is also categorized as a non-specific defence.

Human body uses a lot of non-specific cellular and chemical devices in its second line of defence. The four most important of these are; the germ-killer cells of blood, antimicrobial proteins, the inflammatory response, and the temperature response.

21.2.1- Germ-Killer Cells of Blood

The most important non-specific defence is provided by the germ-killer cell of the blood. They attack and kill the microbes. These cells patrol the bloodstream and wait for invaders. There are three basic kinds: macrophages, neutrophils, and natural killer cells.

1. Macrophages

Macrophages develop from monocytes (a type of white blood cells). A macrophage ingests a bacterium, by endocytosis, and kills it by its lysozyme enzymes. Some macrophages are fixed within some organs e.g., lungs, liver, spleen, and brain. But most of them patrol in the blood, lymph and interstitial fluid.

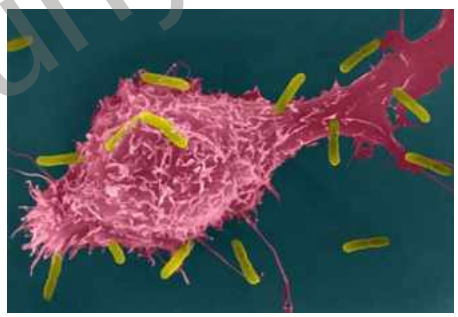

Figure 21.4: A macrophage attacking microbes

2. Neutrophils

Neutrophils are a type of white blood cells. They use three methods for killing pathogens. The first method is phagocytosis. In the second method, they release soluble anti-microbial chemicals on pathogens. In the third method, they generate and release network of DNA fibres. These fibres bind with pathogenic bacteria and kill them.

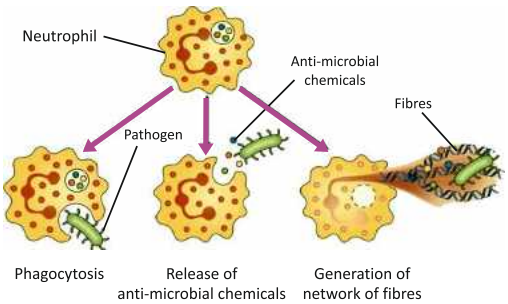

Figure 21.5: Methods of neutrophils to kill pathogens

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3. Natural Killer Cells

These are a type of T-lymphocytes and are also called **cytotoxic T-cells**. They do not kill invading microbes, rather they detect and attack the body cells that have been infected with viruses. They release special proteins, called **perforins**, which insert into the membrane of the target cell. It results in the formation of a pore in the membrane of target cell. This pore allows water to rush into the target cell, which then swells and bursts. They are also able to detect cancer cells, which they kill before the cancer cells have a chance to develop into a tumour.

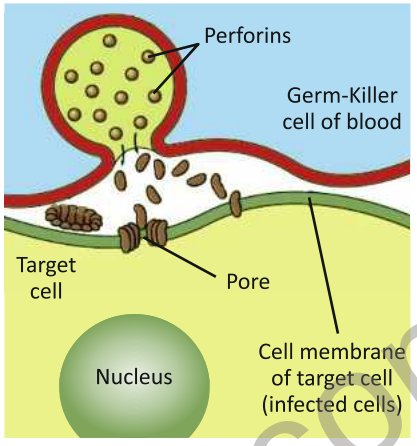

Figure 21.6: Natural killer cell attacking an infected cell

For Information

The patrolling cells (macrophages, neutrophils, and natural killer cells) do not attack their own body because all the normal cells of body contain a surface protein marker that identifies them. This protein is called self-protein. The germ-killer cells of blood do not attack the cells which have self-proteins. They only attack the cells that lacks such proteins.

21.2.2- Protective Proteins

Two main groups of proteins participate in the non-specific defence of the body.

1. Complement system proteins

Special proteins produced in the liver make the **complement system** of proteins. This system consists of more than a dozen different proteins that circulate in the blood in an inactive state. When they encounter a microbe, they become active in a sequence. The first protein activates the second and so on. The final five proteins form a **membrane-attack complex** (MAC) into the plasma membrane of microbe. This complex punctures the plasma membrane of microbe. So, fluids and salts move inside microbe resulting in its swelling and bursting.

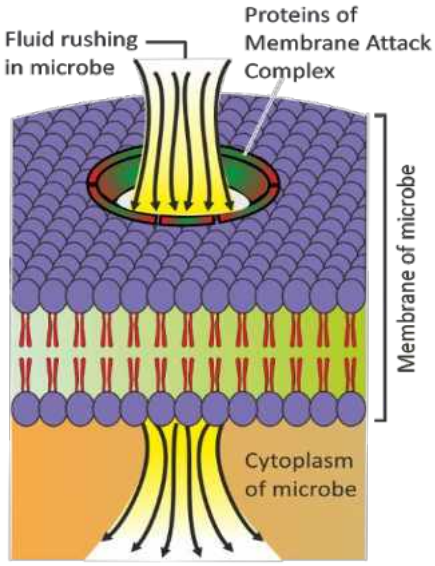

Figure 21.7: The Complement system of proteins and their functioning

2. Interferons

Interferons are another class of protective proteins that play a key role in body's defence. They are released by virus-infected cells. They cause nearby cells to enhance

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their anti-viral defences. Interferons belong to the large class of proteins known as **cytokines**. These proteins work for communication between cells to trigger the protective defences of the immune system. Interferons also activate other immune cells, such as natural killer cells and macrophages.

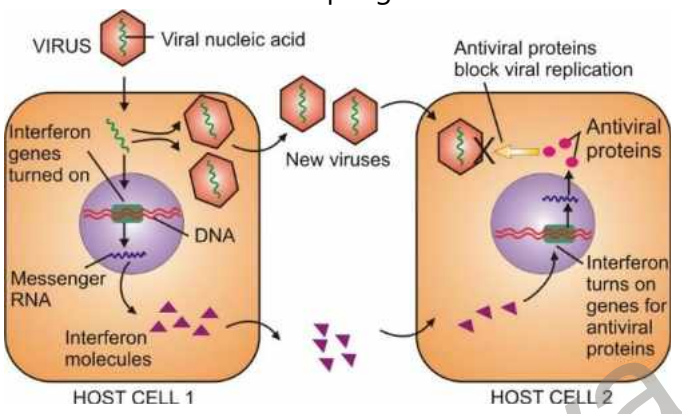

Figure 21.8: The interferon mechanism to alert other cells

21.2.3- Inflammatory Response

It is one of the most generalized non-specific responses to infections. Infected or injured cells release chemical signals such as **histamines** and **prostaglandins**. These chemicals cause local expansion of blood vessels. In this way, the capillary blood flow is increased at the site of injury or infection. The capillaries also become more permeable. It results in redness, swelling and warmth at the spot. This condition is called **inflammation**. The heat makes the site unfavourable for microbial growth. It also promotes healing and raises mobility of white blood cells. More phagocytes (macrophages and neutrophils) migrate from the blood to the inflamed tissue, where they engulf microbes. Monocytes arrive and release chemicals to kill the microbes and then macrophages clean up dead microbes, cells, and debris. The inflammatory response is often strong enough to stop the spread of microbes.

For Information

The pus associated with some infections is a mixture of dead or dying neutrophils, broken down tissue cells, and dead pathogens. In some cases (for example, arthritis) inflammation occurs in the absence of infection or injury. This is an example of misdirected immune response.

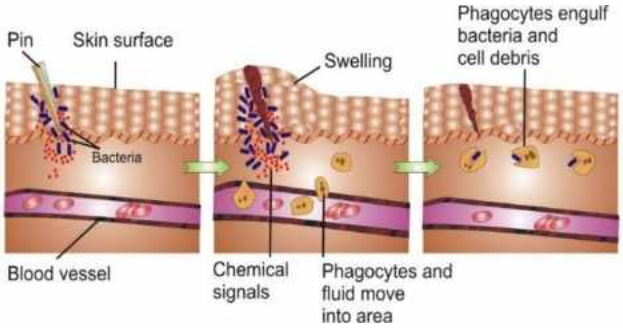

Figure 21.9: The inflammatory response

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21.2.4- Temperature Response

When macrophages attack the invading microbes, they release chemicals called **pyrogens**. The pyrogens pass through the bloodstream to the brain and reach the hypothalamus. It is the body's thermostat. Here, the pyrogens stimulate the hypothalamus to boost the body's core temperature above the normal value of 37 °C (98.6 °F). It results in the rise in body temperature. The higher-than-normal temperature is known as **fever (pyrexia)**. Fever acts as a defence mechanism against microbes. It hinders the ability of bacteria and viruses to reproduce. Some microbes (e.g., *Streptococcus pneumonia*, *Influenza*) are temperature-sensitive and are killed at high fever. Fever also enhances the speed of the actions of neutrophils and macrophages. During fever, the body releases more cytokines (interferons).

For Information
In general, temperatures greater than 103 °F (39.4 °C) are considered dangerous, and those greater than 105 °F (40.5 °C) are often fatal.

21.3- Third Line of Defence

Sometimes, microbes overcome the first and second lines of defence. When this happens, they face a third line of defence. The third line of defence is provided by the **immune system**. It is the specific defence, which means that it targets specific germs and acts especially against them. The third line of defence (immune system) can remember previous invaders and responds quickly if they enter the body again.

Types of Immunity

Non-specific defence (first and second line of defence) is present at birth. So, it is also called **inborn** or **innate immunity**. While the specific defence is acquired during growth and it is called **acquired immunity**. The acquired immunity is further of two types i.e., active and passive. In **active immunity**, the body itself prepares antibodies against the antigens of microbes. While, in **passive immunity**, the individual is given antibodies to combat specific microbes. Passive immunity is short-lived.

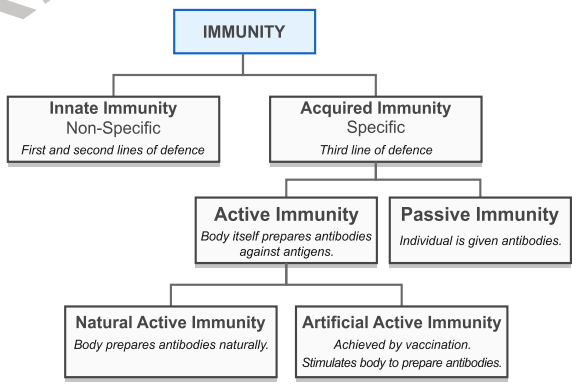

Figure 21.10: Types of immunity and their mechanisms

Active immunity may be natural or artificial. In **natural active immunity**, body prepares antibodies naturally when exposed to antigens. It is the most effective form of immunity. **Artificial active immunity** is achieved by administering weakened

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antigens in the body (vaccination). In this case, the weak antigens stimulate the body to prepare antibodies against the real antigens.

21.3.1. Components of the Immune System

The immune system is not localized to one place in the body. Rather, it is composed of a lot of individual cells that rush to the infection site to combat invading microbes. These cells are the white blood cells which arise in the bone marrow and circulate in blood and lymph. They are also found in lymph nodes, spleen, liver, thymus, and bone marrow. There are three main kinds of white blood cells i.e., monocytes, T cells, and B cells. T and B cells are collectively called **lymphocytes**.

1. Monocytes

They are a type of WBCs and are produced in lymphoid tissues. They circulate in blood and also enter the interstitial fluid. Here, they mature into **macrophages**. The macrophages phagocytose and destroy the microbes. They also produce proteins that activate the helper T cells. Monocytes also mature into other kinds of phagocytes, which remain in liver, spleen, lymph nodes, and other tissues. They also help to clear cellular debris and assist in repairing tissue damage.

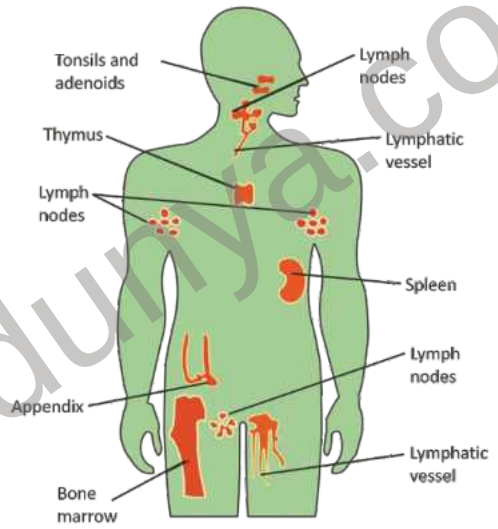

Figure 21.11: Major organs of the immune system

2. T Cells

Like other white blood cells, T cells also develop from the stem cells in the bone marrow. They migrate to the thymus, a small grey gland located just above the heart. There, they mature and develop the ability to identify foreign molecules (antigens) present on the invading microbes. Tens of millions of different T cells are made. Each T cell is specialized in recognizing one particular antigen. No invader can escape being recognized by at least a few T cells. There are four main kinds of T cells.

1. Helper T cells, which initiate the immune response
2. Cytotoxic T cells, which break the cells that have been infected by microbes
3. Inducer T cells, which supervise the development of T cells in the thymus
4. Suppressor T cells, which terminate the immune response

3. B Cells

The B cells also develop in bone marrow but unlike T cells, they do not travel to the thymus for maturity. They complete their maturation in the bone marrow. From

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there they are released to circulate in the bloodstream and lymph. Individual B cells, like T cells, are specialized to recognize particular antigens. When a B cell encounters an antigen, it begins to divide into many new cells called **plasma cells**. Each plasma cell produces an antibody that sticks to the antigen, marking it for destruction.

21.3.2- Cell-Mediated and Antibody-Mediated Immunity

We have learned that when microbes attack body, macrophages provide the immediate response by engulfing them. This response cannot eliminate many microbes, but it gives time for the immune system (third line of defence) to respond.

When a macrophage engulfs a microbe, it also produces special proteins for the helper T cells. When the helper T cells recognize these proteins through their receptors, they are activated. The activated helper T cells initiate two parallel immune responses i.e., cell-mediated immunity, and antibody-mediated immunity.

1. Cell-mediated immunity

The cell-mediated immunity, carried out by T cells, protects the body from virus infections and cancer, killing abnormal or virus-infected cells. In cell-mediated immunity, cytotoxic T cells recognize and destroy infected body cells. Their mechanism of killing is the same as that of natural killer cells i.e., they puncture the membranes of the host target cells (infected cells or abnormal cancer cells). Important events in cell-mediated immunity are as follows.

1. **Proliferation:** When a helper T cell is activated, it produces soluble chemicals e.g., T cell growth factor. This factor starts the proliferation of all types of T cells and thus large clones of T cells are formed, capable of recognizing the antigens.
2. **Activation:** A second factor is secreted by the activated helper T cell, which attracts macrophages to the site of infection.
3. **Induction:** Helper T cells activate inducer T cells in the thymus, which trigger the maturation of lymphocytes into T cells.
4. **Attack:** The receptors present on the surface of cytotoxic T cells recognize the infected body cells and so these T cells bind with the infected cells. They disrupt the membrane of the infected cells, and thus the cells burst.
5. **Suppression:** Suppressor T cells block the response of cytotoxic T cells. The population of suppressor T cells multiplies more slowly than do the cytotoxic T cells. Their low initial numbers prevent them from blocking the cytotoxic attack. After 1 to 2 weeks, however, the number of suppressor T cells rises to the point where they are able to shut down the cytotoxic T cell response.
6. **Memory:** After suppression, a population of T cells persists, probably for the life of the individual. These helper and cytotoxic T cells are now called memory cells. And they provide a rapid response to any later encounter with the microbe again.

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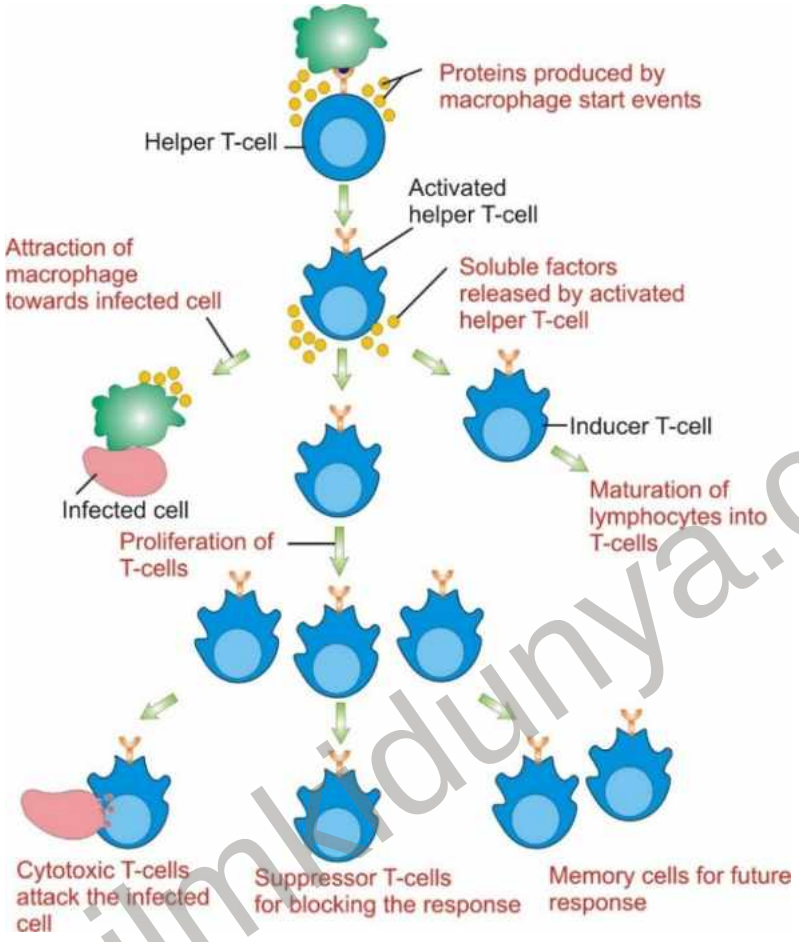

Figure 21.12: Events in cell-mediated immunity

2. Antibody-mediated immunity

When helper T cell is stimulated, it does not activate the cell-mediated immunity only. Raher, it also activates a second, stronger defence, called antibody-mediated or humoral immunity. The key players in this immunity are the B cells. They do not attack the antigens directly. Rather, they mark them for destruction. They also inactivate the toxic molecules. Important events in antibody-mediated immunity are as follows.

Proliferation: Each B cell binds to the antigens through the B receptor proteins on its surface. Helper T cells recognize such antigen-bound B cells and bind with them. Then, the helper T cells release proteins that stimulate the B cell to proliferate. As a result, a large number of cells called **plasma cells** are produced from each B cell.

> Antibodies constitute about 20% by weight of the total protein in blood plasma.

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Differentiation and Secretion: Most plasma cells stop reproducing and start producing more B receptor proteins. The B receptor proteins are secreted and now called as antibodies. The plasma cells secrete a great number of antibodies in only a few days.

A vaccine stimulates the antibody production and formation of memory cells. Vaccines are made from killed or weakened pathogens that cause antibody production.

Attack: Antibodies do not destroy an antigen directly. Rather, they mark it for destruction by one of three mechanisms.

1. ***Complement:*** Some antibodies, when attached to an antigen, activate the proteins of the complement system. These proteins penetrate the membrane of the antibody-coated antigen and make holes in it. Water is drawn into the antigen cell, causing it to swell and burst.
2. ***Macrophages:*** Some antibodies, when attached to an antigen, are recognized by a macrophage, which ingests and breaks it.
3. ***Killer cells:*** Killer cells are similar to natural killer cells, except that these possess receptors that recognize antibody-coated antigens. When a killer cell encounters an antibody-coated antigen, it binds and kills the cell.

Immune System and Gene Therapy

Malignant melanoma is a lethal skin cancer. A special type of WBC called tumour-infiltrating lymphocyte (TIL) normally attacks a cancerous tumour. But, is not strong enough to control the tumour. Genetic engineers are trying to insert a gene into TIL. The gene will enable TIL to make a protein called tumour necrosis factor (TNF). This protein kills tumour cells. When genetically engineered TIL will be returned to patient's blood, they will enter a tumour and produce TNF to kill it.

Suppression: After several weeks, the suppressor T cells shut down the antibody response.

Memory: Some members of the clone of plasma cells do not produce antibodies. Instead, they persist as memory B cells. These cells provide a rapid **secondary response** to any later encounter with the same antigen.

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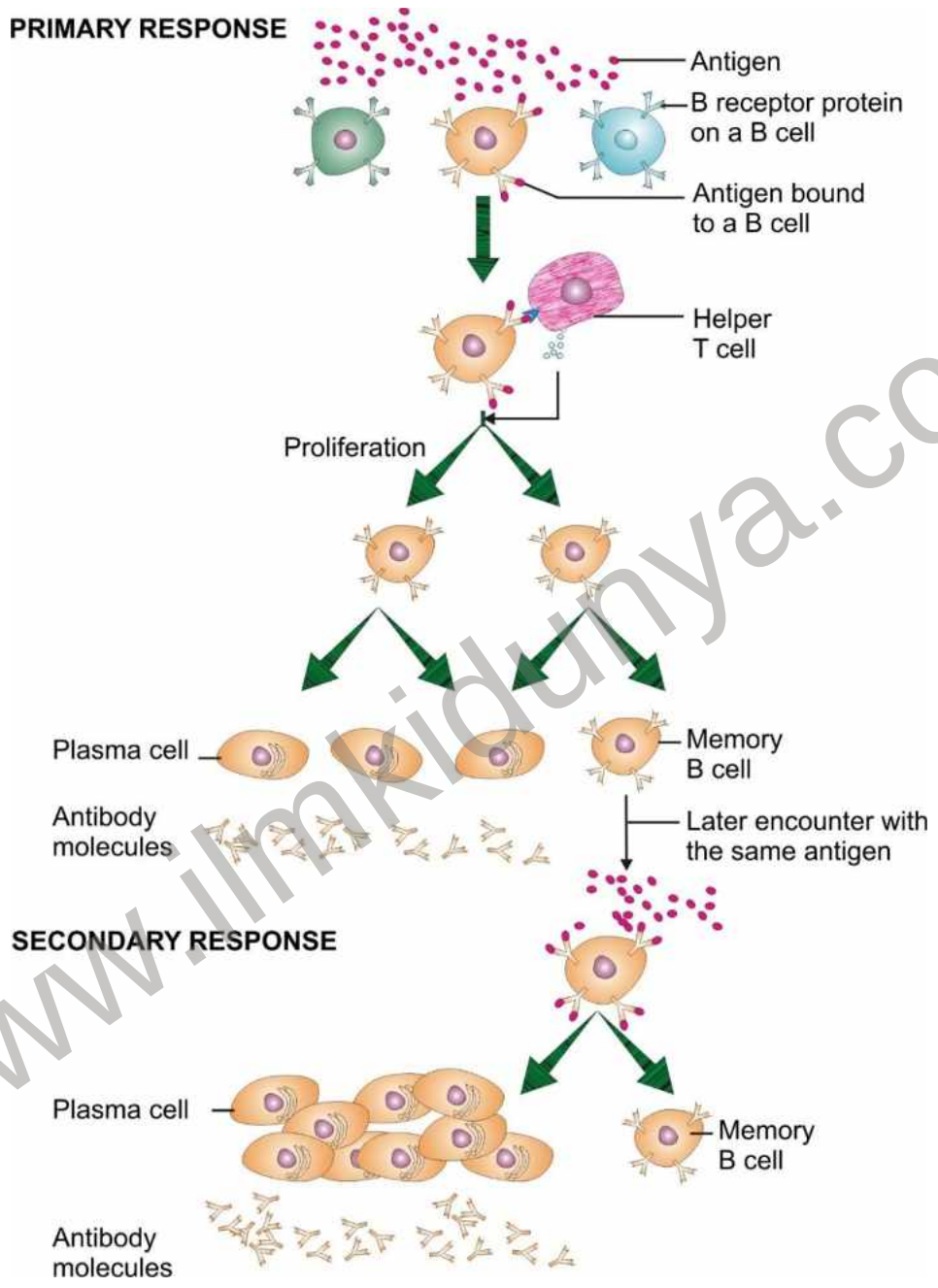

Figure 21.13: Events in antibody-mediated immunity

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Structure of Antibody

An antibody is a Y-shaped protein. It is composed of four polypeptide chains; two identical long (heavy) chains and two short (light) chains. The four chains are held together by disulfide bridges. Each chain has a constant region (similar in all the antibodies of the same class) and a variable region (differs among antibodies). The variable regions on one heavy and one light chain of each arm of Y form a specific binding site. Different antibodies have different variable regions. That is why, antibodies are specific i.e., cause the destruction of specific antigen that stimulated their production.

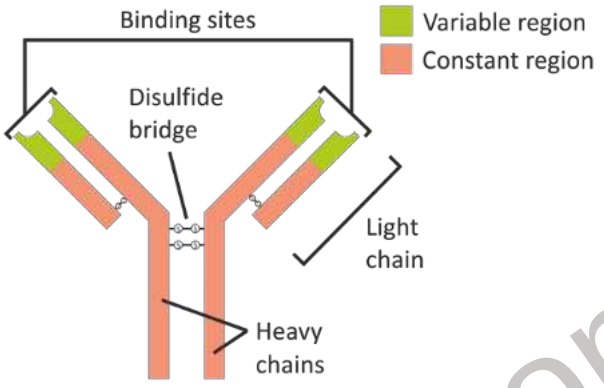

Figure 21.14: Structure of antibody

Monoclonal Antibodies

Only a small portion of antigen molecule fits into an antibody's binding site. This portion is called **determinant**. In some cases, many different antibodies can attach with a typical antigen. In such cases, each antibody fits into a different portion of antigen surface. This type of antibody is called **polyclonal antibody**.

On the other hand, there are antibodies which can attach with only one determinant. These are called **monoclonal antibodies**. In 1984 Cesar Milstein and George Kohler were awarded Nobel Prize for learning how to produce a monoclonal antibody. In their experiment, they mixed plasma cells (that produce antibodies) with cancer cells, called myelomas. They found some new cells growing and dividing in the culture. These cells, called **hybridomas**, used the genes of the plasma cell to make antibodies

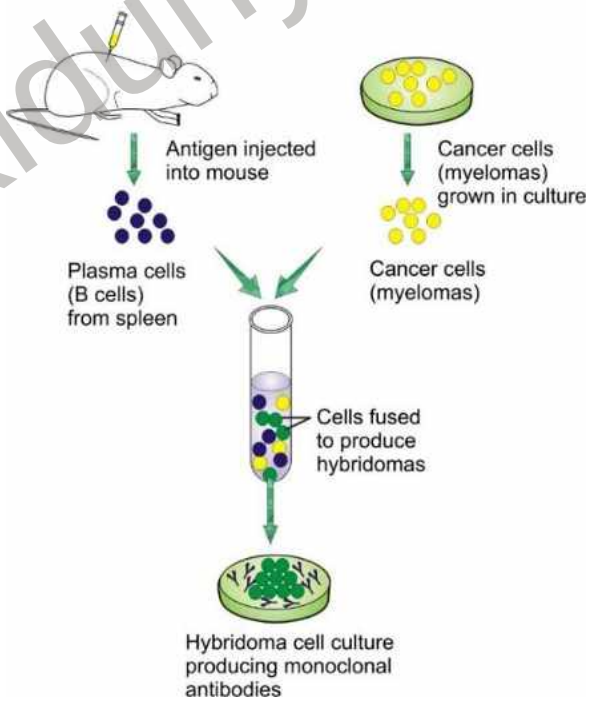

Figure 21.15: Monoclonal antibodies

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and used myeloma genes to grow and divide. The hybridoma cells continued to produce the antibody in which the plasma cells had specialized. They obtained rapidly growing cell hybridoma cells which were producing monoclonal antibodies.

Monoclonal antibodies have proved very important, because they can be used to purify specific molecules from complex mixtures. Interferon was purified in this way. In medicine, monoclonal antibodies are used as vehicles for delivering specific therapies. There is a research underway for antigens that occur only on cancer cells, against which radioactive monoclonal antibodies could be targeted to selectively kill cancer cells.

21.3.3- Disorders of Immune System

1- Allergies

Allergies result from the hypersensitivity of immune system to weak antigens that do not cause an immune response in most people. Allergens are the substances that cause allergies e.g., dust, molds, pollen, cat dander, certain foods, and some medicines (such as penicillin).

After exposure to an allergen, some people make a kind of antibody called **E-antibodies**. These antibodies bind to mast cells (usually found in connective tissues surrounding blood vessels). Mast cells release **histamines**, which start the inflammatory response. It causes dilation of blood vessels, leakage of fluid, and other responses changes like sneezing, runny nose, itching etc. In some individuals the histamines can causes life-threatening anaphylactic shock, in which swelling makes breathing difficult. A treatment of common allergies includes antihistamine drugs that block histamines and give temporary relief. Many allergies are also treated by injecting the extracts of the antigens in patients. This treatment is called **desensitization**. This method produces antibodies called **G-antibodies** in the blood. When an allergen enters, it is readily killed by G-antibodies before facing E-antibodies.

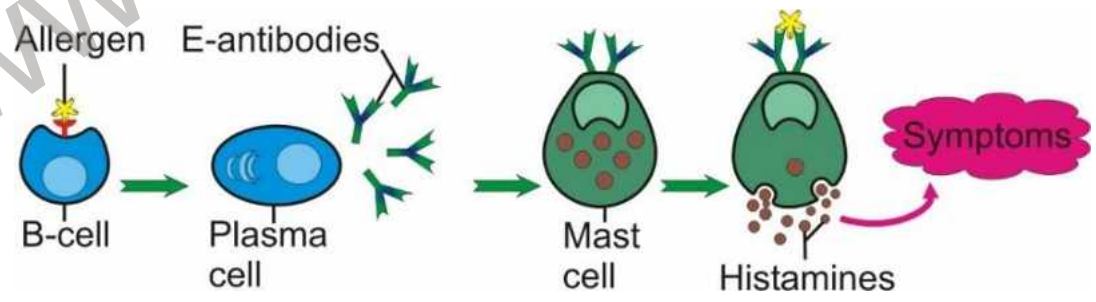

Figure 21.16: Events of allergic response

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2- Autoimmune Diseases

Autoimmune diseases result when the immune system is unable to distinguish the body-cells and foreign materials. In this case, the immune system attacks and destroys cells and tissues of the body. The following are some autoimmune diseases.

- **Myasthenia gravis (MG** is a muscle weakness in which patients produce antibodies against acetylcholine receptors on their own skeletal muscle cells. The antibodies bind with the muscle receptors. So, the muscles do not respond to acetylcholine. The nerve impulse cannot communicate the muscle.
- **Systemic Lupus Erythematosis (SLE)** is an autoimmune disease in which the patient forms antibodies against their own connective tissues and major organs.
- **Rheumatoid arthritis:** In this type of arthritis, the patients forms antibodies against the cartilaginous tissue of joints and damage them.
- **Type I diabetes (Juvenile diabetes)** is an autoimmune disease in which antibodies destroy the insulin-producing cells in the pancreas. Most medicines for treating autoimmune diseases suppress immunity in general.

2- Transplant Rejection

Normally the T and B cells of immune system attack and kill only the foreign molecules and cells. It is due to the presence of **self-proteins** on cell surfaces of each person. A particular set of **self-proteins** is like the molecular "fingerprints" recognized by own T and B cells.

Identical twins have a 100% self-proteins match. The best matches are going to occur within a family. The preference order for transplants is identical twin > sibling > parent > unrelated donor. Chances of an unrelated donor matching the recipient are 1 in 100,000-200,000.

When a person receives an organ transplant, there are chances that the self-proteins present on the donated organ do not match the self-proteins of the recipient. As a result, recipient's T cells may attack the cells of the transplanted organ and B cells produce antibodies against its proteins. This problem is called as transplant rejection. To minimize rejection, doctors look for a donor whose self-proteins are matching the recipient's self-proteins, as closely as possible. They also use drugs to suppress the recipient's immune system against the transplanted organs. But these drugs also reduce the ability to fight infections.

There are two approaches to prevent transplant rejection. In one approach, monoclonal antibodies are used to destroy the T cells that attack the transplant. In another approach, stem cells are used to make a new immune system that recognizes the transplant as self.

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EXERCISE

SECTION 1: MULTIPLE CHOICE QUESTIONS

1. Which of these are known for stimulating an immune response?
   (a) Pathogens (b) Antigens
   (c) Antibodies (d) Histamines
2. Which of the following is a part of the innate immune system?
   (a) T lymphocytes (b) B lymphocytes
   (c) Skin and mucous membranes (d) Antibodies
3. Which of these is not involved in body's non-specific defence?
   (a) Natural killer cells (b) Inflammatory response
   (c) Antibodies (d) Complement system
4. Skin offers an impenetrable barrier to microbes. It is categorized as;
   (a) Non-specific defence (b) Specific defence
   (c) Acquired immunity (d) Passive Immunity
5. What is the difference between B cells and T cells?
   (a) B cells make antibodies, and T cells destroy infected cells
   (b) B cells and T cells do the same work
   (c) B cells attack germs directly, and T cells make antibodies
   (d) B cells fight germs in body fluids, and T cells work inside cells
6. How do the neutrophils kill microbes?
   (a) By engulfing and digesting them (b) By producing antibodies
   (c) By releasing hormones (d) By carrying oxygen to cells
7. Which group of chemicals is involved in inflammatory response?
   (a) Antibodies (b) Histamines and prostaglandins
   (c) Prostaglandins and pyrogens (d) Antibodies and pyrogens
8. Which type of white blood cell is responsible for producing antibodies?
   (a) Neutrophils (b) B lymphocytes (B cells)
   (c) Macrophages (d) Natural killer cells
9. What types of drug are not prescribed to a recipient of organ-transplantation?
   (a) Drugs that suppress immunity (b) Drugs that strengthen immunity
   (c) Anti-inflammatory drugs (d) Anti-histamine drugs
10. Cell-mediated immunity works best against;
    (a) Viruses infected cells and toxic molecules
    (b) Viruses and toxic molecules
    (c) Virus infected cells and cancer cells
    (d) Toxic molecules and cancer cells

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SECTION 2: SHORT QUESTIONS

1. Define the roles of the different types of T cells.
2. Skin is an important component of our defence system. Comment.
3. State how the digestive and respiratory tracts play a role in body's defence.
4. How the natural killer cells play role in body's defence?
5. Define pyrogens and state their effect of hypothalamus.
6. What do you mean by autoimmune diseases?
7. Differentiate between;
   - Antigen and antibody
   - B cells and T cells
   - Macrophages and neutrophils
   - Complement system proteins and interferons
   - Innate and acquired immunity
   - Active and passive immunity
   - Natural active immunity and artificial active immunity
   - Cell-mediated and Antibody-mediated immunity

SECTION 3: LONG QUESTIONS

1. Describe the role of macrophages and neutrophils in killing bacteria.
2. Explain how the proteins of the complement system kill bacteria and how the interferons inhibit the ability of viruses to infect cells.
3. Inflammation is one of the most generalized nonspecific defences. How?
4. How are monocytes, T-cells and B-cells the components of the immune system?
5. Differentiate the two types of acquired immunity (active and passive immunity).
6. Describe the roles T-cells in cell-mediated immunity.
7. Describe the role of B-cells in antibody-mediated immunity.
8. Define allergies. Correlate the symptoms of allergies with the release of histamines.
9. How are T-cells and B-cells related to transplant rejections?

INQUISITIVE QUESTIONS

1. Rationalize the inflammatory response in arthritis as an example of a misdirected immune response.
2. Justify why the physicians prescribe antipyretic drugs, when fever is a nonspecific defence against microbial infections.
3. Justify why physicians prescribe antihistamine therapy to the patients of runny nose or skin rashes.
4. Describe the discovery of monoclonal antibodies and justify how this accomplishment revolutionized many aspects of biological research.

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