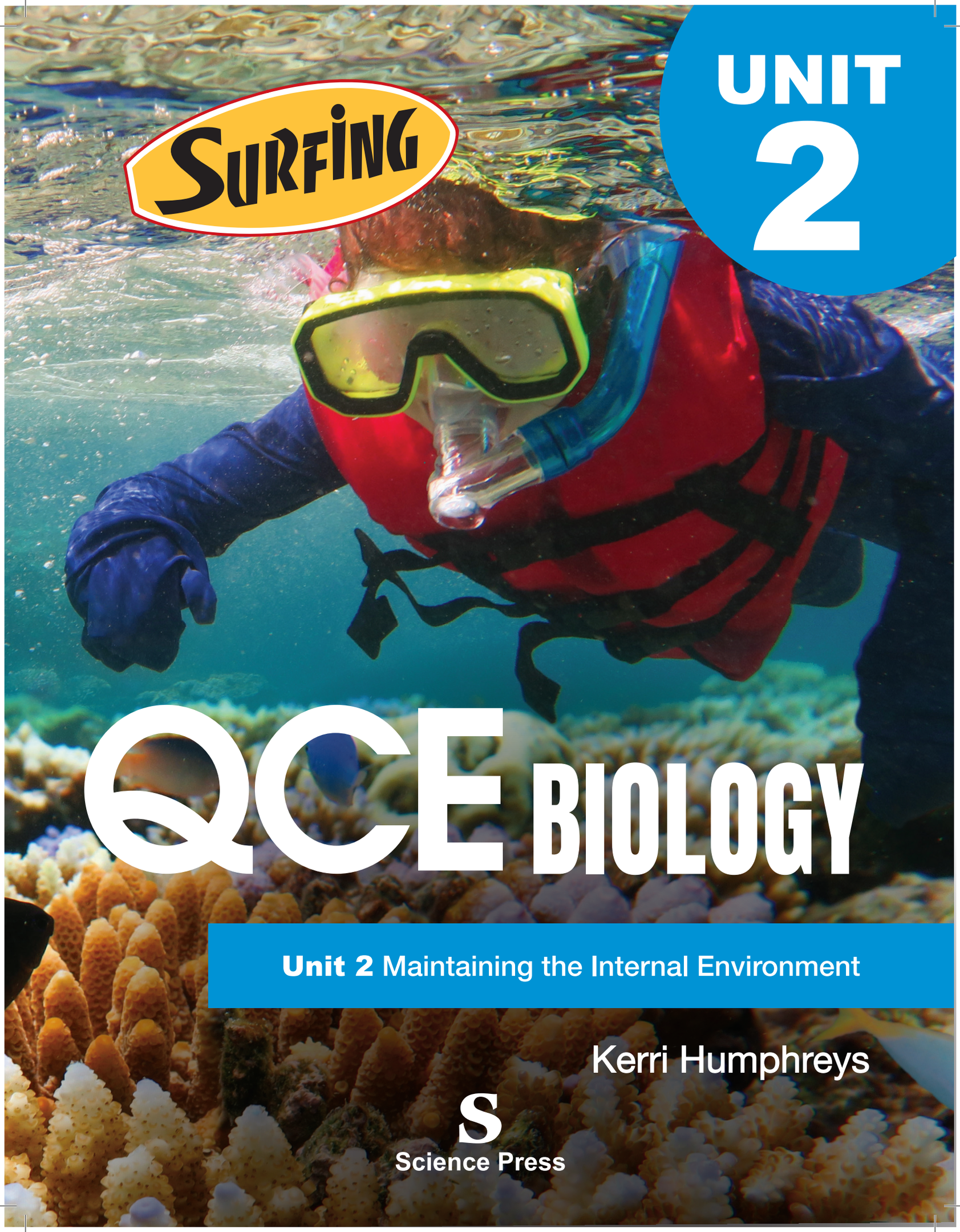

UNIT 2

QCE BIOLOGY

Unit 2 Maintaining the Internal Environment

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Science Press

First published 2026

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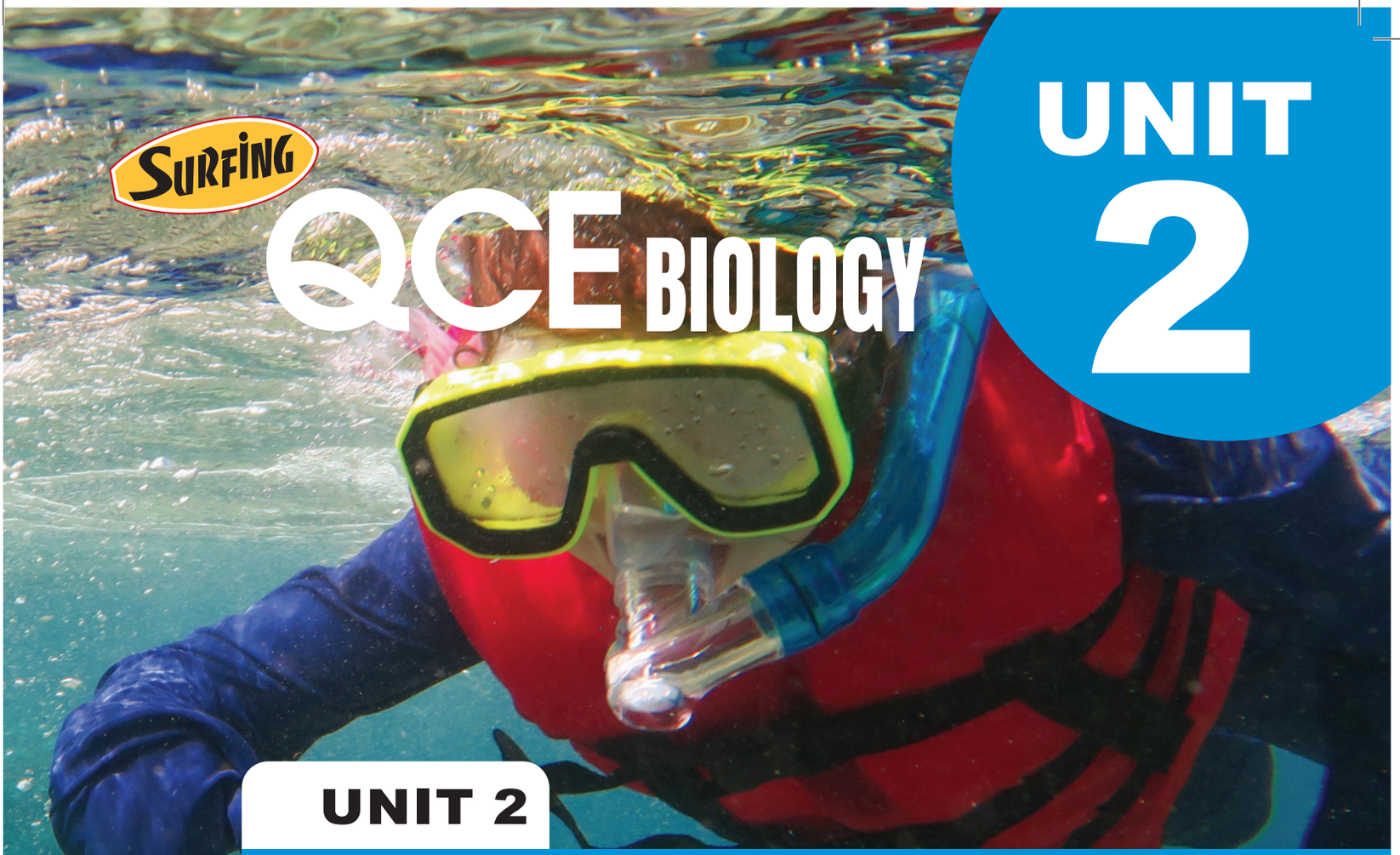

SURFING

QCE BIOLOGY

UNIT 2

UNIT 2

Maintaining the Internal Environment

In this unit you will:

- Explore the ways biology is used to describe and explain the responses of homeostatic mechanisms to stimuli and the human immune system.
- Understand that personal and communal responses involve lifestyle choices and community health.
- Develop scientific skills and conceptual understanding in homeostasis, the immune system and the relationship between global, community and individual immunity.
- Examine data to analyse strategies that may have personal and communal consequences.
- Investigate historical and current epidemics and pandemics.
- Explore immunisation, quarantine, management strategies and travel preparation for both local and international journeys.

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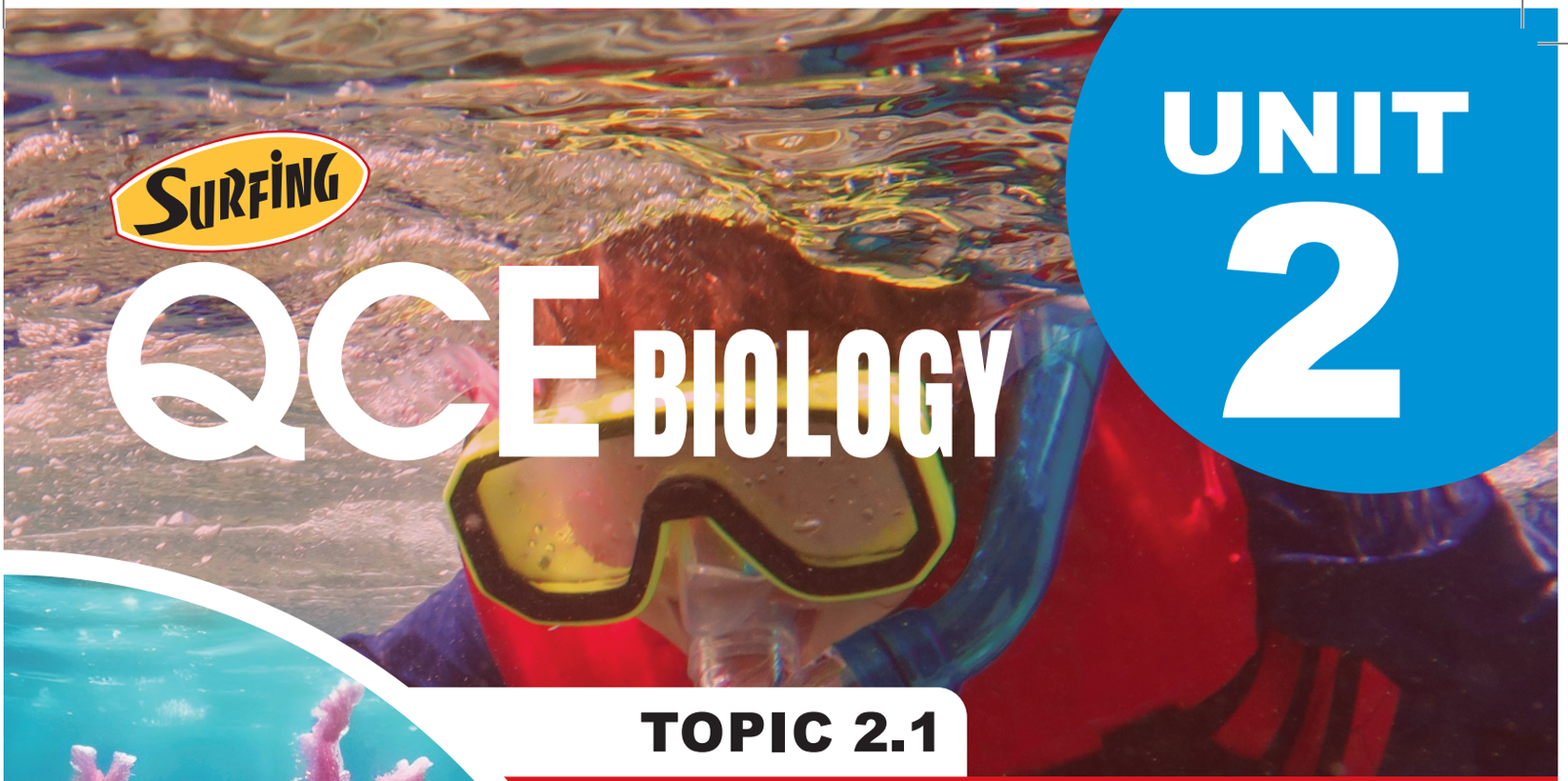

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QCE BIOLOGY

UNIT 2

TOPIC 2.1

Homeostasis

In this topic you will:

- Investigate homeostasis and the stimulus-response model including feedback control mechanisms.
- Determine metabolic activity as catabolic or anabolic and explain why changes in conditions alter enzyme activity.
- Describe neurons and the passage of a nerve impulse in terms of action potential.
- Recognise hormonal control through the endocrine system with cell sensitivity related to the number of receptors.
- Describe how receptor binding activates signal transduction mechanisms.
- Explain thermoregulatory mechanisms of endotherms.
- Explain osmoregulation mechanisms maintaining water balance in plants and animals.
- Conduct an experiment on the distribution of stomates and guard cells.

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1 Assumed Knowledge Topic 2.1

QUESTIONS

1. What is meant by the term 'metabolism'?
2. Distinguish between anabolic and catabolic reactions.
3. Distinguish between the internal and external environment of an organism.
4. Define receptor and effector.
5. List the five main senses found in animals.
6. Name the five sense organs found in animals.
7. The diagram shows most of the sense receptors found in the skin.

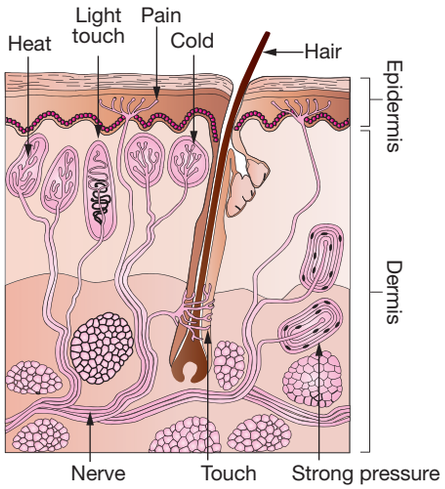

Figure 1.1 Senses in the skin.

Identify the different types of receptors found in the skin.

8. Define homeostasis.
9. Distinguish between an endotherm and an ectotherm.
10. Outline the function of the nervous system.
11. The nervous system is often divided into the central nervous system and the peripheral nervous system. Compare these two systems.
12. What is meant by ambient temperature?
13. In an experiment, what is a control?
14. What is the endocrine system?
15. Briefly describe the lymph system.
16. Define a hormone.
17. Define osmosis.
18. Distinguish between a structural, behavioural and physiological adaptation.
19. What is an enzyme?
20. What is meant by tolerance limits?
21. Define osmoregulation.
22. What is proprioception?
23. Describe the function of stomates.
24. Draw a flow chart diagram to show the stimulus-response pathway.
25. The following diagram shows a cross-section of the human brain.

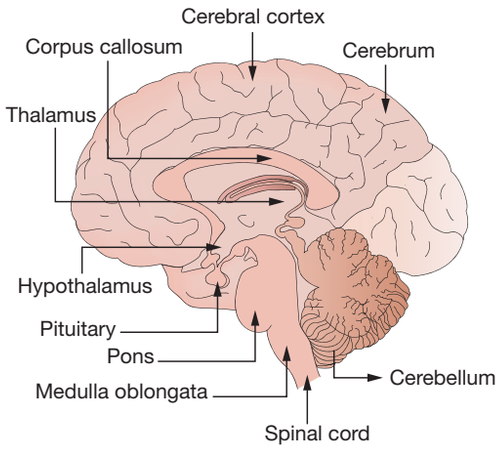

Figure 1.2 Cross-section of human brain.

Outline the function of the spinal cord and cerebrum.

26. The following diagram shows the distribution of body water.

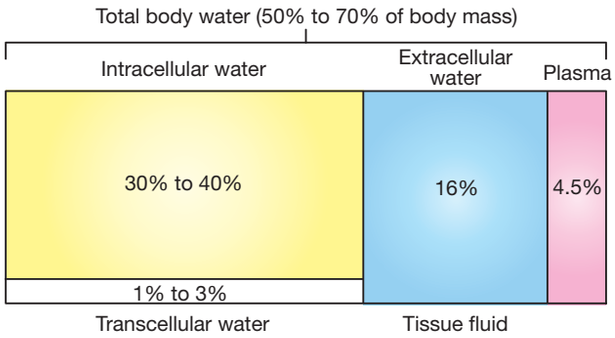

Figure 1.3 Distribution of body water.

Describe why water is an important molecule in organisms.

27. The following diagram shows a cross-section of a typical leaf.

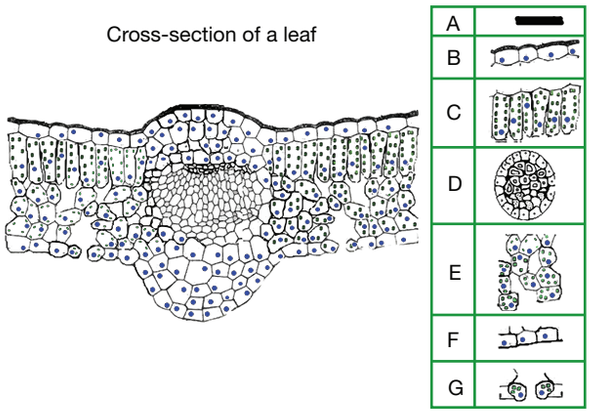

Figure 1.4 Cross-section of a leaf.

Identify the parts of the leaf A, B, C, D, E, F and G.

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6 Endocrine System

The **endocrine system** is a body system composed of different endocrine glands that are ductless glands which secrete hormones directly into the bloodstream or body fluids.

Hormones are chemical signals that travel in the blood and body fluids and act on specific target cells to cause a specific response for internal communication and regulation and maintaining homeostasis. Many hormones are relatively slow acting with their effects being long lasting. The target cells have receptors for that particular hormone which means that the hormone circulating in the bloodstream will only affect cells in particular locations.

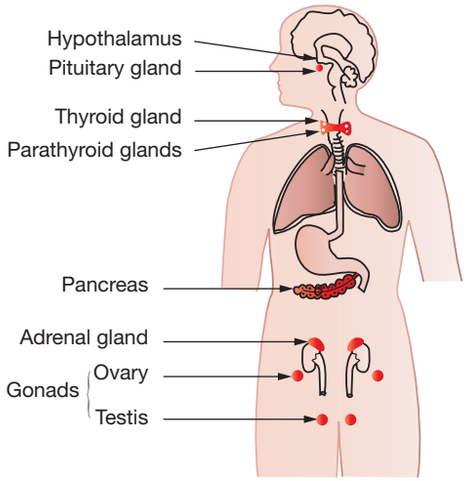

Figure 6.1 Endocrine system.

Homeostasis is the maintenance of a constant internal environment within narrow limits, e.g. homeostasis maintains constant conditions for blood pH, carbon dioxide concentration, blood glucose concentration, body temperature and water balance.

Types of hormones

There are three main types of hormones – the peptide hormones, the steroid hormones and the amine hormones. The structure of the hormone determines if it is water soluble (hydrophilic) or fat soluble (hydrophobic), e.g. polypeptide hormones such as insulin are water soluble while steroid hormones such as cortisol are lipid soluble. The amine hormones include both water soluble hormones, e.g. adrenaline and lipid soluble hormones, e.g. thyroxine.

Table 6.1 Some human hormones.

Hormone	Synthesis site	Target cell/ organ	Effect
Prolactin	Anterior pituitary	Breast gland cells.	Milk production.
Growth hormone (GH)	Anterior pituitary	All body cells, e.g. liver.	Growth, protein, carbohydrate and lipid metabolism.
Thyroid stimulating hormone (TSH)	Anterior pituitary	Thyroid.	Thyroid produces thyroxine.
Adrenocorticotrophic hormone (ACTH)	Anterior pituitary	Adrenal cortex.	Glucocorticoid production.
Follicle stimulating hormone (FSH)	Anterior pituitary	Male testes. Female follicles.	Male production of sperm. Maturation of primary follicle.
Luteinising hormone (LH)	Anterior pituitary	Male testes. Female Graafian follicle.	Male – production of testosterone. Female ovulation, formation of corpus luteum.
Antidiuretic hormone (ADH)	Hypothalamus	Kidney collecting ducts and distal tubule.	Water reabsorption when body water levels low.
Oxytocin	Hypothalamus	Ducts of breast. Uterus.	Milk ejection, labour contractions.
Testosterone	Testes	Testes, body cells.	Sperm production, male secondary sexual characteristics.
Oestrogen	Ovaries – follicles, corpus luteum	Uterus – endometrium. Body cells.	Development of endometrium, female secondary sexual characteristics.
Progesterone	Ovaries – corpus luteum	Uterus – endometrium.	Maintaining endometrium.
Thyroxine	Thyroid	Body cells.	Speed up chemical reactions.
Calcitonin	Thyroid	Bone cells. Kidneys.	Reduces loss of calcium from bones, inhibits reabsorption of calcium and phosphate.
Parathyroid hormone	Parathyroid	Bones. Kidneys. Intestines.	More reabsorption of calcium into blood, e.g. from urine and more absorption from intestines.
Adrenaline and noraderenaline	Adrenal medulla	Liver, heart.	Prepares body for fight or flight.
Aldosterone	Adrenal cortex	Kidneys – nephron.	More reabsorption Na^+ and water and more K^+. secretion.
Cortisol	Adrenal cortex	Liver.	Stimulates gluconeogenesis.
Insulin	Beta cells in pancreas	Body cells, e.g. liver muscle.	Lowers blood glucose levels with synthesis of glycogen.
Glucagon	Alpha cells in pancreas	Liver.	Raises blood glucose levels and glycogen breakdown.

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64 Lymphatic System

The lymphatic system can be considered to be a part of the vascular system as it removes interstitial fluid from tissues and takes it to the heart paralleling veins. This is a one-way movement of fluid from the tissues to the blood circulation. The lymphatic system also plays an important role in the immune response.

When tissue fluid is absorbed into lymph capillaries it is called **lymph**. Since proteins and large particulate matter cannot be absorbed directly into blood capillaries, the lymphatic system is important as it moves these substances away from the tissue spaces.

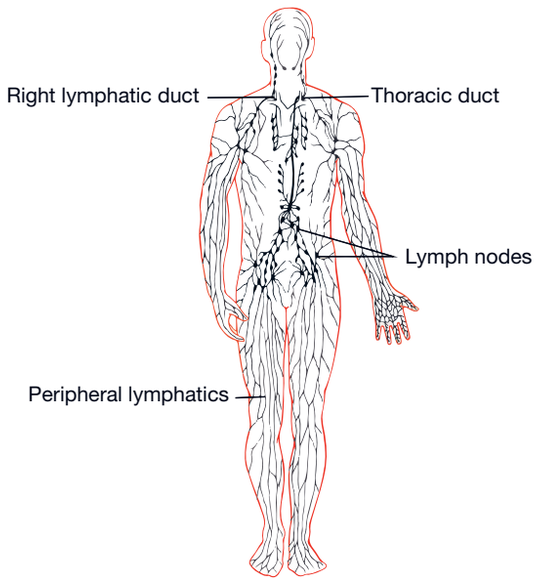

Figure 64.1 Lymphatic system.

Composition of lymphatic system

The organs in the lymphatic system include lymphatic vessels, lymph nodes, spleen and thymus.

Lymphatic vessels are veins and capillaries which carry lymph and lymphocytes from peripheral tissues to the cardiovascular system. Lymphatic drainage from the lower limbs, pelvis, abdomen, left upper side of the thorax, neck and head drains into the **thoracic duct** while lymphatic drainage from the right upper limb and right side of the thorax, neck and head drains into the smaller **right lymphatic duct**. Compared to blood capillaries lymphatic capillaries are larger in diameter and more irregular. The walls of lymph veins are thinner than the walls of blood vessel veins with valves in both types of veins preventing backflow. Unlike blood vessels which form a continuous circuit in the body the lymph capillaries begin at blind ends in the tissues. Interstitial fluid seeps into the lymph capillaries which connect to larger and larger ducts.

The walls of the lymph capillaries are held in place to the surrounding connective tissue by fibres that keep the capillary open. Gaps between the cells allow interstitial fluid and proteins and particulate matter such as cell debris and bacteria to be pushed into the lymph capillary by bulk flow. In mammals lymph is moved by the contractions of body muscles with the valves stopping backflow.

Lymph nodes are bean shaped structures with a fibrous capsule that vary in size from 0.1 to 2.0 cm depending on location and the health of the person. Infections and cancer can cause enlargement of lymph nodes. Lymph nodes contain connective tissue with interspersed white blood cells, e.g. lymphocytes and macrophages which filter, capture and destroy foreign pathogens.

Function of lymphatic system

The lymphatic system has several functions.

- Tissue fluid is collected and returned to the bloodstream.
- Transports fats absorbed from the digestive tract to the bloodstream.
- The walls of lymph vessels are permeable to proteins so that blood proteins lost by leakage from blood capillaries are returned to the blood in the lymph. This maintains normal osmotic balance between blood and tissue which is highly important as without the return of the proteins a person would die within about 24 hours.
- Lymph nodes filter tissue debris and invading pathogens from the lymph and phagocytes destroy and remove these substances.
- Non-digestible particles, e.g. dust and soot that phagocytes cannot remove are stored in lymph nodes.

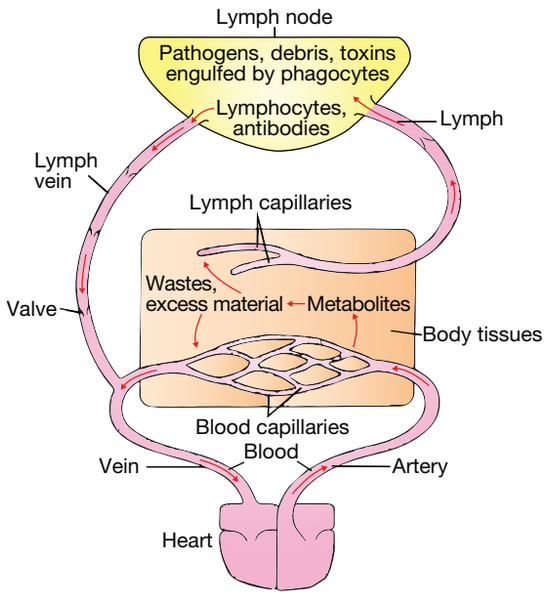

Figure 64.2 Lymphatic circulation.

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Lymphatic circulation begins at blind-end lymph capillaries in tissues and ends when lymph empties into venous blood circulation via the thoracic ducts.

Malfunction of lymphatic system

If major lymph vessels become blocked, the protein concentration in tissue fluid can rise and upset the osmotic balance in the tissues. This will cause less fluid to be reabsorbed by blood capillaries leading to excessive swelling in the tissue and severe oedema.

QUESTIONS

1. Define lymph.
2. Explain why movement in the lymphatic system is a one-way movement.
3. Construct a table to compare lymph vessels with blood vessels.
4. Identify the organ components of the lymphatic system.
5. Construct a table to summarise how the lymphatic system interacts with the cardiovascular system, the digestive system and the immune system.
6. The diagram shows a lymph valve.

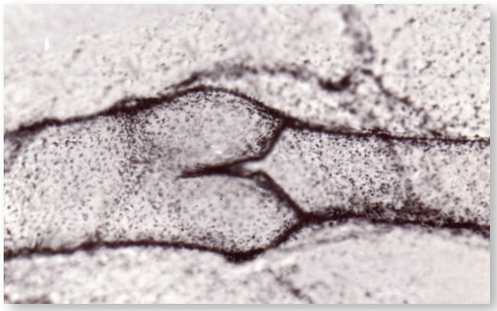

Figure 64.3 Lymph valve.

Where are lymph valves and what is their function?

7. In mammals blood flow in arteries relies on the beating of the heart to maintain blood pressure. What assists the flow of lymph in the lymphatic system?
8. Describe the size and shape of lymph nodes.
9. List the main functions of the lymphatic system.
10. Explain why the recycling of blood proteins is an important function of the lymphatic system.
11. (a) Explain why an immobilised limb will frequently swell in a condition called oedema.
    (b) What treatment is recommended to prevent oedema occurring?
12. Explain how the release of histamines in the inflammation response affects the flow of lymph.
13. Elephantiasis is a chronic condition caused by parasites blocking the lymph vessels in the lower legs. Explain why this condition causes massive enlargement of the legs.
14. The diagram shows the movement of lymph through a lymph node.

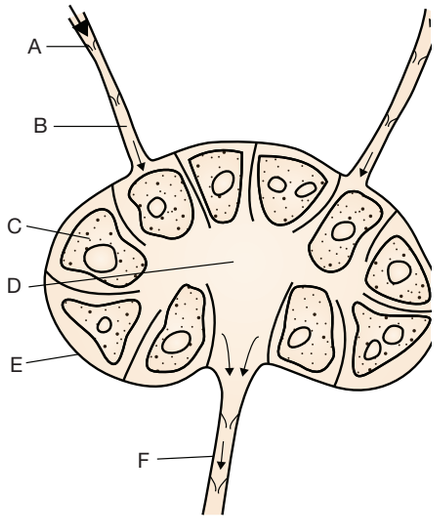

Figure 64.4 Movement through a lymph node.

Identify parts A, B, C, D, E and F.

Use the following diagram for the next TWO questions.

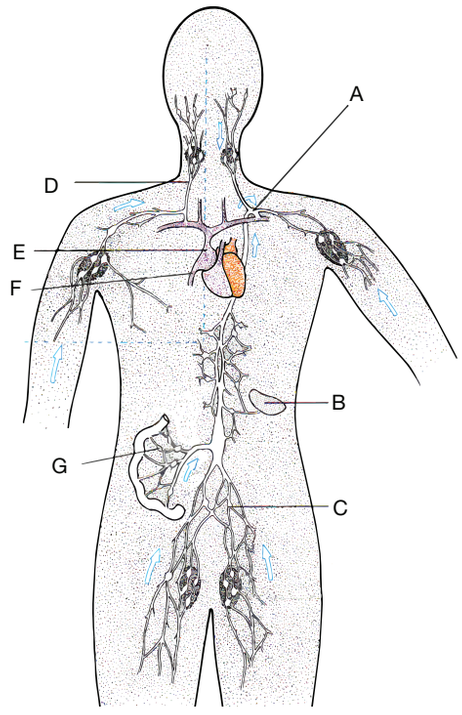

Figure 64.5 Lymphatic system.

15. Which lymph vessel receives lymph from the lower limbs, pelvis, abdomen, left upper side of the thorax, neck and head?
    (A) Part A (B) Part B (C) Part C (D) Part D
16. Peyer's patches have large lymphoid follicles that aid defence against microbes in food. Which shows the location of Peyer's patches?
    (A) Part A (B) Part B (C) Part C (D) Part G

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Questions Scene 1

1. What causes:
   (a) Tetanus?
   (b) Diphtheria?
   (c) Dysentery?
2. How do each of these pathogens enter the human body?
3. How do bacteria reproduce?
4. Explain why Tetani has an identical twin.

SCENE 2 – IN THE BLOOD

(The three friends meet again in the blood)

Tetani Isn't this wonderful fun? Causing disease is SO, SO sick.

Histo How are you making this person sick?

Tetani I've produced a neurotoxin that binds to the synapses and interferes with nerve impulses to cause muscular spasms.

Diphtheriae Speak English, please.

Histo She means that she will try to make the jaw muscles lock up.

Diphtheriae Does that mean she's won our contest and will cause the most damage?

Histo Never. I've been very active and will cause terrible pain to this person.

Tetani Do tell. What have you been doing, Histo?

Histo I was licked off a finger with sugar from a donut and then I hatched in the intestine, invaded the intestine lining and fed on the blood and caused abscesses.

Diphtheriae That doesn't sound very impressive.

Histo I caused severe, continuous diarrhoea, and THAT was impressive.

Diphtheriae OK, you can have a few points for that. Are you going to do anything else?

Histo To get some points, I thought I may invade the liver and cause amoebic hepatitis.

Tetani What have you done, Diphtheriae?

Diphtheriae I released a toxin into the blood and will cause a fever, headache and vomiting all within 5 days.

Tetani Excellent, you certainly work fast.

Diphtheriae I sure do.

Macro Invaders, Invaders!!! Help! Help!

Diphtheriae Who's this?

Macro I'm Macro, a macrophage.

Tetani Well, diffuse off Macro. We are in the middle of a contest.

Macruu I came as fast as I could, Macro. What seems to be the problem?

Macro We have been invaded by these three antigens and the immune system must come to the rescue.

Histo Leave me alone Macro. What are you doing?

Macro Come on Histo, I have you now. I'm taking you to the nearest lymph node.

(Macro transports Histo away)

Macraa Hello, Hello. What do we have here, Constable Macruu?

Macruu Antigen invaders, Sir.

Macraa Well, what are you waiting for? You take the bacteria and I'll get the protozoan.

Macruu Right, Boss.

(Macruu and Macraa engage Tetani and Diphtheriae in a fierce struggle, but eventually the two antigens are taken into custody and taken to the nearest lymph node)

Questions Scene 2

1. Describe the symptoms caused by:
   (a) Tetanus bacteria.
   (b) Diphtheria bacteria.
   (c) Dysentery protozoan.
2. What happens when an antigen enters the body?
3. Where do macrophages take the antigens?

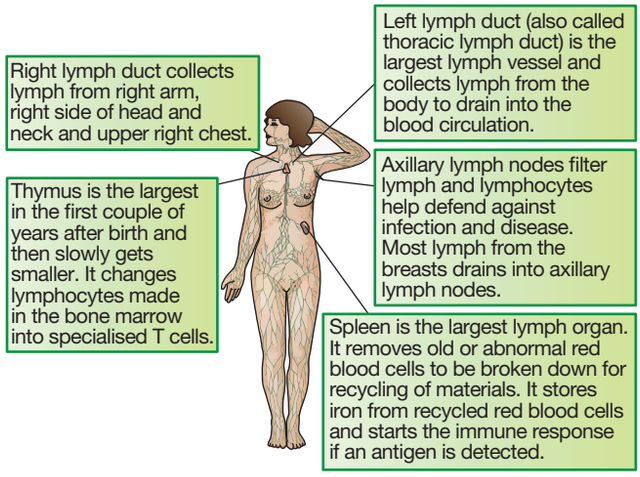

Figure 65.1 Lymphatic system.

SCENE 3 – THE LYMPH NODE

(The three friends meet again, this time in a lymph node. They are brought before Letty, the lymph node Commandant)

Histo They got you two also, did they?

Tetani They certainly did. They are very efficient, these white knights.

Letty Prisoners, be QUIET!

Tetani Yes, Madam.

Letty Step forward, prisoners, and be identified.

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67 Leucocytes

The three main cellular components of blood are red blood cells (erythrocytes), white blood cells (leucocytes) and platelets. Since the red blood cells and platelets do not have nuclei, the leucocytes are the only fully functional cells in circulation. Leucocytes can be divided into **granulocytes** which have secretory granules in their cytoplasm and **agranulocytes** which do not have granules in their cytoplasm and have a one-lobed nucleus. Under a microscope leucocytes can be distinguished from each other by the size and shape of the nucleus, the staining features of the cytoplasm, the regularity of the cell border and the cytoplasmic inclusions.

All blood cells come from **pluripotent haematopoietic stem cells** in bone marrow. In adults only the spine, ribs and pelvis and proximal ends of long bones make new blood cells. Active bone marrow is a red colour while inactive bone marrow is a yellow colour and contains many fat cells. Cytokines, e.g. interleukins and colony stimulating factors stimulate the production and development of blood cells. **Leucopoiesis** is the process of white blood cell production and development and once a leucocyte matures it cannot undergo mitosis and cell division. **Leukaemias** are a group of diseases caused by an excess or lack of white blood cells.

Leucocytes play a major role in defence against foreign invaders with most of their activity carried out in body tissues rather than in the bloodstream.

There are five types of mature leucocytes – eosinophils, basophils, neutrophils, monocytes and lymphocytes. Monocytes that leave the bloodstream develop into macrophages in body tissues.

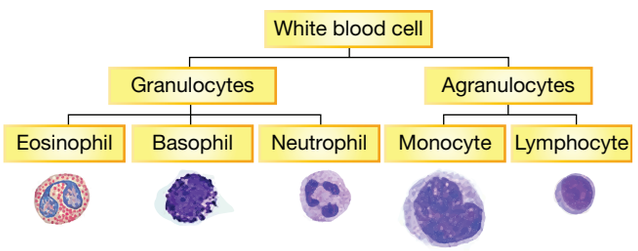

Figure 67.1 Types of leucocytes.

Phagocytes

Granulocytes and monocytes are formed only in bone marrow and are phagocytic ingesting invading organisms. Of these white blood cells it is mainly the neutrophils and macrophages that attack and destroy invading bacteria, viruses and other agents identified as foreign.

Chemotactic substances cause both monocytes and neutrophils to move to the source of the substance, e.g. they move up the concentration gradient of toxins produced by the invading virus or bacteria, towards the degenerative products produced by the inflamed tissue, towards the source of complement proteins and towards the source of reaction products caused by the clotting in an inflamed area.

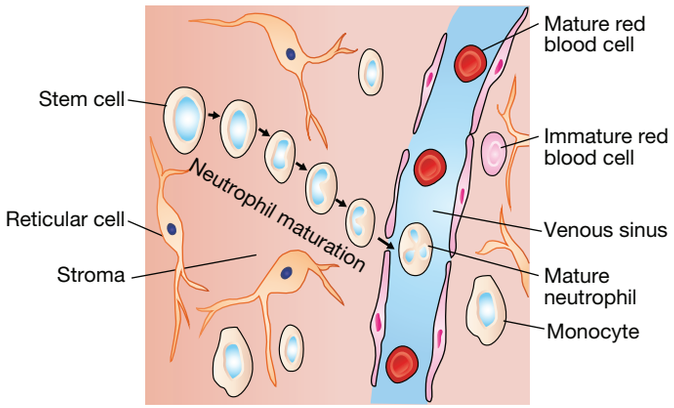

Figure 67.2 Neutrophil development in red bone marrow.

Neutrophils are the most abundant type of granulocyte and mature neutrophils can attack and destroy bacteria in both the bloodstream and tissues. They can move through the pores of blood vessels and chemotaxis causes them to move to an area of tissue damage. After formation mature neutrophils usually spend 4 to 8 hours circulating in the bloodstream and then 4 to 5 days in tissues. A neutrophil is one of the first cells to respond and move to the site of inflammation. They attach to a particle and send pseudopodia around the particle to enclose the particle in a phagocytic vesicle. Lysosomes and other cytoplasmic granules move to the vesicle and begin digestion of and breakdown of the phagocytised particle. One neutrophil can phagocytise between 3 to 10 bacteria before it is inactivated and dies.

Monocytes in the bloodstream are immature cells and when they move into tissues they swell in size to become mature macrophages. Macrophages are more powerful phagocytes than neutrophils with a longer life span, able to phagocytise up to 100 bacteria and can engulf larger particles than neutrophils. After phagocytising particles a macrophage can extrude any residual product and keep functioning for several months. Some macrophages become attached to tissues, e.g. fixed tissue macrophages are found in the spleen, lymph nodes and other tissues of the lymphatic system as well as in the kidney, bone, liver, peritoneal cavity, placenta, connective tissues and skin. In the lymph nodes macrophages trap bacteria, viruses and other foreign agents preventing them from spreading to other parts of the body. During an infection the lymph nodes in the neck and armpit can become swollen due to the increased activity of the immune response. Macrophages are also important in muscle repair, growth and regeneration and in wound healing.

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Dendritic cells are a type of phagocyte that become antigen presenting cells as part of the immune response.

Lymphocytes

Lymphocytes have a non-granular cytoplasm and are the main cells involved in the acquired immune response. Some lymphocytes are specialised to produce antibodies while others directly attack foreign invaders.

Lymph nodes

Lymph nodes are organs located along lymph vessels. They are a mass of spongy tissue separated into compartments by connective tissue. Lymph nodes filter lymph and trap and remove foreign particles such as micro-organisms and tissue debris from the fluid. There are single lymph nodes throughout the body and clusters of nodes in particular areas, e.g. neck, armpits and groin. The cortical regions of a lymph node contain reticulum cells that are the germinating centres of lymphocytes.

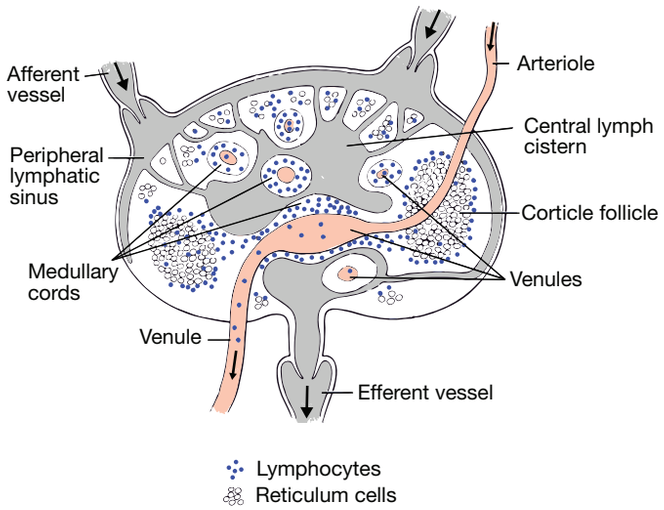

Figure 67.3 Lymph node.

QUESTIONS

1. Name the three main cellular components of blood.
2. Distinguish between granulocytes and agranulocytes.
3. What features distinguish the leucocytes from each other?
4. (a) What is the source of all blood cells?
   (b) What is the location of these sources in adult humans?
5. Distinguish between red and yellow bone marrow.
6. What is the main function of leucocytes?
7. Where is most of the activity of leucocytes carried out?
8. What will stimulate the production and development of blood cells?
9. Define leucopoiesis.
10. What is leukaemia?
11. Name the five main types of leucocytes.
12. (a) What is phagocytosis?
    (b) Which leucocytes are phagocytic?
13. (a) Define chemotaxis.
    (b) Identify the source of some chemotactic substances that attract phagocytes.
14. Explain how a neutrophil acts against bacteria and viral particles.
15. What is the life expectancy of a neutrophil?
16. Construct a table to compare neutrophils and macrophages.
17. What are dendritic cells?
18. What are lymphocytes?
19. What are lymph nodes?
20. Which of the following all have granular cytoplasm?
    (A) Neutrophil, eosinophil, basophil.
    (B) Monocyte, neutrophil, lymphocyte.
    (C) Lymphocyte, eosinophil, macrophage.
    (D) Basophil, macrophage, monocyte.
21. If you observed a stained smear of blood under high power light microscope, what would be the likely identity of a very large cell with a large non-lobed nucleus that was over twice the size of the red blood cells?
    (A) Neutrophil. (B) Macrophage.
    (C) Lymphocyte. (D) Basophil.
22. Which of the following leucocytes is not phagocytic?
    (A) Neutrophil. (B) Macrophage.
    (C) Lymphocyte. (D) Eosinophil.
23. Tissue macrophages are found at lymph nodes. What is the function of these macrophages?
    (A) Increase the rate of respiration.
    (B) Produce antibodies against foreign particles.
    (C) Storage of antibodies.
    (D) Trap and phagocytise foreign particles.
24. An adult human has about 7000 white blood cells per microlitre of blood. The pie chart shows the percentage of different leucocytes found in blood.

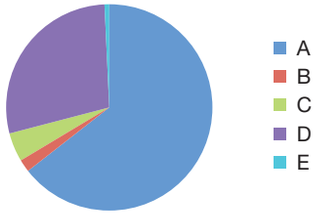

Figure 67.4 Leucocytes in blood.

Which of the sections refers to neutrophils?
(A) Section A.
(B) Section B.
(C) Section C.
(D) Section D.

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68 Mast Cells and Dendritic Cells

Dendritic cells and mast cells are important components of the innate (non-specific) immune response to an antigen. They have roles in the inflammatory response.

Dendritic cells

Dendritic cells are a type of phagocyte that become antigen presenting cells as part of the immune response. They are mainly in lymphatic tissues and skin and are particularly efficient in presenting antigens to naive T helper cells as part of the primary immune response. They link the innate response to the acquired immunity response.

Specialised Langerhans cells first described by Paul Langerhans are found on the surface of the skin in the dermis and epidermis and come from cellular differentiation of monocytes. These cells take up and process microbial antigens to become functional antigen presenting cells.

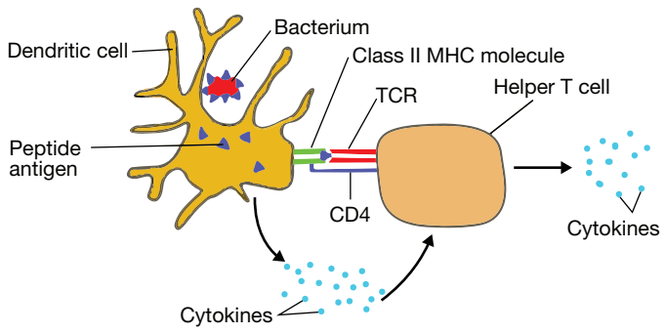

Figure 68.1 Dendritic cell engulfs bacterium and presents antigen.

After a dendritic cell phagocytises a bacterium it will move a fragment of the protein (peptide) antigen to the cell surface in **antigen presentation**. It will then display the antigen fragments with a class II **major histocompatibility complex (MHC) molecule**. T cells can recognise displayed antigens bound to MHC molecules. Most helper T cells have a surface protein called **CD4** which recognises and binds with the class II MHC molecule aiding the **T cell receptor** (TCR) on the helper T cell. The helper T cell now activated will secrete several different cytokines that stimulate other lymphocytes and help begin the acquired immune response.

Mast cells

Mast cells are a type of body cell in vertebrates that produce histamine and other molecules that trigger the inflammatory response. Mast cells are found in connective tissue and under mucous membranes that line the digestive tract and airways so when an injury occurs the mast cells release the stored histamine triggering dilation and increased permeability of the nearby capillaries.

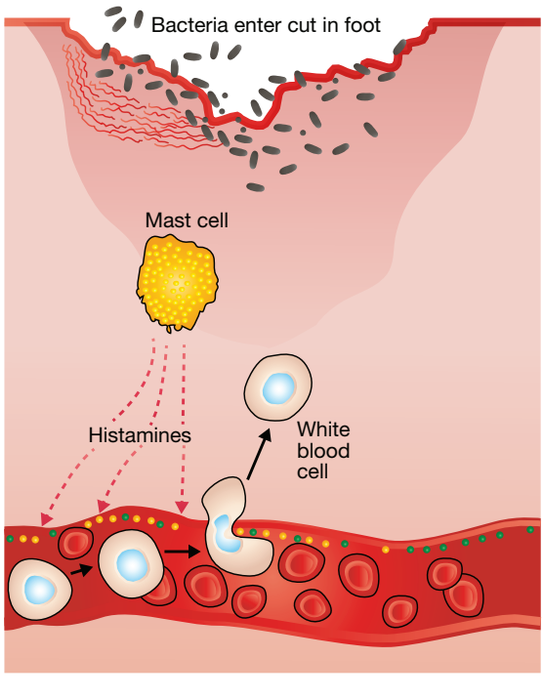

Figure 68.2 Mast cell releases histamine when tissue is damaged.

Histamine is an organic nitrogenous compound that is stored in the granules in mast cells and is the active molecule that initiates the inflammatory response. It is released when mast cells degranulate and it causes the opening pores in capillaries and the dilation of blood vessels. This causes plasma proteins to escape into the tissue leading to swelling and oedema of the tissue. In nasal passages the inflammatory response causes a stuffy, runny nose and antihistamine drugs are used to treat this symptom.

QUESTIONS

1. Identify the importance of dendritic cells and mast cells.
2. What is a dendritic cell?
3. What is the location of most dendritic cells?
4. Define an antigen presenting cell.
5. What are MHC molecules?
6. What is CD4?
7. What is a mast cell?
8. Where are most mast cells found?
9. Outline the function of mast cells.
10. What is histamine?
11. (a) How is histamine involved in the inflammatory response?
    (b) What is the function of antihistamines?
12. Which chemical plays a significant role in anaphylactic shock?
    (A) Penicillin. (B) Histamine.
    (C) Heparin. (D) Glucose.

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70 B Lymphocytes

B lymphocytes (B cells) are white blood cells, formed in the bone marrow like all white blood cells from pluripotent haematopoietic stem cells. They mature in the bone marrow and are released into the bloodstream where they circulate through blood and lymph. The lymphocytes are found concentrated in lymphoid tissue such as lymph nodes, tonsils, Peyer's patches and spleen. B cells provide antibody mediated (humoral) immunity.

Humoral immunity does not develop until after invasion by a pathogen or toxin. Most invading organisms are first phagocytosed and partially digested by **macrophages**. The macrophages become **antigen presenting cells** and also secrete cytokines, e.g. interleukin to promote growth and reproduction of specific lymphocytes. The antigen is bound and displayed on the **major histocompatibility complex** (MHC) molecule which is a cell surface molecule.

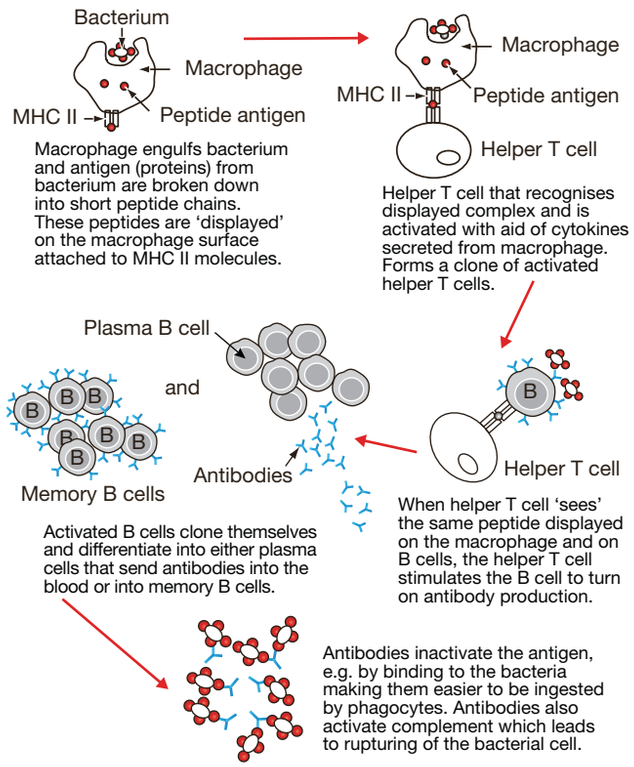

Figure 70.1 Antibody production.

B lymphocyte receptor

A B lymphocyte has antigen receptors for only one type of antigen and all the receptors recognise the same epitope. The **epitope** (also called the antigenic determinant) is the small accessible region of an antigen to which the antibody or antigen receptor binds. A single B lymphocyte can have about 100 000 antigen receptors.

The B cell receptor is a Y shaped molecule with the long tail of the Y shape consisting of two heavy chains that are anchored in the plasma membrane of the cell. Each B cell receptor has two antigen binding sites – one at each end of the short arms of the Y shape. Secreted antibodies are similar to the B cell receptors having the same Y shape and same antigen binding sites but antibodies lack the transmembrane regions that are present in the B cell receptors.

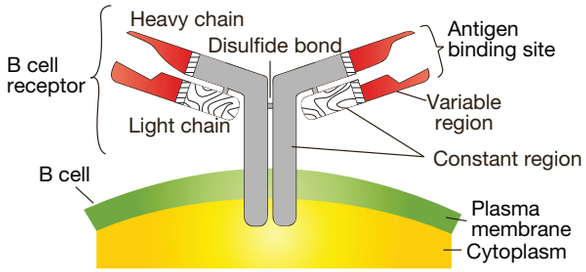

Figure 70.2 B cell receptor.

B lymphocyte activation

The presence of an antigen activates B cells to divide and differentiate into either plasma cells or memory cells. **Plasma cells** secrete immunoglobulins called antibodies with a shape compatible to that of the antigen. The antibody can bind with its particular antigen to form an antibody-antigen complex. This binding destroys the antigen by either immobilising it, or by blocking and neutralising an active binding site of the antigen, or by causing the antibody-antigen complexes to agglutinate and clump which makes them easier to eliminate by phagocytosis.

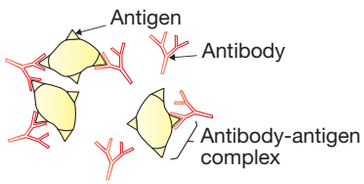

Figure 70.3 The antigen binding site at the end of the short arm of the antibody binds with the antigen to form an antibody-antigen complex.

Immunoglobulins

There are five classes of immunoglobulins (antibodies).

- **IgM** antibodies are the first circulating antibodies to appear after an infection and last a short time. They cause the antigens to agglutinate.
- **IgG** are the most abundant antibodies and activate complement proteins. They easily leave the blood vessels and enter tissues. They pass immunity from the mother to the foetus across the placenta, and from the mother to the baby in breast milk.

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- **IgA** are present in external secretions such as tears, saliva and milk. They are found in mucous membranes and prevent the attachment of viruses and bacteria to the epithelium.
- **IgD** are found on the surface of antibody forming B cells, probably acting as antigen receptors.
- **IgE** cause the release of histamine to begin the inflammatory response and play a role in the expulsion of parasites such as intestinal worms. The release of histamine and other chemicals can cause an allergic reaction.

Memory B cells

The memory B cells remain in the body for a long time and can identify that particular antigen. A later infection of the same antigen will be detected by the memory cells, which cause a rapid, large scale production of the needed antibody. Memory cells provide immunity.

Interaction between B and T lymphocytes

B cells cannot function properly without T cells. Helper T cells induce B cells to divide and produce large numbers of clones (clonal selection), resulting in millions of B cells dedicated to destroying a particular antigen. T cells stimulate the production of antibodies by B cells. Interaction occurs between the B and T lymphocytes either through cell contact between the two cell types, or the production of chemicals by the T cell (such as interleukins) which induce activated B cells to divide.

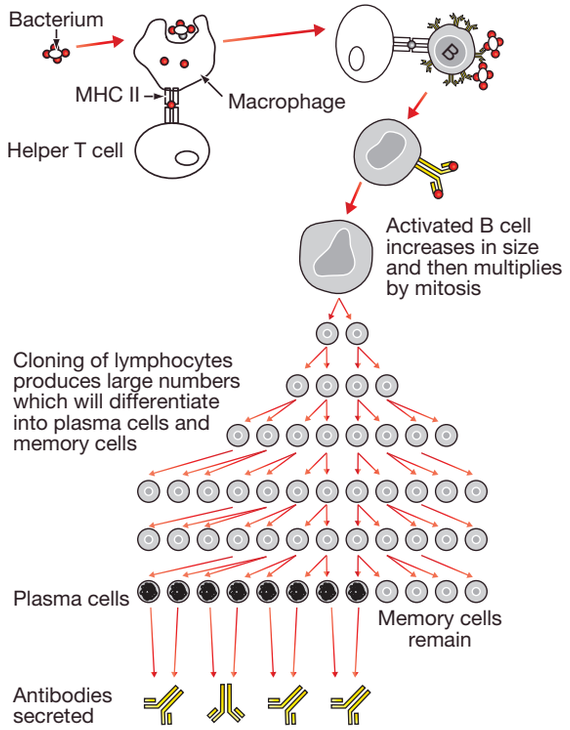

Figure 70.4 Clonal selection of lymphocytes.

Clonal selection of lymphocytes

Clonal selection is a process in which an antigen binds to and activates only the lymphocytes that have the specific receptor for that antigen. Since many lymphocytes are needed these specific lymphocytes are then cloned to produce great numbers. The clones will differentiate into effector cells (e.g. plasma cells for B lymphocytes) and memory cells that are specific for that antigen.

QUESTIONS

1. Where are B cells formed and where do they mature?
2. When does a specific humoral immunity develop?
3. Outline the first step that usually occurs when a new pathogen or toxin enters the body.
4. What is the major histocompatibility complex?
5. What is the epitope?
6. Describe the B lymphocyte receptor.
7. Compare a B lymphocyte receptor to an antibody.
8. What happens when B cells are activated?
9. Outline the function of plasma cells.
10. Explain how B lymphocytes act against an antigen.
11. Construct a table to summarise the five classes of immunoglobulins.
12. What is the function of memory B cells?
13. What is clonal selection?
14. Describe how B and T lymphocytes interact.
15. Describe a possible mechanism which allows the interaction between B and T cells.
16. Which of the following best fits the formation of antibodies?
    (A) When B cells stimulate T cells to begin manufacture.
    (B) When T cells stimulate B cells to begin manufacture.
    (C) Only in response to a malfunction in the immune system.
    (D) After a full course of antibodies in an immunisation program.
17. What are B lymphocytes?
    (A) White blood cells, mainly effective against viruses.
    (B) White blood cells which can recognise antigens.
    (C) A type of phagocyte which can engulf and destroy pathogens.
    (D) Red blood cells, mainly effective against bacteria.
18. Which of the following is caused by the production of antibodies?
    (A) Acquired immunity.
    (B) Passive immunity.
    (C) Inflammatory response.
    (D) An individual to suppress tissue rejection.

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72 T Lymphocytes

T lymphocytes (T cells) form in the bone marrow but mature in the thymus gland where they are 'programmed' for a specific antigen. The thymus is a gland above the heart in the thoracic cavity.

T cells provide cell mediated immunity, being particularly effective against body cells which have been infected by a virus or other pathogen. There are several different types of T cells; killer T cells (cytotoxic T cells), helper T cells and suppressor T cells. Memory T cells recognise the antigen if it is reintroduced.

Note that **natural killer (NK) cells** are part of the **innate** defences. They attach to cells that are affected by virus or cancer and once bound to the cell cause apoptosis. Natural killer cells act more rapidly than other lymphocytes and secrete antiviral cytokines.

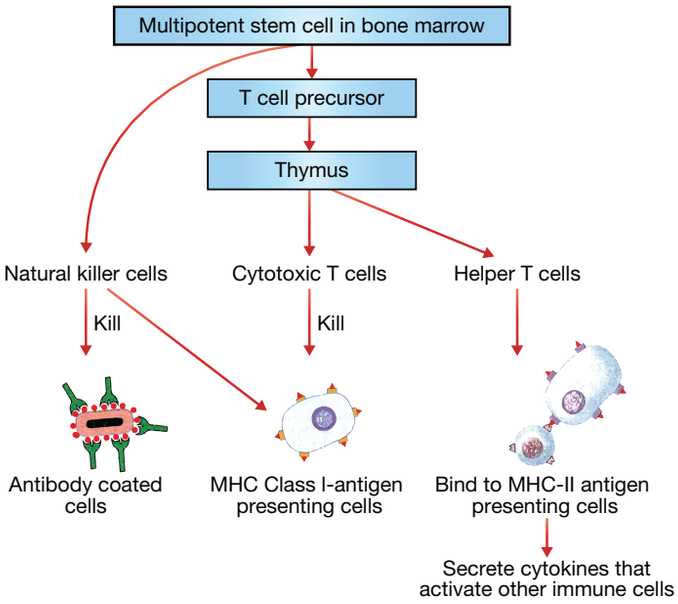

Figure 72.1 Types of leucocytes.

T lymphocyte receptors

The receptors for T lymphocytes are two polypeptide chains linked by a disulfide bridge that have a transmembrane region that locks the receptor into the plasma membrane of the cell. The outer tip of the molecule has an antigen binding site. The T lymphocyte receptors can detect antigens presented by **major histocompatibility complex** (MHC) molecules. These molecules are found on many types of cells, e.g. class I MHC molecules are on almost all nucleated body cells and if cancerous will attract cytotoxic T cells signalling the cell is to be destroyed while class II MHC molecules are mainly on macrophages, dendritic cells and B cells which become antigen presenting cells attracting helper T cells.

The T cell receptors are most important as the sites where the antigen binds to the T cell. There can be as many as 100 000 receptors sites on a single T cell.

T cells recognise cells infected with a virus as the viral proteins are moved to the cell surface and displayed by MHC molecules. The immune system recognises such cells as non-self and T cells are sent to destroy it.

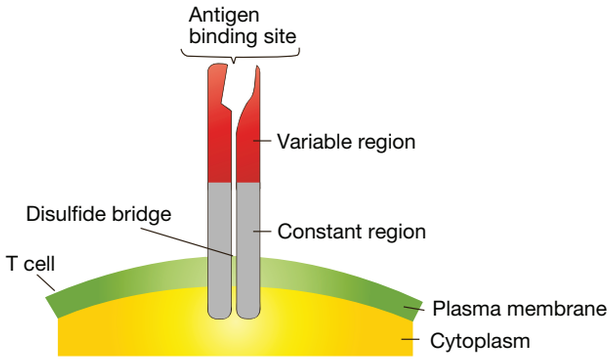

Figure 72.2 T cell receptor.

Types of T lymphocytes

Cytotoxic T cells destroy cells identified as non-self. They are a direct attack cell that can kill some micro-organisms and the body's own cells. They are formed when either a macrophage or an infected cell displays the antigen. The T cell binds with the infected cell and inserts a toxic chemical called **perforin**, a protein which causes the cell membrane to rupture, and the cell lyses. They also release **granzymes** which are digestive enzymes that enter the cell through perforin channels. They also target cancer cells and foreign graft cells.

Helper T cells (T4 cells) stimulate the production of plasma cells and help activate B cells to produce antibodies. Many B cells cannot go into production unless T4 cells for that antigen have first acted. Helper T cells secrete chemicals, such as lymphokines and interleukins, which cause activated B cells or T cells to divide and also stimulate macrophages for phagocytosis. The production of killer T cells is stimulated by T4 cells.

Suppressor T cells (T8 cells) turn off the immune response and suppress the production of antibodies. Some believe they are actually a form of helper T cell. The ratio of T4 to T8 cells is important in the diagnosis of AIDS.

Memory T cells have immunological memory allowing a faster and stronger response to a previously encountered antigen. When a T cell clone is activated by an antigen some of the new clones move to lymphoid tissue throughout the body to be preserved in the tissues. This means that re-exposure to that particular antigen, anywhere in the body will lead to an immediate recognition of the antigen and activation of the adaptive immune response.

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76 Active and Passive Immunity

Specific or **acquired immunity** occurs during the lifetime of an individual with exposure to specific inducing agents, e.g. pathogens.

Immunological memory is the ability of memory cells to recognise antigens after first exposure to the antigen.

The **primary response** occurs with the initial exposure to the antigen when T lymphocytes and B lymphocytes become sensitised to the antigen. It usually takes a while, e.g. up to 14 days for antibodies to appear in blood serum.

The **secondary response** occurs for the second or subsequent exposure to the antigen with memory cells allowing a more rapid production of antibodies and sensitised T lymphocytes that can destroy the pathogen before symptoms of the disease can occur.

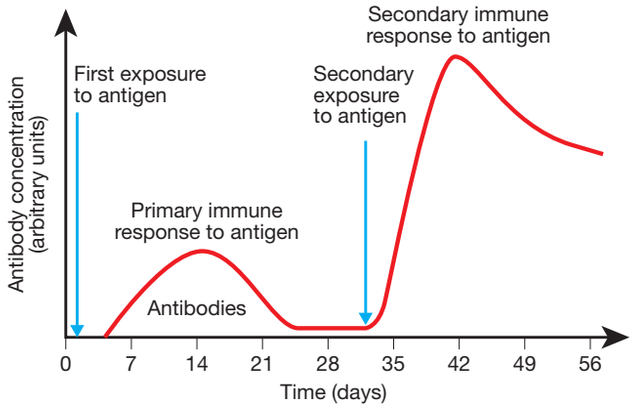

Figure 76.1 Graph of antibody production for primary and secondary immune response.

Types of acquired immunity

Active immunity occurs when the individual is exposed to any antigens and produces antibodies. Active acquired immunity is long lasting and be induced by natural or artificial means. **Naturally induced active immunity** occurs when a person gets the disease and is active after they have recovered from the disease. **Artificially induced active immunity** occurs when an individual is exposed to an antigen in immunisation.

Passive immunity occurs when an individual is given the antibodies to fight an infection and does not become able to produce their own antibodies for that antigen at a later date. **Naturally induced passive immunity** occurs when antibodies pass from mother to baby, e.g. via the placenta, colostrum or milk giving the baby immunity a degree of defence against specific pathogen to which the mother has been exposed. **Artificially induced passive immunity** occurs when an individual is given antibodies in an injection or serum, e.g. hepatitis antibodies if a person contracts hepatitis.

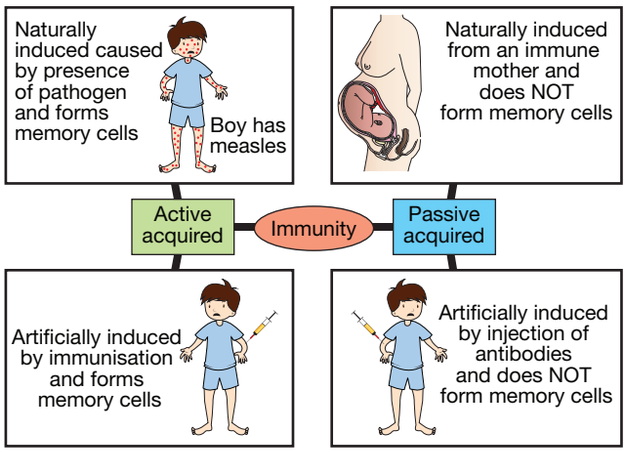

Figure 76.2 Types of immunity.

QUESTIONS

1. What is meant by acquired immunity?
2. Define immunological memory.
3. Describe the primary immune response.
4. Describe the secondary immune response.
5. Construct a table to summarise the different types of passive and active immunity.
6. In the 17th century after visiting the Ottoman Empire as wife of the British ambassador to Turkey Lady Mary Wortley Montague injected bits of smallpox scabs, e.g. pus into healthy children to protect them from smallpox in a process called variolation. Her brother had died of smallpox aged 20 years old and she survived a smallpox infection in 1715 when aged 26 years old. Identify the type of immunity provided by variolation and explain why Lady Mary Montague was such a strong supporter for this procedure.
7. When given antibodies, e.g. that originate from non-human sources some people develop serum sickness which is similar to an allergic reaction, e.g. with fever, itching, rash and swollen lymph nodes. Identify the type of immunity provided when given antibodies and explain why serum sickness can occur in some people.
8. In a graph showing the body's response to infection by a pathogen, there is a sharp rise in the concentration of antibody in the blood after the entry of the pathogen. What causes this rise in antibody concentration?
   (A) The pathogen is the antibody and it reproduces once in the host.
   (B) The pathogen acts as an antigen and activates B lymphocytes to secrete antibodies.
   (C) The pathogen acts as an antigen and activates T lymphocytes to secrete antibodies.
   (D) The pathogen is engulfed by macrophages which secrete specific antibodies.

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Anton de Bary, a German botanist, carried out a series of experiments showing the role of the fungus in the blight. He grew potato plants in cool, wet environmental conditions that favoured blight. To some plants he applied sporangia from blighted plants, but others he kept as 'control' plants and applied no fungus. Only the plants inoculated with the fungus became blighted. His work led to the study of plant diseases by other scientists.

Figure 77.3 Potato with potato blight.

QUESTIONS

1. When studying plant shoots or leaves, what signs indicate the presence of pathogens or insect pests?
2. Describe two defence mechanisms of plants to prevent the entry of pathogens.
3. Identify the causative agent in the late blight of potatoes.
4. Describe how late blight of potato first appears on the plant.
5. Explain how tubers are destroyed.
6. Explain why the potato blight was not common until the mid 19th century.
7. Outline why the Irish famine of 1846 to 1851 led to plant pathology.
8. According to tradition, a French grape farmer in Medoc wanted to stop passers-by stealing his grapes. He put some lime in water and threw it over his grapes. The grapes still looked edible. So he added some blue copper sulfate to the mixture and put up a sign stating that the grapes had been covered in a poisonous mixture which could harm or even kill people. The French scientist Millardet is credited with realising that grapes covered with this mixture, called the Bordeaux mixture, were not affected by powdery mildew. Using this information, design a controlled experiment to show that the Bordeaux mixture prevents powdery mildew on grapes. Describe the results you would expect.
9. Black spot is a fungal disease which attacks a number of plants. It is very common on roses in warmer parts of Australia such as Queensland and NSW. The leaves develop fuzzy brown or black spots with a yellowish surround. Suggest why this disease is more prevalent in damp, humid weather and how gardeners could treat their roses.
10. The fungus *Leptosphaerulina trifoli* causes pepper spot on lucerne, white clover and red clover with brown spots developing on leaflets. Suggest why most damage occurs when infection occurs on young growth after hay cutting.
11. Eucalypts are attacked by many fungi. Dieback of primary branches and crown thinning is caused by *Armillaria luteobubalina*. Death quickly follows. What would be the most likely appearance of fungal attack?
    (A) White mat of fungal mycelium under the bark.
    (B) Black spots on the leaves.
    (C) Leaves with large galls.
    (D) Tightly folded leaves.
12. Some plant diseases, called physiogenic plant diseases, are caused by deficiencies in essential plant nutrients. Nitrogen deficiency first appears in the older leaves which turn pale green, then yellow while phosphorus deficiency causes the plant to flower less and produce fewer seeds. How would you determine if a plant disease was caused by a nutrient deficiency or was an infectious disease?
    (A) Study leaf samples under the light microscope to show the presence of viruses.
    (B) Spray the plant with a fungicide to see if it recovers.
    (C) Take soil samples and identify the nutrients present.
    (D) Analyse the soil and measure the concentration of nutrients.
13. Some plants develop lumps of plant tissue that are small outgrowths caused by fungi, bacteria, nematodes, viruses, mites or insects. The shape of this lump and its position on the plant often indicates which pest is involved. What is the name of these lumps?
    (A) Corms. (B) Scale. (C) Blight. (D) Galls.
14. Humans have caused the spread of plant diseases to other countries, for example the spread of Dutch elm disease to forests of the USA. Dutch elm disease is caused by the fungal pathogen *Ophiostoma ulmi*, with a vector – the bark-eating beetle – transmitting the fungus from tree to tree. Australia has strict quarantine rules and Melbourne has some of the world's healthiest remaining elm trees. The beetle has recently been discovered near Melbourne. What is the most probable reason for the presence of the beetle near Melbourne?
    (A) Evolution of an Australian species into this species.
    (B) It flew to Australia from another continent.
    (C) Importing live cattle containing cysts of the beetle.
    (D) Ineffective spraying of imported products.

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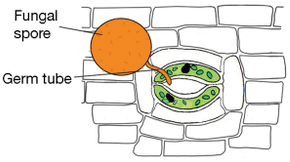
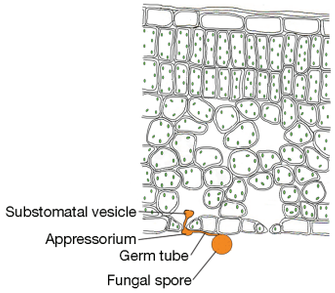
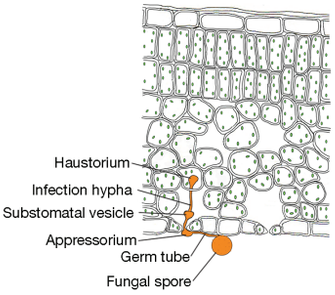
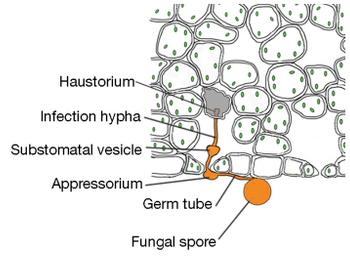
78 Plant Chemical Defence

Plant antimicrobial peptides (AMPs) are used in defence against pathogens. They have been found in leaves, stems, roots, seeds, and flowers.

Defensins are naturally occurring, small, cysteine-rich proteins that are produced by many groups, e.g. plants, invertebrates, and vertebrates for defence against bacteria and fungi. All plants have defensins. The plant defensins have a stronger activity against fungi compared to animal defensins which have stronger antibacterial activity.

Plant defensins can interact with specific components of the cell membrane, e.g. phospholipids and membrane permeability which triggers intracellular signalling cascades.

Table 78.1 Fungal rust infection of plant.

Diagram	Description
	Rust spore lands on leaf and germ tube grows on the leaf surface to enter via a stomate or breaks through the epidermal cell wall.
	An appressorium (flattened 'pressing' organ) forms across the stomate and infection hypha grows into the air space to form a substomatal vesicle.
	An infection hypha grows from the substomatal vesicle and penetrates into a mesophyll cell to form a haustorium which absorbs water and nutrients, e.g. amino acids and glucose from the plant.
	Fungal effector proteins are released. The plant detects the proteins and responds by releasing receptor proteins that bind to the effector proteins and induce apoptosis of the infected cell.

Plant cell surface receptor-like proteins can detect pathogens and host danger signals to trigger an immune response, e.g. against *Phytophthora* water moulds.

Plant ethylene receptors defend against pathogens as the ethylene regulates phytohormones for promoting abscission and senescence of mature leaves, inhibition of young leaf growth and controlling cell division and expansion.

QUESTIONS

1. (a) What are defensins?
   (b) Which groups produce defensins?
2. Compare the activity of plant and animal defensins.
3. What is one action of plant defensins?
4. What is the function of plant cell surface receptor-like proteins?
5. How do plant ethylene receptors act in defence against pathogens?
6. Myrtle rust (*Puccinia psidii*) was first detected in Australia in NSW in April 2010. It has spread rapidly across Australia infecting Myrtaceae plants, e.g. eucalypts, bottlebrushes, tea trees and lilly pilly. The powdery yellow spores are carried by the wind, water, insects, animals and contaminated articles used by humans. The rust affects growing leaves, shoot tips and young stems with a heavy infection destroying soft host tissues. Infection in natural forests has affected regeneration and growth of new seedlings and young trees altering the balance of the ecosystem.
   (a) How would you know that a plant was infected with a rust fungus?
   (b) How is the rust spread?
   (c) What damage is caused by the rust?
   (d) If a rust spore lands on a young leaf, what is the most likely entry point into the leaf after germination?
   (e) In a plant cell, the rust fungus forms a feeding outgrowth called a haustorium. Haustoria secrete effector proteins to promote further infection. Discuss how the plant responds to the effector proteins.
7. Which plants produce defensins?
   (A) Only monocotyledons.
   (B) Only dicotyledons.
   (C) Only vascular plants.
   (D) All plants.
8. How do plant ethylene receptors aid plant defence?
   (A) Promote young leaf growth.
   (B) Promote leaf abscission.
   (C) Cause bending of new growth towards the light.
   (D) Cause stem growth against gravity.

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79 Plant Bacteria and Insect Pests

Bacterial pathogens can cause many plant diseases, e.g. they cause galls, root rot, hairy root, cankers, wilts, leaf spot, blight, soft rot, bud blast, bulb rot, stem scab, root nodules, black venation, cutting rot and bulb rot.

Bacterial pathogens include species from *Agrobacterium, Clavibacter, Rhodococcus, Erwinia, Pseudomonas, Xanthamonas* and *Streptomyces*. In Victoria the bacterium *Pseudomonas cichorii* caused an outbreak of blight and leaf spot on Gerbera in 2008 and an outbreak of *Pseudomonas viridiflava* caused a bacterial blight on Lobelia.

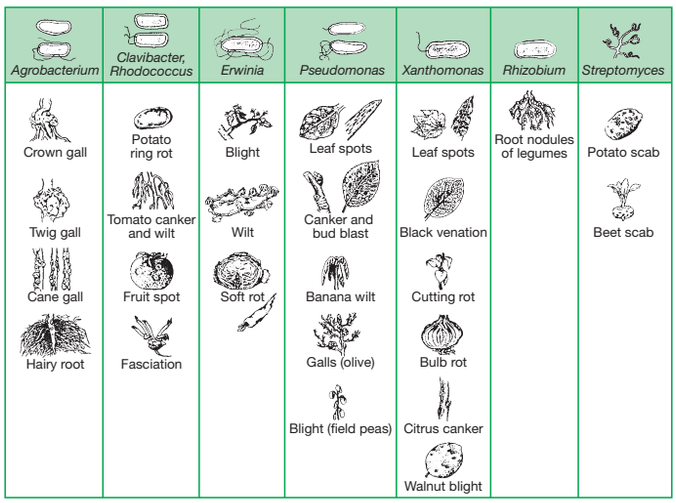

Figure 79.1 Bacterial plant pathogens.

Bacteria can enter plants in many ways – passive entry can occur by movement through natural openings such as open stomates, hydrathodes (leaf secretory tissue that secretes water), lenticels, nectaries or through abrasions or wounds on leaves, stems or roots and feeding insects can introduce pathogens.

Fire blight

Fire blight is an infectious bacterial disease that requires a range of measures for effective treatment. The bacteria causes rapid necrosis (death of cells) and symptoms depend on the part of the plant that is infected, e.g. causes dead discoloured areas on stems, blossoms are water soaked and grey-green and leaves and shoots turn brown then black and bend over producing the typical 'shepherd's crook shape. It can kill young trees in a single season.

If fire blight is suspected in Victoria the Exotic Plant Pest Hotline needs to be called.

The bacterium is found in winter at the edge of cankers and in warm conditions multiplies causing some to ooze in sticky droplets to the surface where they are picked up by rain, wind or insects which transmit the bacteria to other flowers. Entry is through wounds by insect feeding or natural openings, e.g. nectaries.

Figure 79.2 Fire blight on pears.

Root nodules

Some nitrogen fixing bacteria, e.g. genus *Rhizobium* have a close symbiotic relationship with the roots of higher plants, e.g. legumes in the pea family. The bacteria assume a form called bacteroids. The symbiotic bacteria release quantities of the fixed nitrogen into the plant's cytoplasm in the form of amino acids. This means plants with nodules can grow on infertile soils, e.g. *Acacias* in Australia.

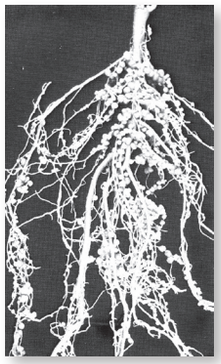

Figure 79.3 Root nodules on a legume.

The nitrogen fixing bacteria often produce surplus nitrates which increases the fertility of the soil. Growing legumes can reduce the need for fertilisers for later crops in the field and growing legumes with root nodules in the field can increase the protein yield of a crop. Planting legumes is often part of a crop rotation scheme for fields under cultivation.

Case study – insect pests: aphids

Aphids, greenflies or plant lice belong to the order *Homoptera* and are small soft bodied insects with piercing mouthparts that can feed on plant sap. Many backyard gardeners in Sydney have problems with aphids feeding on their rose plants. The aphids cause direct damage by feeding, but also act as a vector transmitting viruses. They feed by inserting their mouthpart (stylet) into the plant and the pressure of sap in the phloem forces the sap into the stylet and gut of the aphid.

Science Press ISBN 978-085583-8775

82 Experiment – Antibiotics and Bacterial Growth

Several microbiology students wished to investigate the effects of different antibiotics on the growth of bacteria.

Student actions

The students investigated different bacteria and decided to use *Staphylococcus epidermidis* a gram positive bacteria that is part of the normal human skin flora and *Escherichia coli* a gram negative bacteria that is found in the lower intestine of humans and other endotherms.

The students also investigated different antibiotics. They found that penicillin based antibiotics prevent cross-linking in peptidoglycan cell walls and are thus effective against gram positive bacteria which have simple cell walls with a large amount of peptidoglycan and are not so effective against gram negative bacteria which have a cell wall that consists of an outer membrane and thin layer of peptidoglycan as well as a cell membrane. They also found that streptomycin is a broad spectrum drug that inhibits protein synthesis preventing bacterial growth and eventual death of both gram negative and gram positive bacteria.

The students prepared four Petri dishes with sterile nutrient agar following all safety guidelines and using correct aseptic microbial techniques. They labelled two *S. epidermidis* and the other two *E. coli*.

The students moistened a sterile cotton swab in a *S. epidermidis* broth then rubbed the swab across the entire surface of the two plates labelled *S. epidermidis*. The swab was then placed in a container of disinfectant.

They then used the same method to cover the entire surface of the other two plates with *E. coli*.

They collected two antibiotic discs of **Ampicillin** and two antibiotic discs of **Streptomycin**. Using sterile forceps and aseptic techniques they placed two discs each labelled with their different antibiotic sensitivity on one plate labelled *S. epidermidis* and on one plate labelled *E. coli*. Each antibiotic disc was lightly pressed with the tips of the forceps to ensure that each adhered to the agar surface. The plates were closed and sealed with Parafilm.

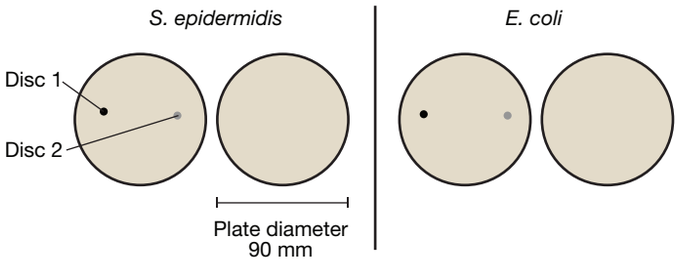

Figure 82.1 Four agar plates set up.

The students thoroughly washed their hands with disinfectant soap before leaving the work station.

All four plates were inverted and incubated at 25°C for 24 hours.

After incubation the plates were examined and the diameter of any clear areas around each disc were measured using a millimetre ruler and tabulated. The plates with no discs were observed for any clear areas.

The plates were not opened and were disposed of in a biohazard bag.

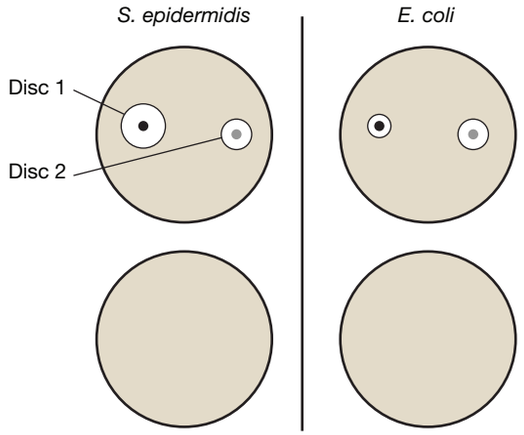

Figure 82.2 Experiment results.

QUESTIONS

1. Suggest why the students chose a gram negative bacteria and a gram positive bacteria.
2. What is the difference in action of the two chosen antibiotics?
3. Explain why the students set up plates with each culture without any discs.
4. Explain why the students needed to thoroughly wash their hands with disinfectant soap before they left the work station.
5. Measure the clear zone around each disc and knowing the diameter of the Petri dish is 90.0 mm complete the following table to show the student results.

Table 82.1 Tabulated experiment results.

Antibiotic disc	Diameter of zone of Inhibition (mm)	
	S. epidermidis	*E. coli*
Disc 1		
Disc 2		
Plate no discs		

6. From the results identify which antibiotic was in disc 1 and which antibiotic was in disc 2.
7. Identify some of the safety guidelines and sterile techniques that are needed when handling microbes.

Science Press ISBN 978-085583-8775

83 Antiviral Drugs and Antibiotics

Antibiotics

Antibiotics are antimicrobials that kill or inhibit the growth of bacteria. Antibiotics can be naturally produced by other micro-organisms or can be semisynthetic or synthetic.

Broad spectrum antibiotics are effective for killing many types of bacteria while **narrow spectrum antibiotics** target disease-causing bacteria.

Bacteriostatic antibiotics do not kill the bacteria, but inhibit their growth allowing the immune system to overcome the infection. Many bacteriostatic antibiotics interfere with protein synthesis, e.g. tetracycline, chloramphenicol, macrolides and spectinomycin.

Bactericidal antibiotics destroy bacterial pathogens. Bactericidal antibiotics can target outer cell walls, inner cell membranes or metabolic pathways of bacteria, e.g. penicillins and polymixins.

Table 83.1 Mode of action for some antibiotics.

Mechanism	Description	Examples
Inhibition of cell wall synthesis	Antibiotics, e.g. penicillin inhibit the formation of peptidoglycan cross-links for the cell walls of bacteria. This weakens the cell walls and without strong cells walls, the new bacterium is unable to survive.	Penicillin used to treat syphilis and *Staphylococcus* infections. Cephalosporin is used to treat typhoid fever.
Inhibition of protein synthesis	Antibiotics block some stage of protein synthesis by attacking the bacterial ribosomes. They can bind to the bacterial ribosomes (70S) but do not attack the ribosomes in human cells (80S). Some cause the misreading of the genetic code which leads to the synthesis of abnormal proteins.	Erythromycin is used for respiratory tract infections and for people who have an allergy to penicillin. Chloramphenicol is a broad band antimicrobial active against Gram positive and Gram negative bacteria and anaerobes usually used topically, e.g. eye infections. Neomycin is in many topical medications, e.g. creams, ointments.
Inhibition of nucleic acid synthesis	Rifampicin inhibits DNA dependent RNA polymerase preventing initiation of transcription, especially in Gram positive bacteria. Antibiotics affect the synthesis of DNA/RNA or attach to DNA/RNA so they cannot be read. Anthracyclines inhibit DNA and RNA synthesis by intercalating between base pairs of the DNA/RNA strand and prevent cell replication.	Rifampicin (semisynthetic) used to treat the meningitis form of tuberculosis. Anthracyclines can kill or inhibit bacteria but due to toxicity to humans are never used to treat infections but are used to treat a wide range of cancers.

Antiviral drugs

Antiviral drugs are antimicrobials that are used to cure or control virus infections. Most antiviral drugs do not kill viruses; they inhibit their replication. Viruses can only replicate and metabolise inside a host cell. Some antiviral drugs prevent the virus entering the cell. Others prevent the virus releasing its DNA or RNA into the cell nucleus or prevent it splicing its genetic material into the host cell DNA. Antiviral drugs include anti-herpetic agents, e.g. Helpin, Famvir and anti-influenza agents, e.g. Relenza, Tamiflu.

Antiseptics

Antiseptics are antimicrobial chemicals that are applied to living tissue or skin to reduce the likelihood of infection, sepsis or putrefaction, e.g. alcohols are used to disinfect skin before injections. Chlorhexidine is a skin antiseptic or is used to treat inflammation of the gums. Antiseptics kill and/or prevent microbial growth and prevent infection.

Antiseptics are mild chemicals less effective than disinfectants but not damaging to skin and mucous membranes. Toxicity means they cannot be ingested. Since many are mild chemicals all endospores may not be killed, and the resistant forms multiply leading to a population that is not affected by the antiseptic.

Disinfectants

Disinfectants are antimicrobial agents that are applied to non-living objects to destroy micro-organisms, e.g. hypochlorite is used to purify swimming pools. They are extremely effective in sterilising surfaces, e.g. sterilisation in hospitals of medical tools, in dental surgeries, kitchens, bathrooms and floors. Choice of disinfectant depends upon the need, e.g. some have a wide spectrum and kill nearly all organisms while others kill a small range of pathogens. Disinfectants cannot be used on living tissue, or ingested. If all endospores are not killed, the resistant forms multiply leading to a population that is not affected by the disinfectant.

QUESTIONS

1. Distinguish between antibiotics and antiviral drugs.
2. Distinguish between broad spectrum and narrow spectrum antibiotics.
3. Distinguish between bacteriostatic and bactericidal antibiotics.
4. Construct a table to evaluate the use of antiseptics and disinfectants giving advantages and disadvantages of each and an evaluation.

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84 Rational Drug Design

Rational drug design is a process that looks for new medications based on a biological target. The drug is usually a small organic molecule that is complementary in shape and charge to its target which it activates or inhibits, e.g. a target protein such as an enzyme. The action of the drug has a therapeutic effect aiding the patient. This method of investigation is used by many pharmaceutical companies as it reduces the need of 'chance' findings as occurred with Alexander Fleming discovering the action of penicillin.

Each rational drug design starts with the **proteomics** which is the study of the proteome – the full protein set encoded by the genome. The proteome of normal cell cells are compared with the proteome of cells from a particular disease state.

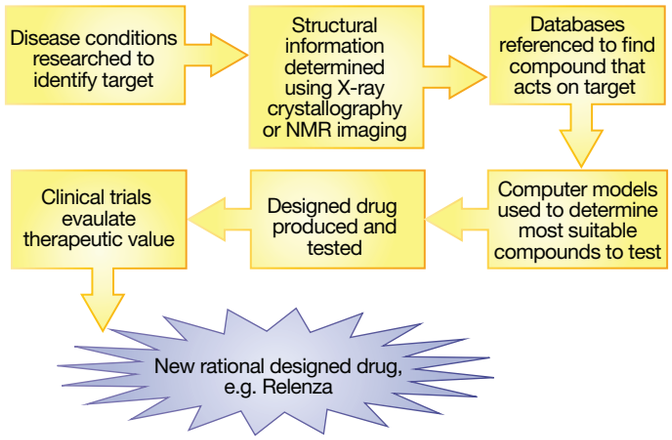

Figure 84.1 Rational drug design.

Computer modelling

Drug design often involves computer modelling techniques in **computer aided drug design**. Research into the structure and activity of the specific target molecule allows researchers to modify the target molecule to see if the changes have therapeutic value. The 3-D shape of the target molecule gives the shape to which the potential drug needs to attach and physical models in **structure based drug design** are used to predict the functionality of changes in the structure of the molecule. Different parameters can be set in the computer modelling to predict properties of the changed molecule, e.g. binding ability, electronic properties before the molecule is produced. Once suitable forms have been found possible forms can be produced reducing the time, effort and cost of research.

Steps in rational drug design

There are three basic steps in drug design.

Target, e.g. receptor or enzyme is identified relating to a particular disease state. There are many macromolecules involved in disease and many have specific signalling pathways. The disease can cause a disruption or imbalance in the signalling pathway leading to specific symptoms. With knowledge about differences between normal cell proteins and diseased cell proteins, a suitable target can be chosen. The target needs to be disease modifying, e.g. enzyme activator or inhibitor. The target must not affect any other 'off target' molecules which will cause side effects of the potential drug.

Target is fully characterised giving structural information about the given target. In this process the target is usually cloned and produced and purified to establish a screening assay which is used to determine the 3-D structure. Magnetic resonance spectroscopy and X-ray crystallography are used to find the structure of molecules.

Molecule chosen – screening libraries that contain information about the structure and functionality of biomolecules are searched to find possible molecules that will suitably interact with the target. The chosen molecule is then designed so that it binds to the target to either restore normal signalling pathway or inhibit a dysfunctional biomolecule. The design should make a compound that can be taken as a drug, e.g. form an oral medicine with suitable chemical and metabolic stability and have few toxic effects.

Relenza

Relenza is an antiviral drug developed by the Australian biotech firm Biota Holdings. It is a neuraminidase inhibitor used in the treatment of influenza caused by influenza A and B viruses. Neuraminidase is a viral enzyme that is part of the mechanism that causes newly formed virions in an infected host cell to be released from the host cell. Relenza acts by blocking the active site of neuraminidase using competitive inhibition to stop the host cell releasing the virions and spreading the infection.

Critics of rational drug design

Critics argue that the most significant discoveries have been by chance and this formula approach to discovery may limit development and restrict initiatives.

QUESTIONS

1. What is rational drug design?
2. Identify the main features of rational drugs.
3. What is the expected effect of a rational drug?
4. How is computer modelling involved?
5. Outline the three main steps in rational drug design.
6. (a) What is Relenza?
   (b) How does Relenza work?

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85 Pathogen Transmission

A **pathogen** is an organism, virus, viroid or prion that causes disease. The invading cellular and non-cellular pathogens are a source of non-self antigens.

Most pathogens are micro-organisms such as bacteria, viruses, protozoans or fungi. All viruses are potential pathogens as they can only metabolise and reproduce inside a host cell.

A **parasite** benefits by living on or in a host which is harmed.

Pathogens can come from the external environment, e.g. entering the body from the air, with ingested food and water, through wounds or cuts in the skin or they can be endogenous are originate from within an organism, tissue or cell.

The transmission or communicability of an infectious disease depends on the way the pathogen leaves its host and how it enters a susceptible person.

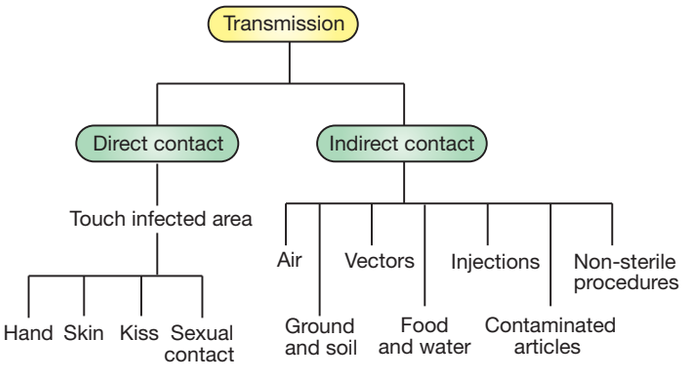

Figure 85.1 Disease transmission.

Direct contact

Direct contact involves touching the affected area of the infected person with the contagious person passing the pathogen to another person. The communicable person may be showing symptoms of the disease or may be a symptomless carrier. There are many diseases that can be transmitted by both direct and indirect contact, e.g. measles is caused by the measles virus and is spread by contact with saliva or nasal secretions of an infected person but can also be spread through contact with surfaces that have droplets from coughs and sneezes from an infected person.

Direct contact can involve touching skin, handshaking, kissing and sexual contact. Diseases include *Herpes simplex* infection, chickenpox, warts, ringworm, scabies, head lice, *Staphylococcus aureus* infection, syphilis, HIV infection, gonorrhoea, pubic lice and conjunctivitis.

Indirect contact

There are many ways a pathogen can be transmitted from person to person through indirect contact.

- **Airborne diseases** – involves inhalation of bacterial spores, or droplets containing pathogens from coughing, sneezing or speaking. Airborne diseases include chickenpox, common cold, diphtheria, influenza, measles, meningitis (bacterial), mumps, rubella, tuberculosis and whooping cough.
- **Food and water** – involves eating or drinking contaminated food which can be caused by lack of proper sewage treatment of water supplies, poor hygiene, use of sewage as a fertiliser. Diseases include *Cryptosporidium* infection, *Giardia* infection, hepatitis A, meningitis (viral), worms, *Salmonella* infection, cholera, typhoid and botulism.

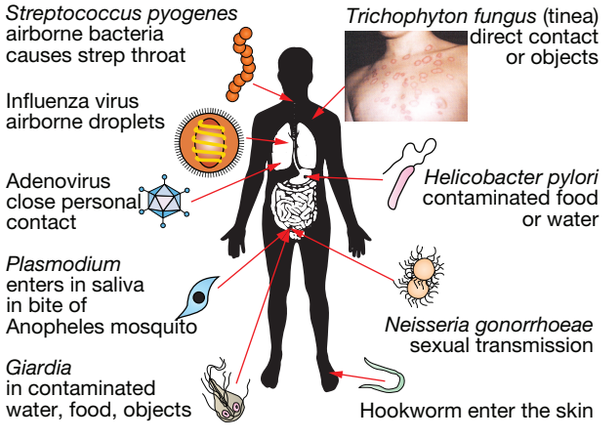

Figure 85.2 Pathogens in humans.

- **Vectors** – involve transmission through other organisms that carry the pathogen from one person to another or from an infected animal to another person. The vector may or may not be an intermediate host of the pathogen. Parasitic life cycles can be complex involving more than one host. The intermediate or secondary host is usually the organism in which the parasite goes through the larval stage or undergoes asexual reproduction. Many different organisms act as vectors, e.g. the following.
  - **Arthropods** – e.g. houseflies, cockroaches carry pathogens on their feet and bodies and contaminate food and water, e.g. houseflies transmit anthrax, typhoid and cholera. While mosquitoes, fleas, lice and tick carry pathogens internally and transmit them while feeding, e.g. Lyme disease is transmitted in the bite of a tick.
  - **Mammals** – many different mammals can transmit pathogens to infect humans, e.g. dogs transmit rabies and hydatid tapeworms and pigs transmit pork tapeworm.
  - **Birds** – certain birds can transmit avian influenza, e.g. water birds and parrots can transmit psittacosis.

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Pasteur, vaccinations and the germ theory

The germ theory became firmly established with Pasteur's work with anthrax.

Anthrax is a deadly disease of sheep, horses and cattle. In 1876, Robert Koch isolated the anthrax bacillus (*Bacillus anthracis*) and suggested that sick animals be killed or burned as the bacterial spores survived for months in contaminated fields. Pasteur decided to work on anthrax and added one drop of blood from a sheep dying of anthrax to 50 mL of sterile culture and grew the culture. He repeated the dilution 100 times so that none of the original culture cells were present and found that the final culture was as active as the first in causing anthrax. This showed that the bacteria caused the disease as only the reproducing bacillus could escape the dilution.

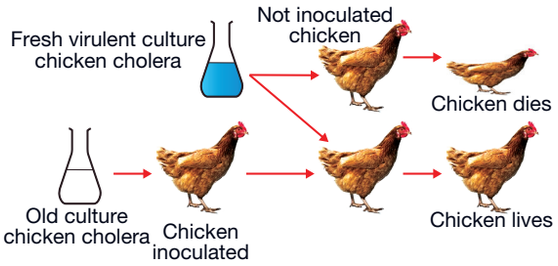

Figure 87.3 Louis Pasteur and chicken cholera.

His work with **chicken cholera** made Pasteur believe he could find a vaccine for anthrax. He tried several techniques such as oxidation and ageing, and eventually developed a vaccine.

His work was challenged by a well known veterinarian, causing Pasteur to conduct a **public test**. Using 25 sheep as controls and 25 vaccinated, all sheep were given a lethal dose of anthrax. There was intense publicity. On 5 May 1882, two days after the final inoculation, all control sheep were dead and all vaccinated sheep were alive and healthy. The publicity educated the world about microbes and vaccination.

QUESTIONS

1. Define a pathogen.
2. Although Pasteur is known as the father of microbiology and immunology, how did he begin his science career?
3. Why did Pasteur begin investigating the production of wine?
4. What was the current belief about fermentation when Pasteur began investigating wine production?
5. Describe the three clues that allowed Pasteur to solve the puzzle of fermentation.
6. What did Pasteur conclude?
7. State the 'germ theory of disease'.
8. Describe the role of Pasteur in identifying the causes of disease.
9. Describe pasteurisation and explain why it is used.
10. What is spontaneous generation?
11. In Pasteur's classic experiment, identify the control and explain how it is used.
12. Explain why Pasteur boiled each broth.
13. Explain why the flask with the swan neck did not develop mould or bacteria.
14. What is anthrax?
15. Who discovered the microbe that causes anthrax?
16. Describe Pasteur's experiment that confirmed the germ theory of disease.
17. How did Pasteur develop a vaccine?
18. Explain how microbes and vaccination came to the attention of the public.

The following information is to be used for the next THREE questions. In 1864 Louis Pasteur carried out a series of experiments where he put broth into several special S shaped flasks.

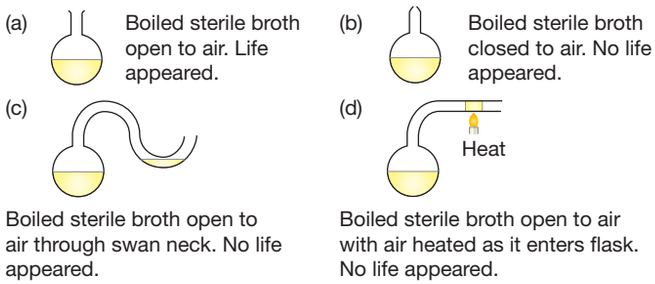

Figure 87.4 Louis Pasteur's flasks of broth.

19. Which theory was disproved by this experiment?
    (A) Spontaneous generation.
    (B) Microbes caused disease.
    (C) Life forms are made up of cells.
    (D) Decay is caused by micro-organisms.
20. If the swan neck is broken off the flask, what would you would expect to happen to the experimental broth?
    (A) Remain sterile.
    (B) Decay due to microbes from the air reaching the broth.
    (C) Decay due to microbes already present in the broth now multiplying.
    (D) Decay only when the contents of the swan neck are deposited into the flask.
21. In Pasteur's experiment, the broth in the flask with the swan neck did not decay because:
    (A) Boiling the broth in step one prevents decay.
    (B) The temperature changes along the length of the swan neck kill bacteria.
    (C) Bacteria were trapped in the swan neck.
    (D) The swan neck prevents oxygen reaching the broth.
22. In 1882 Louis Pasteur carried out a public test to show the benefit of vaccination. What did he use in this public test?
    (A) Dogs with cholera. (B) Dogs with anthrax.
    (C) Sheep with cholera. (D) Sheep with anthrax.

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88 Robert Koch and Pathogens

A **pathogen** is an organism, virus, viroid or prion that causes disease. Robert Koch made significant contributions to scientific understanding making people aware of micro-organisms as causative agents in disease. He was a pioneering bacteriologist and established the rules of procedure for proving a particular micro-organism is the cause of a particular disease.

Robert Koch

Robert Koch was a German physician who took his medical degree in 1866 and served as a surgeon in the Prussian army. He became a country physician in Wollstein where many cattle and sheep died of anthrax. Anthrax has the characteristics of boils and swelling of the spleen and oedema, and death occurs quickly. Learning of Pasteur's theory that micro-organisms caused disease, he investigated, and in 1876 discovered the anthrax bacillus. He isolated these bacteria from other bacteria present and injected them into healthy mice. The mice developed anthrax similar to the sick sheep. Koch's work was supported by Pasteur, and his study methods were so successful he was invited to the University of Breslau and then to Berlin.

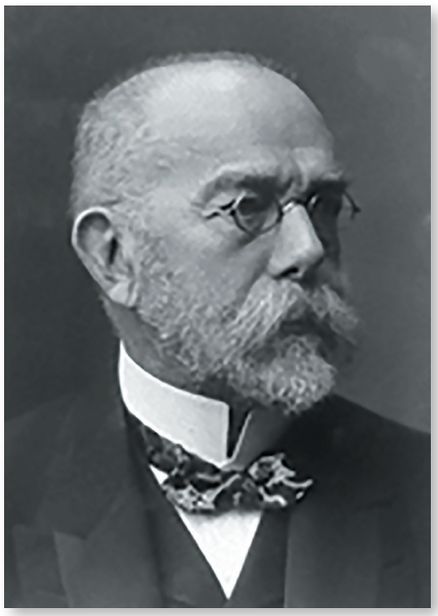

Figure 88.1 Robert Koch (1843-1910).

In 1877 Koch described techniques of fixing, staining and photographing bacteria. He used dyes to stain bacteria and began using solid gels such as gelatin instead of sticky liquid mediums. The bacteria formed colonies on the agar and were easier to isolate.

Julius Richard Petri (1852-1921) invented a special glass dish with lid to hold the agar media. The gelatin was replaced with agar.

In 1884, Koch made a list of criteria needed to prove that a particular organism causes a particular disease. These are known as **Koch's postulates**.

In 1891, Koch was appointed Director of the Institute of Infectious Diseases in Berlin and by 1892 he and his pupils had found the agents causing diphtheria, typhoid, tetanus and tuberculosis.

Koch pioneered bacteriology and was awarded the Nobel Prize for Medicine in 1905 for developing tuberculin to test for tuberculosis.

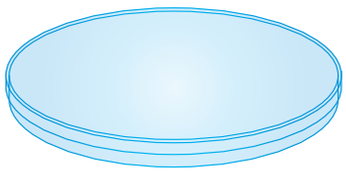

Figure 88.2 Petri dish invented by Julius Richard Petri.

Koch's postulates

1. The organism believed to be the cause of the disease must always be present when the disease occurs.
2. The organism must be isolated from the host and grown in pure culture.
3. Organisms from the pure culture, when inoculated into healthy, suitable, susceptible hosts must produce the disease.
4. The organism must be reisolated, grown in pure culture and compared with the organism first injected.

This basic bacteriological technique is still used today to identify the pathogen of an infectious disease.

QUESTIONS

1. What is anthrax?
2. Describe Robert Koch's contribution to our understanding of anthrax.
3. Outline some of the techniques developed by Koch to study micro-organisms.
4. How did Julius Richard Petri assist bacteriology?
5. Outline Koch's postulates.
6. How was Koch rewarded for his work?
7. Explain how Koch's postulates can be used to identify the causative organism of an infectious disease.
8. No one has been yet able to culture on artificial media the bacteria (*Treponema pallidum*) that causes syphilis.
   (a) How could they decide that *Treponema pallidum* causes syphilis?
   (b) Does this mean that Koch's postulates are invalid?
9. Describe the circumstances that led to a named micro-organism being identified as the causal agent of a particular disease.

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VIVAS (Vaccination Information and Vaccination Administration System) is administered by Queensland Health and details are available on the Queensland government website about vaccination records, distribution and use of vaccines.

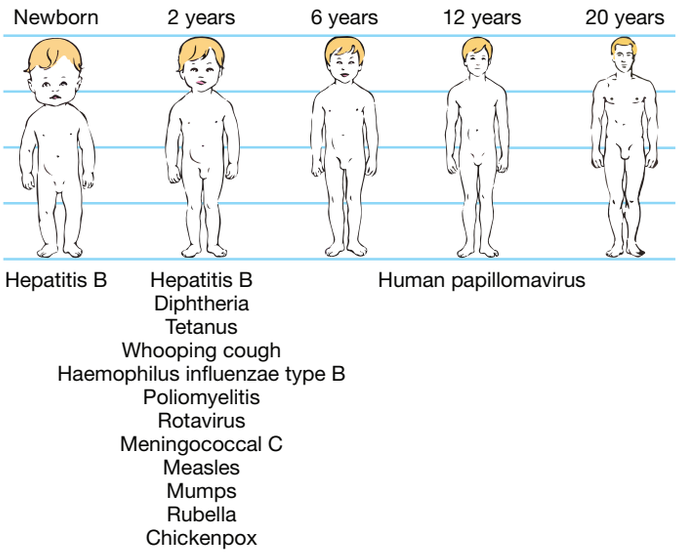

Figure 94.2 Beginning of immunity due to vaccinations.

Disease incidence

The **Australian government** regularly carries out **national health surveys** and estimates the **burden of disease** for the nation and different states and territories. These statistics are available on the government website.

Immunisation rates are also provided by the Australian government on their website showing rates for individual diseases, e.g. polio, DTP (diphtheria, tetanus and pertussis (whooping cough)) and varicella (chickenpox) as well as the rates of people fully immunised. These rates are also divided into cohorts, e.g. 12 to 15 months, 24 to 27 months, 60 to 63 months.

The **World Health Organisation** (WHO) provides data concerning the incidence, prevalence and mortality due to diseases for all regions of the world as well as rates of immunisation with data available on their website.

The collected data shows that there has been a significant decrease in the number of deaths due to infectious diseases since the introduction of childhood vaccinations. The data also shows that when there are drops in immunisation levels against particular diseases, e.g. measles and whooping cough, outbreaks and epidemics of these diseases occur.

QUESTIONS

1. What is meant by the population at risk in an epidemiological study?
2. Define disease incidence.
3. What is meant by disease prevalence?
4. List some factors that can affect the health of a person and thus affect the functioning of their immune system.
5. Why do researchers need to know about the persistence of a pathogen in a host?
6. Distinguish between transmission by direct and indirect contact.
7. Distinguish between vertical transmission and horizontal transmission of a pathogen.
8. Use an example to show how mobility of individuals affects disease incidence in a population.
9. What is involved in immunisation?
10. (a) What is the Australian Immunisation Register?
    (b) Why is it important to have a national immunisation register?
11. How can a person show their immunisation status?
12. Compare immunisation status requirements for child care, primary school and high school in Queensland.
13. Where can you find data about immunisation rates and disease incidence?
14. What has data shown about the relationship between vaccination and disease incidence?
15. Discuss how age can affect the functioning of the immune system and level of immunity.
16. How do vaccinations prevent disease?
    (A) They stimulate T cells to produce antigens.
    (B) They stimulate B cells to produce antibodies.
    (C) They stimulate infected cells to seal off the pathogen.
    (D) They increase primary barriers preventing pathogen entry.
17. Which process is responsible for the long term success of vaccination for an individual?
    (A) Production of memory cells.
    (B) Regular production of antigens by host.
    (C) Establishment of passive acquired immunity.
    (D) Establishment of more effective physical barriers.
18. The table shows the features of two pathogens.

Table 94.1 Two pathogens.

Feature	Pathogen X	Pathogen Y
Living conditions	Can survive outside a host	Must have a host to survive
Reproduction	Can reproduce without a host	Uses host genetic material and organelles to reproduce
Susceptibility to antibiotics	Can be killed or inhibited by antibiotics	Cannot be killed by antibiotics.

From this data you can deduce that pathogens X and Y are respectively:
(A) Fungus and bacteria. (B) Virus and bacteria.
(C) Bacteria and virus. (D) Virus and fungus.

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