Preface

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Team Slim Academy

P.S. This summary has been written based on the author's own interpretation. It remains a summary and should be seen as a supplement to the required study materials — not a replacement

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Adaptive Immune System and Viruses

ILOs (directly sourced from the course manual by the University of Manchester)

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2. Delineate the functional anatomy of the immune system (lymph nodes, spleen, thymus, and bone marrow)*
3. Describe the origin of B and T cells
4. Explain the activation of B and T cells in the adaptive immune response
5. Define the role of B and T-cells in producing specific antibodies
6. Use labelled diagrams to demonstrate the structure of antibodies and how they interact with antigens
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15. Apply the biopsychosocial model to sexual health, including diagnosis and management of sexual health problems
16. Discuss the GMC guidance document 'Confidentiality: good practice in handling patient information'
17. Describe the ways in which GP surgeries and hospital settings can work to maintain patient confidentiality
18. Discuss the duty to balance the rights of the individual with the need to prevent harm to others
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20. Give examples of circumstances where it might be necessary to breach a patient's confidentiality

*ILO 2 will be covered in a separate anatomy booklet

Chapter 1 - Role of the innate immune system in infection

Introduction

This chapter discusses the inflammatory response, key cell types in early infection.
The innate immune system responds immediately upon pathogen entry, typically within the first 12 hours. In contrast to the adaptive immune system, which provides specific, long-lasting responses and immunological memory, the innate immune system is rapid, broad, and non-specific. It employs a combination of physical barriers, chemical defenses, and various specialized cells and molecules to contain and control infections.

Inflammatory Response
Upon tissue injury or infection, the innate immune system initiates an inflammatory response. This process increases blood flow, recruits immune cells to the affected site, and results in localized heat and swelling. These physiological changes facilitate containment of the infection and enhance the effectiveness of immune cell activity.

Key Cell Types in Early Infection

- Macrophages reside in tissues, phagocytose pathogens, and secrete cytokines such as TNF-α, IL-1, and IL-6 to recruit additional immune cells. They play roles in both acute and chronic immune responses;
- Neutrophils are the most prevalent leukocytes and are rapidly recruited to infection sites, where they engulf pathogens, create reactive oxygen species, and release proteolytic enzymes. Neutrophils also form neutrophil extracellular traps (NETs) to immobilize microorganisms;
- Dendritic Cells capture antigens and present them to T lymphocytes, to help bridge innate and adaptive immunity. They secrete IL-12 to promote Th1 differentiation and regulate autoimmunity;
- Natural Killer (NK) Cells identify and eliminate virus-infected or malignant cells through cytotoxic mechanisms;
- Mast Cells release histamine and cytokines such as IL-4 and IL-5, which amplify inflammation and recruit additional immune effectors;
- Eosinophils specialize in defense against certain pathogens, releasing cytotoxic proteins such as major basic protein (MBP) and eosinophil cationic protein (ECP), thereby modulating tissue responses and immunity;
- Basophils contribute to allergic responses and augment inflammatory processes.

Cytokines and Pyrokines
Cytokines are critical mediators of immune communication, orchestrating inflammation, hematopoiesis, and coordination of immune responses. Key examples include:

- Tumor Necrosis Factor-alpha (TNF-α): Induces inflammation and recruits immune cells;
- Interleukins (ILs): Activate lymphocytes, induce fever, and regulate immune responses;
- Interferons (IFNs): Inhibit viral replication and activate NK cells;
- Pyrokines, such as IL-1β and IL-18, are specific cytokines that induce fever, thereby contributing to the host defense against infection.

Additional Innate Immune Components
Complement System: This cascade of proteins opsonizes pathogens, enhances antibody function, and mediates direct lysis of microbial membranes.

Pattern Recognition Receptors (PRRs): Receptors such as Toll-like receptors (TLRs) detect pathogen-associated molecular patterns (PAMPs) or damage-associated molecular patterns (DAMPs), initiating prompt innate immune responses.

Slim Summary!

- Physical, chemical, and biological barriers prevent pathogen entry;
- Inflammation recruits immune cells and enhances pathogen clearance;
- Key cells include macrophages, neutrophils, dendritic cells, NK cells, mast cells, eosinophils, and basophils;
- Cytokines and pyrokines coordinate immune responses and induce fever;
- Complement proteins and pattern recognition receptors detect and eliminate pathogens.

Chapter 2 - Anatomy of the lymphatic system

This will be covered in a separate anatomy booklet.

Chapter 3 - Origin of B and T cells

Introduction

This chapter discusses the origin and development of B and T cells. Their development shapes their roles in adaptive immunity and ensures their specific and regulated responses in adaptive immunity.

Origin and Development of T-Cells

T-cells originate from hematopoietic stem cells in the bone marrow, and they mature within the thymus, which is an organ located in the anterior mediastinum. Immature T cell precursors called thymocytes migrate from the bone marrow to the cortex of the thymus, and they express both CD4 and CD8 surface markers so they are called double-positive which allows them to interact with MHC molecules later in their development. At the same time, these DP thymocytes have a newly rearranged T-cell receptor (TCR). This TCR is produced through VDJ recombination, a process that randomly mixes variable (V), diversity (D), and joining (J) gene segments. This allows the T cell to recognize a wide variety of antigens presented by MHC molecules. After immature T-cells arrive in the thymus cortex as double positive cells, they undergo positive selection, this ensures that T cells are able to recognize the body's own MHC molecules so it is able to function properly in the immune system. Thymocytes that do not recognise self-MHC will undergo apoptosis as they will not be able to identify antigens that invade the body, making them functionally useless. If a thymocyte's CD8 receptor binds MHC class I, the CD4 receptor is downregulated and the cell commits to becoming a cytotoxic ($CD8^+$) T-cell committed to killing infected cells if they see antigens presented by MHC Class I. If its CD4 receptor binds MHC class II, the CD8 receptor is downregulated and it becomes a helper ($CD4^+$) T-cell only help other immune cells if they see antigens presented by MHC class II.

Thymocytes that survive positive selection in the thymic cortex migrate toward the corticomedullary junction, where they undergo negative selection. During this, thymocytes that bind too strongly to self-antigens on MHC Class 1 or Class 2 by bone marrow antigen presenting cells are eliminated. This process prevents autoimmunity by deleting thymocytes with high affinity for self-antigens. Only about 2% of all developing thymocytes survive both positive and negative selection. The surviving mature but naïve T-cells then exit the thymus and circulate to lymph nodes, where they remain until activated by antigen-presenting dendritic cells.

Once activated, T-cells differentiate into specialised effector T-cells guided by cytokine signals. $CD8^+$ T-cells differentiate into cytotoxic T-cells activated by IL-2 secreted by activated $CD4^+$ Th1 cells. They **kill infected cells** by releasing perforins (create holes in the target cell membrane), granzymes (trigger apoptosis), and granulysin (antimicrobial). Effector cytotoxic T-cells produce additional IL-2, engaging the JAK/STAT pathway, which drives clonal expansion and full differentiation. These cells leave the lymph node and target infected cells displaying antigen on MHC class I.

$CD4^+$ helper T-cells also differentiate into specialized subsets based on cytokine exposure. Th1 cells form in response to IL-12 during viral or intracellular infections; they secrete IFN-γ and IL-2, which activate macrophages and promote cytotoxic T-cell responses. IL-23 further enhances cytotoxic T-cell production and Th1 expansion, although IL-10 can suppress excessive Th1 activity. Th2 cells, promoted by IL-4, support B-cell proliferation and antibody production, while

IL-10 helps suppress Th1 differentiation. Th17 cells produce IL-17, which recruits neutrophils to infection sites, and IL-22, which stimulates epithelial cells to release antimicrobial peptides. Regulatory T-cells (T-regs), which arise naturally in the thymus or are induced at mucosal surfaces, function to limit tissue damage and prevent autoimmunity through secretion of IL-10 and TGF-β once pathogens are cleared.

Origin and Development of B cells

B-cells originate from lymphoid progenitor cells in the bone marrow, where they undergo maturation before migrating to secondary lymphoid organs, including lymph nodes, Peyer's patches, and the spleen. Pre-B cells remain in the bone marrow to complete their maturation. Once B-cells finish developing, they enter the circulation as mature, antigen-sensitive B-lymphocytes, continuously going between the bloodstream, lymph nodes, spleen, and other lymphoid tissues in search of antigen. Mature B-cells express characteristic surface markers, including CD19, CD20, CD21, and carry IgM and IgD as their membrane-bound immunoglobulin receptors. They also express MHC class II, B7, and CD40, enabling them to act as antigen-presenting cells for T-helper cells. Upon encountering antigen and receiving appropriate signals—particularly from T-helper cells via CD40–CD40L interaction and cytokines such as IL-4 and IL-5—B-cells undergo activation, proliferation, and differentiation into antibody-secreting plasma cells.

An important feature in B cell development is that they generate a vast antibody diversity to be able to recognize a variety of potential pathogens through VDJ recombination. During this process, the Variable (V), Diversity (D), and Joining (J) gene segments of the immunoglobulin undergo rearrangement to create unique antigen-binding sites. This recombination begins when double-stranded DNA breaks occur at recombination signal sequences (RSS) flanking each V, D, and J region. The enzymes RAG-1 and RAG-2 recognize these recombinant signal sequences and start cutting DNA at those sites, allowing countless possible combinations that generate various unique B-cell receptors (BCRs). This mechanism ensures that the immune system can recognize an enormous range of potential pathogens.

Because VDJ recombination is random, immature B-cells often generate receptors that bind to self-antigens that could cause auto-immunity. To maintain self-tolerance, developing B-cells undergo negative selection in the bone marrow. If a B-cell binds strongly to self-antigen, it may undergo receptor editing, to eliminate self-reactivity. If editing fails, the cell may undergo clonal deletion, in which apoptosis removes the potentially harmful lymphocyte. Alternatively, some self-reactive cells become anergic, remaining alive but nonfunctional until they eventually die in peripheral tissues. B-cells that do not react to self-antigen in the bone marrow, or that successfully edit their receptors, exit into the periphery as mature, naïve B-cells ready to respond to foreign antigens.

When mature B cells encounter antigen in secondary lymphoid organs such as lymph nodes, Peyer's patches, and the spleen, they receive help from CD4 T helper cells, typically via CD40-CD40L interactions and cytokines like IL-4 and IL-5. This triggers activation, proliferation, and differentiation into plasma cells, which secrete antibodies, or memory B-cells for long-term immunity.

After activation in secondary lymphoid tissues, B-cells undergo somatic hypermutation within germinal centers, a process that improves antigen-binding affinity. AID (activation-induced

deaminase) introduces mutations in the variable region of the antibody gene. B-cells with higher-affinity antibodies are selected for survival. Class-switch recombination (CSR), also driven by AID, changes the antibody isotype→ IFN-γ promotes switching to IgG, IL-4 to IgE, and TGF-β to IgA, especially in mucosal tissues. During the primary immune response, B-cells initially produce IgM, with IgG appearing later through somatic hypermutation. In the secondary immune response, memory B-cells rapidly produce large amounts of IgG within 1–3 days, compared to the slower 4–7-day primary response.

Slim Summary!

- B-cells mature in the bone marrow while T-cells mature in the thymus, and both undergo receptor formation and selection to ensure self-tolerance and antigen specificity.
- After maturation, both cell types circulate to secondary lymphoid organs, where they can be activated to produce effective immune responses and long-lasting memory.

Chapter 4 - Activation of B and T cells

Introduction

T and B cells are central to the adaptive immune response, with T-cells eliminating infected cells and directing immune responses, while B-cells produce antibodies to neutralize extracellular pathogens. Activation of these cells requires antigen recognition, co-stimulation, and cytokine signaling, leading to clonal expansion, differentiation, and formation of memory cells for long-term immunity.

The adaptive immune system is characterized by specificity, memory, and discrimination between self and non-self. Unlike the innate immune response, which reacts immediately, adaptive immunity becomes active within 4–96 hours after infection and relies primarily on T and B lymphocytes. These cells respond to particular antigens and generate long-term immunity through memory cell formation.

T-Cell Activation in the Adaptive Response

T-cell activation begins when antigen-presenting cells (APCs) which are mainly dendritic cells capture pathogens and break down the pathogen and display antigen fragments on their surface using MHC molecules. Antigens presented on MHC class I activate $CD8^+$ cytotoxic T-cells, while antigens on MHC class II activate $CD4^+$ helper T-cells. Then the T cell receptor binds to and recognises the antigen-MHC complex. This recognition alone is insufficient, T-cells require a second signal known as co-stimulation which acts like a safety check to prevent accidental activation. APCs express the molecules CD80 and CD86, which bind to CD28 on T-cells. This triggers the activation of T-cells by triggering intracellular signaling pathways such as the PI3K/AKT pathway. This promotes rapid T-cell division from a naive state into its effector form. Cytokines then decide what type of helper T cell as they have different supportive actions. Through activation, CD8 T cells start killing infected cells.

Once activated, T-cells undergo differentiation shaped by cytokines released by APCs. For example, IL-12 drives differentiation into Th1 cells, which enhance macrophage activity and support $CD8^+$ T-cell responses. IL-4 promotes Th2 differentiation, which supports B-cell activation and humoral immunity. IL-6 and TGF-β together promote Th17 differentiation, producing IL-17 and IL-22 to recruit neutrophils and strengthen epithelial defenses. TGF-β alone favors the generation of regulatory T-cells, which limit immune responses. Simultaneously, IL-2, produced by activated T-cells, drives clonal expansion, allowing the immune system to generate large populations of antigen-specific T-cells. $CD8^+$ cytotoxic T-cells, once fully activated, become capable of killing infected cells by releasing perforin(make holes) and granzymes(cause cell death).

B-Cell Activation in the Adaptive Response

The humoral branch of adaptive immunity relies on antibodies produced by B-cells to eliminate pathogens outside of the cell, such as bacteria and viruses circulating in bodily fluids. In contrast, T-cells kill intracellular pathogens. Antibodies neutralize pathogens by blocking their ability to infect host cells, binding and inactivating toxins, facilitating opsonization (where antibodies mark

pathogens for phagocytosis), activating the complement system, and promoting agglutination that clusters pathogens for easier clearance.

B-cell activation typically depends on Th2 helper cells. Once a B-cell binds antigen through its membrane-bound antibody, it internalizes the antigen, processes it, and presents it on MHC class II. Activated Th2 cells recognize this antigen–MHC II complex through their T cell receptor. For full activation, the Th2 cell provides a co-stimulatory interaction, as it offers the molecule CD40L which binds to CD40 on the B-cell. Th2 cytokines, IL-4, IL-5, and IL-13 drive B-cell proliferation, differentiation, and class switching, enabling the production of different antibody isotypes. B-cells initially produce IgM, then switch to other classes such as IgG, IgA, or IgE, depending on the immune requirements.

Within secondary lymphoid organs, activated B-cells enter germinal centers, specialized structures where affinity maturation occurs. During this process, somatic hypermutation improves antibody binding, affinity selection picks the best cells, and class switching changes the antibody type B-cells producing higher-affinity antibodies are selected for survival, while low-affinity cells undergo apoptosis. Activated B-cells either become plasma cells, which secrete large quantities of high-affinity antibodies, or memory B-cells, which provide lifelong immunity and provide rapid protection from future infections.

Slim Summary!

- T-cell activation involves antigen recognition via MHC, co-stimulation, and cytokine-driven differentiation into effector types like Th1, Th2, Th17, regulatory T-cells, or cytotoxic $CD8^+$ cells.
- B-cell activation depends on Th2 helper signals, antigen binding, class switching, and germinal center processes, resulting in plasma cells that secrete antibodies and memory B-cells for long-lasting immunity.

Chapter 5 - Role of B and T cells in producing specific antibodies

Introduction

This chapter discusses production of specific antibodies during an adaptive immune response is a coordinated and highly regulated process involving both B-cells and T-cells. These two immune cells work together to ensure that antibodies that are produced have a high affinity for antigens and are suited to the type of pathogen entering the body. This process not only clears active infections but also provides immune memory for rapid defence against future exposures.

Role of B-Cells in Producing Specific Antibodies

Plasma Cells

Activated B-cells differentiate into plasma cells, which are the primary factories for antibody production. B-cells undergo structural changes by expanding their endoplasmic reticulum, ribosomes, and Golgi apparatus to support the enormous amount of protein synthesis required for antibody secretion. Plasma cells initially produce IgM, the first antibody released during a primary immune response. IgM is a large pentameric molecule, allowing it to bind strongly to pathogens even before affinity maturation occurs; it is especially effective at activating the complement system, giving an early defence while the immune response develops.

As the immune response continues, T-cell signals stimulate B-cells to undergo class switching, enabling the production of other antibody classes such as IgG, IgA, and IgE. Each of these antibodies plays a specialised role. IgG is the most abundant antibody in the bloodstream, it neutralises viruses and toxins, opsonises pathogens for phagocytosis, activates complement, and can even cross the placenta to protect newborns. IgA is the dominant antibody at mucosal surfaces such as the respiratory, digestive, and reproductive tracts. Often produced as a dimer with a protective secretory component, IgA prevents pathogens from attaching to and penetrating epithelial barriers, giving frontline defence in saliva, tears, breast milk, and mucus. IgE is produced in smaller amounts and binds tightly to mast cells and basophils. It plays a central role in fighting parasitic worms and helps recruit eosinophils; however, when triggered inappropriately, it can lead to allergic responses due to histamine release. Thro nbugh these antibody classes, plasma cells ensure that the body has a rapid and versatile supply of antigen-specific molecules capable of neutralising pathogens, marking them for destruction, or preventing their spread.

Memory B-Cells

Once an infection has been cleared, a portion of the activated B-cells become memory B-cells. These cells express markers such as CD27, CD19, CD20, and often switched antibody types like IgG or IgA. During their formation, they undergo somatic hypermutation, refining their antigen-binding sites so that they possess greater affinity for the pathogen. Memory B-cells persist in the body for many years, sometimes for life, and are primed to respond extremely rapidly upon re-infection. Because they require less activation and can quickly proliferate and differentiate into plasma cells, they allow the body to mount a secondary immune response that is much faster, stronger, and more efficient than the primary one. This is the basis for long-term immunity after natural infection or vaccination.

Role of T-Cells in Helping Produce Specific Antibodies

T-cells, especially $CD4^+$ helper T-cells, play an essential supportive role in enabling B-cells to produce high-affinity, class-switched antibodies. Without T-cell help, B-cells would mostly produce IgM and would not undergo class switching, meaning the immune response would be significantly weaker and less specific.

Th1 Helper T-Cells

Th1 cells differentiate in response to IL-12 released by dendritic cells and macrophages. They produce cytokines such as IFN-γ, which activate macrophages and promote B-cell class switching towards IgG subclasses that are particularly good at opsonisation and complement activation. This makes Th1 responses crucial for immunity against intracellular pathogens such as viruses and certain bacteria, where high-affinity IgG is necessary for clearing infection and enhancing phagocytic killing.

Th2 Helper T-Cells

Th2 cells develop when IL-4 is present, and they support B-cells through cytokines such as IL-4, IL-5, and IL-13. These signals encourage B-cell proliferation, plasma cell differentiation, and class switching towards antibody types like IgE and, in some mucosal environments, IgA. The CD40L–CD40 interaction between Th2 cells and B-cells is especially important for enabling class switching and affinity maturation. Th2-driven antibody responses are essential for defending against parasitic infections, but they also play a central role in allergic conditions due to their promotion of IgE production.

Th17 Helper T-Cells

Th17 cells arise in the presence of IL-6, IL-23, and TGF-β, and although they do not directly stimulate antibody production, they create inflammatory environments that support effective immune responses. By producing IL-17, Th17 cells recruit neutrophils to infection sites and strengthen epithelial barriers through the induction of antimicrobial peptides. This process indirectly enhances antibody effectiveness, particularly during fungal infections and extracellular bacterial infections where strong barrier defence is essential.

Regulatory T-Cells (Tregs)

Regulatory T-cells develop when TGF-β and IL-2 are present and are characterised by the transcription factor FoxP3. They help maintain immune balance by releasing IL-10 and TGF-β, which suppress excessive activation of both B-cells and T-cells. By down-regulating overly aggressive immune responses, Tregs prevent tissue damage and help avoid autoimmune conditions in which antibody production could become misdirected toward the body's own tissues.

Cytotoxic T-Cells ($CD8^+$ T-Cells)

Although $CD8^+$ cytotoxic T-cells do not directly contribute to B-cell activation, they play an important supporting role in clearing infections. They recognise and destroy virus-infected cells displaying antigen on MHC class I molecules, limiting the spread of intracellular pathogens.

Additionally, they produce IFN-γ, which can indirectly enhance some IgG responses. By controlling intracellular pathogens, cytotoxic T-cells allow antibodies to effectively target extracellular stages of infection.

Memory T-Cells

Memory T-cells, including both $CD4^+$ and $CD8^+$ subsets, persist after infection and respond rapidly upon re-exposure to the same antigen. They require less co-stimulation for activation and can quickly produce cytokines that support memory B-cell activation. This rapid signalling helps accelerate the production of high-affinity antibodies, ensuring that the secondary immune response is faster, more powerful, and more specific than the primary response.

Slim Summary!

- B-cells produce specific antibodies through differentiation into plasma cells, which secrete IgM initially and later class-switched antibodies (IgG, IgA, IgE), while memory B-cells ensure rapid and high-affinity secondary responses upon re-infection.
- T-cells, particularly $CD4^+$ helper subsets (Th1, Th2, Th17, and Tregs), provide essential support for B-cell activation, class switching, and regulation, while $CD8^+$ cytotoxic and memory T-cells help control infections and enhance antibody effectiveness indirectly.

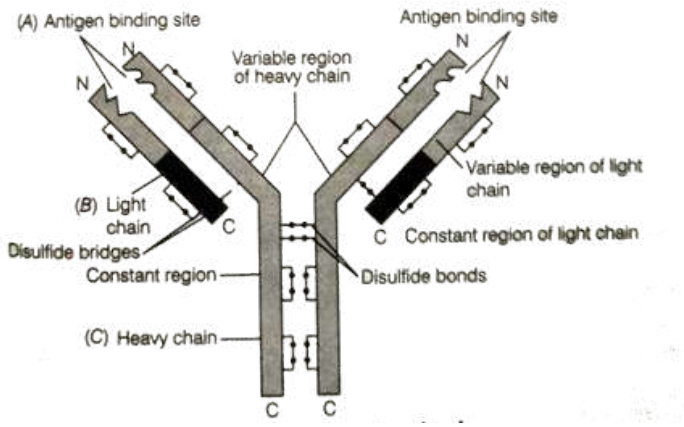
Chapter 6 - Structure of antibodies

Introduction

This chapter describes the structure of antibodies and how they interact with antigens. Antibodies, also known as immunoglobulins, are specifically shaped proteins produced by plasma B cells.

Structure of an antibody

An antibody is formed of two heavy chains and two light chains arranged in a Y-like configuration. The chains are joined together by disulphide bonds and bridges. These disulphide bonds are found in the hinge region, joining together the heavy chains, and they also found joining the light chains to the heavy chains. There is a constant region, which does not vary in structure between antibodies, that is found towards the tail end of each chain, and a variable region. The variable region is found towards the upper end of the chains where the antigen-binding site is. This region varies between antibodies, so that they can bind specifically to the different antigens of different pathogens.



Structure of an antibody. *Source: storage.googleapis.com*

Main antibody classes

There are different classes of antibody which have slightly different structures and features for specific functions. The different classes have different arrangements of their heavy chains.

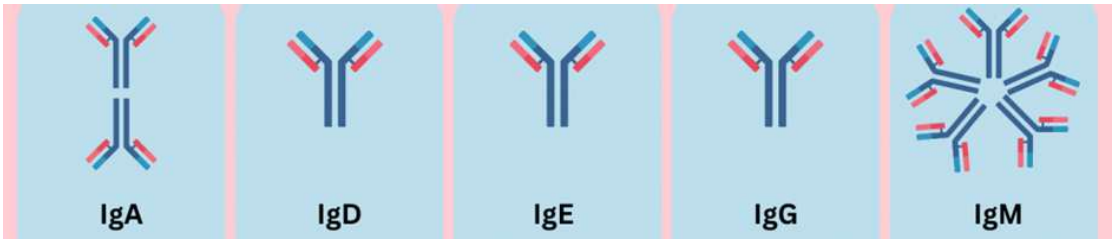
IgG antibodies have the typical Y-like antibody structure, and they dominate the secondary immune response. They are found in the blood and tissue and are also able to be transferred across the placenta during pregnancy to the fetus. Their functions include fixing complement proteins, binding to phagocytes, and neutralising toxins.

IgM antibodies are made up of five antibody molecules joined together at their tails, allowing for ten binding sites. This means that these antibodies have a high avidity as they can bind to several antigens at once and they dominate the primary immune response. They are found in the blood and, like IgG antibodies are also involved in fixing complement. They are also found on B cells as they can be used as receptors.

IgA antibodies are formed of two antibodies joined at their tails. They are secreted in mucus, saliva and tears, and therefore allow protection at mucosal surfaces.

IgE antibodies have extended heavy chains to form a longer constant region. These antibodies bind to mast cells and basophils and are therefore involved in allergic reactions. They also play an important role in defending against parasitic infections.

IgD antibodies have a typical antibody structure and are found on B cells as receptors. They also activate IgM antibodies to be released.

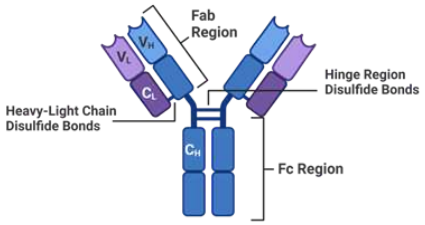


Classes of antibodies. *Source: sciencenotes.org*

Interaction with antigens

As mentioned before, antibodies have antigen-binding sites at the tips of the Y-like structure. These are contained within the Fragment Antigen Binding (Fab) regions. These regions have a specific complementary structure to the antigen of a specific pathogen, so that they can bind to the corresponding antigen. Antigens are usually large proteins on the surface of pathogens, but they can also be carbohydrates and lipids.

As well as the Fab regions, antibodies have a Fragment crystallisable (Fc) region at their tail end. This Fc region allows the antibody's effector functions to be initiated, such as opsonisation and complement activation, which will be explained in the next chapter.



Regions of an antibody. *Source: blog.addgene.org*

Slim Summary!

- Antibodies have Fab regions with antigen binding sites and Fc regions which allow its effector functions;
- There are five main classes on antibody, each with different structural arrangements and functions

Chapter 7 - Role of antibodies against infection

Introduction

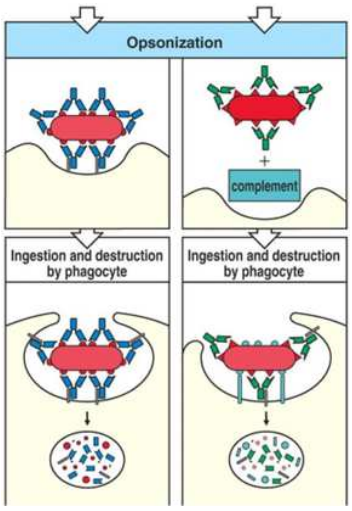
This chapter describes how antibodies protect against microbiological infection by facilitating processes such as phagocytosis and complement activation.

Blocking of binding

Antibodies can bind to toxins released by pathogens as well as their antigens. Binding to toxins neutralises them as they can no longer bind to receptors on body cells. They also cause agglutination of pathogens, which is when antibodies bind to both pathogen antigens via the Fab regions and other antibodies at the same time via the Fc regions. This causes a clump of pathogens to form and prevents the pathogens from moving to body cells and binding to them.

Opsonisation and phagocytosis

Opsonisation is when antibodies coat a pathogen. This marks it for phagocytosis because a series of complement proteins (C1, C4, C2, C3b) can bind to the Fc domain of the antibody. The phagocytes have receptors for complement proteins such as C3b and can consequently bind to the attached proteins on the pathogen. This all occurs in the **classical pathway** of complement activation. Phagocytes can also recognise and bind directly to the Fc domain of antibodies. Therefore, overall, the binding of antibodies attracts a large number of phagocytes, such as neutrophils and macrophages, to bind to the pathogen. Phagocytosis would then be carried out, and the pathogen would be engulfed, digested and destroyed. This process is particularly important for immunity against encapsulated bacteria.



Opsonisation and phagocytosis. *Source: blogspot.com*

Slim Summary!

- Antibodies can bind to toxins, antigens and also complement proteins to facilitate processes such as opsonisation and phagocytosis and destroy pathogens

Chapter 8 - Fever

Introduction

This chapter describes the body's responses that result in fever.

Fever or pyrexia is an increase in body temperature which is triggered by resetting the hypothalamic thermostat. When an infection by a pathogen is detected, cytokines are secreted by various immune cells such as macrophages, which induce fever. These are known as **pyrogens**, and they act as messengers to signal the hypothalamus to raise the body's temperature. Examples of pyrogens include IL-1β, IL-6, IL-18, and TNF-α.

The hypothalamus acts like a thermostat for the body and sends signals to body tissues to cause an increase in body temperature. Responses include shivering and increased metabolism to generate heat, and also vasoconstriction to reduce heat loss.

The increase in body temperature makes the body's environment less favourable for bacterial growth and replication and it helps to speed up the immune response by immune cells.

Slim Summary!

- Fever involves resetting the hypothalamic thermostat;
- Fever is triggered by the release of pyrogenic cytokines that signal the hypothalamus to raise the body's temperature.

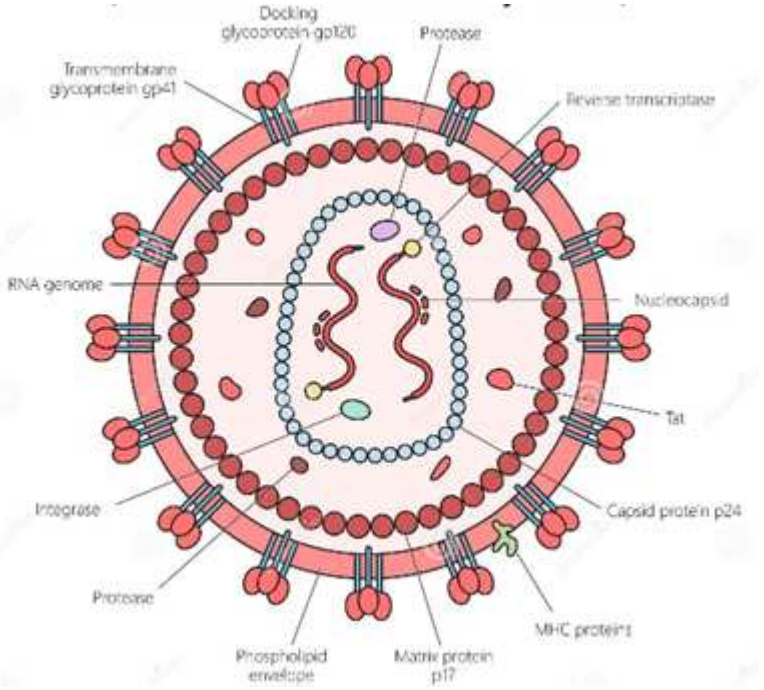
Chapter 9 - Life cycle of a retrovirus

Introduction

This chapter describes the structure and life cycle of a retrovirus and uses HIV as an example. Human immunodeficiency virus is a retrovirus in the family retroviridae. This means that it possesses the enzyme reverse transcriptase which allows single-stranded viral RNA to be transcribed into double-stranded viral DNA. This DNA can be integrated into the host genome to allow the virus to replicate itself using the host cell's machinery. The virus can be transmitted by unprotected sex, pregnancy, infected injection equipment and blood transfusions.

Structure of HIV

HIV has an outer **lipid envelope** with several attachment proteins on the surface, including **gp120** and **gp41**. There is a **matrix** layer underneath this which is made up of **p17** matrix proteins. The inner protein **capsid** holds the virus' genetic information in the form of two strands of single-stranded RNA along with several enzymes including **reverse transcriptase**, p32 integrase and p10 protease. The functions of these enzymes are important in replication of the virus. The capsid is made up of structural proteins including **p24**.

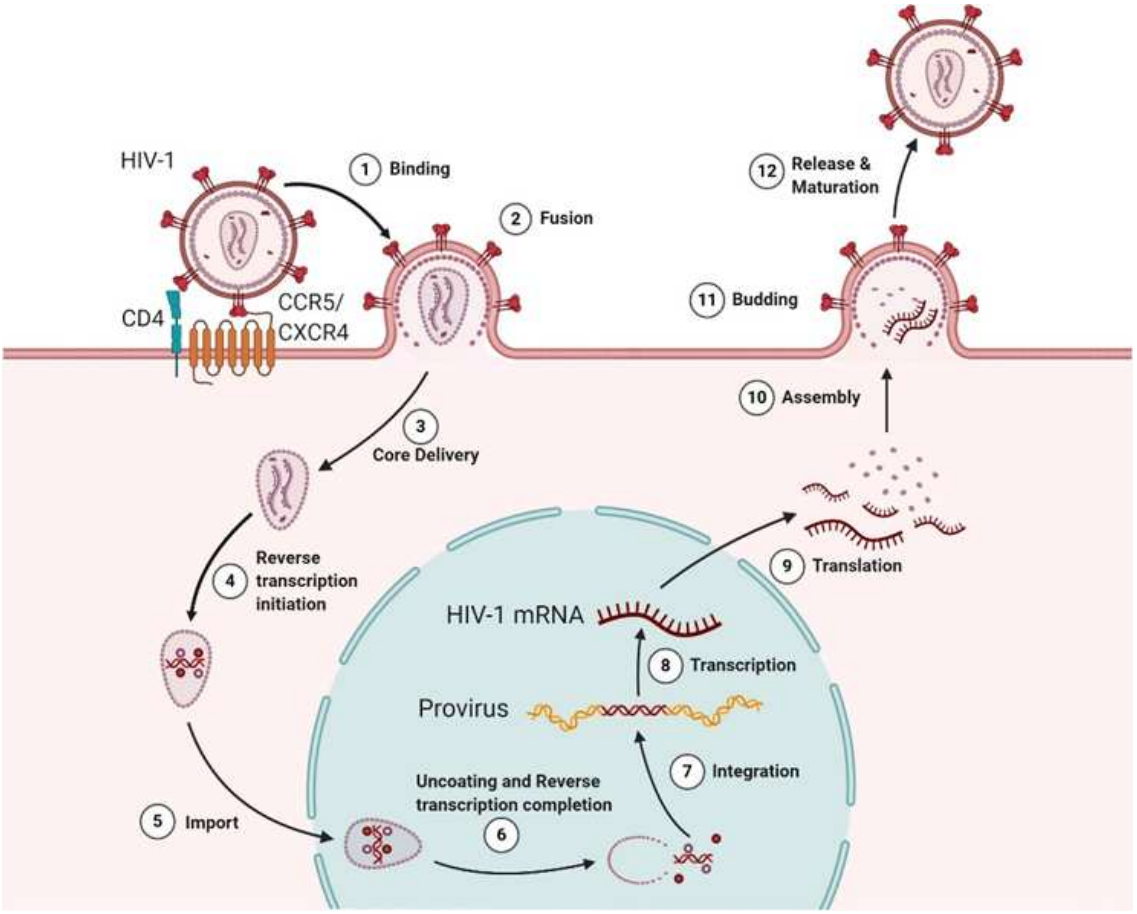


Structure of HIV. *Source: dreamstime.com*

Life Cycle of HIV

HIV begins its infection of a host by attaching to **CD4** receptors on CD4 T-lymphocytes. The **gp120** protein on the lipid envelope specifically binds to the CD4 receptors. The initial binding triggers conformational changes in the gp120 protein, which allow it to bind further to co-receptors such as CCR5 and CXCR4. The viral envelope then fuses with the cell membrane after the binding of the gp41 protein. This allows the viral core, including the RNA and enzymes to be released into the cytoplasm of the cell.

Reverse transcription by the reverse transcriptase enzyme then occurs, which is when the single-stranded viral RNA is converted into double-stranded viral DNA. This viral DNA is then transported across the nucleus and is integrated into the host cell DNA via the action of p32 integrase. The virus can then replicate itself via protein synthesis using the host cell's machinery. The p10 protease enzyme cleaves new polyproteins to create a mature infectious virus, and the virus can then be released and spread to more cells. To leave the cell, it buds from the cell membrane.



Life cycle of HIV. *Source: www.frontiersin.org*

Slim Summary!

- The basic structure of HIV includes a lipid envelope with attachment proteins, a matrix, and a protein capsid enclosing the strands of RNA;
- HIV binds to and uses the cellular machinery of CD4 T cells to complete protein synthesis and replicate itself.

Chapter 10 - Diagnosis and monitoring of HIV

Introduction

This chapter discusses the diagnosis and monitoring of HIV infection rely on a combination of sensitive tests and ongoing evaluation to ensure early detection, effective treatment, and prevention of disease progression. These strategies include screening, confirmatory tests, viral load and CD4 monitoring, resistance testing, and assessment for co-infections.

Screening Tests (Initial Diagnosis)

HIV diagnosis begins with highly sensitive screening tests that detect viral antigens, antibodies, or both. The preferred first-line test is the 4th-generation HIV Ag/Ab combination assay, which detects HIV-1/2 antibodies and the p24 antigen, allowing early detection before antibodies form.

Confirmatory Tests

Reactive screening tests require confirmation to rule out false positives. The HIV-1/2 antibody differentiation immunoassay is the standard confirmatory test, identifying whether the infection is HIV-1 or HIV-2. If results are unclear, an HIV nucleic acid test (NAT) detects viral RNA directly, particularly useful for acute infection or discordant results.

Nucleic Acid Testing (NAT)

HIV RNA PCR measures viral RNA in the blood and can detect infection within 10–12 days post-exposure. NAT is used for early diagnosis, resolving indeterminate tests, detecting infection in newborns, and monitoring viral load during treatment.

$CD4^+$ T-Cell Count (Immune Status Monitoring)

CD4 counts assess immunosuppression and risk for opportunistic infections. Normal ranges are 500–1500 cells/μL, while counts below 200 cells/μL indicate severe immunodeficiency and define AIDS. Regular monitoring tracks immune recovery on antiretroviral therapy (ART).

Viral Load Monitoring (Treatment Response)

Viral load measures HIV RNA and is the key marker for treatment effectiveness. ART should reduce viral load to undetectable levels; rising levels may signal treatment failure, non-adherence, or resistance. Testing is done at diagnosis, 2–8 weeks after starting ART, and every 3–6 months thereafter.

Resistance Testing (Genotypic or Phenotypic)

Resistance tests detect viral mutations that reduce drug susceptibility. Genotypic tests analyze viral sequences for known mutations, while phenotypic tests examine viral behavior in the presence of drugs. These tests guide ART selection and modification, especially after treatment failure.

Additional Monitoring Tests

Routine tests include full blood counts, liver and kidney function, and screening for co-infections like hepatitis B/C, tuberculosis, and other STIs. Optional tests, such as CD8 counts or the CD4/CD8 ratio, provide extra insight into long-term immune recovery. Together, these tests ensure accurate diagnosis, effective treatment, and ongoing monitoring of HIV patients.

Slim Summary!

- HIV diagnosis begins with high-sensitivity screening tests, such as 4th-generation Ag/Ab assays, followed by confirmatory antibody differentiation tests or nucleic acid testing (NAT) to detect early or ambiguous infections.
- Monitoring of HIV-infected patients involves CD4 counts to assess immune status, viral load to evaluate treatment response, resistance testing to guide ART, and additional investigations for drug toxicity and co-infections.

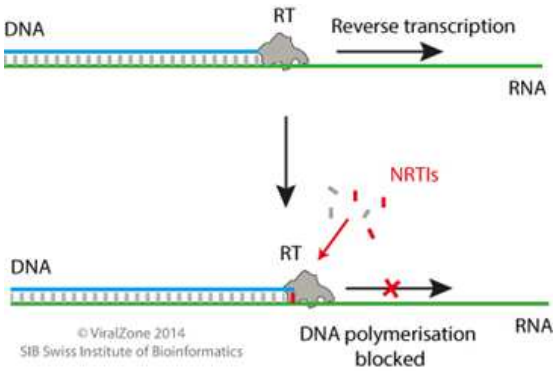
Chapter 11 - Pharmacological treatment of HIV

Introduction

This chapter describes the mechanisms of anti-viral and anti-bacterial drugs used in the treatment of HIV. **Anti-retroviral therapy (ART)** with various drugs is the main treatment for HIV. It is usually lifelong, as HIV can't be fully cured, but it can be very effective at suppressing viral replication and eventually allow the virus to be undetectable and untransmissible.

Nucleoside Reverse Transcriptase Inhibitors (NRTIs)

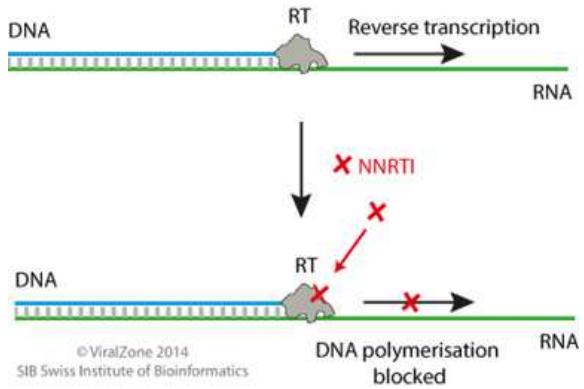
NRTIs are nucleoside analogues of DNA nucleotides. They terminate the chain sequence by binding in the DNA chain, which is being formed by reverse transcriptase. This interrupts the formation of viral DNA and prevents viral replication. This is **competitive inhibition** as the drug molecules mimic normal nucleosides so are able to bind in the chain. Examples of NRTIs include tenofovir, abacavir, emtricitabine and lamivudine.



Mechanism of NRTIs. *Source: ViralZone*

Non-nucleoside Reverse Transcriptase Inhibitors (NNRTIs)

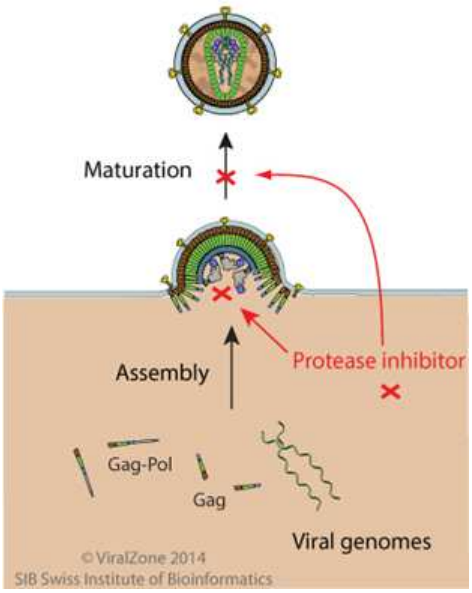
NNRTIs change the structure of the active site of reverse transcriptase, which is **non-competitive inhibition**. This prevents reverse transcriptase from forming the DNA, so reverse transcription is inhibited, and the virus cannot replicate. Examples of these include nevirapine, efavirenz, rilpivirine and etravirine.



Mechanism of NNRTIs. *Source: ViralZone*

Protease inhibitors

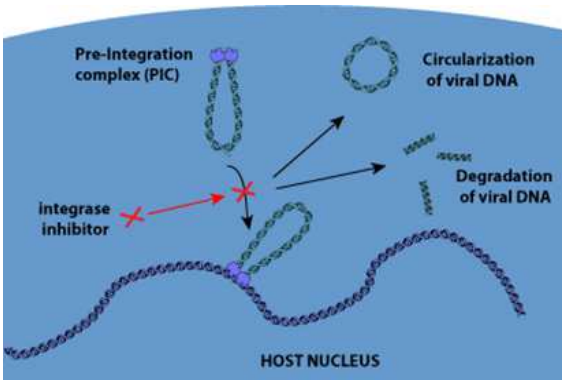
Protease inhibitors bind to the catalytic site of HIV protease enzymes and prevent the cleavage of viral polyprotein into mature functional proteins. This prevents viral proteins from becoming functional and the replication of the virus cannot be completed. Examples of protease inhibitors are darunavir and atazanavir.



Mechanism of protease inhibitors. *Source: ViralZone*

Integrase Strand Transfer Inhibitors (INSTIs)

These drugs block the integration of retroviral DNA into the host DNA by circularising it. They cause the viral DNA strand to attach one of its ends to the other and form a circle, which prevents it from integrating into the host DNA. This means that protein synthesis cannot occur and the virus cannot replicate itself. INSTIs tend to be well-tolerated compared to the other drugs, and they have less drug interactions. Examples of them include raltegravir, dolutegravir, elvitegravir and cabotegravir.



Mechanism of INSTIs. *Source: ViralZone*

Other anti-retroviral drug groups

There are also other drugs which inhibit specific processes in the replication of HIV to prevent it. These include capsid inhibitors, fusion inhibitors, CCR5 inhibitors and attachment/post-attachment inhibitors. These drugs are less commonly used but are still available as treatment options.

Choosing an ART regime

The treatment regime for HIV usually involves combination therapy of multiple drugs that target viral replication. It usually consists of **two NRTIs** and one other drug out of an **NNRTI/protease inhibitor/INSTI**. The doctor and patient should consider lifestyle factors, drug-drug interactions and genetic barriers to resistance when prescribing antiretroviral drugs. HIV can become resistant to certain drugs, and the resistant strains may be transmitted.

Prophylaxis and preventing HIV

For those who may have been recently exposed to the virus and are at high risk of catching it, there is a three-drug regime which can be started within 72 hours of exposure. These drugs are tenofovir, emtricitabine and raltegravir. They are taken for 28 days. Some individuals who are at high risk of catching HIV and are frequently exposed may have pre-exposure prophylaxis, which involves taking a single tablet containing tenofovir and emtricitabine.

Anti-bacterial drugs

Those with poorly treated HIV would be at high risk of contracting opportunistic infections as CD4 count becomes very low. The treatment for these infections involves antibiotics. There are several types of antibiotics which target different bacterial cell processes. These were covered in Chapter 20 of Theme 3. Some antibiotics are more commonly used in HIV, for example trimethoprim-sulfamethoxazole (TMP-SMX) is often used to treat pneumocystis pneumonia.

Slim Summary!

- The main drugs used in ART include NRTIs, NNRTIs, protease inhibitors, and INSTIs;
- Antiretroviral drugs may be given as prophylaxis to those at high risk of exposure.

Chapter 12 - Opportunistic infections with HIV and AIDS

Introduction

This chapter discusses the opportunistic infections such as Pneumocystis pneumonia, tuberculosis, toxoplasmosis, cytomegalovirus, candidiasis, and others found in advanced HIV/AIDS which is caused by profound immunosuppression due to severe $CD4^+$ T-cell depletion. These infections, along with direct HIV-related organ damage, lead to significant morbidity and mortality in untreated or advanced disease.

Opportunistic Infections in Advanced HIV (AIDS)

In advanced HIV infection, severe depletion of $CD4^+$ T cells leads to profound immunosuppression, making individuals highly susceptible to a range of opportunistic infections (OIs). These infections occur when pathogens that are normally harmless take advantage of a weakened immune system, and they collectively contribute significantly to AIDS-related morbidity and mortality. One of the most common OIs is **Pneumocystis pneumonia (PCP)**, caused by *Pneumocystis jirovecii*. PCP produces severe inflammation within the lungs, impairing oxygen diffusion and leading to symptoms such as chronic dry cough, fever, fatigue, hypoxia, and potentially respiratory failure. Another major opportunistic pathogen is *Mycobacterium tuberculosis*. HIV-1 is the strongest known risk factor for developing active tuberculosis, which typically affects the respiratory system but can disseminate to the brain, bones, liver, and lymph nodes. TB presents with chronic cough (often with blood), fever, night sweats, and weight loss, and may cause lung cavitation, granuloma formation, and widespread systemic inflammation.

Toxoplasmosis, caused by *Toxoplasma gondii*, primarily affects the central nervous system (CNS) in people with AIDS. It produces brain abscesses, swelling, and tissue destruction, resulting in raised intracranial pressure, seizures, and focal neurological deficits. **Cytomegalovirus (CMV)**, a herpesvirus, also commonly reactivates in severe immunosuppression. CMV can affect the retina (causing visual impairment and blindness), the gastrointestinal tract (abdominal pain and diarrhoea), and the lungs (cough and fever), and its ability to break down mucosal barriers further predisposes patients to secondary infections. **Candidiasis**, caused by *Candida* species, manifests as white fungal plaques in the mouth, esophagus, or vagina; esophageal candidiasis is especially associated with advanced immunodeficiency.

Other significant opportunistic infections include **Cryptococcosis**, caused by *Cryptococcus neoformans*, which frequently involves the CNS. It can lead to meningitis, altered mental status, brain impairment, and seizures. **Mycobacterium avium complex (MAC)** is another late-stage infection that typically becomes systemic, spreading through the blood to the liver, spleen, and lymph nodes. MAC causes chronic inflammation, fever, weight loss, and severe anaemia. Additionally, **Kaposi sarcoma**, driven by human herpesvirus-8 (HHV-8), is a defining malignancy of AIDS. It produces characteristic purple or red vascular lesions on the skin, lymph nodes, and internal organs, and may result in breathlessness when the lungs are involved, as well as gastrointestinal bleeding when it affects the digestive tract.

Beyond infections, HIV itself directly injures multiple organ systems. Advanced disease can cause **HIV-associated encephalopathy**, **nephropathy**, **cardiomyopathy**, endocrine dysfunction such as **hypogonadism**, and gastrointestinal damage leading to malabsorption and chronic diarrhoea. Together, these opportunistic infections and HIV-related organ injuries represent the

hallmark complications of AIDS, illustrating the profound consequences of immune collapse in untreated or advanced HIV infection.

Slim Summary!

- Severe CD4+ T-cell depletion can cause severe immunosuppression, making patients vulnerable to ooportunistic infections such as Pneumocystis pneumonia, tuberculosis, toxoplasmosis, cytomegalovirus, candidiasis, and others. These infections, along with direct HIV-related organ damage, lead to significant morbidity and mortality in untreated or advanced disease.

Chapter 13 - Behaviour models

Introduction

This chapter describes the models and frameworks of behaviour that help us to understand why people engage in health threatening behaviours. Behaviours can be the way someone acts towards others or in response to a particular situation or stimulus. There are five main theories of behaviour.

COM-B Model

This model theorises how types of capability, motivation and opportunity influence behaviour. **Psychological capability** is having the knowledge about how to perform a certain task, whilst **physical capability** is possessing the skills to perform it. **Physical opportunity** is having the resources for something, whilst **social opportunity** is the availability of social opportunities to do something, and this is influenced by the opinions of others such as friends or family. There are also two types of motivation, which are reflective and automatic. **Reflective motivation** is weighing up the benefits and disadvantages of doing something, whilst **automatic motivation** is a habit or natural response to a cue.

Transtheoretical stages of change model

This is a model which proposes different stages that people go through when trying to change a behaviour such as smoking or drinking. People may not necessarily move through the stages in order and may go back and forth between them throughout the process of changing their behaviour. The first stage is **precontemplation**, where someone has not yet started thinking about changing their behaviour. **Contemplation** is then when someone starts to think about behaviour change. When they start to plan for changing behaviour, this is the **preparation** stage. The **action** stage is when they actively start making changes to their routine and behaviour. **Maintenance** is the continuation of the good behaviour change habits and if this continues for a long period then a stable lifestyle may be reached. However, throughout this process there may be a **lapse**, where someone goes back to their old behaviour once. This could then lead to a **relapse**, where they frequently go back to their old behaviour and no longer maintain good behaviour habits.

PRIME

This model evaluates the complexity of motivation and explores how the internal and external environment can affect decisions about behaviour change. PRIME stands for: Plans, Response, Impulses/Inhibitions, Motives, Evaluations.

Plans are the mental representations of actions when someone intends to do something. **Response** is the behaviour itself and may be due to a certain stimulus. **Impulses and inhibitions** compete with each other, and people may have urges to do certain things. Patients may really want to change, but in the moment the competing impulses and inhibitions might cause an unhealthy behaviour. **Motives** are representations of something attractive or repulsive and can heavily influence whether someone makes a behaviour change or not. **Evaluations** are people using their beliefs and values to analyse and reflect on a situation or behaviour to determine their views on it. Our intentions and beliefs about what is good and bad will only

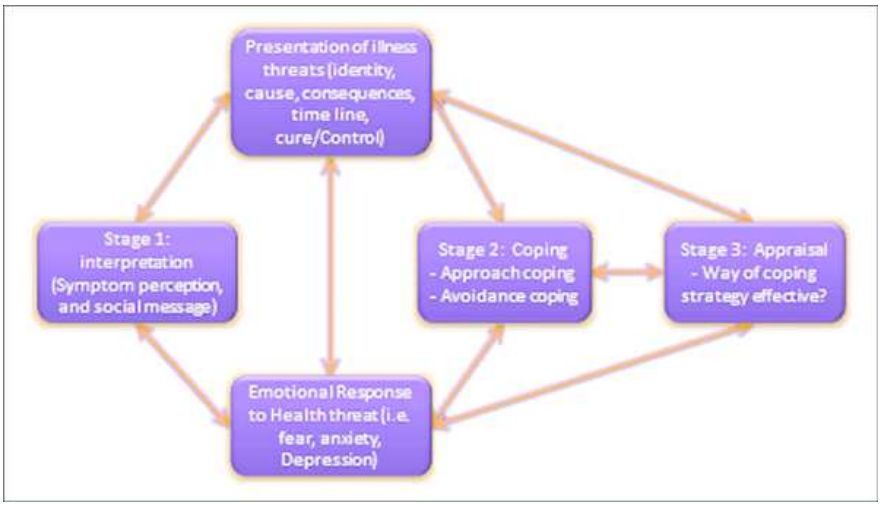
influence our behaviours if they create sufficiently strong wants and needs at the relevant moment.

Health belief model

This model was originally developed to explain the uptake of tuberculosis screening to predict health protective behaviour. It is now used to predict and explain a variety of behaviours, including health threatening and health protective behaviours and adherence to medication. It considers the following factors to determine the likelihood of a behaviour: **susceptibility, severity, costs, benefits, cues to action, health motivation, perceived control, and self-efficacy**.

Self-regulatory model

This model explores how illness representations can impact on health behaviours, so it focuses on how people behave when coping with an illness or diagnosis.



Self-regulatory model. *Source: researchgate.net*

It involves different stages of interpretation, presentation of illness, emotional response, coping and appraisal of coping. The presentation of an illness can include its identity (symptoms), cause, timeline, consequences, and control or curability. People cope with illness in different ways and may appraise or analyse their way of coping. Someone's primary appraisal is an evaluation of the illness or situation itself and how significantly it will affect them, whilst someone's secondary appraisal is the evaluation of their ability to cope with it.

Slim Summary!

- The five main models of behaviour are: the COM-B model, PRIME, the health belief model, and the self-regulatory model;
- They explain different theories for behaviours and responses to stimuli.

Chapter 14 - Adherence, compliance and concordance

Introduction

Compliance, adherence, and concordance describe different ways patients engage with healthcare recommendations, ranging from following instructions to actively participating in shared decision-making. This demonstrates the importance of patient involvement, understanding, and collaboration in achieving effective treatment outcomes.

Compliance refers to the extent to which a patient follows the instructions or recommendations given by a healthcare professional. It implies a largely one-way relationship, where the healthcare provider prescribes treatment and the patient is expected to follow it, often without much discussion. Compliance focuses on whether patients "do as they are told," and non-compliance is typically viewed as a problem on the patient's side.

Adherence expands on compliance by emphasizing the patient's active role in following a treatment plan. Adherence recognizes that patients make choices based on their beliefs, understanding, and circumstances. It reflects how consistently a patient takes medications, attends appointments, or implements lifestyle changes as agreed with their healthcare provider. Unlike compliance, adherence is less judgmental and focuses on supporting patients to follow treatment in real-world contexts.

Concordance is the shared decision-making process between the patient and healthcare professional. Concordance reflects mutual agreement on treatment goals and strategies, taking into account the patient's values, preferences, and beliefs. This concept emphasizes partnership and negotiation, rather than simply instructing the patient.

Facilitation by Healthcare Professionals

Healthcare professionals can promote adherence, concordance, and compliance through several strategies:

Patient Education: Providing clear, understandable information about the condition, treatment options, potential side effects, and expected outcomes empowers patients to follow through on treatment. Understanding improves motivation and adherence.

Shared Decision-Making: Involving patients in treatment decisions fosters concordance.

Supportive Communication: Active listening, empathy, and encouragement help identify barriers to adherence, such as misunderstandings, fear, or socioeconomic challenges. Tailoring communication to the patient's needs promotes adherence and compliance.

Simplifying Regimens: Reducing complexity in medication schedules, using combination therapies, and setting reminders improve practical adherence.

Follow-Up and Monitoring: Regular check-ins allow healthcare providers to assess adherence, address difficulties, reinforce education, and adjust treatment if necessary.

Behavioral and Psychological Support: Encouraging motivation, addressing mental health issues, and using motivational interviewing or reminder systems can enhance patient engagement with their care.

Slim Summary!

- Compliance focuses on whether patients follow healthcare instructions, adherence emphasizes consistent patient engagement with a treatment plan, and concordance reflects a collaborative, negotiated approach between patient and provider.
- Healthcare professionals can facilitate these approaches through patient education, shared decision-making, supportive communication, simplifying regimens, follow-up monitoring, and behavioral or psychological support.

Chapter 15 - Biopsychosocial model and sexual health

Introduction

This chapter discusses the biopsychosocial model and its views on sexual health as shaped by biological, psychological, and social factors. This holistic perspective guides assessment and treatment to address all influences on sexual wellbeing.

The biopsychosocial model emphasizes that sexual health is influenced by biological, psychological, and social factors. Biologically, clinicians consider hormonal function, reproductive anatomy, sexually transmitted infections (STIs), and chronic conditions that can affect sexual function, such as diabetes or cardiovascular disease. Psychologically, mental health conditions like anxiety, depression, body image concerns, and stress can significantly impact sexual desire, performance, and satisfaction. Socially, relationships, cultural norms, family dynamics, and societal attitudes towards sexuality shape sexual behaviors and access to care. Applying this model in diagnosis requires clinicians to take a holistic history covering physical symptoms, emotional wellbeing, sexual practices, and social context. In management, interventions may include medical treatments (e.g., for erectile dysfunction or infections), psychological therapies (e.g., counseling or cognitive behavioral therapy), and social support (e.g., relationship counseling, education on safe sexual practices, or community resources). This approach ensures care is patient-centered, comprehensive, and addresses the full spectrum of factors influencing sexual health.

Slim Summary!

- Biological factors include hormones, anatomy, STIs, and chronic illness; psychological factors involve mental health and stress; social factors cover relationships, culture, and societal norms.
- Management integrates medical treatment, psychological support, and social interventions for comprehensive, patient-centered care.

Chapter 16 - GMC Guidance on Confidentiality

Introduction

The GMC guidance on confidentiality sets out the ethical and legal standards for handling patient information. It ensures that patient data is protected, used appropriately, and shared only when necessary or legally required.

The General Medical Council (GMC) guidance on confidentiality outlines the ethical and legal principles that govern the handling of patient information. It emphasizes that patient information is private and must be protected, and that disclosure is only permissible with the patient's consent, or in exceptional circumstances such as risk of serious harm to the patient or others, or where legally required. The guidance highlights the importance of clarifying with patients how their information will be used, ensuring they understand who may access it, and taking steps to minimize unnecessary sharing. It also stresses the need for secure storage and communication of patient data, whether electronically or on paper, and careful consideration when sharing information with colleagues or third parties. Ultimately, the GMC guidance promotes trust in the doctor-patient relationship and ensures that information is handled responsibly while supporting safe and effective care.

Slim Summary!

- Patient information must remain private, with disclosure allowed only with consent, to prevent serious harm, or when legally mandated, and patients should be informed about how their data is used.
- Secure storage, careful communication, and responsible sharing with colleagues or third parties are essential to maintain trust and support safe, effective care.

Chapter 17 - Ways to maintain patient confidentiality

Introduction

GP surgeries and hospitals use a variety of strategies to protect patient confidentiality and ensure sensitive information is handled appropriately. These measures combine physical, electronic, and procedural safeguards to maintain trust and comply with legal and professional standards.

GP surgeries and hospitals implement multiple strategies to maintain patient confidentiality. Access control ensures that only authorized staff can view patient records, often through secure login systems and tiered permissions. Physical safeguards include locked filing cabinets, private consultation rooms, and restricted areas for sensitive discussions. Electronic safeguards involve encrypted records, password protection, and secure data transfer protocols. Staff are trained on understanding the importance of discretion and professional conduct when discussing patient information. Policies such as **minimum necessary disclosure** ensure that only relevant information is shared for clinical purposes. Additionally, procedures are in place for handling breaches, consent documentation, and anonymization of data used for research or audits. Combined, these measures help build patient trust, comply with legal standards, and protect sensitive personal information in healthcare settings.

Slim Summary!

- Confidentiality is maintained through access controls, secure storage, private consultation areas, encrypted electronic records, and staff training.

Chapter 18 - Rights of the individual and preventing harm

Introduction

This chapter discusses the duty to balance the rights of the individual with the need to prevent harm to others in the context of confidentiality.

Patients have a right to confidentiality and keeping information about their health private. However, if the patient has a communicable or transmissible disease which could be passed to others, there is potential to cause harm to people who are at risk of catching the disease. In the case of HIV, which can be transmitted sexually, sexual partners of the patient with HIV would be at risk. Therefore, in some cases, doctors would have a duty to inform people at risk of catching the disease about the health status of the patient.

In order to balance the rights of the patient, doctors should make every effort to inform the patient of the risks to those around them and encourage them to share the information themselves with those at risk or at least give consent for the doctor to. Even if the patient cannot be convinced to consent to break confidentiality, the doctor would still have a duty to inform those at risk of catching the disease, as potential harm could be caused. These situations balance the ethical pillars of autonomy and non-maleficence, as patients have a right to keeping their information private, but sometimes doing this may cause harm to others and this must be prevented.

Slim Summary!

- Individual patient rights and patient autonomy is important to keep, but sometimes the need to prevent harm to others (non-maleficence) takes precedence.

Chapter 19 - Ethical imperative for confidentiality

Introduction
This chapter explains the ethical imperative for confidentiality in the doctor-patient relationship and its limits.

There is an ethical imperative for confidentiality, as patients have a right to keep information about their health private. This is supported by the ethical pillar of autonomy, as the patient's independence is respected. Patients must give explicit consent to release their information to others when they have the capacity to do so. Respecting the patient's right to confidentiality is not only a duty of a healthcare professional, but it also builds a good relationship with the patient, and the patient is more likely to trust the professional with sensitive information in the future.

In court cases regarding patient care and information, courts will protect the confidentiality of all adults with capacity who refuse to have their information shared. There are several pieces of legislation regarding protection of patient's data, including the Data Protection Act, the NHS Confidentiality Code of Conduct and the Freedom of Information Act.

There are, however, circumstances where confidentiality may be breached, and these will be discussed in the next chapter.

Slim Summary!

- Patients have a right to confidentiality and keeping their information private, so doctors have a duty to respect this and not breach confidentiality unless absolutely necessary.

Chapter 20 - Breaching confidentiality

Introduction
This chapter discusses circumstances where it may be necessary to breach a patient's confidentiality. If a healthcare professional is unsure about breaking confidentiality, then they should consult a senior colleague before doing so. When breaking confidentiality, robust notes should also be kept to document and justify the reasoning for breaking confidentiality.

As discussed previously, if there is potential harm caused to others by not breaking confidentiality, then it may be necessary to break it. An example of this is when a patient has a sexually transmissible disease (STD) such as HIV and they are sexually active. Whilst the HIV is transmissible, sexual partners of the patient would need to be informed of the risk to them as potential harm could be caused. The patient should be informed of this requirement and encouraged to tell their partner themselves about their condition. However, if the patient refuses the doctor would still have a duty to inform the partner as there is potential risk and harm. If the patient has taken the relevant medications and their HIV becomes undetectable (and therefore untransmissible), then it would not be necessary to breach confidentiality as the virus can no longer be transmitted by the patient to their partner.

In cases of communicable diseases, it may be in the public interest to breach confidentiality, because there is a high risk of harm to the general public. Therefore, healthcare professionals and courts may have a duty to release patient information in order to protect the general public. The amount of information needed to be shared should be assessed, and ideally patients should be encouraged to consent to the release of their information. Confidentiality should still be respected as much as possible, so only the necessary information about their health should be released. It may also be in the public interest to breach confidentiality in cases of work accidents and food poisoning to prevent potential harm being caused to others.

Patients may also lack capacity to consent to a breach of confidentiality, for example if they are unconscious. If this happens in an emergency situation, it may be necessary to breach confidentiality and inform next of kin about the situation. This is so that any relevant information about their health can be obtained and imperative decisions about treatment can be made.

Slim Summary!

- It is necessary to breach confidentiality where there is potential harm caused to the general public by disease transmission, such as with STDs and communicable diseases;
- Patients may also lack capacity to consent to breaching confidentiality in emergency situations, so it may be necessary to inform next of kin.

Afterword

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