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Defence Against Disease

IB Biology - Revision Notes

Pathogens

Types of Pathogen

- A disease is an **illness or disorder** of the **body or mind** that leads to **poor health**
- Each disease is associated with a set of **signs and symptoms**
- A pathogen is any microorganism that **causes disease** in another organism (e.g. in plants or animals)
- Many **microorganisms** are pathogens including:
  - **Bacteria**
  - **Fungi**
  - **Protists**
  - **Viruses**
- **Not all species** within these groups (apart from the viruses) are pathogens, as many bacteria, fungi and protists are **harmless** and **do not cause disease**
- However, **all viruses are pathogenic** as they can **only exist** by living inside the living cells of other organisms (or by using these cells to create more viruses)
- **No archaea** are known to be pathogenic in humans
- Pathogens cause **communicable diseases** which means they transfer from a diseased host to a healthy organism during infection, in other words the disease is infectious
- Examples of such diseases include:
  - tuberculosis
  - athletes foot
  - malaria
  - cholera
- **Non-communicable** diseases are non infectious diseases such as
  - cancer
  - cardiovascular disease
  - malnutrition

Infectious & Non-infectious Diseases Table

Term	Definition	Example
Infectious diseases	These are diseases caused by organisms known as pathogens. They are sometimes called communicable diseases as they are passed from infected to uninfected people (they are transmissible). Some also affect animals and are passed from animals to humans.	◦ Cholera ◦ Malaria ◦ HIV/AIDS ◦ Tuberculosis (TB)
Non-infectious diseases	These are long-term, degenerative diseases that are not caused by pathogens. Examples include diseases of the gas exchange and cardiovascular systems, inherited or genetic diseases, deficiency diseases caused by malnutrition, and mental diseases.	◦ Lung cancer ◦ Chronic obstructive pulmonary disease ◦ Sickle cell anaemia ◦ Cystic fibrosis

NOS: Careful observation can lead to important progress

- **Observations** have lead to many **medical breakthroughs** in the treatment of disease
- These observations have allowed a **deeper understanding of diseases** and the pathogens that cause them
- Knowledge of symptoms, incubation times and transmission mechanisms are all important in order to implement measures to control the spread of a disease
- The optimum scenario is to eradicate the disease completely

Cholera

- Cholera is a **water** and **food-borne** disease caused by the bacterium ***Vibrio cholerae***
- Cholera can be **transmitted** when people are exposed to **contaminated** water, either through consumption or through bathing

- The disease is common where **people do not have access to proper sanitation (clean water supply) and uncontaminated food**
- Infected people pass large numbers of the **bacteria in their faeces**
- If these faeces contaminate the **water supply** (due to lack of proper sewage treatment), or if infected people **handle food** or **cooking utensils** without washing their hands, then the bacteria are transmitted to uninfected people

- In 1854 a Cholera outbreak in **Soho** in London lead to the death of over **500 people in a month**
- A local doctor, John Snow, **observed** the clinical presentation of the disease after encountering an outbreak in a mining village in 1832, and so was familiar with the **symptoms and mechanisms for transmission**
- His prior **experience provided a fundamental insight** which helped him to identify the cause of the outbreak in Soho
- He mapped the cases of cholera and traced them all back to **one water pump**
- The pump handle was **removed** and the outbreak came to an end
- Later it was noted that the water pump was positioned only a few feet from a **cesspit** which was **contaminated with *Vibrio cholerae***
- John Snow's careful observations facilitated the **control of this spread of cholera** in this situation and provided useful evidence which became incorporated into the **'germ' theory** of disease which revolutionised sanitation in the 19th century

Childbed fever

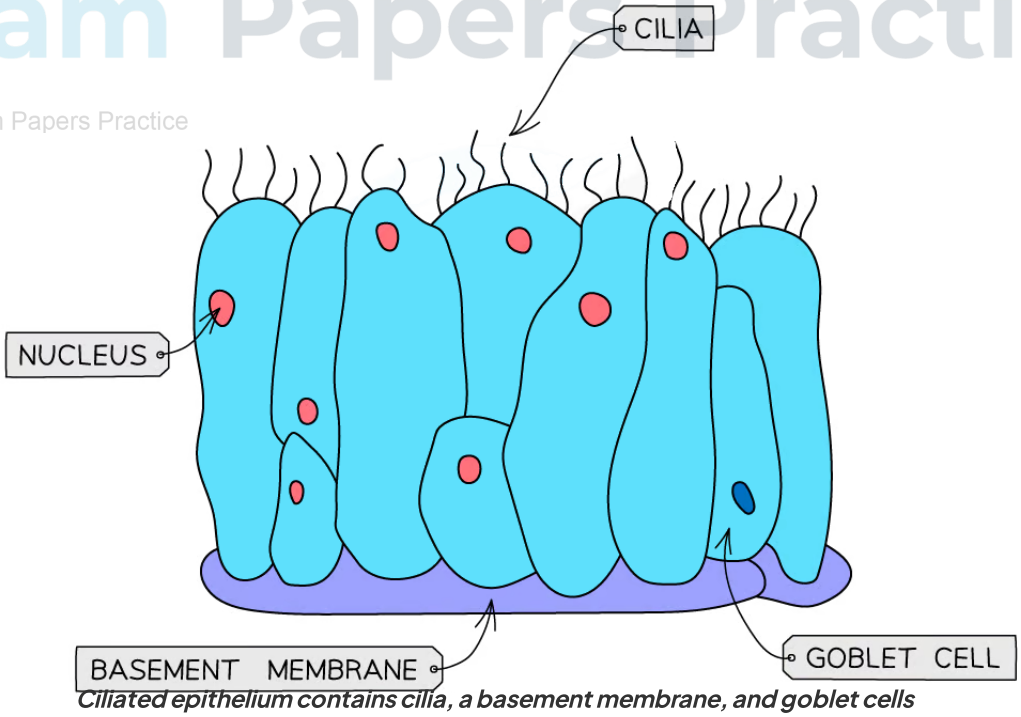
- Puerperal fever, also known as childbed fever, is a **bacterial** infection of the female reproductive tract after **childbirth**
- Transmission of the disease occurs through **direct contact** during the delivery process
- Childbed fever was the most common cause of death associated with childbirth in the 19th century
- A Hungarian physician, **Ignaz Semmelweis**, observed that there were a greater number of deaths in one maternity ward compared to another
- On closer investigation, Semmelweis noted that obstetricians and medical students who took part in **autopsies** (of women who had died from childbed fever) went on to deliver babies in the maternity ward **without washing their hands**
- He found a **correlation** with the **number of deaths** in the ward and the number of **autopsies** carried out leading him to suggest a link between handling the corpses and the number of new cases
- Semmelweis suggested that **particles** were being **transferred** from the corpses to the women on the maternity ward
- He initiated a **mandatory hand washing policy** for all those involved and later also began washing the medical instruments
- These precautions lead to a clear **decline in patient deaths** from childbed fever and informed the foundations of routine hand washing routines in healthcare
  - Such processes are fundamental, particularly in hospitals, to the **control of many transmissible diseases**

Barriers to Pathogens: Skin & Mucous Membranes

Skin as a Barrier to Pathogens

- The **skin and mucous** membranes form a **primary defence** against pathogens that cause infectious disease
- **Skin** is the largest organ of the body and is covered in **microorganisms** that usually cause no issues, as they can't enter the body. Skin provides:
  - A **tough physical barrier** that prevents entry of pathogens into our bodies
  - Cuts in the skin are sealed by formation of **blood clots** to prevent entry of pathogens
  - **Chemical protection** through the production of **sebum** from the sebaceous glands of the hair follicles
    - Sebum is a chemical responsible for maintaining a **low skin pH** which inhibits the growth of microorganisms
- **Mucous membranes** are found lining vulnerable areas which may be a route for pathogens into the body
  - This includes the **airways**, areas around the **reproductive organs** (foreskin and vagina) and the **digestive system**
- The membranes contain **goblet cells** which **produce mucus** containing glycoproteins
  - Microorganisms and particles become **trapped** by the mucus
  - The mucus is then **swept along by the cilia** of the ciliated epithelium upwards and is swallowed
  - The mucus and any microorganisms will then be **swallowed** and destroyed by the acid in the stomach or **expelled**, therefore preventing infection
  - Mucus also contains **lysozyme enzymes** which have **antibacterial** properties, providing more protection from invading microorganisms

Cilia and Goblet Cells Diagram



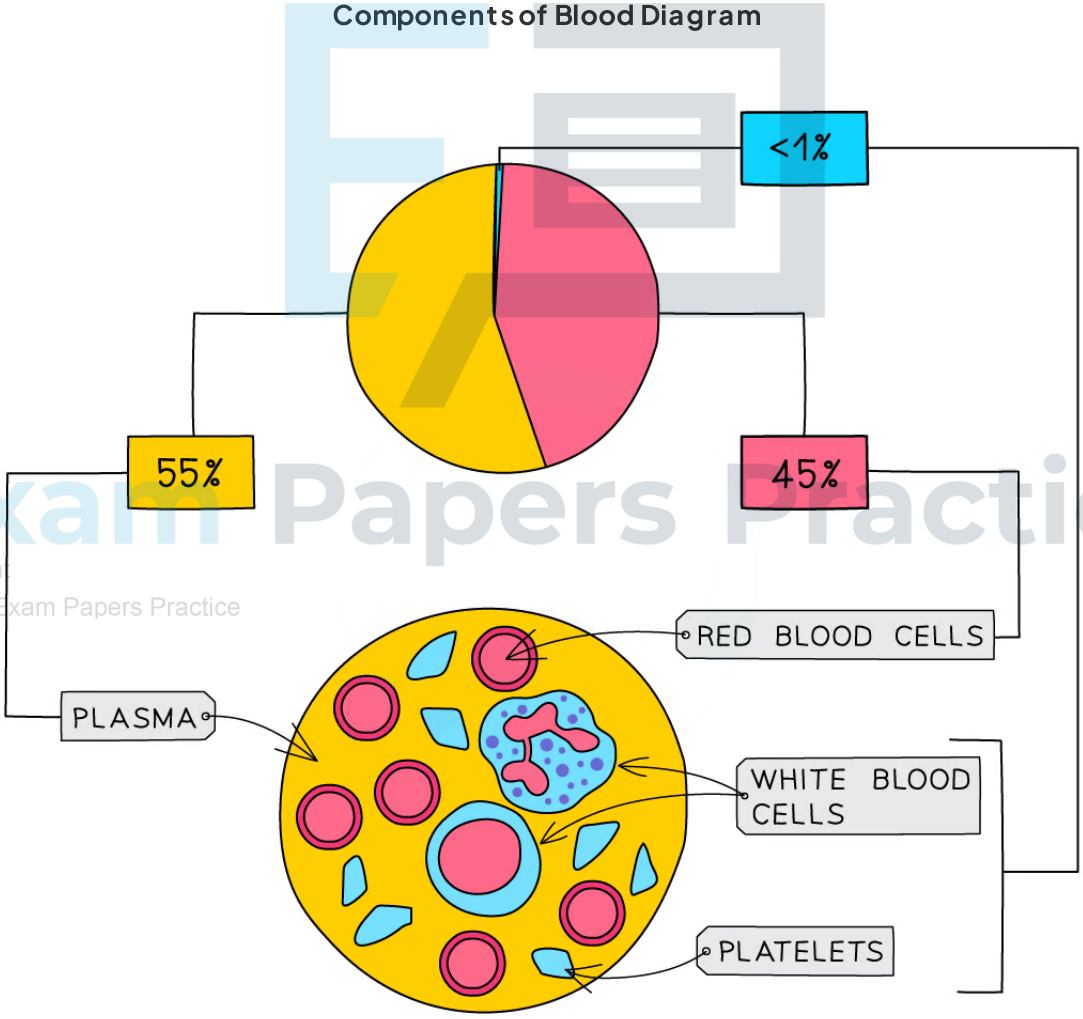
Ciliated epithelium contains cilia, a basement membrane, and goblet cells

Blood Clotting

The Process of Blood Clotting

Platelets

- When the skin is cut, microorganisms have an entry point to get into the body
  - The first line of defence is compromised
- In order to minimise the risk of substantial **blood loss** and entry of **unwanted microorganisms**, the blood starts to clot and **seal the wound**
- In response to **blood vessel damage**, platelets form a temporary **plug** to stem bleeding
  - Platelets are **cellular fragments** that make up one component of the blood
- They release chemicals called **clotting factors** that trigger a **chemical cascade** which results in blood clotting

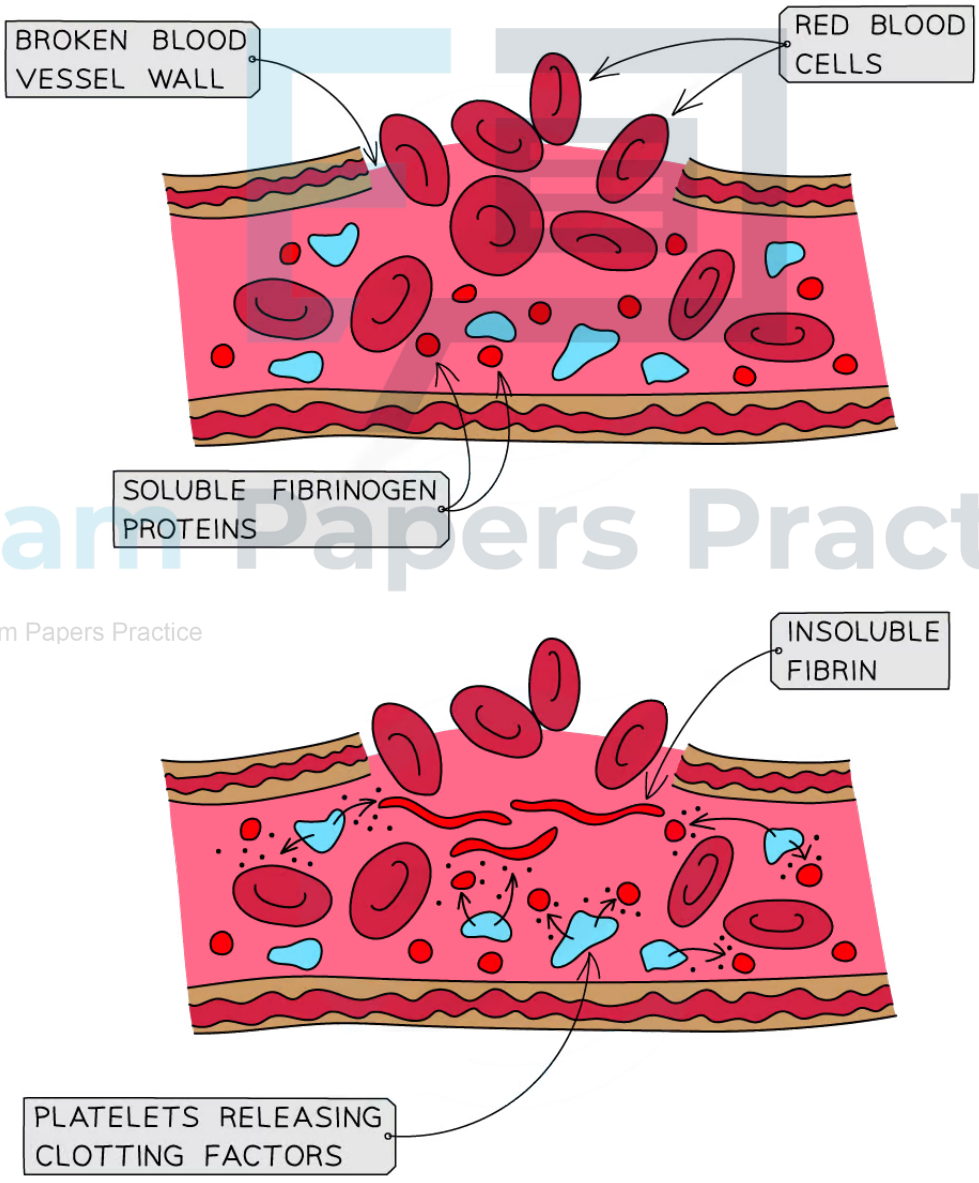


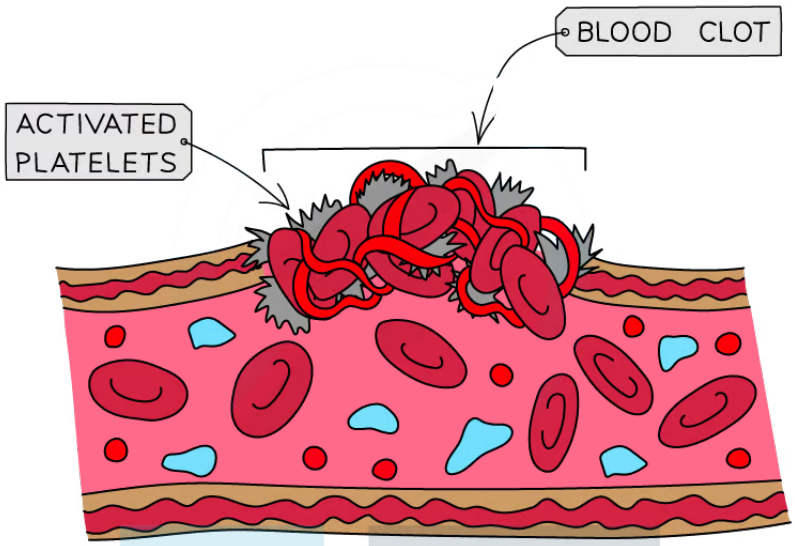
The blood is made up of 4 key components; plasma, red blood cells, white blood cells and platelets

Blood clotting process

- The chemical cascade, triggered by the clotting factors, involves a large number of steps and several plasma proteins
  - First of all, the **clotting factors** stimulate the release of the enzyme **thrombin**
  - Thrombin catalyses the conversion of the soluble protein **fibrinogen** into **fibrin**, which is insoluble
  - Fibrin forms a **mesh** that traps more platelets and blood cells to prevent entry through the wound
    - A small initial stimulus is **amplified** to produce a large amount of fibrin so that the wound is quickly sealed
  - Exposure to air results in the hardening of the mesh to create a **scab**

Blood Clot Formation Diagram





Blood clotting involves a chemical cascade process

The Immune System

The Immune System: Innate vs Adaptive

The innate immune system

- The innate immune system is able to **recognise and respond to** any item that enters the body that is **'non-self'**; these items could be:
  - Bacteria
  - Fungi
  - Viruses
  - Protists
  - Pollen grains
  - Dust
- The innate immune system recognises these non-self items because they display, or act as, **non-self antigens**
  - An antigen is a **molecule that can trigger an immune response**
  - All cells have antigens on their cell surface membranes
  - An individual's own cells will be recognised due to the presence of **self antigens**, while a foreign cell will have **non-self antigens** and so will **initiate an immune response**
  - Items such as pollen grains, or other allergens, may be recognised by the innate immune system as non-self antigens; this leads to the symptoms of allergy
- Individuals are born with the ability to mount an innate immune response to non-self antigens, and the response **does not change during their lifetime**
- The action of phagocytes forms part of the innate immune response; phagocytes will engulf and digest any item that displays non-self antigens
- Innate immune responses are sometimes described as **non-specific** immune responses
  - Innate immune responses are **broad** in nature; they occur in response to **any non-self antigen** and are not specific to any one particular type of antigen

The adaptive immune system

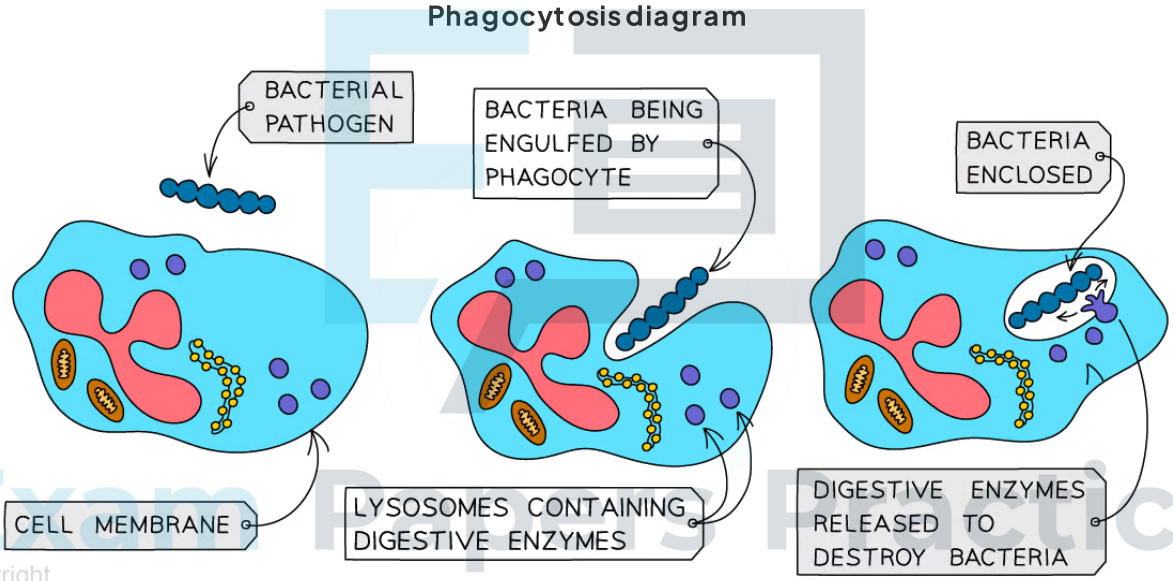
- The adaptive immune system responds to the presence of **specific non-self antigens**, e.g. the antigens of a **particular type of pathogen**
  - When the adaptive immune system first encounters a new type of non-self antigen, a sequence of events occurs that eventually leads to **antibody production** and the presence of **memory cells** in the blood
  - When the adaptive immune system encounters the same type of antigen again, the sequence of events occurs **much more quickly** and produces **many more antibodies**, and the pathogen is destroyed before any symptoms occur
- The adaptive immune system **changes over the course of an individual's lifetime** as they are **exposed** to diff t t f ti
  - A **memory of different pathogens** is built up as exposure occurs; this is known as immunological memory
  - Young babies have no adaptive immunity, and the adaptive immune system **develops with age**
- **Vaccination** makes use of the adaptive immune system by introducing it to new pathogens, therefore speeding up the immune response on the next exposure to the same pathogen
- Adaptive immune responses are sometimes referred to as **specific immune responses**, as they occur due to the presence of **specific antigens**

White Blood Cells

Phagocytes

What do phagocytes do?

- **Phagocytes** are white blood cells that are produced continuously in the **bone marrow**
- They are responsible for **removing dead cells and invasive microorganisms**; a **non-specific immune response**
- Phagocytes move in an **amoeboid** movement to the site of infection and attach to pathogens
  - The **cell surface membrane** of the phagocyte extends out and around the pathogen, **engulfing it** by endocytosis
- They then digest the pathogen using **enzymes** which are stored within lysosomes (in their cytoplasm)



Phagocytic cells ingest pathogens and digest them using enzymes

Lymphocytes

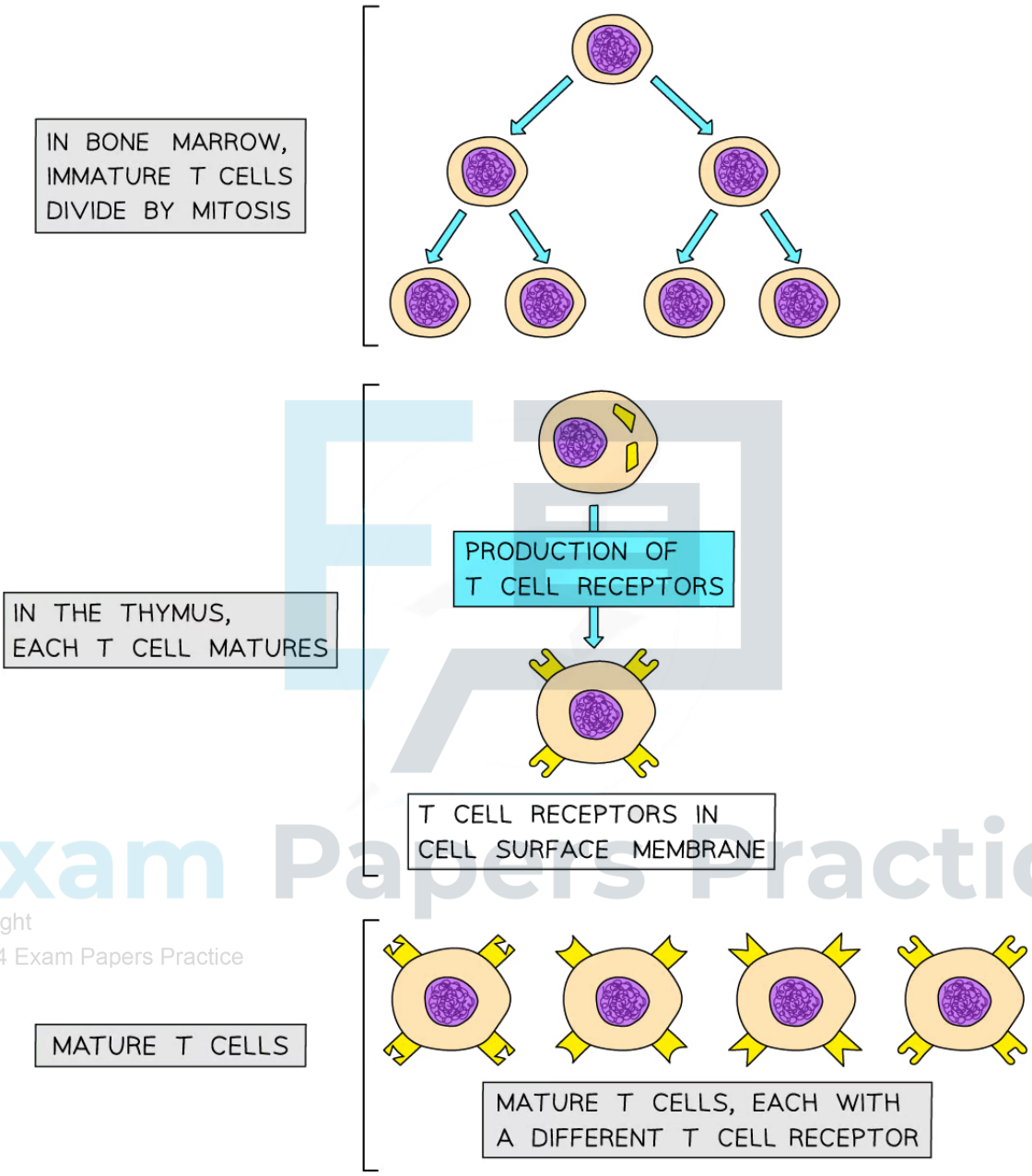
What are lymphocytes?

- There are two types of **lymphocyte** that play a particular role in the specific immune response
  - T cells
  - B cells
- Note that lymphocytes are a type of **white blood cell** found both in the **lymph nodes** and circulating in the **blood**

T cells

- **T cells**, sometimes known as T lymphocytes, are produced in the bone marrow and finish maturing in the **t**hymus, which is where the **T** in their name comes from
- Mature **T cells** have specific cell surface receptors called **T cell receptors**
- These receptors have a **similar structure to antibodies** and are each **specific to a particular type of antigen**

Production of T cells diagram



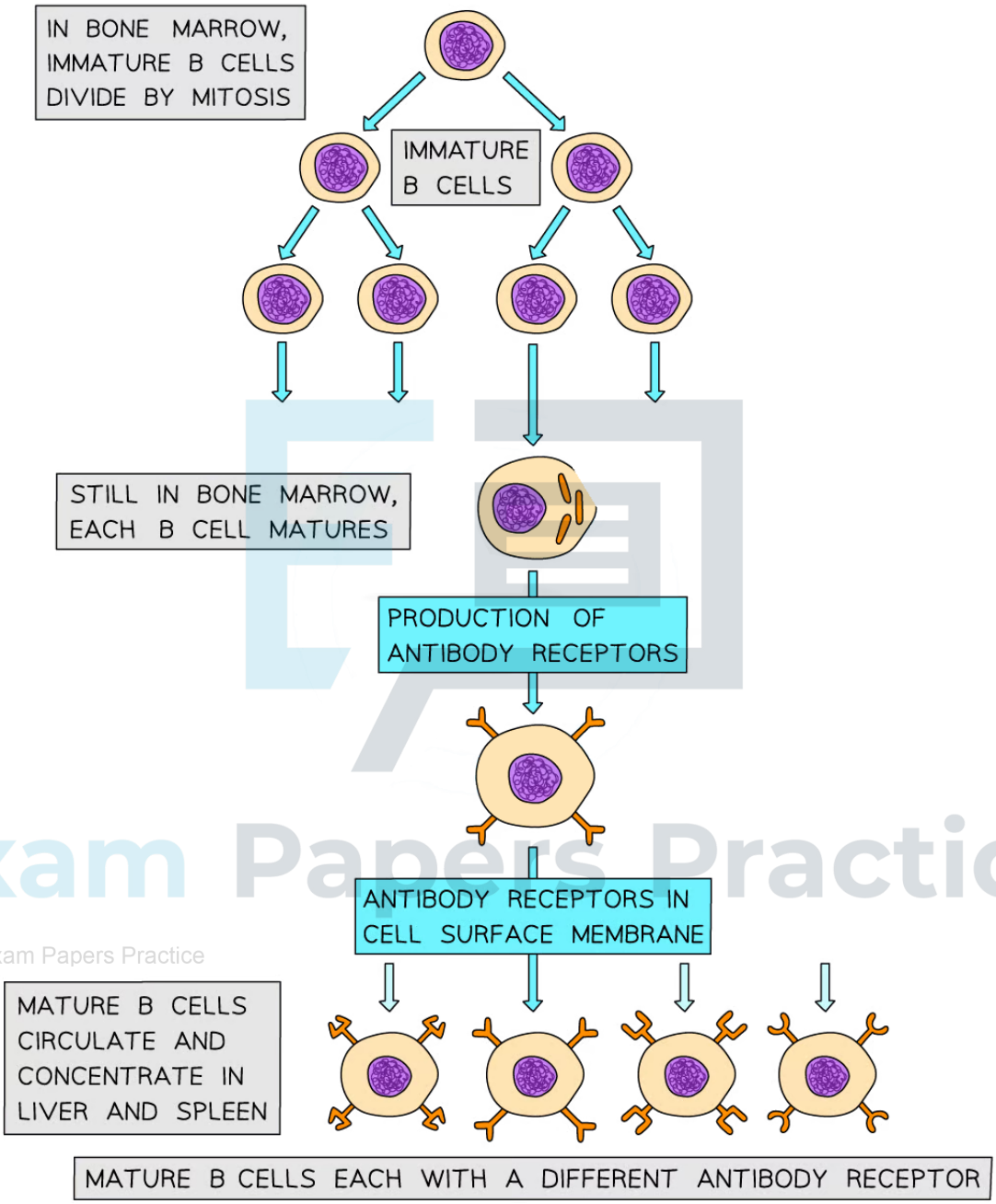
Mature T cells have many different types of receptor on the cell surface membrane; these receptors will bind to different antigens on antigen presenting cells

- T cells are **activated** when they encounter and **bind to their specific antigen** on the surface of an antigen-presenting cell
  - This **antigen-presenting** cell might be a **macrophage,** an **infected body cell**, or the **pathogen** itself
- These activated T cells **divide by mitosis** to increase in number
  - Dividing by mitosis produces **genetically identical cells**, or **clones**, so all of the daughter cells will have the **same type of T cell receptor** on their surface

B cells

- **B cells,** also known as B lymphocytes, are a second type of white blood cell in the specific immune response
  - B cells remain in the **b**one marrow as they mature, hence the **B** in their name
- B cells have many **specific receptors** on their cell surface membrane
  - The receptors are in fact **antibodies**, and are known as **antibody receptors**
  - Each B cell has a **different type of antibody receptor**, meaning that each B cell can **bind to a different type of antigen**

Production of B cells diagram



Mature B cells each have different types of antibody receptors on their cell surface membrane

- If the corresponding antigen enters the body, B cells with the correct cell surface antibodies will be able to **recognise** it and bind to it
  - When the B cell binds to an antigen it forms an **antigen-antibody complex**
- The **binding of the B cell to its specific antigen**, along with the **cell signalling molecules produced by T helper cells**, **activates** the B cell
- Once activated, the B cells divide repeatedly by mitosis, producing many clones of the original activated B cell
- There are **two main types of B cell**
  - **Effector cells,** which differentiate into **plasma cells**
    - Plasma cells produce specific antibodies to combat non-self antigens
  - **Memory cells**
    - Remain in the blood to allow a faster immune response to the same pathogen in the future

Adaptive Immune Response

Antigens

- Every organism has cells with **unique molecules** on the cell surface membrane which act as **markers** to **identify it**
- These unique markers are **macromolecules** and they allow **cell-to-cell recognition**
- The **immune system** has the ability to distinguish between 'self' and 'non-self' based on these molecules
  - **Microorganisms** (both pathogenic and non-pathogenic), such as **bacteria** and **viruses**, trigger an **immune response** as the immune system recognises their markers as being **non-self**
  - Molecules that trigger an immune response in this way are named antigens
  - **Antigens** are found on cell surface membranes of cancer cells, bacterial cell walls, the envelopes of viruses and even pollen grains
  - Some **glycolipids** and glycoproteins on the outer surface of cell surface membranes act as antigens
- **Allergies** are the result of an immune response triggered by antigens on the surface of an **allergen**, such as pollen

Exam Tip

The different **types of pathogen** include viruses, bacteria, fungi and protozoans.

Antigens on red blood cells

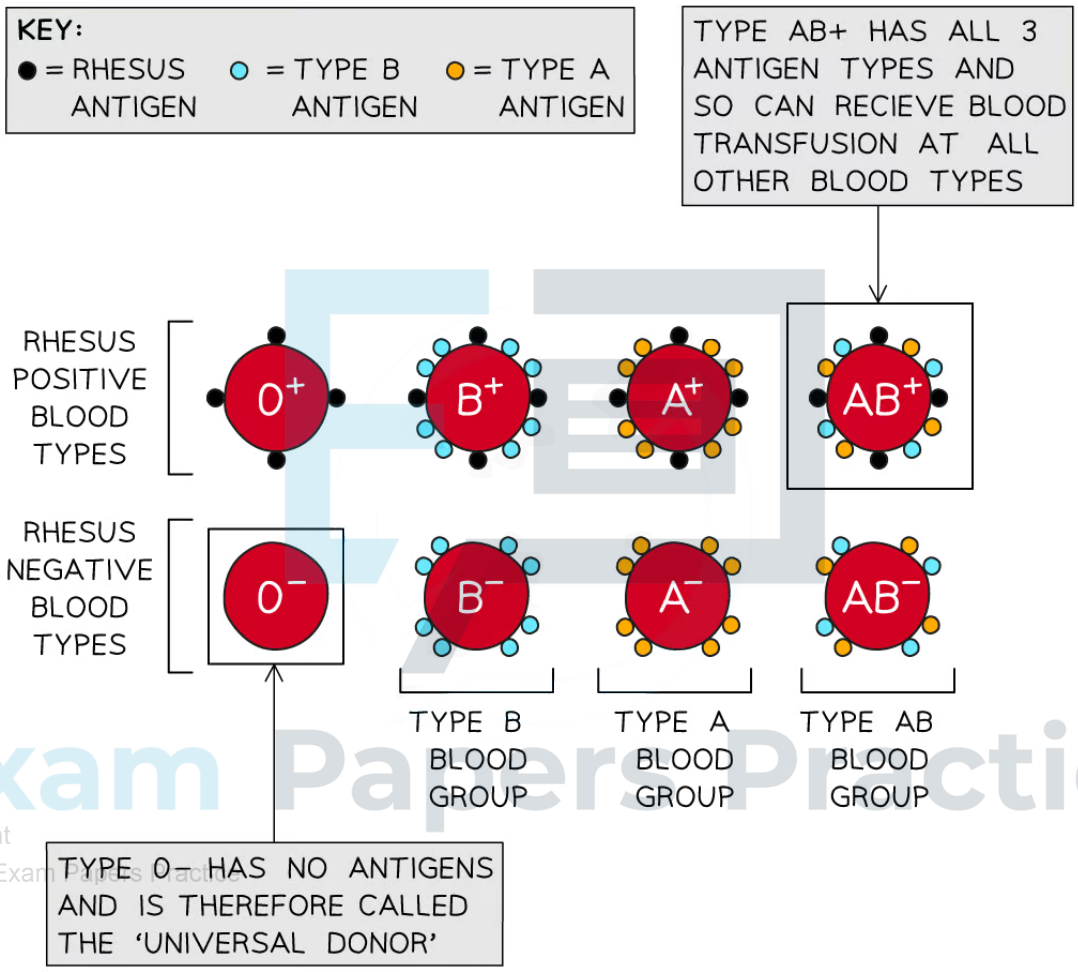
- Red blood cells have **specific markers** on their surface known as **antigens** which **determine the blood group** of an individual
- If a **transfusion** is given to an individual with mismatched blood group, the antigens on the red blood cell surface will trigger an immune response
- There are two **antigen markers** that must be considered:
  - The **ABO marker** - this determines whether the individual is **blood group A, B, AB or O**
  - The **Rhesus (Rh) marker** - this determines whether the individual is rhesus **positive** or rhesus **negative**

Determining ABO blood types

- **Blood type A** has a **type A antigen** consisting of an initial 'H' marker which is modified with another molecule called N-acetylgalactosamine
- **Blood type B** has a **type B antigen** consisting of an initial 'H' marker which is modified with another molecule called galactose

- **Blood type AB** has **type A and B antigens** consisting of two 'H' markers one of which is modified with N-acetylgalactosamine and the other with galactose'
- In **blood type O,** the 'H' marker is not modified and so there are no A or B antigens

Antigens and blood type diagram



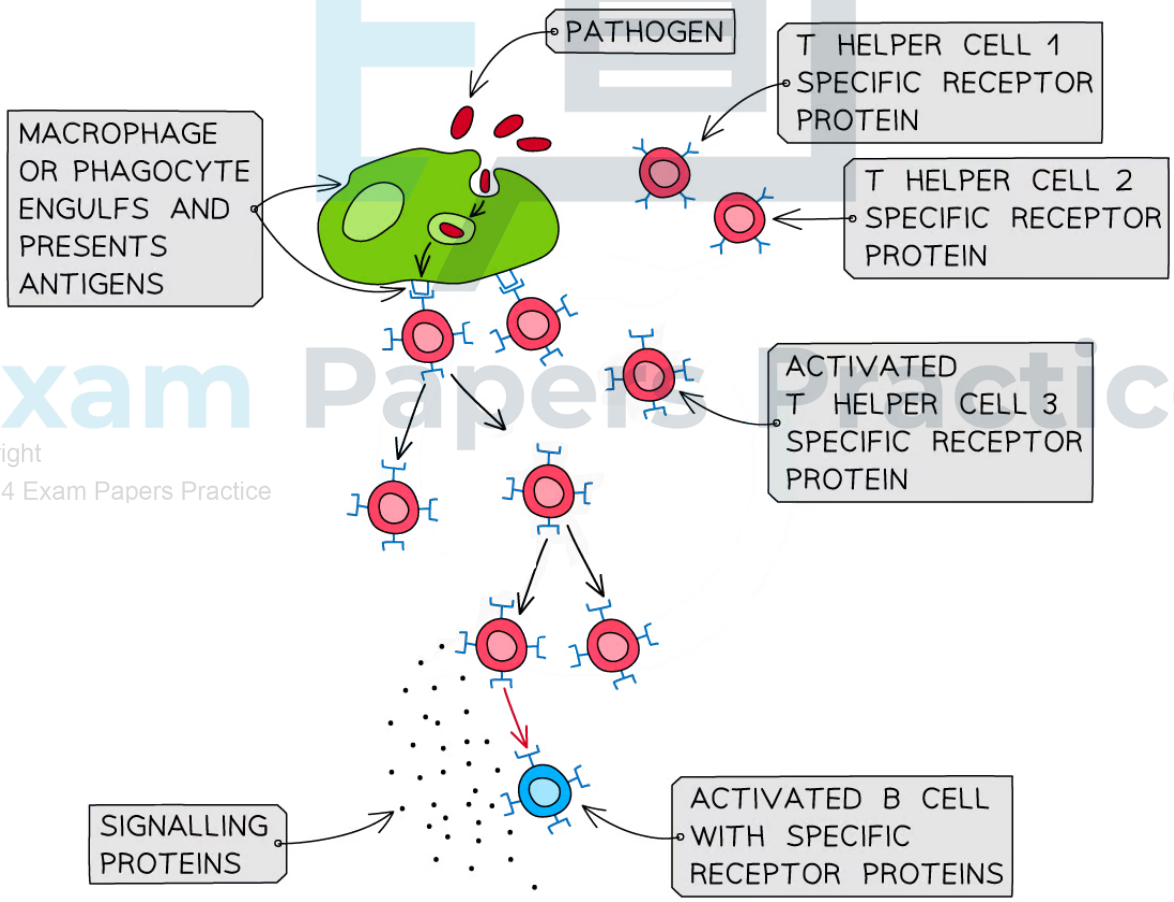
Blood type is determined by the presence or absence of specific antigen markers on the surface of the red blood cells

- If a transfusion is given to someone of an **incompatible blood type,** an immune response will occur due to the presence of antibodies in the recipient's blood that bind to blood cells with non-self antigens
- An immune response may result in **agglutination** of the blood in the blood vessels and **could be fatal**
  - Agglutination is when red blood cells clump together due to the binding of antigens and antibodies
- Blood type must be **compatible** when carrying out a transfusion to prevent **coagulation of blood** in blood vessels

Activation of B-lymphocytes

- T-Helper cells (a type of lymphocyte that responds to specific antigens) and mature B cells (another type of lymphocyte) have specific receptors located on their cell surface membranes
  - These receptors have a **similar structure to** antibodies and are each **specific to one antigen**
  - Note that lymphocytes are a type of white blood cell involved in the specific immune response; there are several different types of lymphocyte
- When phagocytes engulf pathogens, they **present the pathogen antigens** on their own cell surface membrane
  - A cell with non-self antigens on its surface membrane is known as an antigen presenting cell
- The T-helper cell with the **complementary receptor proteins to the antigen** will bind to the antigen and become **activated** by the phagocyte
- **Activated T-helper cells** then bind with **complementary receptors** on the surface membrane of specific **B-lymphocytes**
- On binding, the **T-helper cells** releases **signalling proteins** and **activate these B-cells**
- Once activated, the B cells **clone** themselves to become
  - **plasma cells** which produce **antibodies**
  - **memory cells** which provide **immunity** against future infection from the same pathogen

B cell activation diagram



Antigens activate complementary T-helper cells which go on to activate complementary B-cells

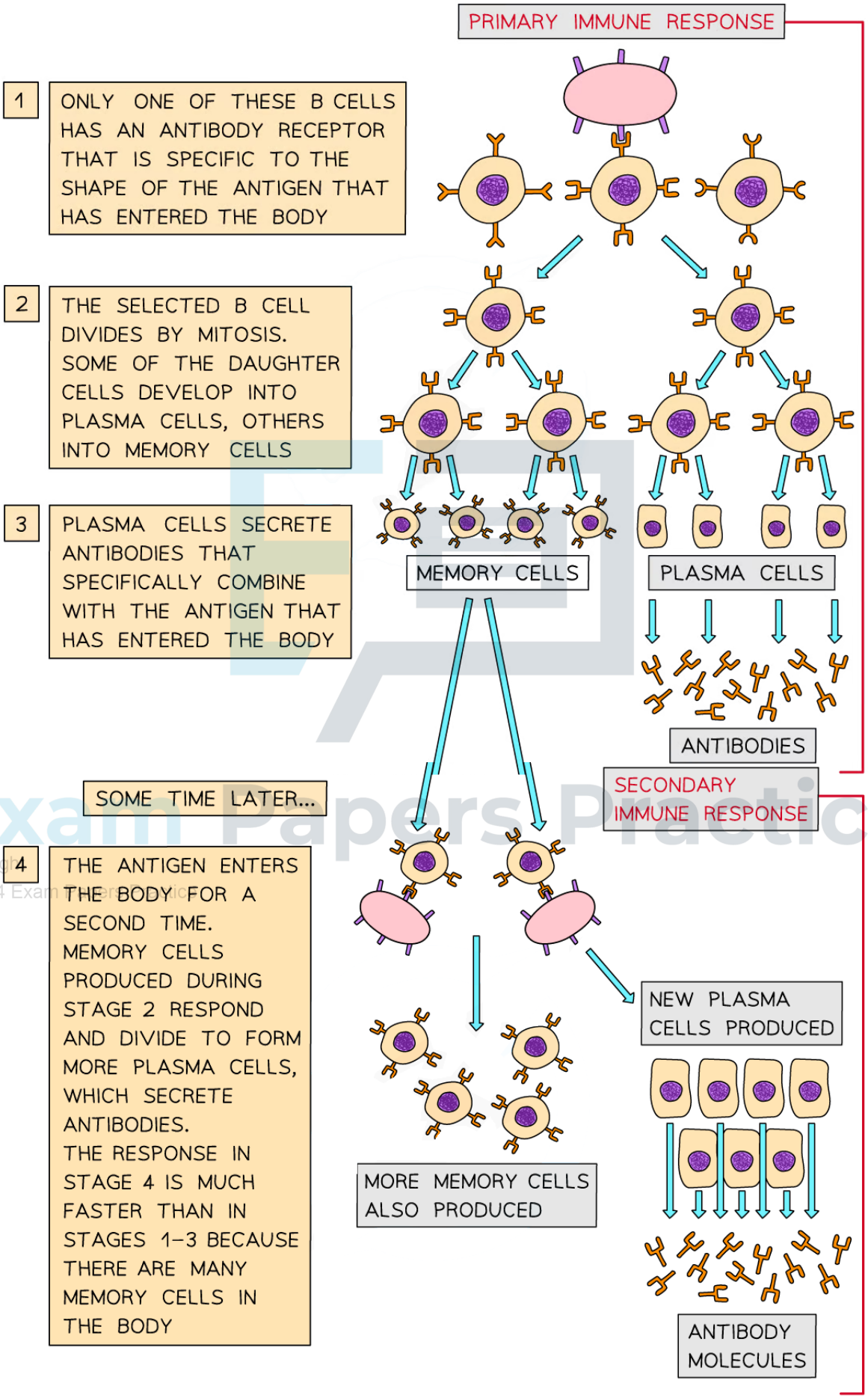
Clonal Expansion

- Once the B cell has been activated, **clonal expansion** can then occur
  - The **activated B-cell divides by** mitosis to create many clones of itself
    - Each **clone** will produce **the exact same antibody**, complementary to the target antigen
- Some of these mature B-lymphocytes differentiate into plasma cells
- The other B-lymphocytes become **memory cells** that remain and circulate in the blood
  - Whilst the antibodies produced by the plasma cells are only present for a matter of weeks or months, memory cells form the basis of **immunological memory** – the cells can last for **many years** and often a lifetime

Memory Cells & Immunity

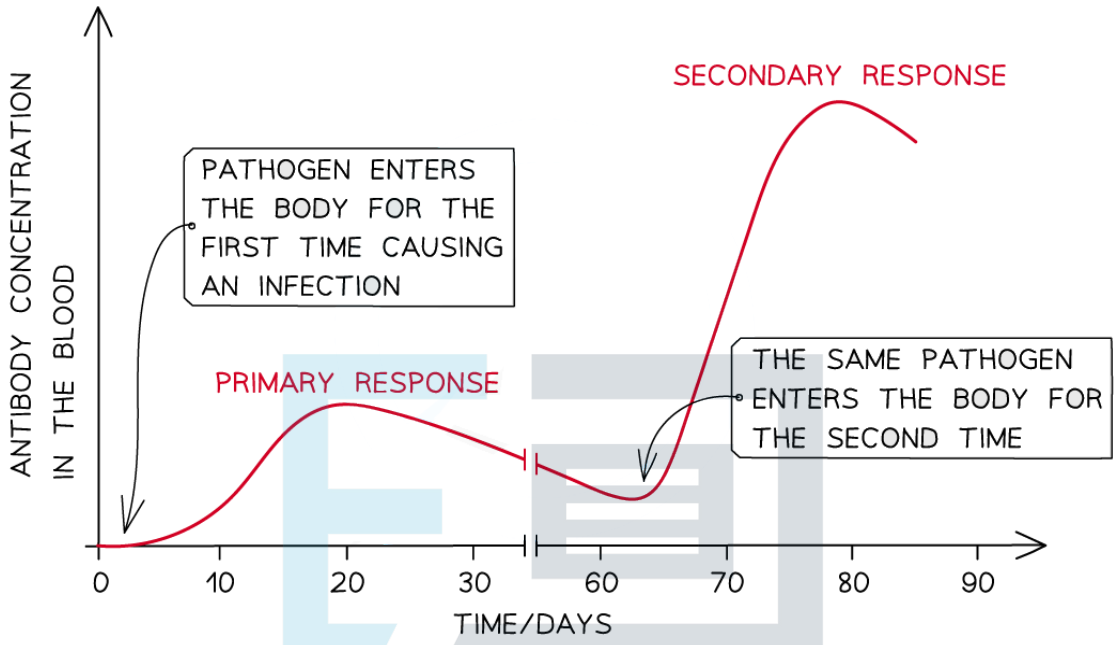
- Immunity is initiated when **exposure to a specific antigen** results in the production of **complementary antibodies** and **memory cells**
- This first exposure to an antigen **triggers the primary immune response**
- The **primary immune response** leads to the **development of immunity** if memory cells and antibodies persist in the bloodstream after the pathogen has been eliminated
- The **secondary immune response** occurs when the **same antigen is found in the body a second time**
  - The **memory cells recognise the antigen, divide very quickly** and differentiate into antibody-producing plasma cells and more memory cells
  - The response to a previously encountered pathogen is, relative to the primary immune response, **extremely fast**
  - This means that the **infection can be destroyed and removed before the pathogen population increases** too much and symptoms of the disease develop

Developing immunity diagram



During a secondary immune response, memory cells that remained in the blood divide very quickly into plasma cells (to produce antibodies) and more memory cells; 2000 antibodies can be produced per second! Whereas a primary response occurs much more slowly.

Primary and secondary immune response graph



The secondary response is much larger and more rapid than the primary response

Exam Tip

Immunological memory (made possible by memory cells) is the reason why catching certain diseases twice is so unlikely. For example, there is only one strain of the virus that causes measles, and each time someone is re-infected with this virus, there is a very fast secondary immune response so they **do not get ill**.

However, some infections such as the common cold and influenza are caused by viruses that are constantly developing into **new strains**. As each strain has different antigens, the primary immune response (during which we often become ill) must be carried out each time before immunity can be achieved.

HIV & AIDS

Transmission of HIV

- Human Immunodeficiency Virus is a retrovirus
- The virus is **unable to survive** outside of the human body; it needs **host cells** in order to **replicate**
- HIV is not transmitted by a vector (unlike in malaria), it is spread by **direct exchange** of body fluids
- This means HIV can be **transmitted** in the following ways:
  - Sexual intercourse
  - Blood donation
  - Sharing of needles used by intravenous drug users
  - From mother to child across the placenta
  - Mixing of blood between mother and child during birth
  - From mother to child through breast milk

HIV Infection

- **HIV** is made up of several key components including **RNA** and the enzyme, **reverse transcriptase,** which is used to produce DNA in the host cell; this classifies HIV as a retrovirus
- HIV infects the body and attacks a type of lymphocyte cell called a **T-helper cell**
- T-helper cells are a key component in the production of antibodies, so HIV **inhibits the body's capacity to produce antibodies**
- In the **early stages of infection**, antibodies are produced to fight HIV, these can be **detected in blood tests**
  - The individual is said to be **HIV positive**

The development of AIDS

- As the **infection progresses**, the ability to produce antibodies significantly reduces
- This renders the immune system unable to fight off other pathogens and so the individual becomes **prone to infection** from other **opportunistic pathogens**
- When the individual is suffering from **several diseases** or conditions at the same time, they are said to have **acquired immune deficiency syndrome** (AIDS)
- **Progression of HIV,** from the initial infection to the development of AIDS, can be slowed down using **anti-retroviral drugs**
  - Due to highly successful drugs, many HIV positive individuals are able to live full-quality lives with normal life expectancies

Exam Tip

HIV and AIDS are not the same thing:

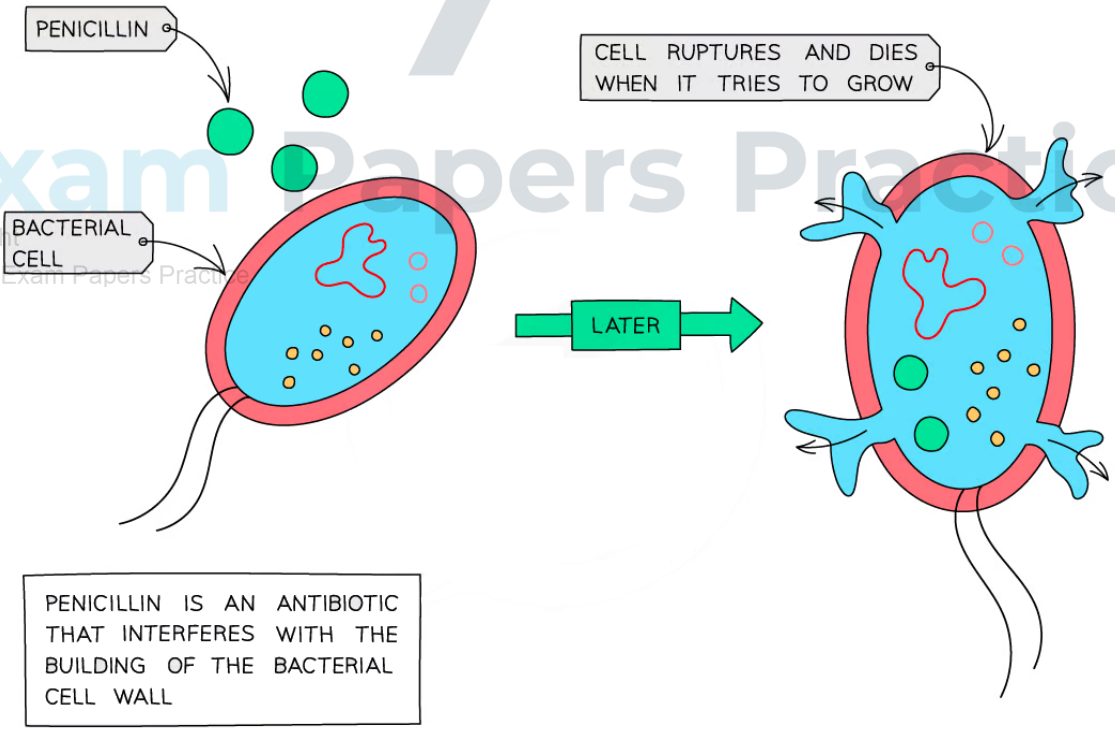
- HIV is the name used for the specific virus that is infecting the host
- AIDS is the acronym used to describe the syndrome that the virus, HIV, causes

Antibiotics

Antibiotics

- **Antibiotics** are **drugs** that **inhibit the growth** of microorganisms
  - Most antibiotics **kill or stop the growth of bacteria** (prokaryotes) but do not harm the cells of the infected organism
  - This is because they block specific processes that occur in **prokaryotic cells** but **do not have the same effect on eukaryotic cells**
- Processes that might be targeted include:
  - Transcription
  - Translation
  - DNA replication
  - Ribosome function
  - Cell wall formation
- Some antibiotics are derived from living organisms such as saprotrophic fungi
  - **Penicillin** is produced by certain fungi in the genus *Penicillium*
  - When growing in the wild the antimicrobial secretions of the fungus helps it to **compete** by killing nearby saprotrophic bacteria
- Antibiotics can also be made synthetically (in a laboratory)

Antibiotic action diagram



Penicillin interferes with the production of bacterial cell walls

- Penicillin is not effective against **all bacteria** (e.g. *tuberculosis*) because the bacteria may have:
  - **Thicker cell walls** which reduce permeability
  - **Enzymes which breakdown penicillin**
- There are many different examples of antibiotics which are effective against a range of bacterial diseases

Antibiotics & viruses

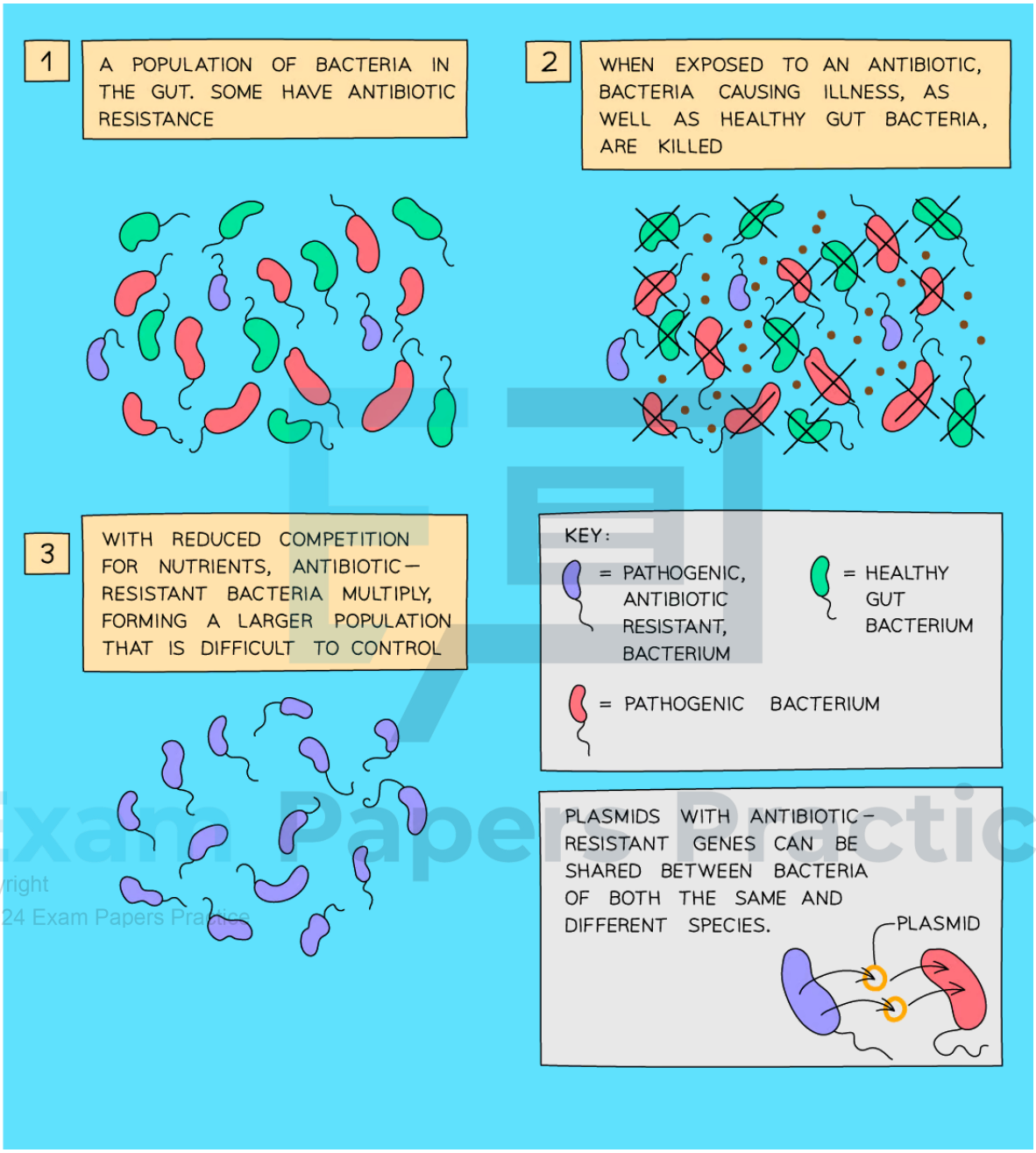
- Antibiotics are ineffective against **viruses** as they are **non-living**
- Viruses are **particles** and not cells
  - They have **no metabolism** or cell structure and therefore cannot be targeted in any of the ways that antibiotics target a bacterial cell
- When a virus **replicates**, it uses the **host cell's mechanisms** for transcription, translation and other metabolic pathways, so not even these processes can be targeted as antibiotics do not bind to the proteins that host cells use in these processes
  - Drugs that would target these processes would **damage the host cells** and cause even more harm
- **Antivirals** are drugs that target viral **enzymes** without harming the host cell

Antibiotic Resistance

Antibiotic Resistance

- Within a bacterial population, there is **variation** caused by mutations (as occurs in populations of all species)
- A chance mutation might cause some bacteria to become **resistant** to an antibiotic (e.g. penicillin)
- When the population is treated with this antibiotic, **the resistant bacteria do not die**
- This means the resistant bacteria can continue to reproduce with **less competition from the non-resistant bacteria**, which are now dead
- Therefore the **genes for antibiotic resistance are passed on** with a much greater frequency to the next generation
  - As bacteria only have one copy of each gene, a mutant gene will have an immediate effect on any bacterium possessing it
- Over time, the whole population of bacteria becomes **antibiotic-resistant** because the antibiotic-resistant bacteria are best suited to their environment
- This is an example of **evolution by** natural selection
- Some pathogenic bacteria have become **resistant to penicillin** as they have acquired **genes that code for the production of the enzyme β-lactamase** (also known as penicillinase), which breaks down penicillin

Antibiotic resistance diagram



Bacteria evolve rapidly as they reproduce and acquire random mutations, some of which confer resistance

The future of antibiotic resistance

- Antibiotic-resistant strains are a major problem in human medicine
- New resistant strains are constantly emerging due to the **overuse of antibiotics**
  - By using antibiotics frequently, humans exert a **selective pressure** on the bacteria, which supports the evolution of antibiotic resistance
- Scientists are trying hard to find **new antibiotics** that bacteria have not yet been exposed to, but this process is expensive and time-consuming
- Some strains of bacteria, such as methicillin-resistant *Staphylococcus aureus* (MRSA), can be **resistant to multiple antibiotics** and they create infections and diseases which are very difficult to treat
- When antibiotics were discovered, scientists thought they would be able to **eradicate** bacterial infections, but less than a century later a future is being imagined where many bacterial infections cannot be treated with current medicines

Measures to avoid antibiotic resistance

- Antibiotic resistance in bacteria is an example of natural selection that humans have helped to develop through **incorrect use or overuse** of antibiotics
- Implementation of certain measures can help to avoid antibiotic resistance. These measures may include:
  - Avoiding prescription of antibiotics for **non-serious or non-bacterial infections**
  - Maintaining **high standards of hygiene** in the hospital environment
  - Minimising use of antibiotics for routine treatment of animals in **agriculture**
  - Development of **new types of antibiotic**

NOS: The development of new techniques can lead to new avenues of research

- The rise of **antibiotic resistance** presents significant challenges within the medical field, as it renders the treatment of specific illnesses more challenging and contributes to **higher mortality rates**
- Addressing antibiotic resistance stands as a **top priority** for the World Health Organization (WHO)
- The future effectiveness in treating common infections and minor injuries hinges upon the development of **novel antibiotics**
- Presently, researchers are making use of **chemical libraries** to craft and produce fresh antibiotics
- Within these screening libraries, there exists a wealth of information about **numerous chemical compounds** possessing **antibacterial characteristics**
- Innovative methodologies like incorporating chemical libraries introduce **promising avenues** for countering the issue of antibiotic resistance

Zoonoses

Zoonosis

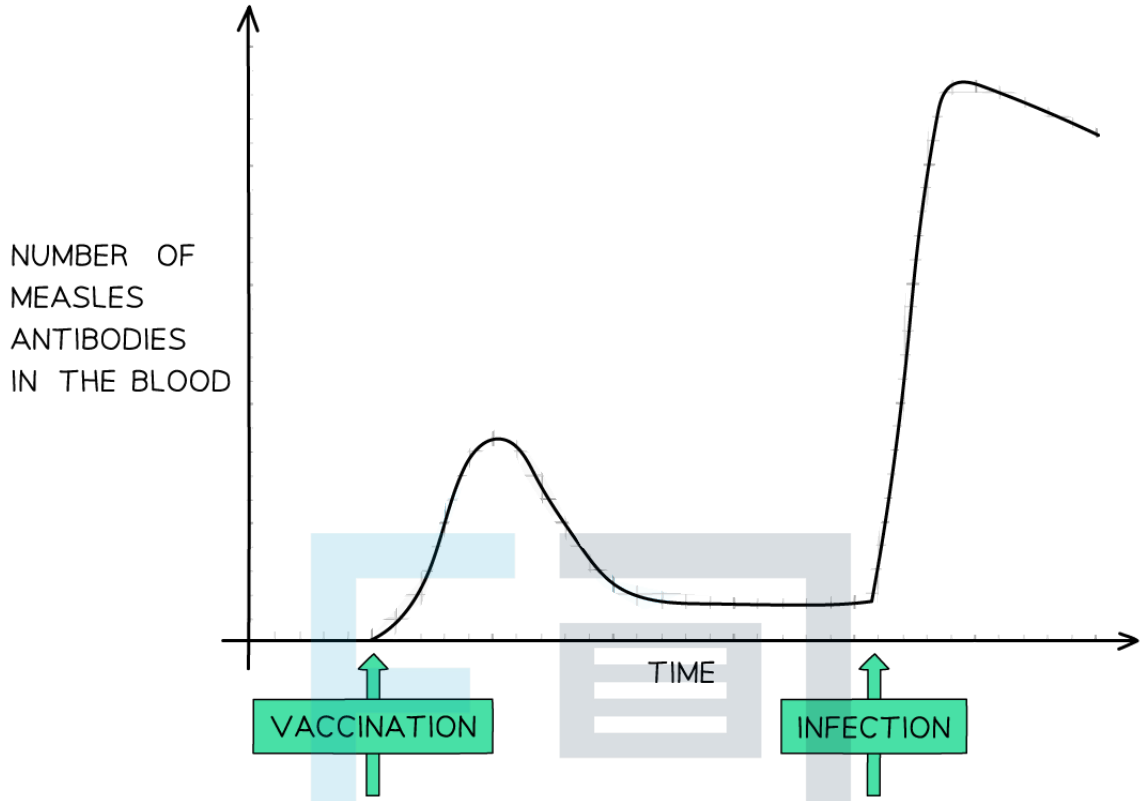
- Some diseases are **species specific** whilst others can **cross species barriers** to infect multiple different species
- Species-specific disease may be **unable to cross the species barrier** for many reasons:
  - If a species does not possess the **necessary receptors** to be at risk of infection
  - If the **body temperature** of the organism doesn't reach temperatures required for the development of the disease
- **Zoonotic diseases are those which can cross the species barrier** from animal to human
- This is a growing **global concern** due to the close relationships between humans and animals meaning the disease may be difficult to control and eradicate
- This may potentially lead to **pandemics** such as that caused by COVID-19
- Animal products may also be affected by zoonotic disease which poses a further issue
- Some zoonotic **diseases** can initially **emerge from animal populations** before developing into human only strains e.g. HIV

Vaccines & Immunity

Vaccines

- A vaccine is a source of antigens **or DNA/RNA** which codes for **antigens**
- The vaccine is introduced into the body to **induce immunity without causing the disease**
- Vaccines cause a **specific immune response** where antibodies are released by plasma cells
- There are different types of vaccine, including
  - **Live attenuated** - these are **weakened versions of the pathogen**
  - **Inactivated** - these are **killed, non-living components of pathogens** or even just the **antigens** alone
- Vaccines are administered either by **injection** or **orally** (by mouth)
  - The vaccinations given by injection can be into a vein or muscle
- Vaccinations produce **long-term immunity** as they cause memory cells to be created
- The memory cells recognise the antigen when re-encountered and produces antibodies, in what is a **faster, stronger, secondary response**

Vaccination & antibodies graph



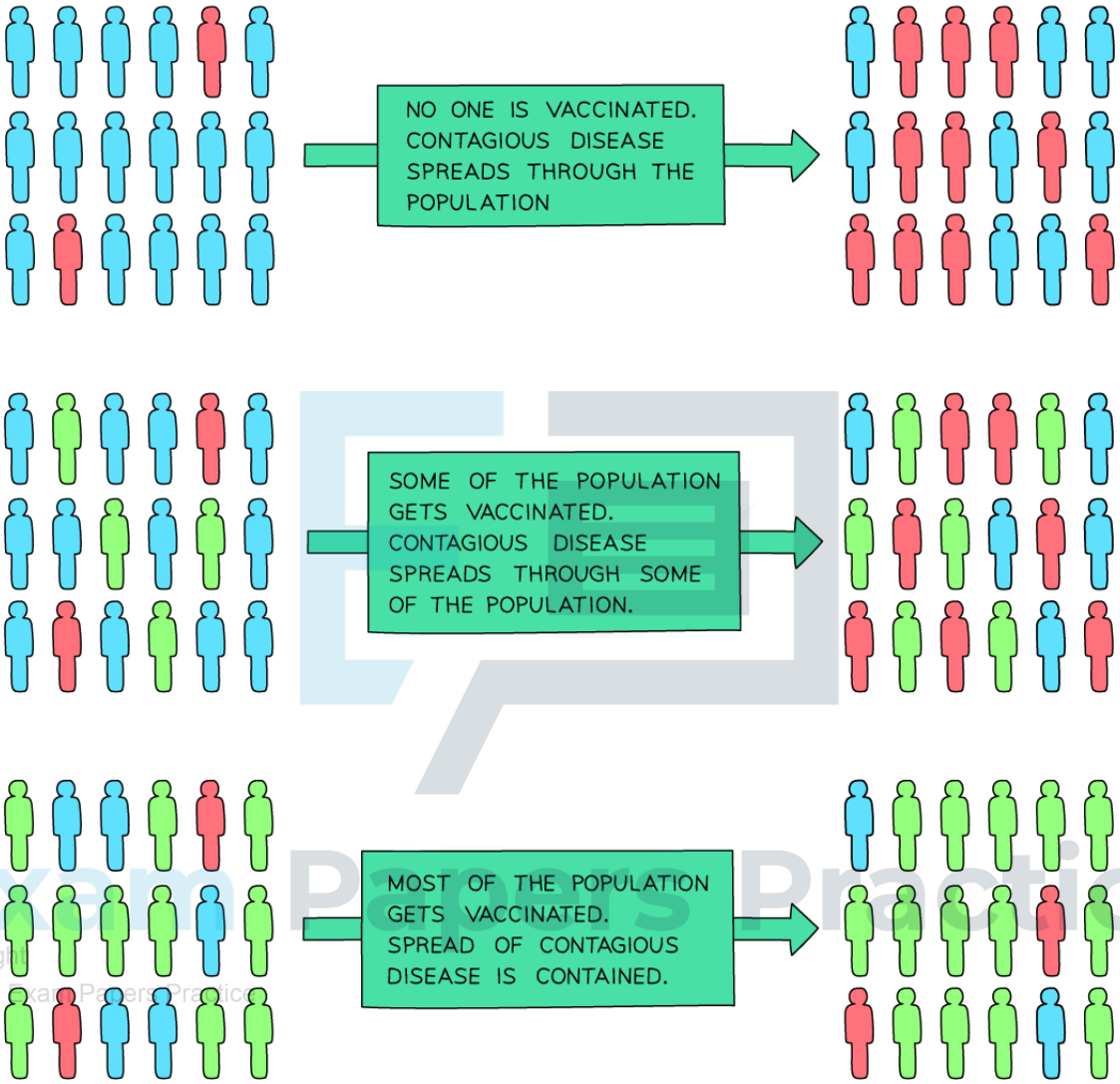
Exam Tip

Remember vaccines trigger the primary immune response (T helper cells trigger B plasma cells to secrete specific antibodies) which leads to the production of memory cells which will give a faster and larger (higher concentration of antibodies) secondary response.

Herd Immunity

- If **a large enough percentage** of the population is vaccinated, it **provides protection for the entire population** because there are **very few places for the pathogen to breed** - it can only do so if it enters the body of an unvaccinated person
- This is known as **herd immunity**
- If the number of people vaccinated against a specific disease **drops** in a population, it leaves the rest of the population at risk of **mass infection**, as they are more likely to come across people who are infected and contagious This **increases the number of infections**, as well as the number of people who could die from a specific infectious disease

Herd immunity diagram



Vaccinating a large enough percentage of the population provides protection for the entire population; this is herd immunity

- **Herd immunity prevents** epidemics **and** pandemics **from occurring in populations**
- This is the reason that many vaccinations are given to **children**, as they are regularly seen by medical practitioners and can be vaccinated early to ensure the entire vaccinated population remains at a high level
- In certain instances, vaccination programmes are run with the aim of **eradicating** certain dangerous diseases, as opposed to controlling them at low levels
- An example of a disease which has been eradicated as a result of a successful vaccination programme is **smallpox**, which was officially eradicated in 1980 after a vaccination programme run by the World Health Organisation since the mid-1950s

NOS: Scientists publish their research so that other scientists can evaluate it

- Data that is collected by scientists, to support theories in their research, is **peer reviewed**; this means that other scientists in the same field can judge the **accuracy and validity** of any conclusions drawn
  - Once research has been published, other scientists may use this research to aid further work
- In some situations the **media** may report on the findings of scientific studies before the full peer review process has been carried out; this can cause issues in **public responses** to new findings, for example:
  - When new medicines or vaccines are tested, the media may report on the side effects before tests are complete
  - The public view may be **biased** towards the media presentation of research, which may not be accurate
  - This can be **damaging** to the **progression and implementation** of any new medicines
- It is **important that the public are aware** of this problem of media reporting on incomplete research, though education on this is often not present in media reports
- When evaluating the introduction of a new medicine or vaccine, scientists tend to use a **pragmatic approach**, meaning that they consider the overall practicalities and effectiveness of a new treatment, rather than the **certainty** of its effect on individuals
  - I.e. a vaccine in testing may be safe and effective, but may result in unpleasant side effects for a **very small number** of individuals; scientists would draw the **overall conclusion** that this vaccine can be rolled out to the public, but an individual receiving the vaccine **would not be certain** that they wouldn't experience any negative side effects
- In the case of **COVID-19 vaccine** development, the pragmatic approach was applied in order to **develop an effective vaccine as quickly as possible**; results of trials showed that the vaccine was safe and effective for the vast majority of people, though there were a small number of individuals who experienced medical difficulties
- Although the vaccine showed a high degree of efficacy, there was a level of **distrust** from the public due to some of the **representation of negative side effects in the media**

Evaluating COVID-19 Data: Skills

Evaluating COVID-19 Data

Calculating percentage difference

- A percentage difference calculation allows comparison of two **directly comparable values** that occur at the **same time**, e.g. the number of COVID-19 cases in two different countries at the same point in time
  - Directly comparable values are values that **mean the same thing**, i.e. the number of COVID-19 *cases* and the number of COVID-19 *deaths* are two different types of value; they are not directly comparable
- Percentage difference is calculated by **dividing the difference between two values by the average of the two values**
- The resulting value is expressed as a percentage

$$\text{Percentage difference} = \frac{\text{difference between two values}}{\text{average of two values}} \times 100$$

Worked example

In mid-July 2023, Europe had 18 392 confirmed cases of COVID-19, while South-East Asia had 1 584 confirmed cases.

Calculate the percentage difference between the number of confirmed cases in Europe and South-East Asia

Step 1: Calculate the difference between the two values

$$18\ 392 - 1\ 584 = 16\ 808$$

Step 2: Calculate the average of the two values

$$\frac{18\ 392 + 1\ 584}{2} = 9\ 988$$

Step 3: Cubstitute numbers into the formula

$$\text{Percentage difference} = \frac{16\ 808}{9\ 988} \times 100$$

$$= 168.3\ \%$$

There was 168.3 % difference between the number of confirmed cases in Europe and South-East Asia in mid-July 2023

Calculating percentage change

- A percentage change calculation allows comparison of **two values from the same data set** at **different times**, i.e. how a factor has changed over time
- Percentage change is calculated by dividing the **difference between an old and a new value**, **divided by the old value**
- The resulting value is also expressed as a percentage

$$\text{Percentage change} = \frac{\text{change}}{\text{original value}} \times 100$$

- If the original number is larger, then the change will be a **percentage decrease**, and if the original number is smaller then the change will be a **percentage increase**

Worked example

In mid-June 2023, Europe had 38 950 confirmed cases of COVID-19, while in mid-July 2023, it had 18 392 cases.

Calculate the percentage change in COVID-19 cases in Europe between June and July 2023.

Step 1: Calculate the change in the number of cases

$$38\ 950 - 18\ 392 = 20\ 558$$

Step 2: Substitute numbers into the equation

$$\text{Percentage change} = \frac{20\ 558}{38\ 950} \times 100$$

$$= 52.8\ \%$$

There has been a percentage change of 52.8 % in the number of European COVID-19 cases between June and July 2023

As the original value was larger than the new value, this can be represented as a negative number (-52.8 %) or described as a percentage decrease