A. VOCABULARY

ANTIGEN

A **foreign** **glycoprotein** on the **surface** of a **pathogen** that **stimulates** the **production** of **specific antibodies**

ANTIBODY

A **glycoprotein** produced by **B-lymphocytes** in **response** to a **specific antigen**, which **attaches** to the **antigen** and **destroys** it

- **B lymphocytes** produce **antibodies**.

LEUCOCYTE

A **white blood cell**, such as:

lymphocytes = produce **antibodies**

phagocytes = **engulf** and **digest pathogens**

- **Erythrocytes** are **red blood cells**.

B. SPECIFIC v NON-SPECIFIC IMMUNITY

NON-SPECIFIC IMMUNITY

Not specific to the **pathogen**

Kill **any type** of **bacteria**

- Skin
- Mucous membranes
- Stomach acid
- Tears
- Phagocytes

SPECIFIC IMMUNITY

Specific to the **pathogen**

Kill **specific types** of **bacteria**

- B cells and antibodies

C. NON-SPECIFIC IMMUNITY

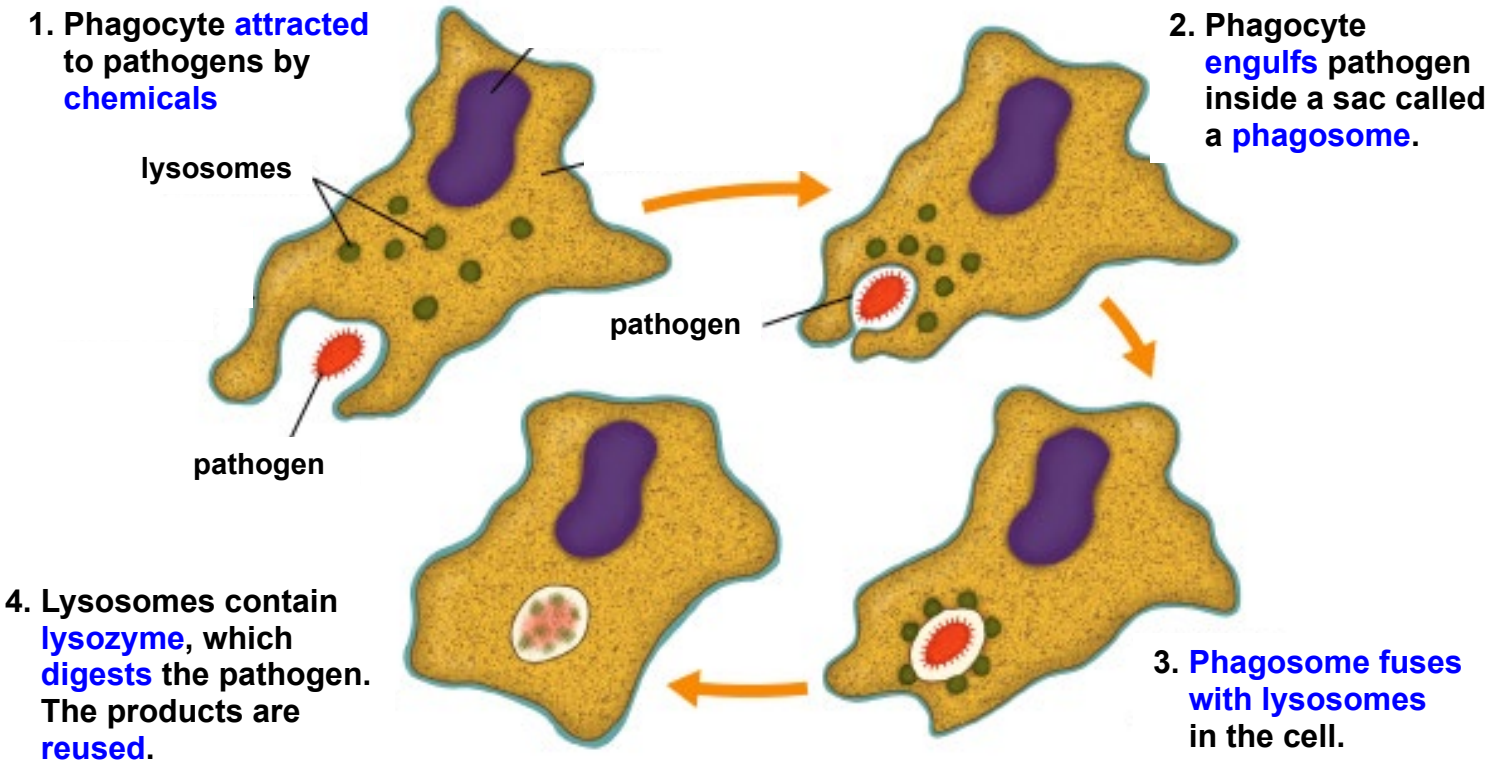
1. SKIN

- tough, impermeable outer layer
- sebaceous glands secrete lactic acid and fatty acids to prevent bacterial growth

2. MUCOUS MEMBRANES

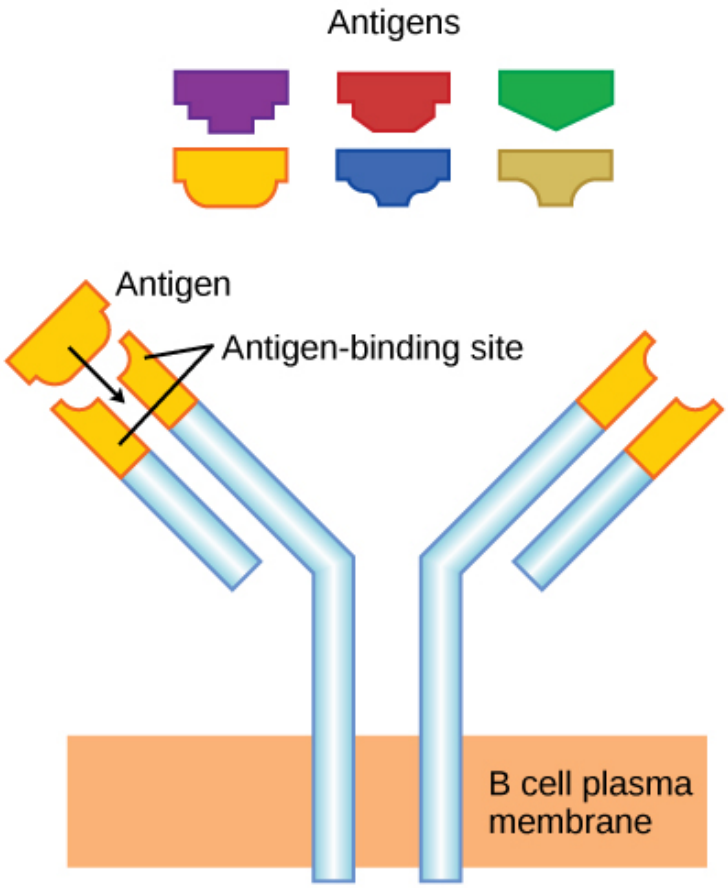
- nose, trachea, urethra, trachea, vagina are lined with these soft areas of skin
- mucus contains lysozyme, which kills bacteria
- mucus traps bacteria and cilia hairs push this up to the throat and out of the trachea

3. PHAGOCYTOSIS



- Phagocytes can also **leave** the **blood** and **enter tissues** to destroy pathogens.

D. WHY EACH ANTIBODY IS SPECIFIC FOR ONLY ONE ANTIGEN



- Antibodies are **proteins (= polypeptides)**
- They have a specific **tertiary structure**
- They have an **antigen binding site** of a **specific shape**
- This can only **attach** to an **antigen** that has a **complementary shape**

E. SPECIFIC IMMUNITY: HOW ANTIBODIES ARE PRODUCED

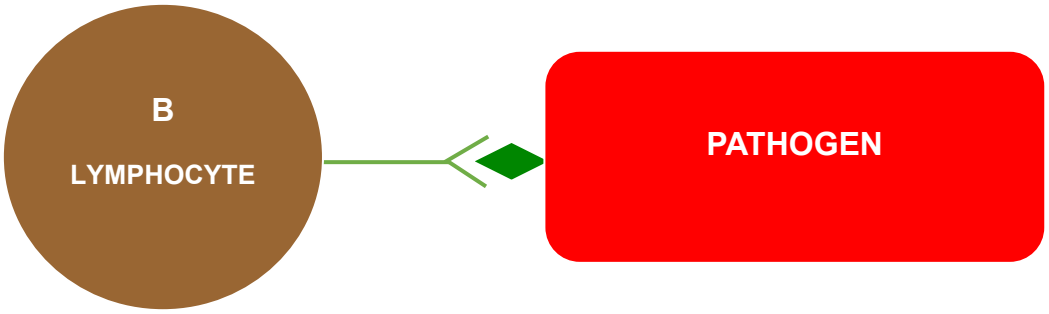
- **Antibodies** are also known as **immunoglobulins**.

1. **Many different types** of **B lymphocytes** exist

Each type **recognises one specific antigen**

They are **stored** in the **lymph nodes**

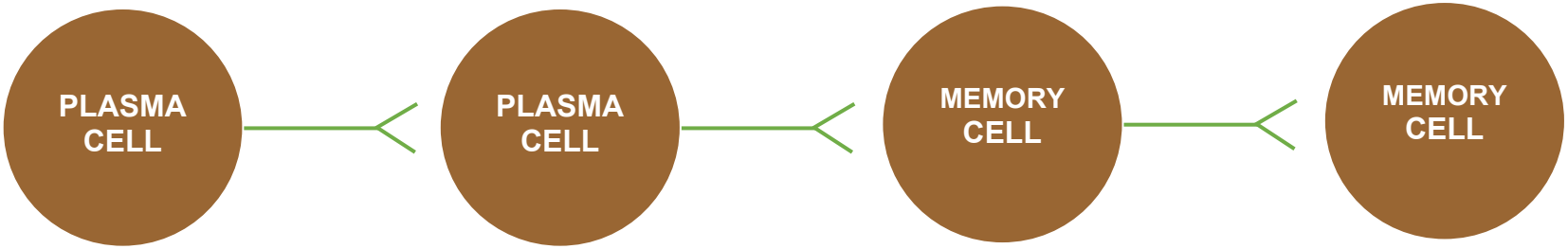
2. A **specific B lymphocyte** that has a **complementary shaped antibody attaches** to the **antigen** on the pathogen



3. **The B-lymphocyte** then **divides by mitosis** to form a **clone** of **cells** that all have the **same-shaped antibody**

Some develop into **plasma cells** and some develop into **memory cells**

= CLONAL SELECTION



4. **PLASMA CELLS produce** and **secrete many specific antibodies** into the **blood** that **destroy** the pathogen
= we **recover**

5. **MEMORY CELLS stay in the blood** for **many years** to give **long-term immunity**. They **clone faster into plasma cells** if the **same antigen** is **met again** at a **later date**
= **more antibodies produced faster** = we **do not get ill**

F. BLOOD CLOTTING

- Blood **proteins** help to form a **clot** to:
  - prevent **further loss of blood**
  - prevent **pathogens** from **entering** the **blood**

START

Platelets or **damaged tissue** release **clotting factors**

such as

prothrombin activator

prothrombin (inactive enzyme) → thrombin (active enzyme)

fibrinogen (soluble) → fibrin (insoluble)

Forms a mesh/net of fibres across wounds

This traps **red** blood cells and dries to form a scab if exposed to air

This:

- **prevents** **further loss of blood**
- **prevents** **pathogens** from **entering** the **blood**

- Like **antibodies** and **lysozyme** enzyme in **phagocytes**, these are all **blood proteins**.
- You **need** to know this for **long-answer questions**, so you can write about all of them.

G. ANTIBIOTICS

What they do

- Chemicals produced by **microorganisms** that **inhibit the growth of** or **kill bacteria**.
- Antibiotics **block processes** that occur in **prokaryotic cells** but not in **eukaryotic cells.**

TARGETS

1. PREVENT CELL WALL SYNTHESIS

- Bacteria will **burst** (lyse)
- Human cells **do not have cell walls**

2. PREVENT PROTEIN SYNTHESIS

- **Eukaryotic ribosomes** (80S) are **structurally different** to **prokaryotic ribosomes** (70S)

3. PREVENT DNA SYNTHESIS

- **Eukaryotic enzymes** are **structurally different** to **prokaryotic enzymes** involved in **DNA replication**

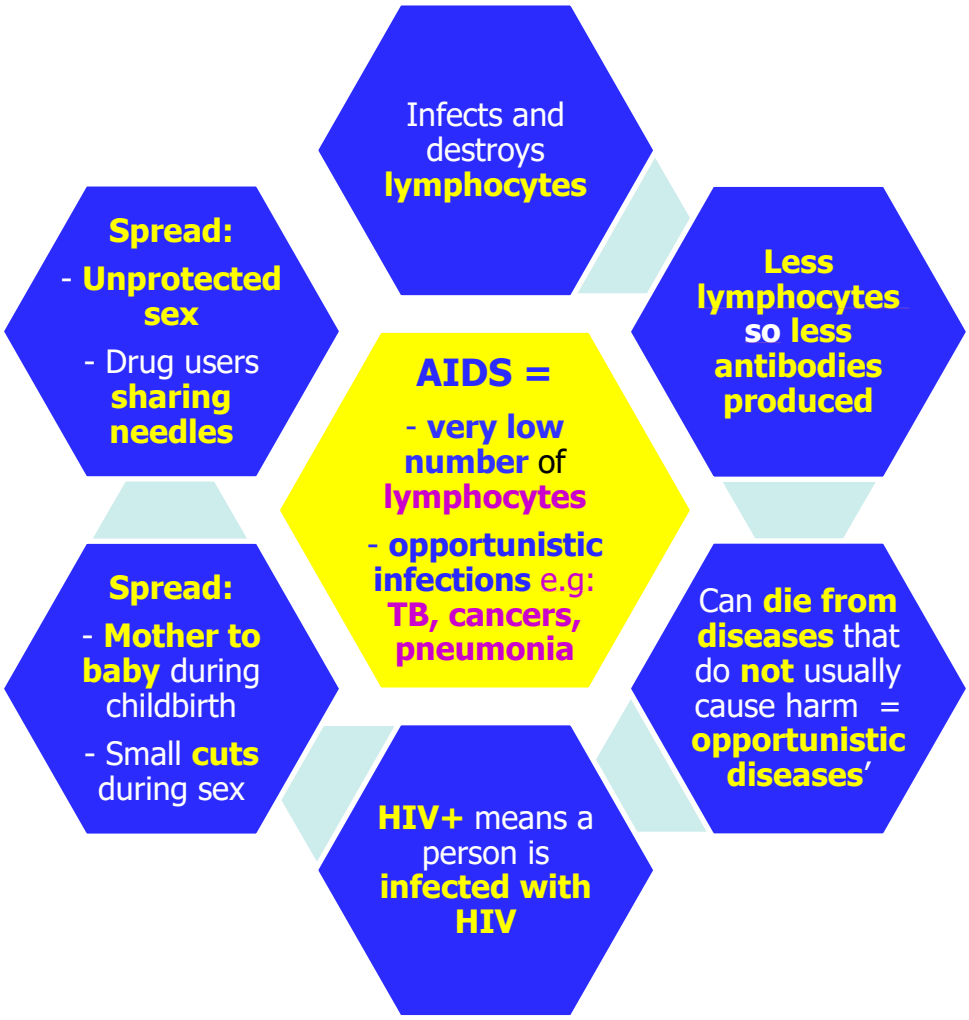
Why do antibiotics kill bacteria but not viruses?

- Viruses **do not have** **organelles** (e.g. cell wall and ribosomes)
- Viruses **do not have** their **own metabolism** (chemical reactions).
- They use the **organelles** in **human cells** to **reproduce**.

There is no way of **inhibiting the virus** without inhibiting human metabolism

H. HUMAN IMMUNODEFICIENCY VIRUS (HIV)

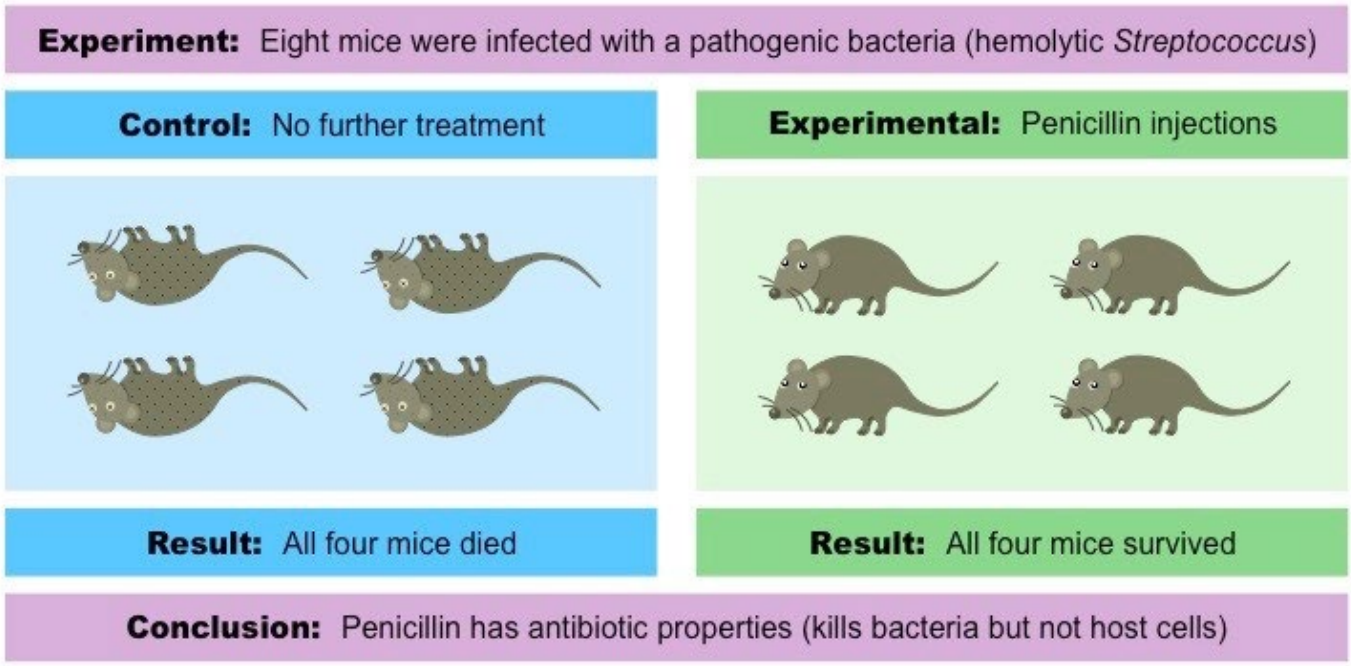
- **HIV** is the **virus** that causes the **disease AIDS**
- **HIV infects** and **destroys white blood cells** called **lymphocytes**
- This means that **less antibodies** are **produced**
- Meaning, people with **AIDS** can **die** of **diseases** that **do not usually harm us** = **'opportunistic infections'**



- **Prevention methods** include:
  - **free condoms**
  - **free needles for drug users**
  - **test blood for HIV**
  - **free and anonymous HIV tests**
  - **education** about **HIV** and **AIDS**
  - **infected pregnant mothers** take **anti-HIV drugs** during **pregnancy**

I. FLOREY & CHAIN'S EXPERIMENTS

- They tested the ability of the antibiotic **penicillin** to treat **bacterial infections** in **mice**.



- Nowadays, the **ethics** of this may be questioned.
- However, it has led to the development of several **synthetic derivatives** of penicillin e.g. amoxycillin, which offer **extra benefits** including **killing a greater variety** of bacteria, **greater stability** and **greater tolerance**.

I. ALEXANDER FLEMING - THE ACCIDENTAL DISCOVERY OF PENICILLIN

- The discovery of penicillin was a fortuitous **accident**, resulting from the **accidental contamination** of a **dish** containing the bacterial pathogen ***Staphylococcus aureus***.
- A ***Penicillium* mould** (fungus) began to **grow** on the **plate** and a **halo** of **inhibited bacterial growth** was observed **around** the **mould**
- Fleming concluded that **the mould was releasing a substance (penicillin) that was killing** the nearby **bacteria**.