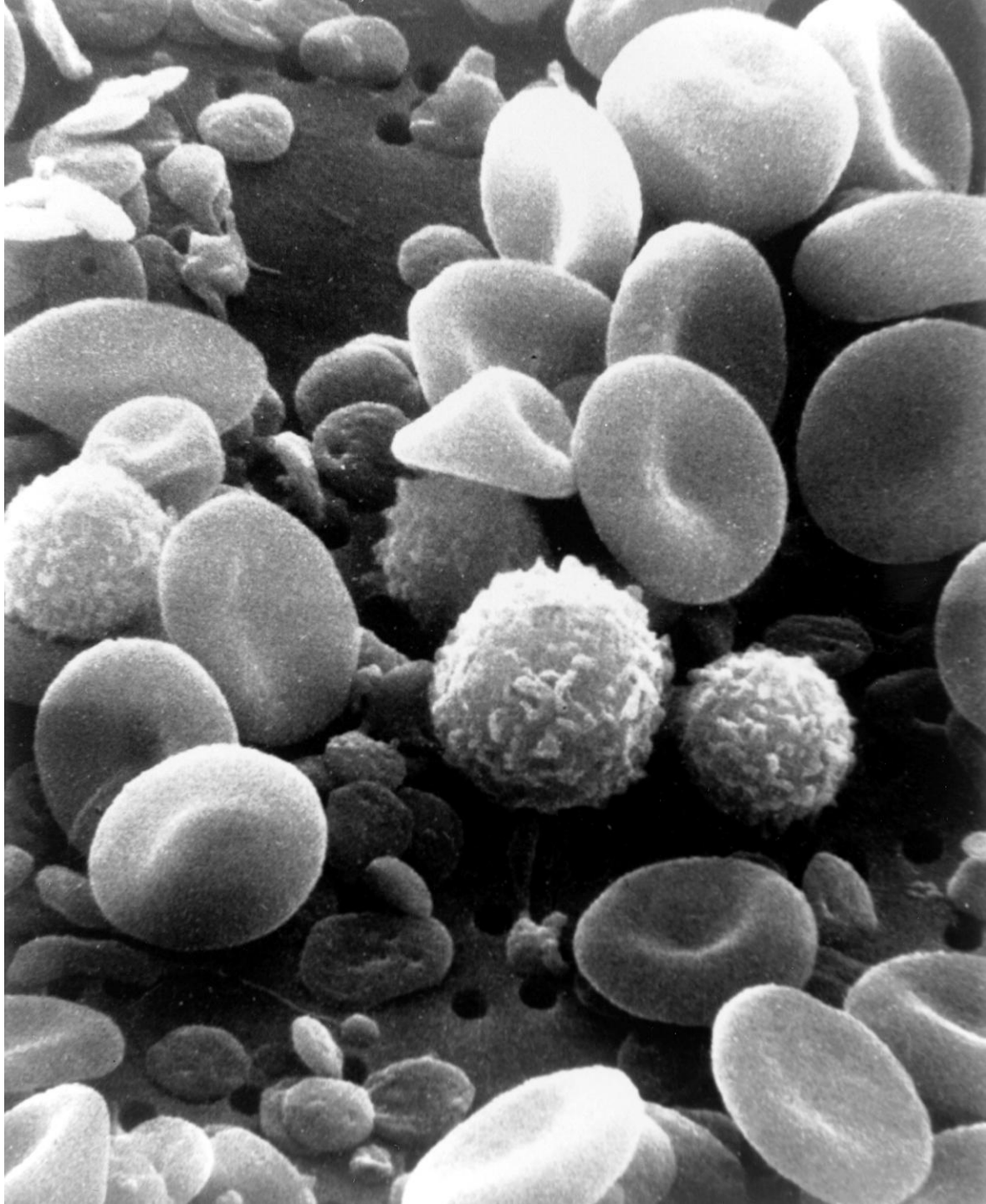
DP Biology – C3.2

# Defence against disease

- How does the body detect pathogens and fight infection?
- What affects how common a disease is in a population?

SL and HL. Textbook pp. 517–547.
Image: human blood cells, scanning electron micrograph. National Cancer Institute, public domain.



# Objectives for each class

| Class | Statements | By the end of the class, you can… |
|---|---|---|
| **1** | C3.2.1–3 | Identify groups of pathogen, and explain skin, mucous membranes and clotting as defences |
| **2** | C3.2.4–7 | Compare innate and adaptive immunity, and describe phagocytes, lymphocytes and antigens |
| **3** | C3.2.8–10 | Explain how B-cells are activated, form clones and give lasting immunity |
| **4** | C3.2.11–14 | Explain how HIV leads to AIDS, why antibiotics fail on viruses and how resistance evolves |
| **5** | C3.2.15–18 | Explain zoonoses, vaccines and herd immunity, and evaluate pandemic data |

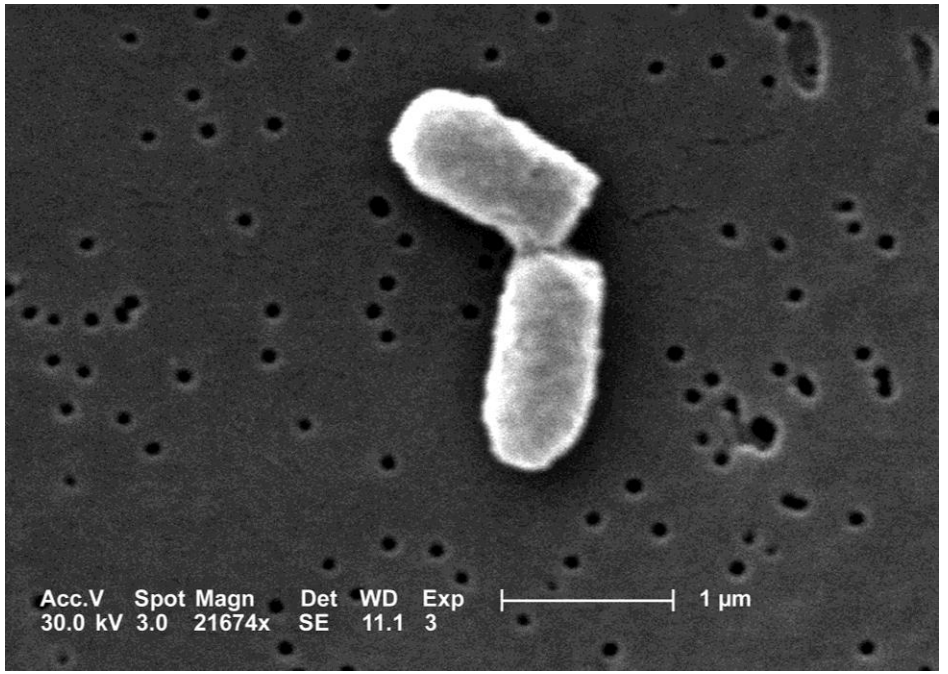
# Overview of this chapter

| Part | Statements | What it covers |
|---|---|---|
| **Getting in** | C3.2.1–3 | Pathogens, skin and mucous membranes, blood clotting |
| **Fighting back** | C3.2.4–10 | Phagocytes, lymphocytes, antibodies and memory cells |
| **When defence fails** | C3.2.11–12 | How HIV is transmitted and how it leads to AIDS |
| **Medicines** | C3.2.13–14 | How antibiotics work and how resistance evolves |
| **Crossing species** | C3.2.15 | Zoonoses, from tuberculosis to COVID-19 |
| **Protecting populations** | C3.2.16–18 | Vaccines, herd immunity and pandemic data |

# Pathogens cause infectious diseases



- A **pathogen** is an organism or virus that causes disease
- The infected organism is the **host**
- **Infectious** diseases can pass from one host to another
- Cancer, malnutrition and inherited conditions are not infectious
- Archaea are not known to cause disease in humans



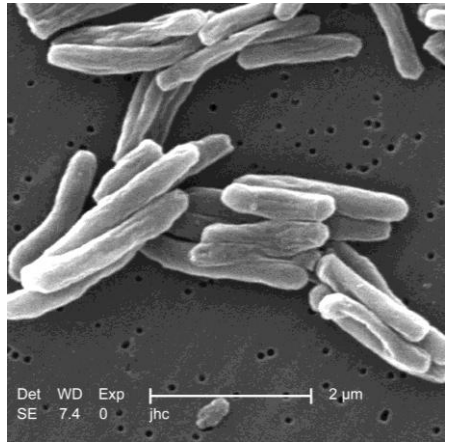

Escherichia coli dividing, scanning electron micrograph. CDC, public domain.

## Activity

Pathogens have to travel between hosts. List as many routes as you can, with one disease for each. Think about air, water, food, touch, body fluids and animal vectors.

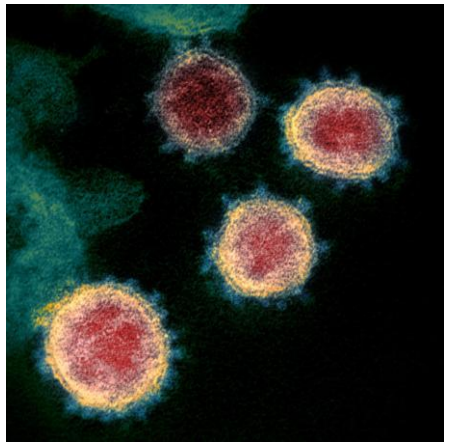
# Five groups of pathogen





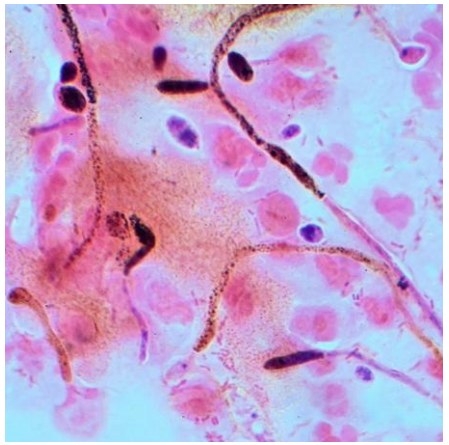

## Bacteria

- Prokaryotes
- Peptidoglycan wall
- Cholera, tuberculosis



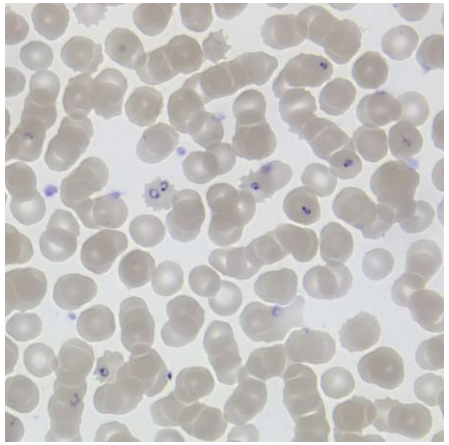
## Viruses

- Not cells
- Replicate only in host cells
- Influenza, measles, HIV



## Fungi

- Eukaryotes
- Chitin cell wall
- Athlete's foot, thrush



## Protists

- Single-celled eukaryotes
- Often carried by vectors
- Malaria



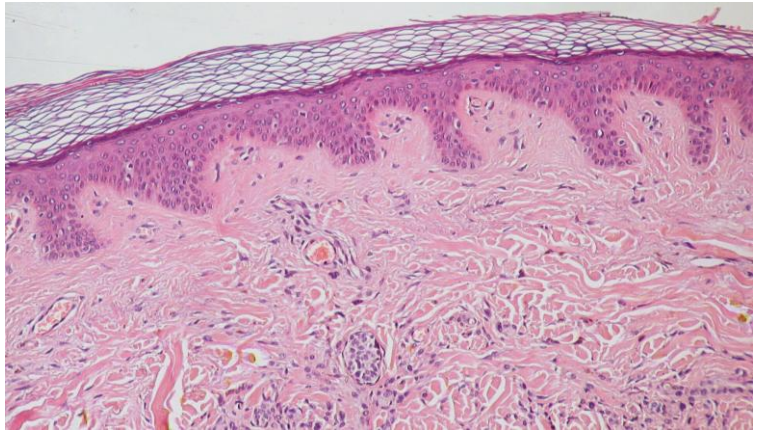
## Parasitic worms

- Multicellular eukaryotes
- Flatworms, roundworms
- Schistosomiasis

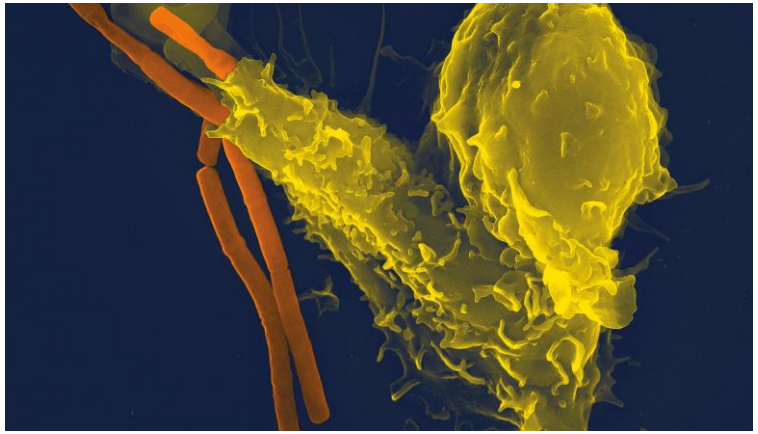
Images, left to right: Mycobacterium tuberculosis (CDC); SARS-CoV-2 (NIAID, CC BY 2.0); Candida albicans (Graham Beards, CC BY-SA 4.0); Plasmodium falciparum in blood (Michael Zahniser, public domain); Schistosoma (David Williams, public domain).

# Three lines of defence

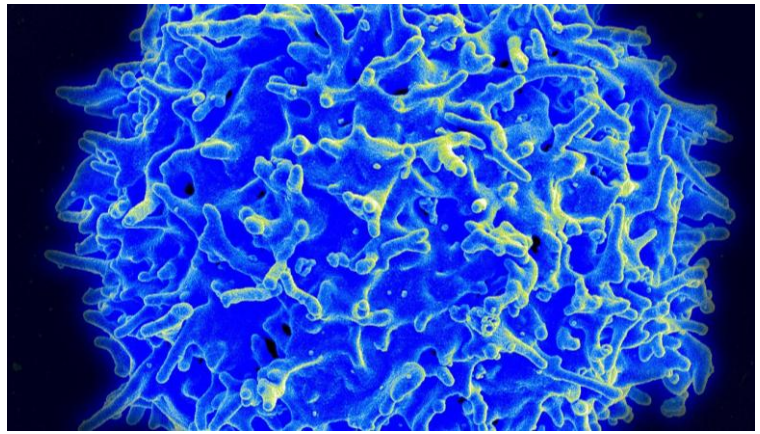
C3.2.4



Skin section. Kilbad, public domain.



Neutrophil engulfing bacteria. Volker Brinkmann, CC BY 2.5.



Human T-lymphocyte. NIAID/NIH, public domain.

## 1. Surface barriers

- Keep pathogens out
- Skin and mucous membranes
- Non-specific

## 2. Innate system

- Starts within hours
- Phagocytes engulf pathogens
- Non-specific

## 3. Adaptive system

- Days at first exposure
- Lymphocytes make antibodies
- Specific, with memory

**Key idea**

Only the third line improves with experience, because it builds a memory of the pathogens it meets.

# Skin: the outer barrier

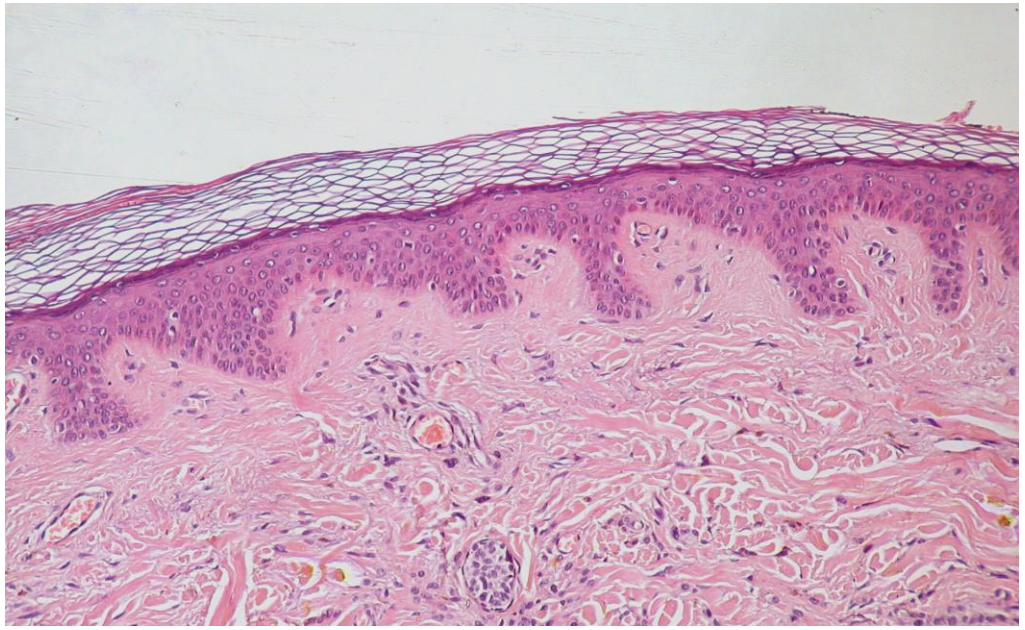
C3.2.2

## Physical

- Outer layer of dead, **keratin**-filled cells
- Lipid seals the gaps between cells
- Shedding removes microbes from the surface

## Chemical

- **Sebum** keeps skin moist and lowers the pH
- Low pH slows the growth of bacteria and fungi
- **Lysozyme** in sweat damages bacterial walls



Human skin, stained section: the pale layer at the top is dead, keratinized cells. Kilbad, public domain.

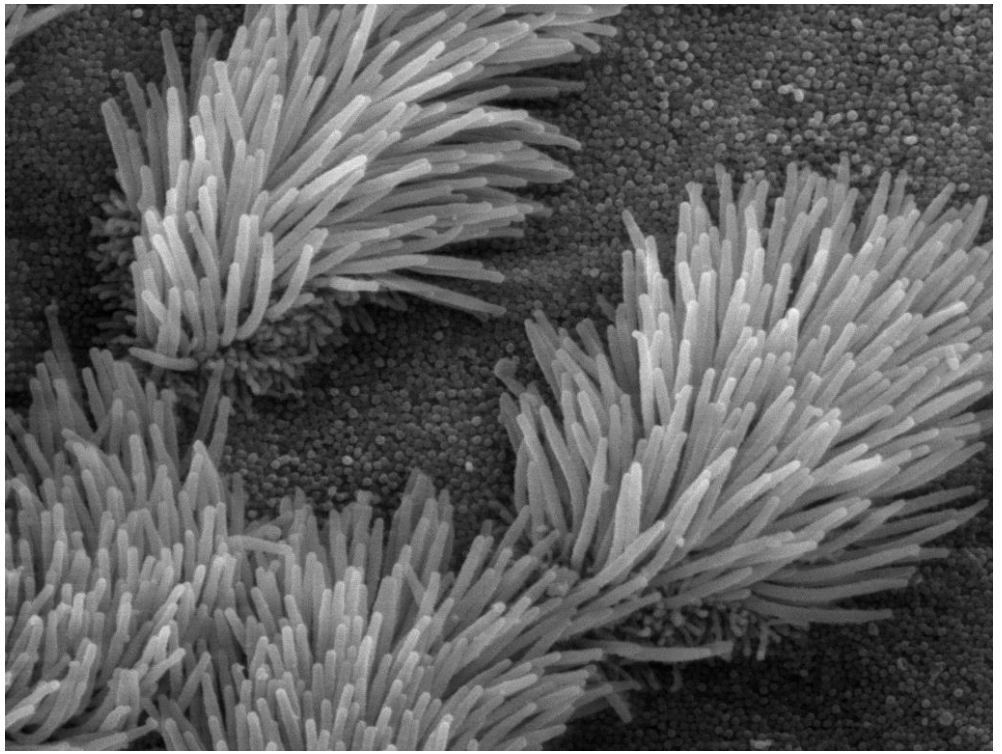
### Exam note

You do not need to draw or label a diagram of skin. You should be able to explain how it acts as both a physical and a chemical barrier.

# Mucous membranes: protecting the inside

C3.2.2

- Line the airways, gut and other body openings
- Thin, moist and living, so more vulnerable than skin
- Sticky **mucus** traps particles and contains lysozyme
- **Cilia** sweep mucus up the airway to the throat
- Mucus is then swallowed or coughed out
- Stomach acid kills many swallowed microbes



Cilia on the lining of the trachea, scanning electron micrograph. Charles Daghlian, public domain.

**Activity**

Suggest why people whose cilia are damaged by smoking get more chest infections.

# Blood clotting seals cuts

C3.2.3

Platelets and damaged tissue release **clotting factors**

↓

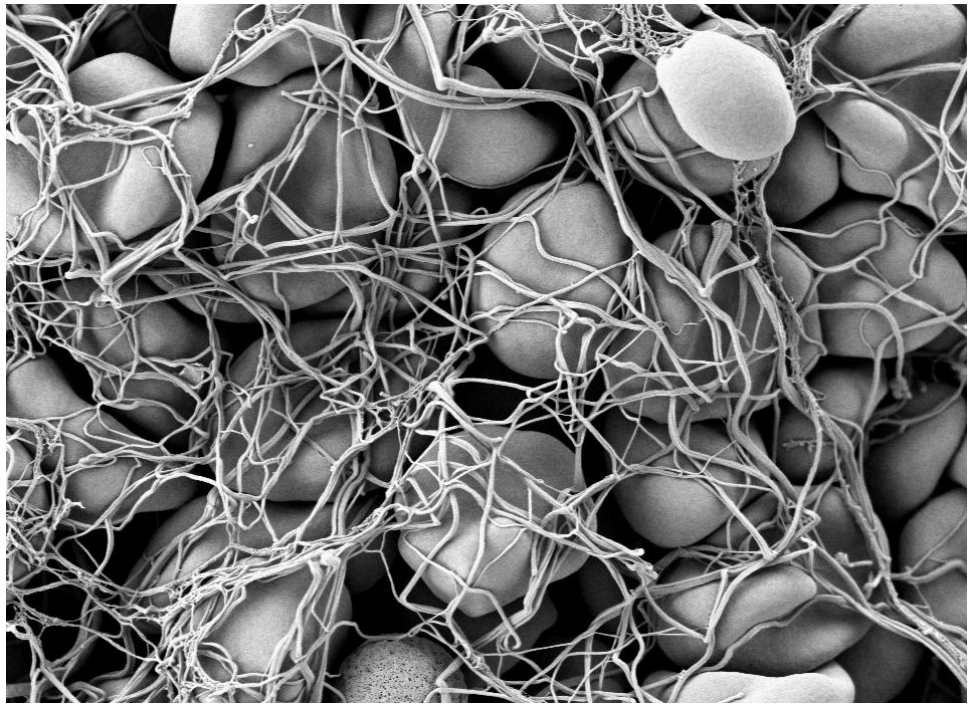
Prothrombin is converted to **thrombin**, an enzyme

↓

Thrombin converts soluble fibrinogen to insoluble **fibrin**

↓

Fibrin fibres form a mesh that traps **erythrocytes**



A real clot: red blood cells trapped in a fibrin network, scanning electron micrograph. Fuzis, CC BY-SA 4.0.

## Common mistake

Fibrinogen and fibrin are not the same: fibrinogen is soluble and fibrin is not. Write that a clot prevents the entry of pathogens, not that it stops diseases getting in.

# Innate and adaptive systems compared

C3.2.4

| | Innate system | Adaptive system |
|---|---|---|
| **Targets** | Broad categories of pathogen | One specific pathogen |
| **Over a lifetime** | Does not change | Builds a memory of pathogens met |
| **Second exposure** | Same response as the first | Faster and much stronger |
| **Cells to know** | Phagocytes | Lymphocytes (B-cells and helper T-cells) |
| **Speed** | Within hours | Days at first exposure |

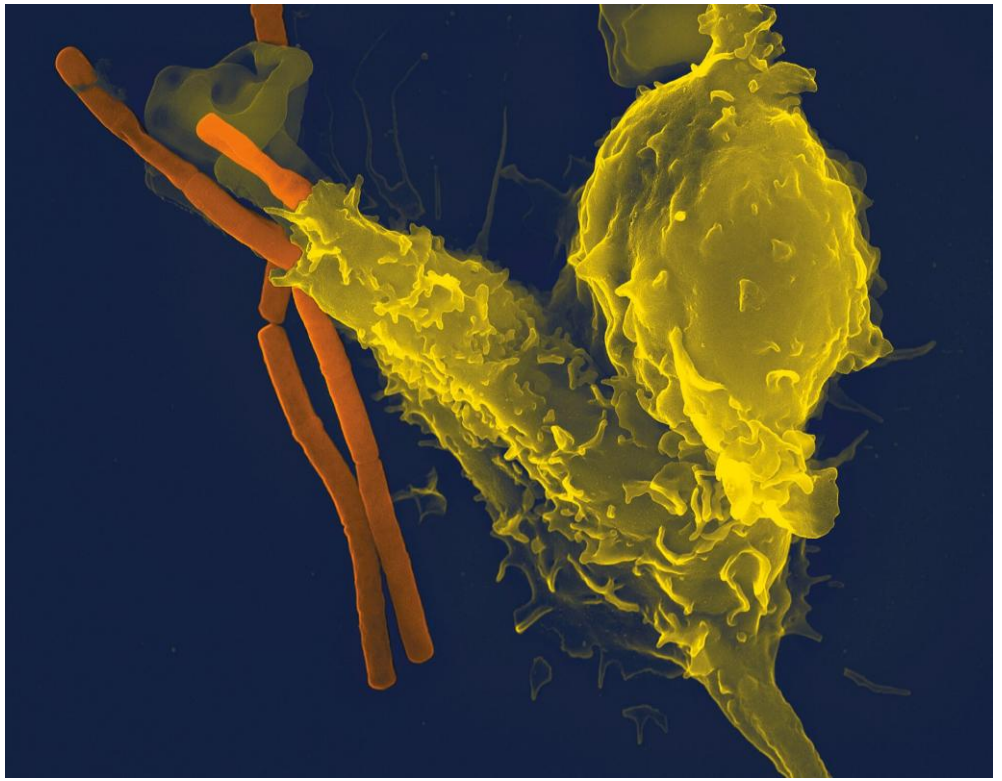
**Exam note**

The only innate component you must know is the phagocyte. Both types of white blood cell circulate in the blood.

# Phagocytes engulf and digest pathogens

C3.2.5

1. Move from blood to the infection by **amoeboid movement**
2. Recognise the **pathogen**
3. Engulf it by **endocytosis**, forming a vesicle
4. A **lysosome** fuses with the vesicle
5. Lysosomal enzymes **digest** the pathogen



Neutrophil (yellow) engulfing anthrax bacteria (orange), coloured scanning electron micrograph. Volker Brinkmann, CC BY 2.5.

**Key idea**

Phagocytes respond to broad categories of pathogen, not one specific type.

# Lymphocytes cooperate to make antibodies

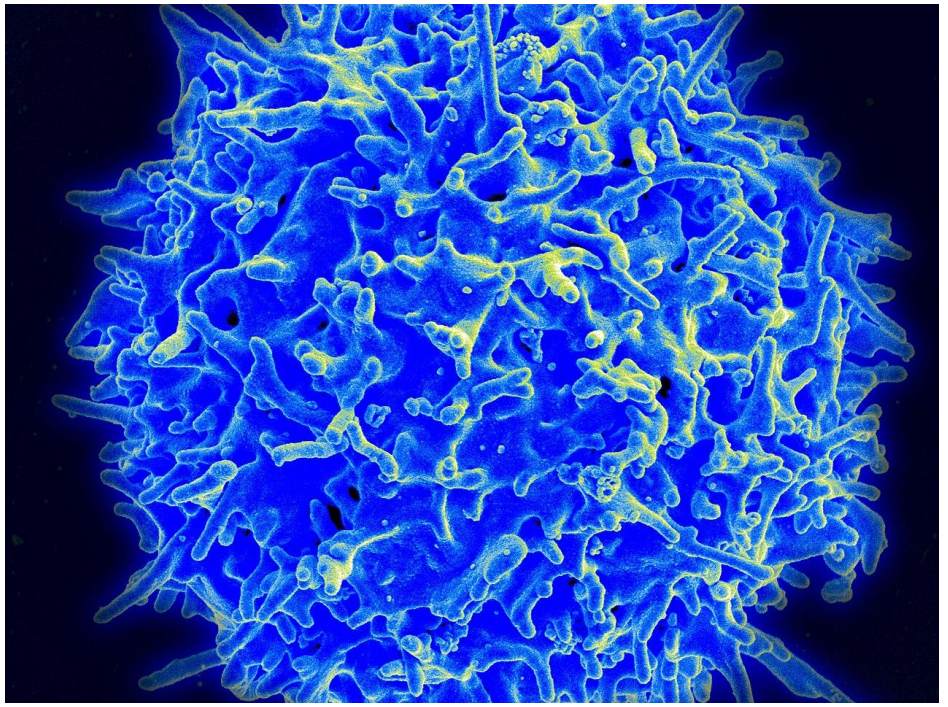
C3.2.6

## B-cells

- Each type makes one specific antibody
- A very large number of different types exist
- Only a few of each type are present at first

## Helper T-cells

- Each type recognises one antigen
- They do not make antibodies
- Their role is to activate matching B-cells



Human T-lymphocyte, scanning electron micrograph. NIAID/NIH, public domain.

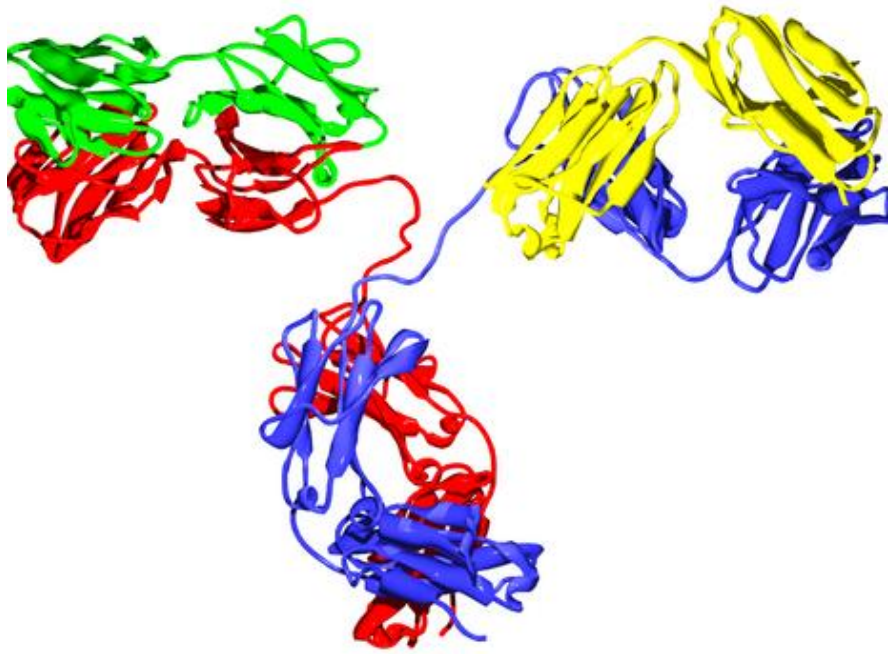
### Key idea

Lymphocytes circulate in the blood and collect in lymph nodes. So many types exist that the body can respond to almost any antigen, but so few match each one that they must multiply first.

# Antigens trigger antibody production

C3.2.7

- An **antigen** is a molecule recognised as foreign
- Usually a glycoprotein or other protein on the outer surface of a pathogen
- An **antibody** is a Y-shaped protein made by B-cells
- The binding site at the tip of each arm fits one antigen



Structure of an antibody, about 10 nm tall, built from crystal structure data (PDB 1IGT). TimVickers, public domain.

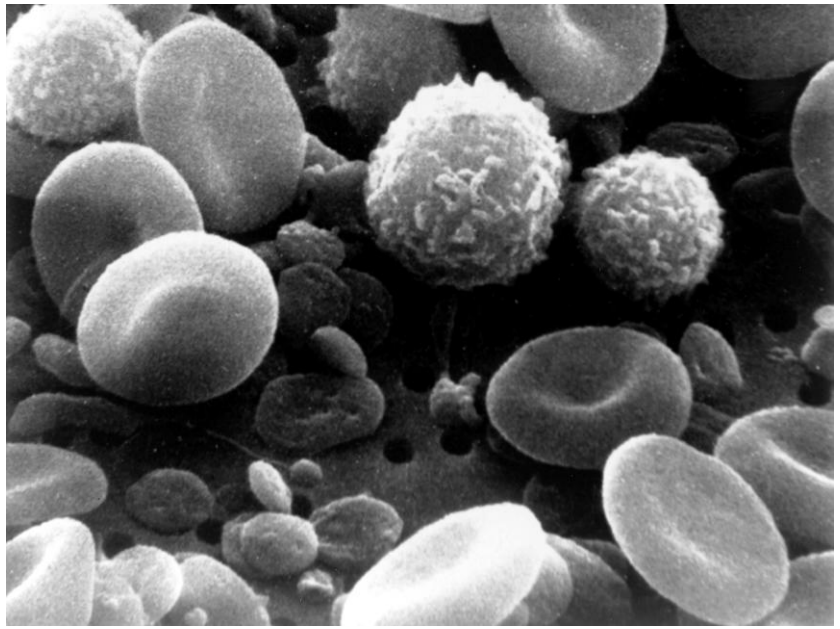
**Common mistake**

Antigen and antibody sound alike. Antigens are on the invader and trigger the response. Antibodies are the proteins your body makes in reply.

# Blood groups are antigens too



| Group | Antigens on red cells | Antibodies in plasma |
|---|---|---|
| **A** | A | Anti-B |
| **B** | B | Anti-A |
| **AB** | A and B | None |
| **O** | None | Anti-A and anti-B |



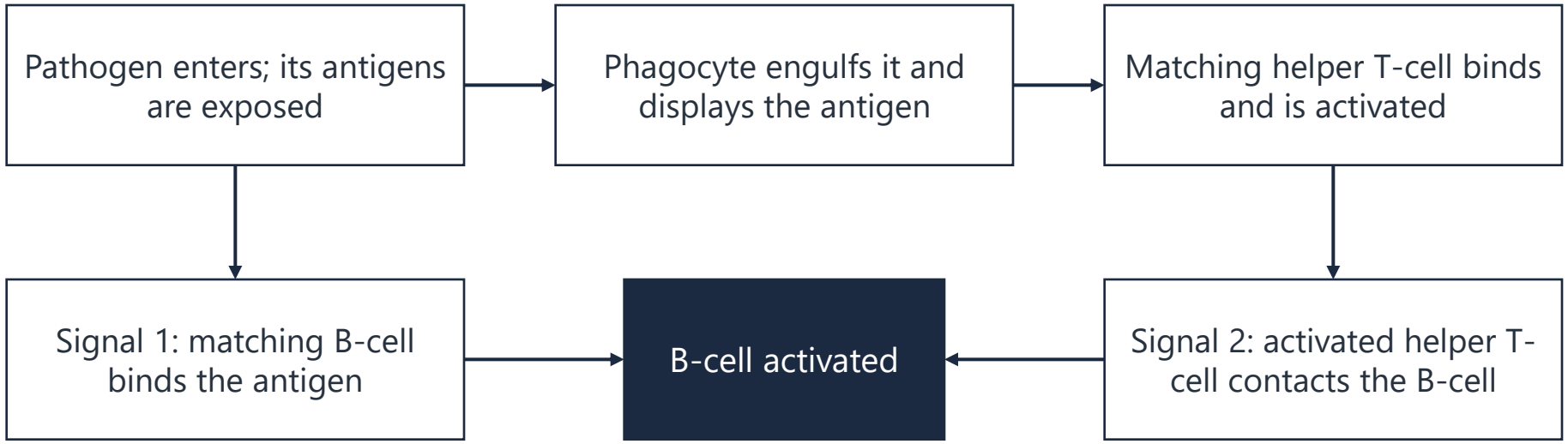
Red blood cells carry these antigens on their surface. National Cancer Institute, public domain.

## Activity

A patient with group A blood needs red blood cells. Which donor groups would be safe, and why? Explain why group O red cells can usually be given to any patient.

# B-cells need two signals

C3.2.8




**Rule**

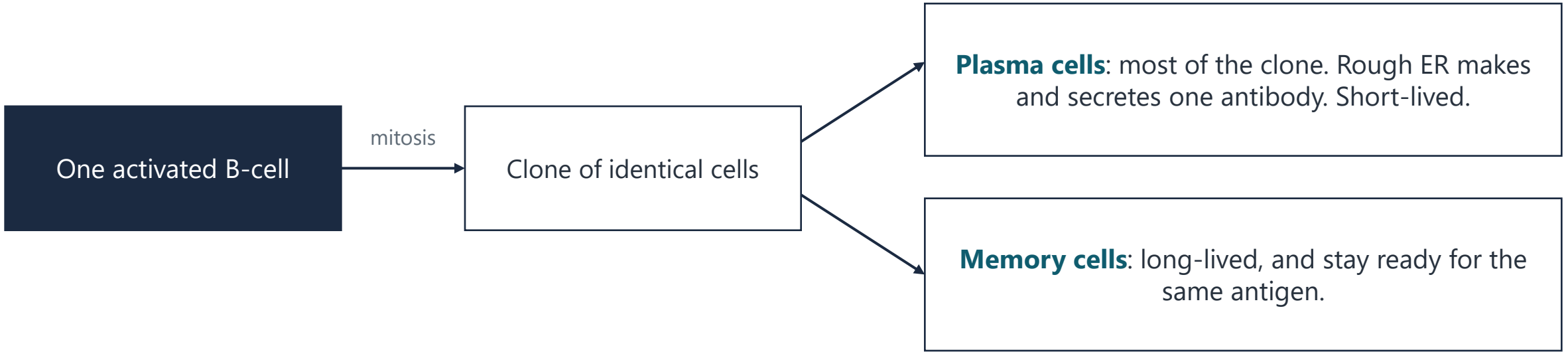
Both signals must relate to the same antigen.

**Common mistake**

Helper T-cells do not secrete antibodies; plasma cells do. A B-cell makes antibodies and memory cells only after it has been activated.

# Activated B-cells multiply

C3.2.9

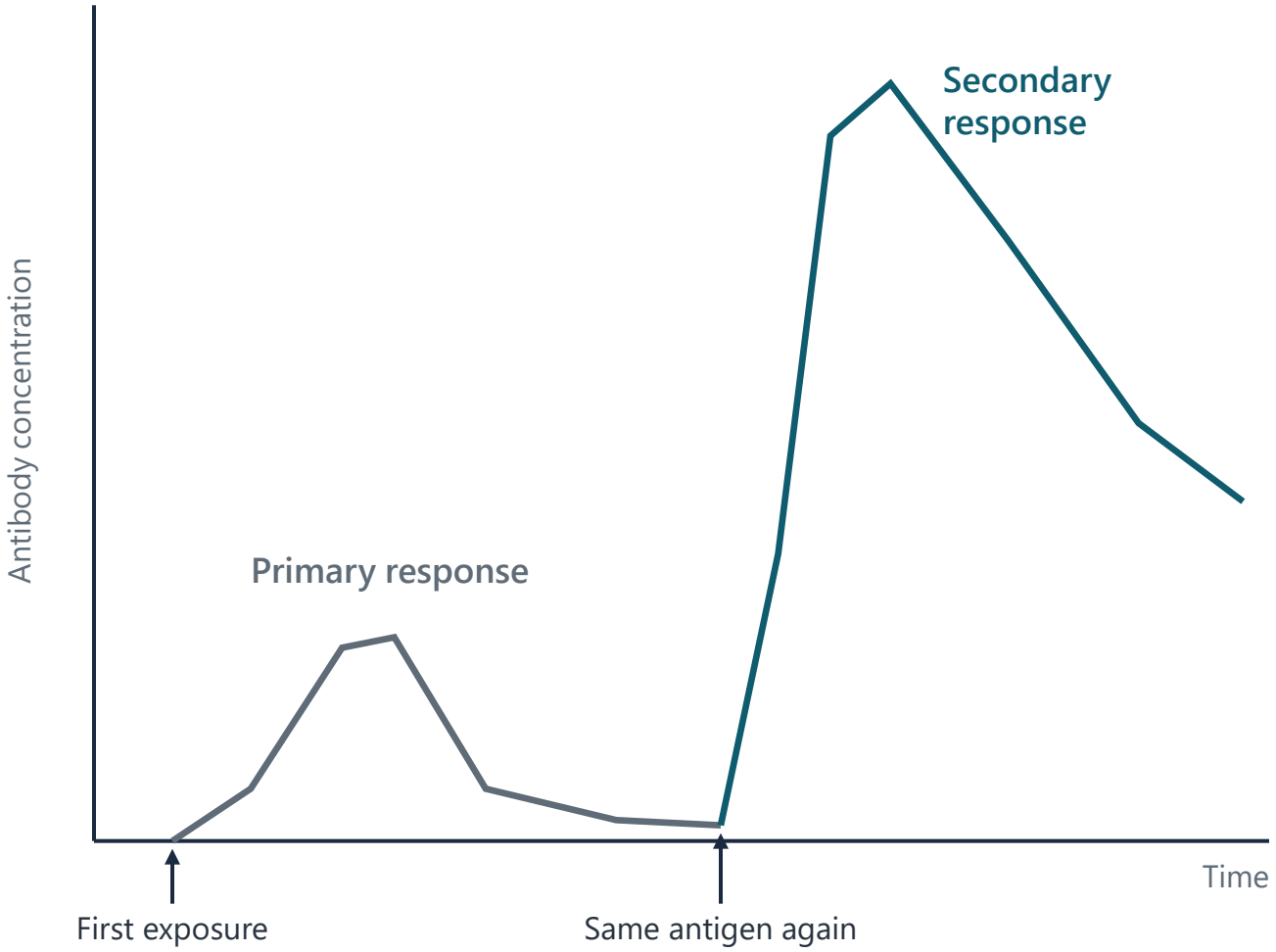



## Key idea

Very few B-cells match any one antigen, far too few to make enough antibody. Dividing first solves this: every plasma cell in the clone secretes the same antibody. This is clonal selection.

# Memory cells give immunity






Schematic, not to scale.

- **Immunity** is the ability to eliminate an infectious disease from the body
- **Memory cells** survive long after plasma cells have gone
- On re-exposure, antibodies are made faster and at a higher level

## Activity

Compare the two curves. Which steps of plasma cell formation are skipped the second time?

# Transmission of HIV

C3.2.11

## HIV passes in body fluids

- Unprotected sexual intercourse
- Shared hypodermic needles
- Mother to baby, including breastfeeding
- Unscreened transfusions or transplants

## HIV does not pass by

- Sharing a glass, plate or towel
- Saliva on a drinking glass
- Hugging or shaking hands
- Mosquito bites

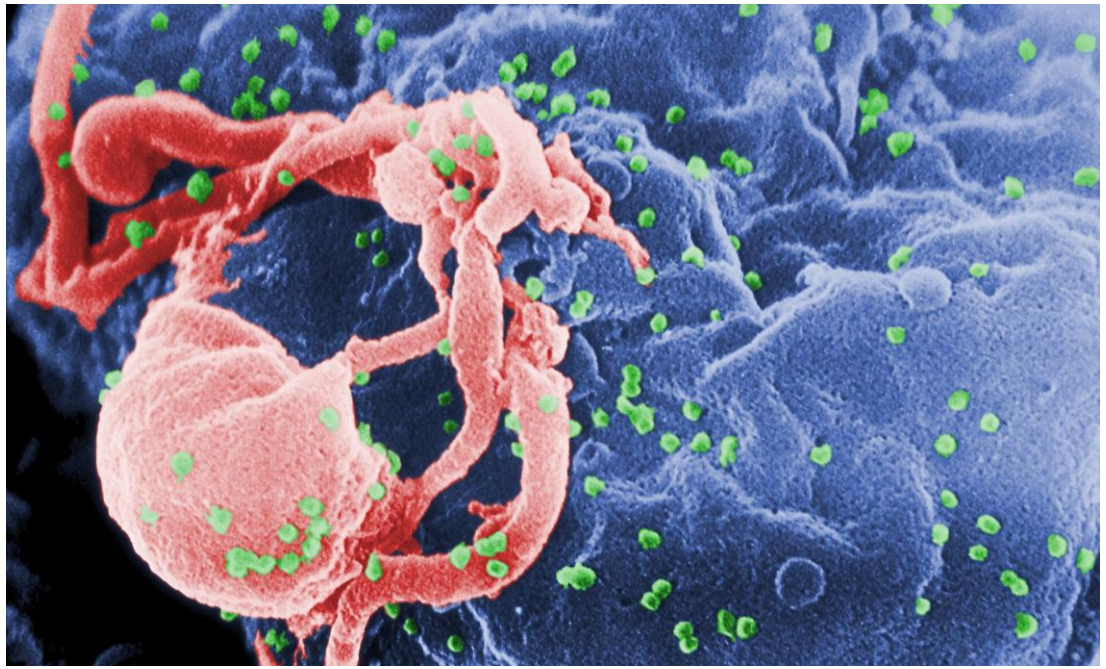
### Implications

Condoms, screening of donated blood and clean needles all interrupt transmission. Knowing the real routes also reduces stigma. Write ‘HIV is transmitted’, never ‘AIDS is transmitted’: HIV is the virus, and AIDS is the condition it can cause.

# HIV infects helper T-cells



1. HIV binds to **CD4** receptors on a helper T-cell
2. Its RNA core enters the cell
3. The enzyme **reverse transcriptase** copies RNA into DNA
4. Viral DNA is inserted into a host chromosome
5. New viruses are made; infected cells are lost



HIV particles (green) budding from a lymphocyte, coloured scanning electron micrograph. CDC, public domain.

### From HIV to AIDS

Fewer helper T-cells mean fewer B-cells are activated, so too few antibodies are made. Opportunistic infections then take hold. This stage is acquired immunodeficiency syndrome (AIDS).

# Antibiotics target bacteria, not human cells

C3.2.13

| Process targeted | In bacteria | In human cells |
|---|---|---|
| **Cell wall synthesis** | Peptidoglycan wall weakens; cell bursts | No cell wall to damage |
| **Protein synthesis** | 70S ribosomes are blocked | Cytoplasmic 80S ribosomes unaffected |
| **DNA and metabolism** | Bacterial enzymes are blocked | Different enzymes, so unaffected |



Penicillium culture, a source of penicillin. Crulina 98, CC BY-SA 3.0.

## Key idea

Antibiotics are safe for patients because prokaryotes and eukaryotes differ. Penicillin, from a fungus, was the first in wide use.

## Activity

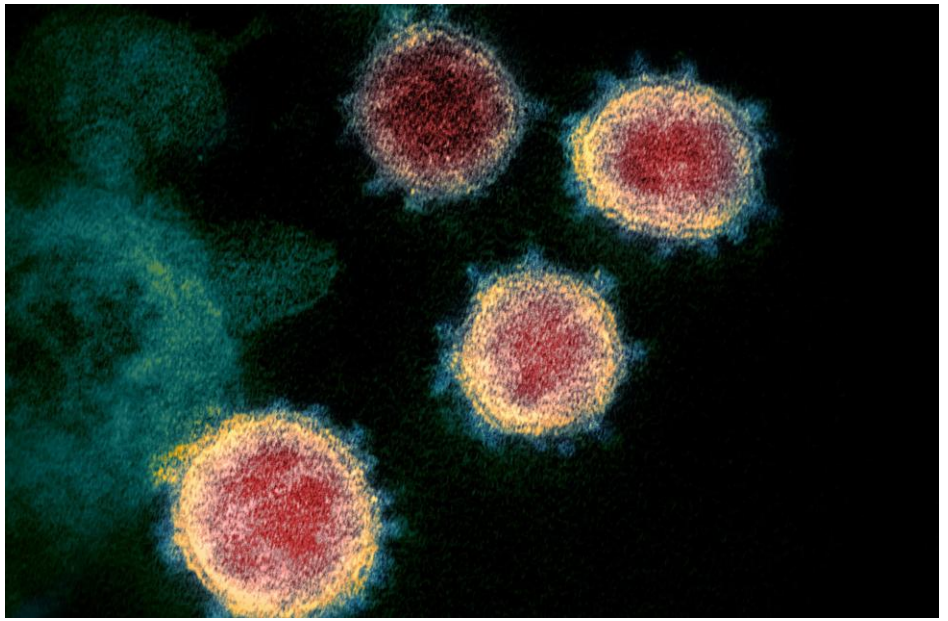
Watch the antibiotics video. Then explain in one sentence why a drug can kill bacteria inside a patient without harming the patient.

# Why antibiotics fail against viruses



- Viruses have **no cell wall**
- They have **no ribosomes** of their own
- They have **no metabolism** of their own
- They use the host cell to make new virus parts

Antiviral drugs are different: they interfere with a viral step, such as reverse transcriptase in HIV.



SARS-CoV-2 particles on a cell, coloured transmission electron micrograph. NIAID, CC BY 2.0.

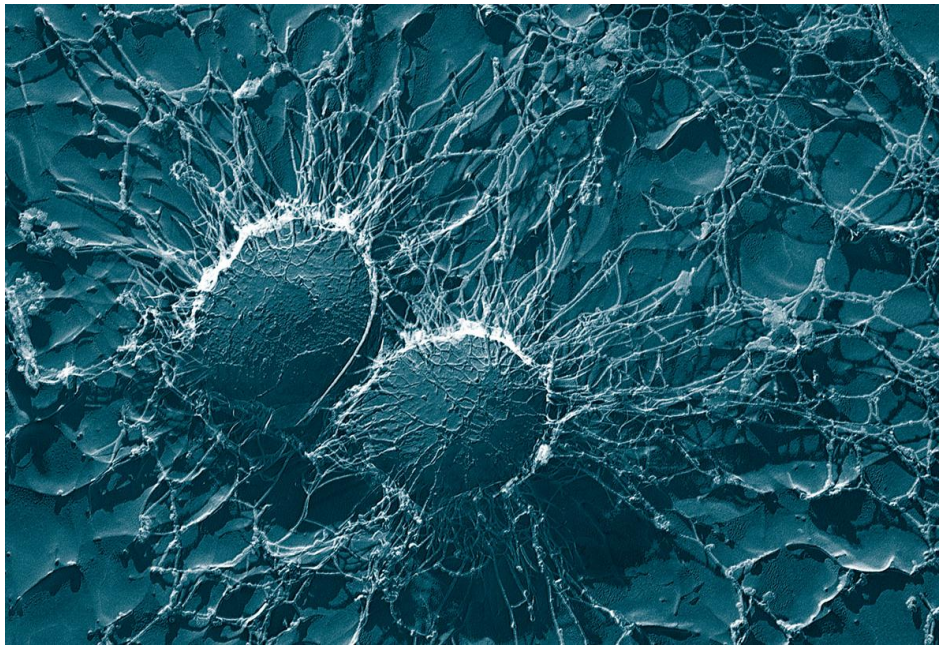
**Common mistake**

Do not write that antibiotics fail because viruses 'hide inside cells'. They fail because they act on bacterial structures and enzymes, which viruses do not have. Antibiotics do nothing for a cold or flu, and using them helps resistance spread.

# Resistance evolves by natural selection

C3.2.14

1. A chance **mutation** makes a bacterium resistant
2. The antibiotic kills susceptible bacteria
3. The resistant cell survives: a **selective advantage**
4. It reproduces with less competition
5. The population becomes resistant



Staphylococcus aureus, the species that includes MRSA, coloured scanning electron micrograph. USDA, public domain.

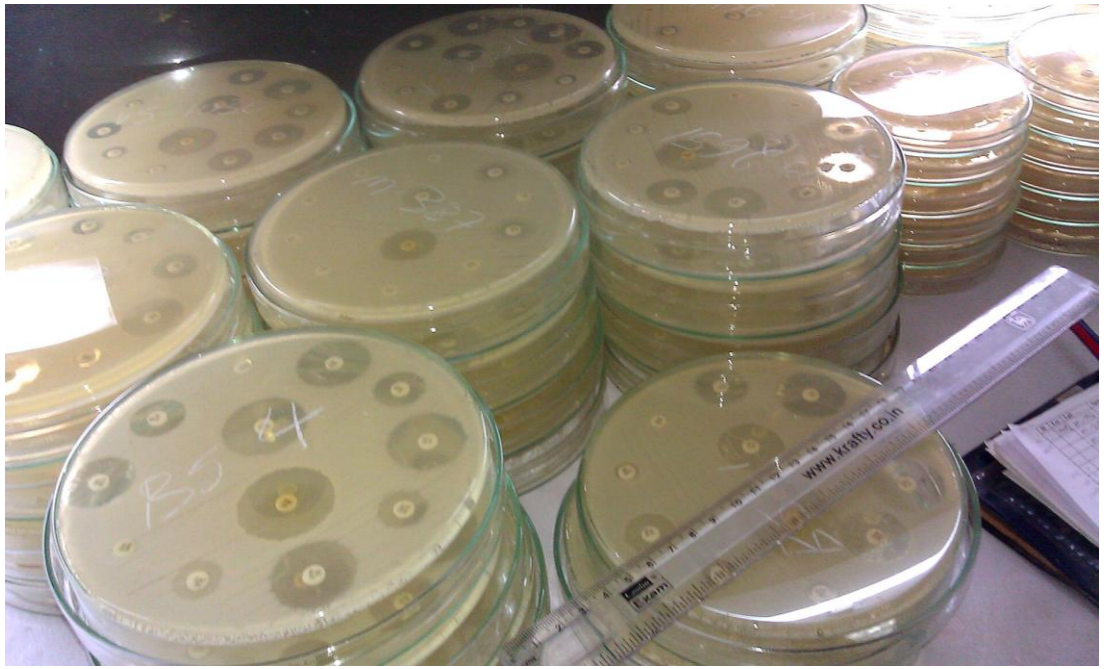
## Key idea

The antibiotic does not cause the mutation; it selects for cells that already carry it. Genes on plasmids can pass between bacteria, so a strain can gather several resistances. MRSA is a well-known multiresistant example.

# Testing resistance in the laboratory

C3.2.14

- Bacteria are spread across agar as a lawn
- Each paper disc contains a different antibiotic
- A **clear zone** around a disc: growth was stopped
- **No clear zone**: the strain is resistant to it
- A multiresistant strain shows few clear zones



Disc diffusion plates with clear zones around the antibiotic discs. Microrao, CC BY-SA 4.0.

### Activity

A patient's bacteria grow right up to all but one disc. Which antibiotic would you try, and what would concern you? Why might a wider clear zone not always mean a better medicine?

# Slowing down resistance

C3.2.14

| Who | What they can do |
|---|---|
| **Doctors** | Prescribe antibiotics only for bacterial infections, and only when needed |
| **Patients** | Take the full course; never share or save antibiotics |
| **Hospitals** | Keep hygiene high to stop resistant strains spreading |
| **Farmers** | Avoid routine use, such as to boost animal growth |

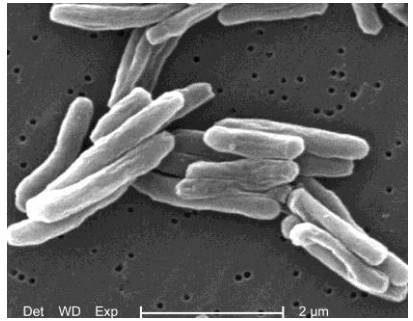
### Key idea

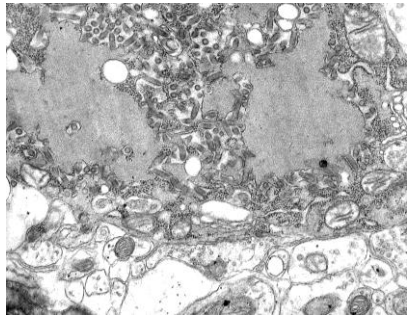
Careful use slows the emergence of multiresistant bacteria. This matters because new antibiotics are hard to find: far fewer are approved now than in the 1980s.

# Zoonoses cross from animals to humans

C3.2.15

| Disease | Pathogen | Animal source | Route to humans |
|---|---|---|---|
| **Tuberculosis** | Bacterium (M. bovis) | Cattle | Raw milk; infected tissue |
| **Rabies** | Virus | Dogs, bats, other mammals | Bite; saliva on broken skin |
| **Japanese encephalitis** | Virus | Pigs, wading birds | Bite of infected mosquito |
| **COVID-19** | Virus (SARS-CoV-2) | Probably bats; route unconfirmed | Now spreads between humans |





M. tuberculosis (CDC, public domain); rabies virus in tissue (CDC/F. Murphy, public domain); Culex mosquito (S. Hinkley and K. Walker, Museum Victoria, CC BY 3.0 AU).

### Why are zoonoses rising?

Closer contact with wild and farmed animals, habitat loss and faster travel all give pathogens more chances to cross and spread.

### Activity

Research one more zoonosis, such as Lyme disease: pathogen, source and route.

# Vaccines stimulate immunity

C3.2.16

## What a vaccine contains

- Weakened or inactivated pathogen, or purified antigens
- **DNA or mRNA** coding for an antigen

## How it works

1. Antigen starts a **primary response**, without the disease
2. Long-lived **memory cells** remain
3. The real pathogen meets a fast secondary response



A microbiologist handling influenza virus in a high-containment laboratory. James Gathany, CDC, public domain.

### Exam note

Vaccines contain antigens, or nucleic acids coding for antigens. They give immunity to a specific pathogen without causing the disease. Not all are injected: some are given by mouth or nasal spray.

# Active and passive immunity

C3.2.16

| | Active | Passive |
|---|---|---|
| **Antibodies** | Made by your own B-cells | Received from another source |
| **Memory cells** | Yes, long-lasting | No, protection fades |
| **Natural example** | Recovering from an infection | Maternal antibodies across the placenta |
| **Artificial example** | Vaccination | Injection of ready-made antibodies |

### Beyond the syllabus

This distinction is not in the C3.2 statements, but the textbook covers it and it makes vaccination easier to explain. Smallpox, declared eradicated in 1980, is the best-known success of a vaccination programme.

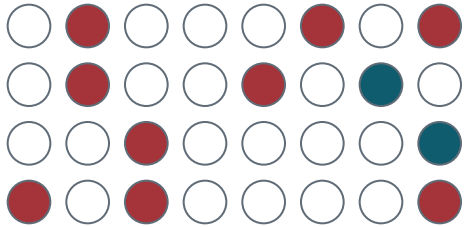
# Herd immunity

C3.2.17

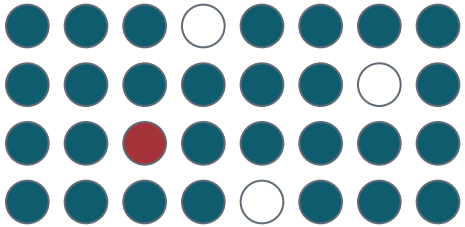


People on the Brooklyn Bridge. Members of a population depend on one another. EazyDee107, CC0.

**Few immune: infection spreads**



**Most immune: infection stalls**



○ Not immune ● Infected ● Immune

The percentage needed is higher for very contagious diseases: up to about 95% for measles.

## Key idea

When enough people are immune, a pathogen rarely reaches a susceptible person, so transmission is greatly impeded. This protects people who cannot be vaccinated, such as those with weakened immune systems.

# Two calculations for pandemic data

C3.2.18

## Percentage change

**(new − original) / original × 100**

Compares one value over time, or a vaccine with a placebo.
A vaccine trial recorded 162 cases with placebo and 8 with vaccine, in groups of similar size:
(162 − 8) / 162 × 100 = 95.1% fewer cases

## Percentage difference

**difference / average × 100**

Compares two values at the same time.
Invented example: 150 and 90 cases per 100 000 in two countries:
(150 − 90) / 120 × 100 = 50%

### Common mistake

Check which one the question asks for. For percentage change, divide by the original value. For percentage difference, divide by the average of the two.

# Evaluate real COVID-19 data

C3.2.18

## Activity: WHO dashboard

1. Open data.who.int/dashboards/covid19
2. Choose your country and find its cases graph
3. Pick a one-month period; note the figures at the start and end
4. Calculate the **percentage change**
5. Choose a second country; find its figure for the same end date
6. Calculate the **percentage difference** between the countries

### Then evaluate

Why might the two countries differ? Think about testing, reporting, population, vaccination and public health rules. How confident can you be that the numbers are comparable?

# Check that you can explain

1. How skin and mucus defend physically and chemically
2. How thrombin and fibrin form a clot
3. How innate and adaptive defences differ
4. How phagocytes destroy a pathogen
5. Why a B-cell needs two signals
6. Why a second infection is usually milder
7. How HIV leads to AIDS
8. Why antibiotics fail against viruses
9. How resistance evolves
10. How zoonoses reach humans
11. How vaccines and herd immunity prevent epidemics
12. How to calculate percentage change and difference

# Exit activity: 5 minutes

**On your own and without notes. Hand this in as you leave.**

1. Distinguish between an antigen and an antibody.
2. A patient asks for antibiotics for the flu. In one sentence, explain why they will not help.
3. Explain why a second infection by the same pathogen is usually milder than the first.

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