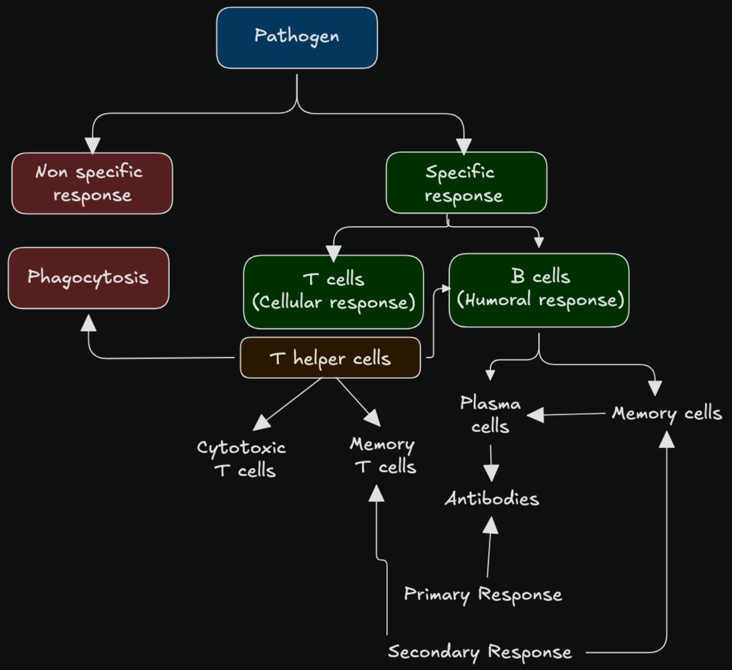
Cell Recognition & the Immune System

AQA A-level Biology · Unit 2 · Topic 3.2

← All slides

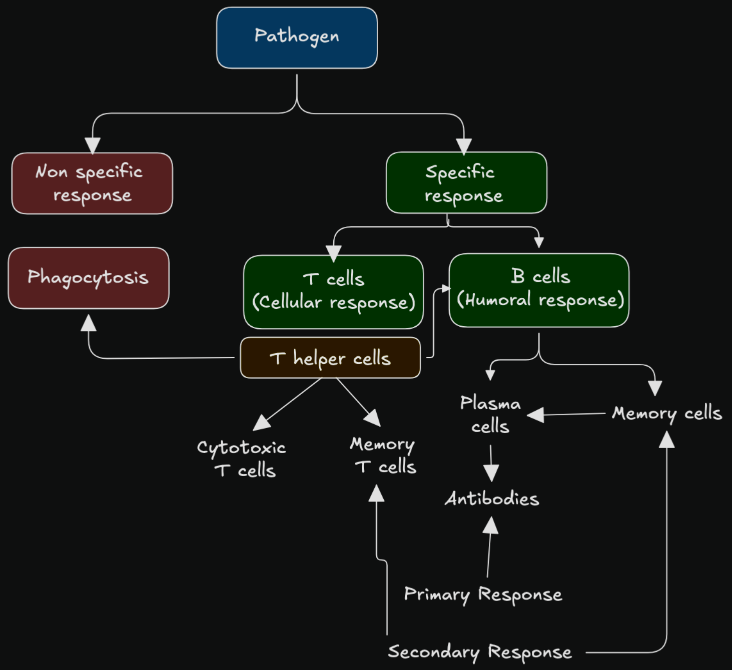
What this covers

- **Antigens** & antigen variability
- **Phagocytosis** – the non-specific response
- **Cellular response** – T lymphocytes
- **Humoral response** – B lymphocytes
- **Antibodies** – structure + how they destroy pathogens
- **Primary vs secondary** immune response
- **Vaccination** & herd immunity
- **Active vs passive** immunity

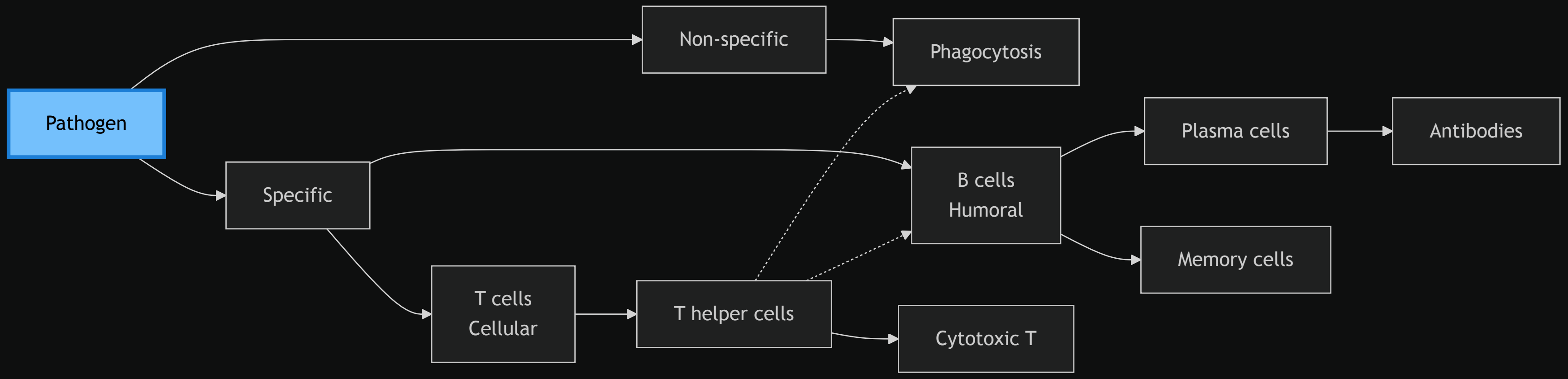


Big picture — two lines of defence

- **Non-specific response** – same response to any pathogen
  - **Phagocytosis** – fast, but not targeted
- **Specific response** – targeted at one particular pathogen
  - **Cellular response** – T lymphocytes
  - **Humoral response** – B lymphocytes + antibodies
  - Slower than the non-specific response



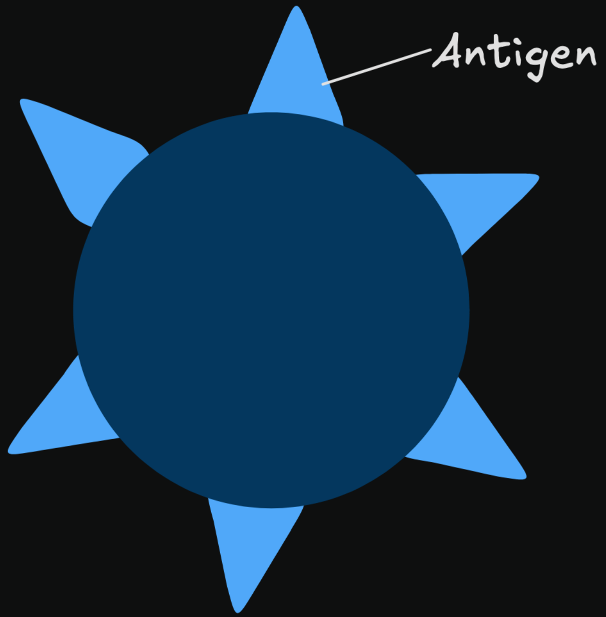
Antigens


Pathogen
Non-specific
Phagocytosis
Specific
B cells
Humoral
Plasma cells
Antibodies
Memory cells
T cells
Cellular
T helper cells
Cytotoxic T

All slides

How does the body recognise a pathogen?

- Every cell has specific molecules on its surface – **antigens**
- They can be **proteins, glycoproteins, or glycolipids**
- The immune system uses them to tell **self** from **non-self**



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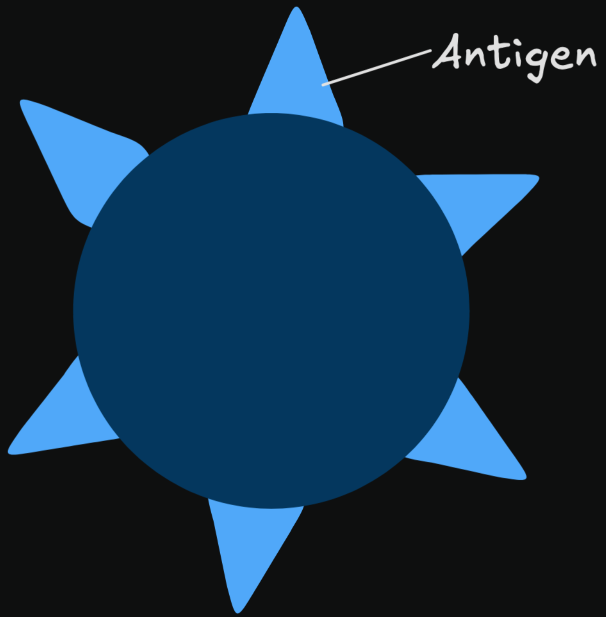
What can antigens (markers) help the immune system identify?

- **Pathogens** – disease-causing organisms (bacteria, viruses, fungi)
- **Cells from other organisms of the same species** – organ transplants
- **Abnormal body cells** – cancer cells, virus-infected cells
- **Toxins** – poisons produced by some bacteria



Try this — what stimulates an immune response?

AQA · 2 marks

Give two types of cell, other than pathogens, that can stimulate an immune response.

Mark scheme — other immune triggers

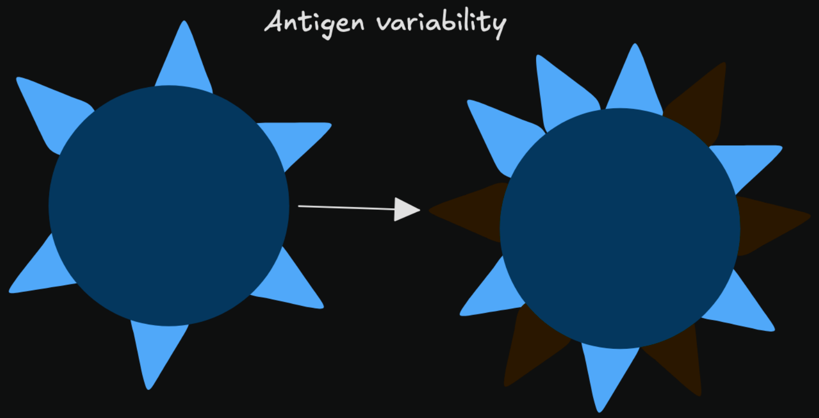
Any **two** of:

1. (Cells from) **other organisms / transplants**
2. **Abnormal / cancer / tumour** cells
3. Cells **infected by virus**

Accept "own cells" if autoimmune response suggested. Accept APCs. Accept "non-self".

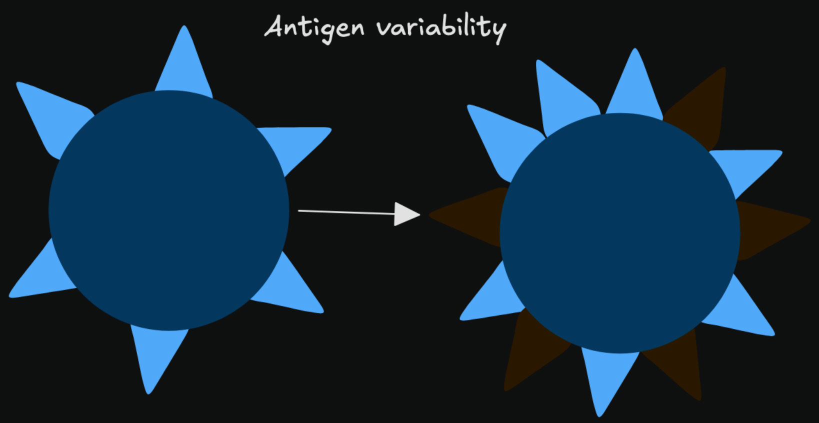
What is antigen variability?

- Some pathogens (e.g. **influenza, HIV**) have antigens that **change frequently**
- Caused by **mutations** in the genes coding for the antigens
- Different "strains" have different antigens



Why do you need a new flu vaccine regularly?

- **Memory cells** from a previous infection no longer recognise the new antigen shape
- You can **catch the same disease repeatedly** (e.g. flu)
- **Vaccines become ineffective** – last year's flu vaccine doesn't protect against this year's strain
- This is why we need a **new flu vaccine every year**
- We discuss memory cells later



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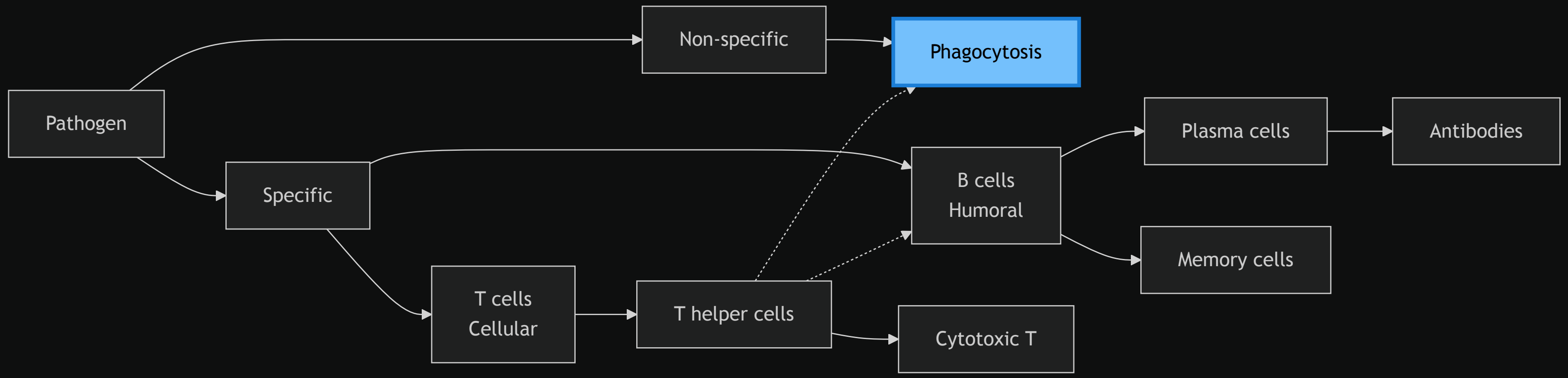
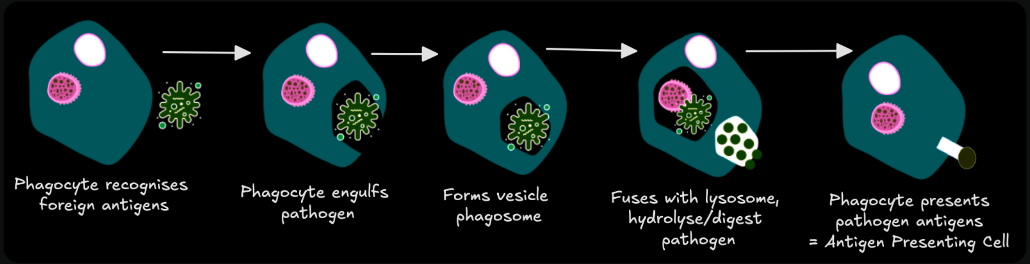
Phagocytosis (Non-specific)



Phagocytosis — the 5 steps

1. Phagocyte **recognises** non-self antigens (chemotaxis)
2. **Engulfs** the pathogen, forming a **phagosome**
3. A **lysosome** fuses with the phagosome
4. **Lysozymes** (hydrolytic enzymes) hydrolyse the pathogen
5. Antigens displayed on the phagocyte's surface, so it becomes an **antigen-presenting cell (APC)**



Try this — phagocytosis recall

AQA · 3 marks

Describe how a phagocyte destroys a pathogen present in the blood.

Mark scheme — phagocyte destroys pathogen

1. **Engulfs** the pathogen *(accept endocytosis; ignore "taken in")* *(1 mark)*
2. Forming **vesicle/phagosome** AND **fuses with a lysosome** *(1 mark)*
3. **Enzymes digest/hydrolyse** the pathogen *(accept lysozymes for "enzymes")* *(1 mark)*

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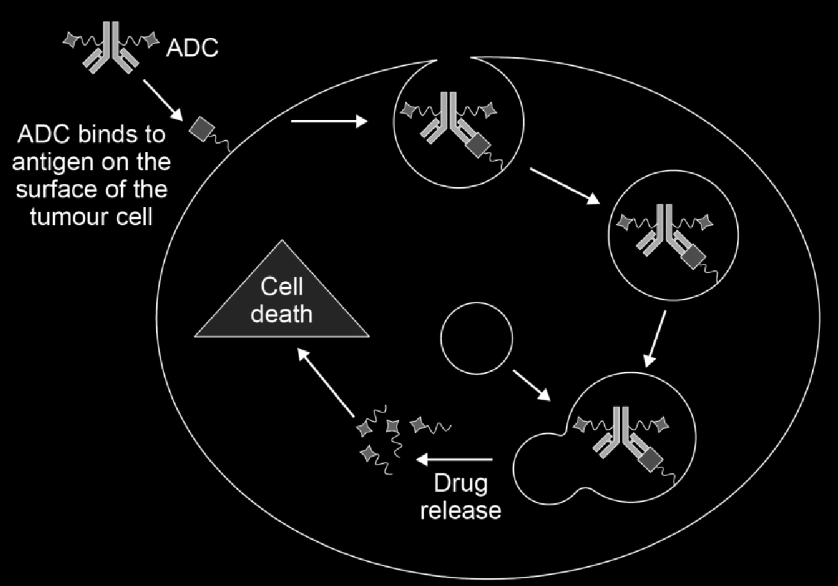
Application — ADCs and tumour cells

AQA Paper 1, 2021 · Q7.1 · 3 marks

ADCs are molecules made of a monoclonal antibody linked to a cancer drug.

Use your knowledge of phagocytosis to describe how an ADC enters and kills the tumour cell.

Figure 8



Mark scheme — ADC mechanism

1. Cell **ingests/engulfs** the ADC (cell membrane surrounds it) *(1 mark)*
2. **Lysosomes fuse** with vesicle/phagosome (containing the ADC) *(1 mark)*
3. **Lysozymes** break down/digest the antibody to release the drug *(1 mark)*

Same 3 phagocytosis points – just applied to ADCs. The drug then kills the tumour cell.

Application — Why do ADCs cause side effects?

AQA Paper 1, 2021 · Q7.2 · 2 marks

Some of the antigens found on the surface of tumour cells are also found on the surface of **healthy human cells**.

Use this information to explain why treatment with an ADC often causes side effects.

Mark scheme — ADC side effects

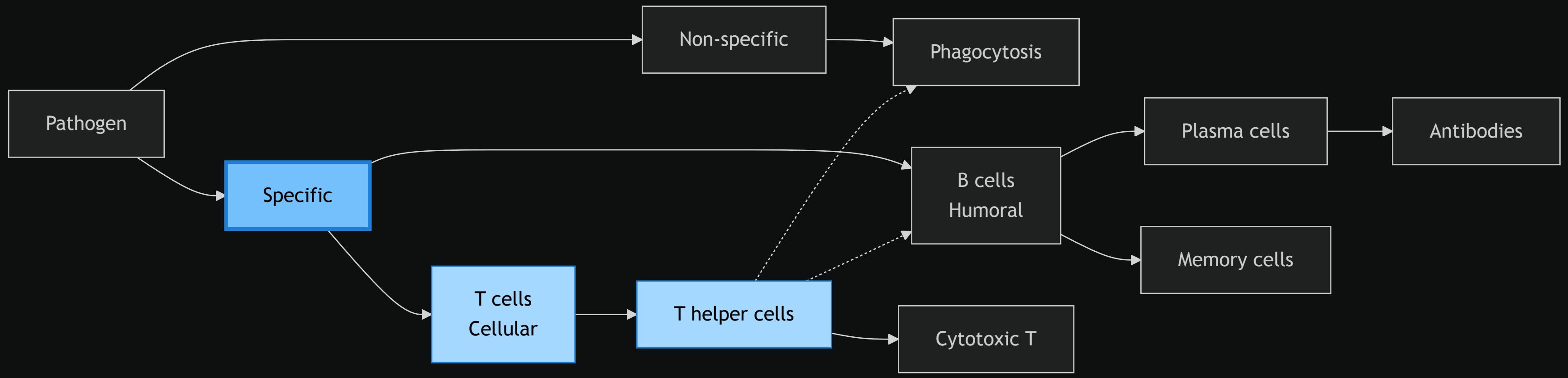
1. ADC will **bind to non-tumour / healthy cells** *(1 mark)*
2. Causes **death/damage of healthy cells**

   OR damage to other organs/systems *(1 mark)*

The drug is delivered into healthy cells too – and kills them just like tumour cells.

Big picture: monoclonal antibodies are powerful in cancer treatment because they target tumour cells far more selectively than chemotherapy – but they're not perfect. We'll come back to this in *Monoclonal Antibodies & ELISA*.

Specific Immune Response



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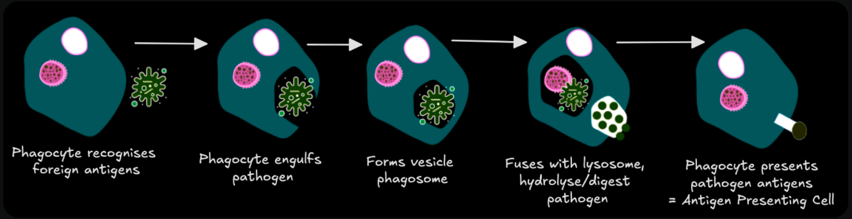
Specific response

After phagocytosis, the body launches a **specific response** targeted at the particular pathogen:

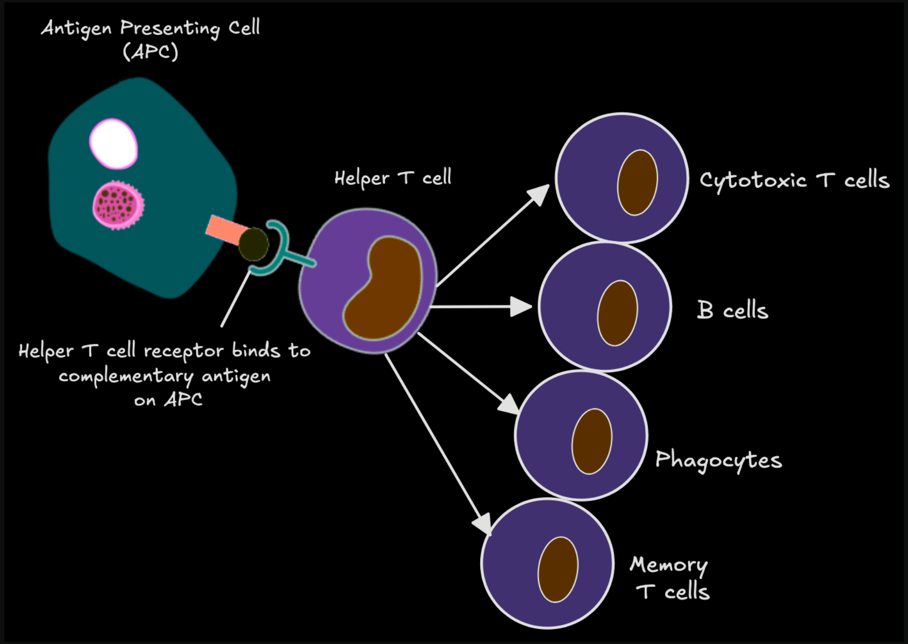
- **Cellular response** – T lymphocytes (T cells)
- **Humoral response** – B lymphocytes (B cells) and antibodies



Phagocyte → APC after engulfing a pathogen

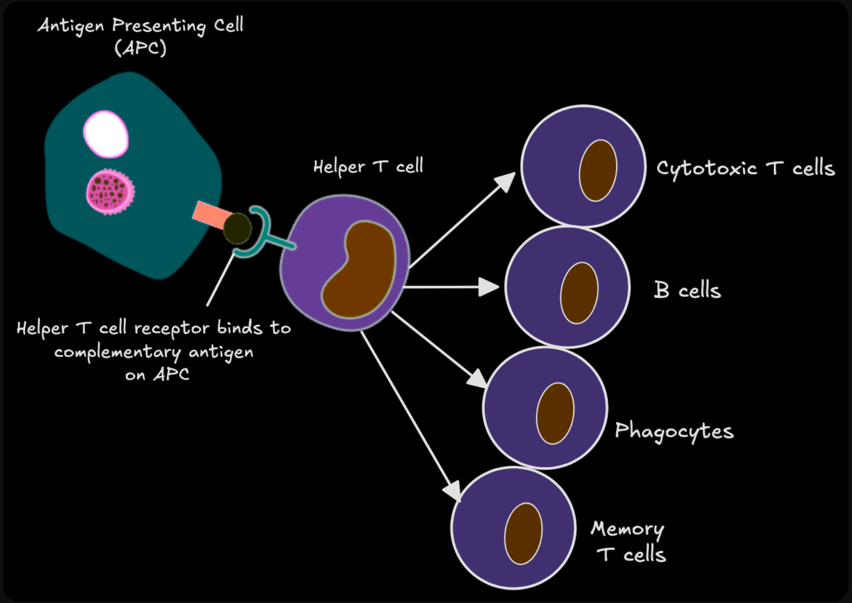
What are T lymphocytes?

- Mature in the **thymus** (T for thymus)
- Respond to antigens displayed on **antigen-presenting cells (APCs)**
- **Helper T cells** activate other cells in the immune response



Cellular response — what happens?

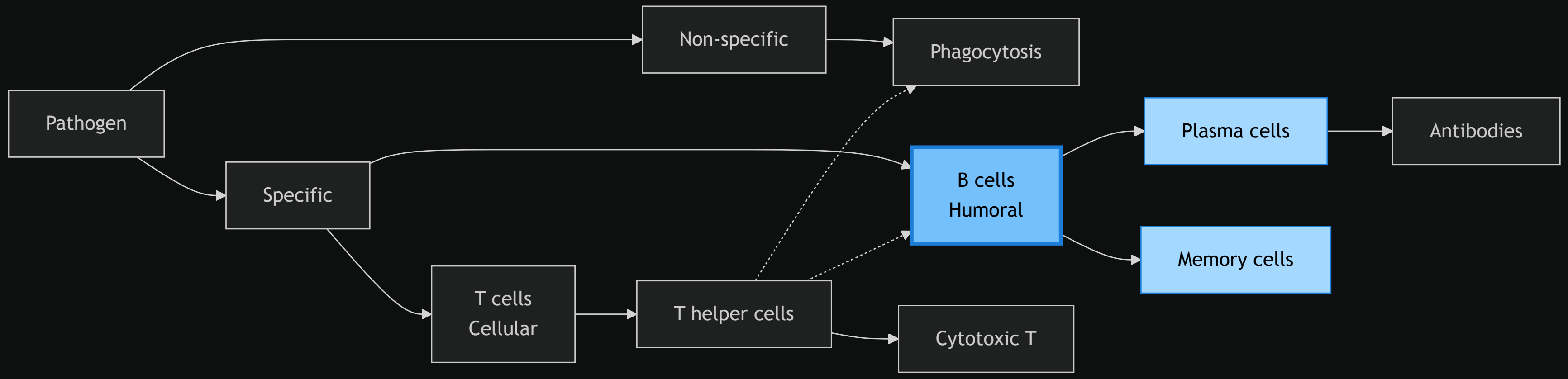
1. **APC** displays the pathogen's antigen
2. **Helper T cell** with complementary receptor binds → **clonal selection**
3. Activates → divides by mitosis → **clonal expansion**
4. Activated helper T cells stimulate:
   - **Cytotoxic T cells**
   - **B cells**
   - **Phagocytes**



How do cytotoxic T cells destroy abnormal cells?

- Target: **abnormal cells** (cancer cells) or **infected cells**
- Release a protein called **perforin**
- Perforin forms **pores** in the cell-surface membrane
- Substances enter/leave uncontrolled → the cell **dies**

Humoral Response (B cells)



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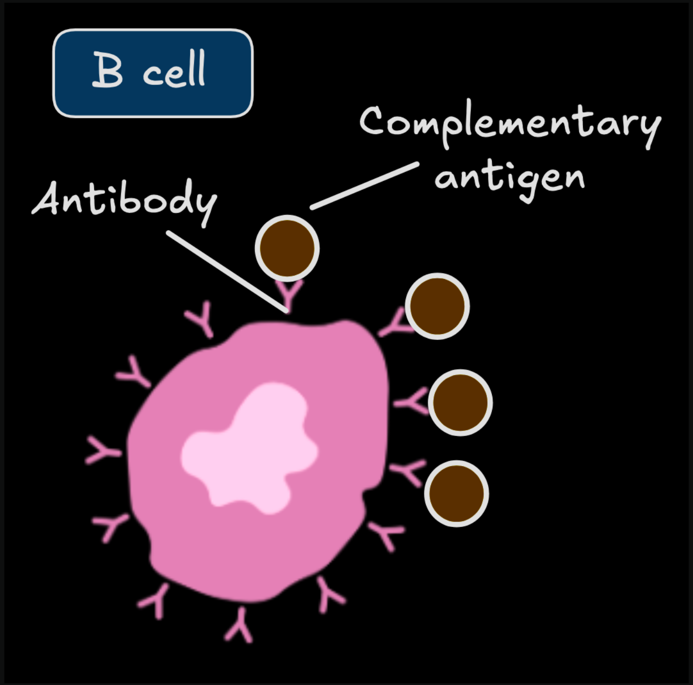
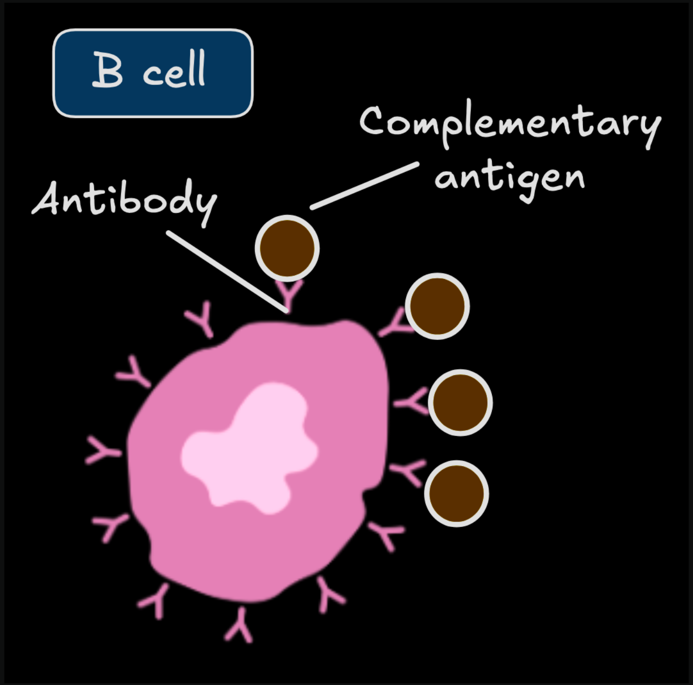
What are B lymphocytes?

- Mature in the **bone marrow** (B for bone marrow)
- Each B cell has a unique **antibody on its surface** (a B cell receptor)
- Complementary to **one specific antigen**



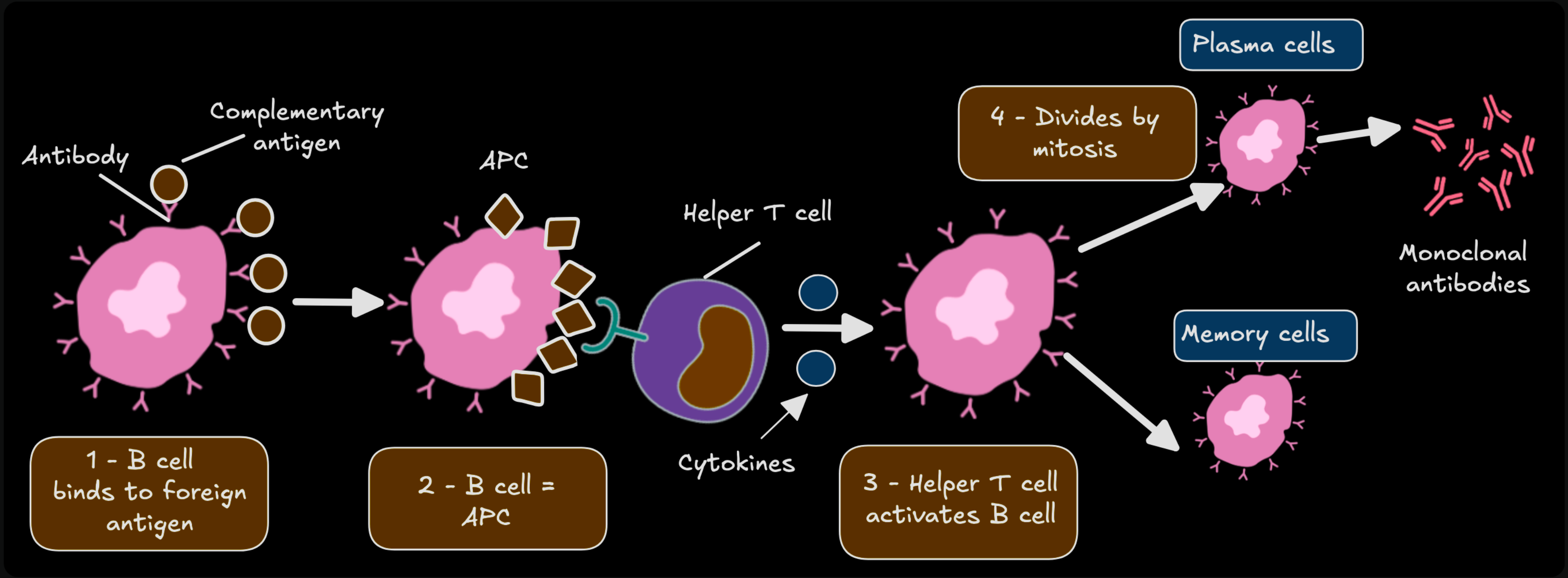
One B cell, one antibody specificity

- Every B cell carries **many identical antibodies** on its surface
- They all bind the **same antigen**
- **Different B cells** carry **different antibodies**
- Collectively: millions of B cells, each able to recognise different antigens



Humoral response — steps 1 & 2

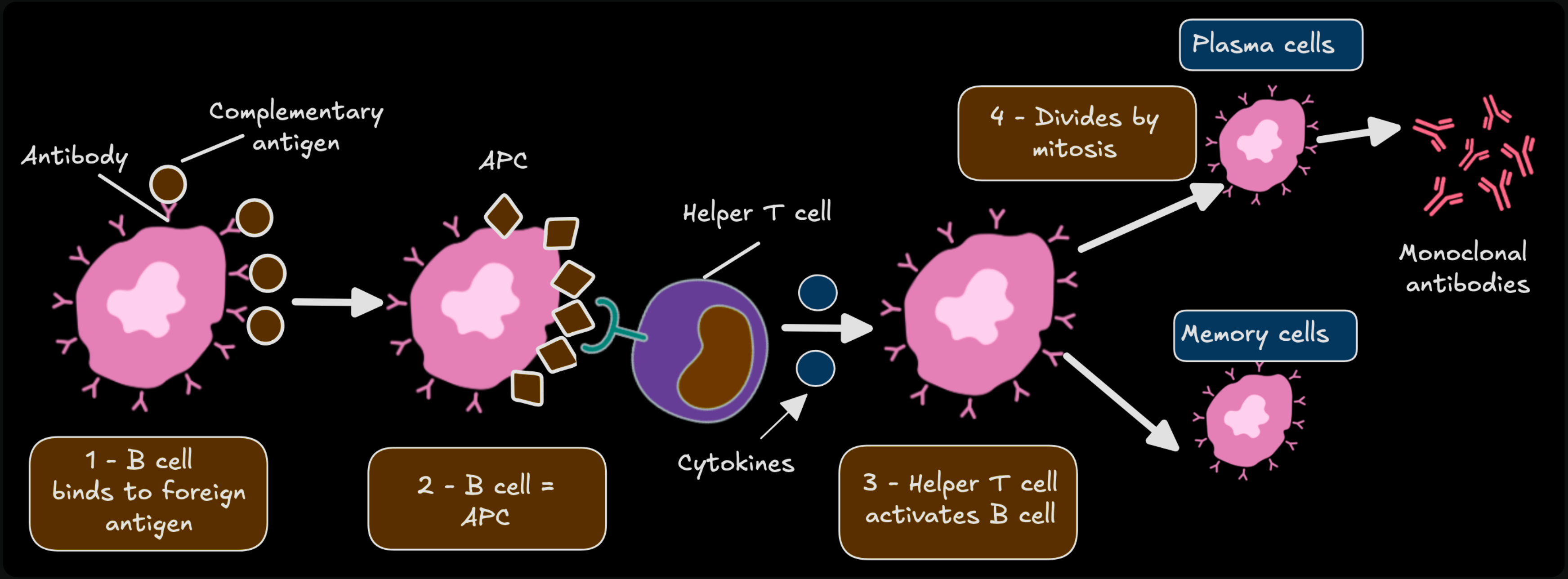
1. B cell receptor binds antigen → **clonal selection**
2. B cell internalises antigen, presents on surface → **APC**



Humoral response — steps 3 & 4

3. **Helper T cell** binds & activates the B cell

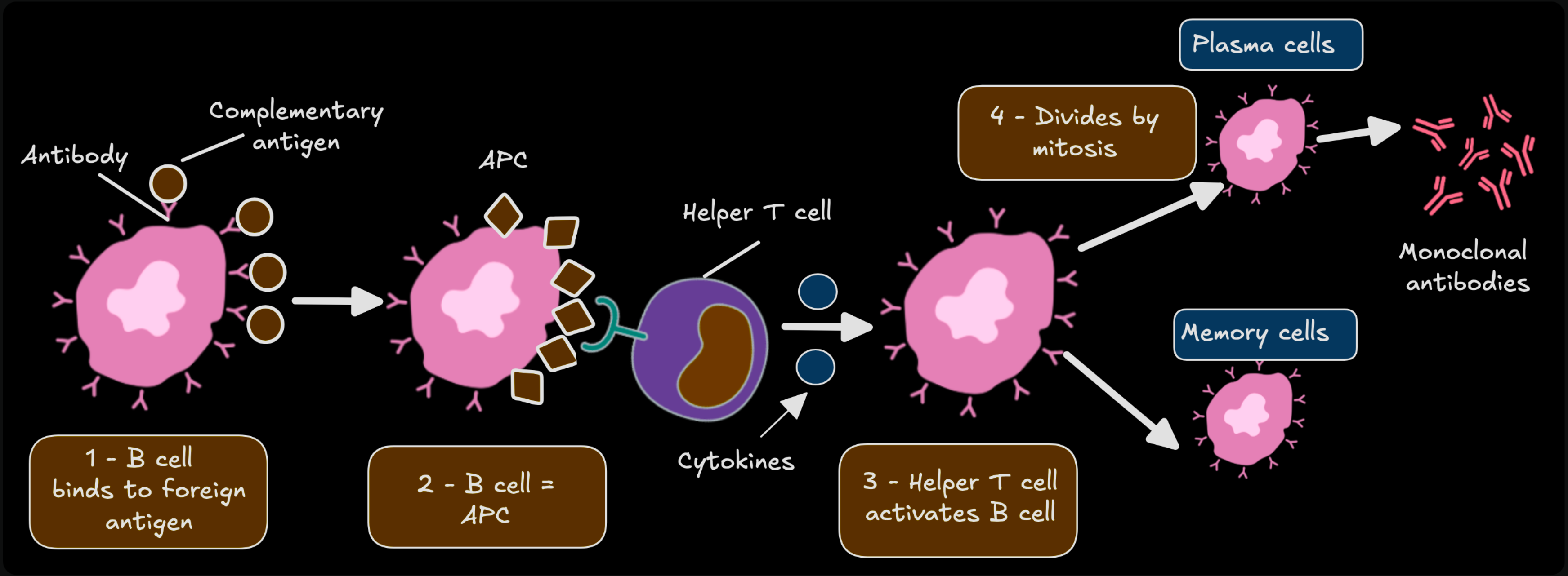
4. Activated B cell divides by mitosis → **clonal expansion**



Humoral response — step 5

5. The clone of B cells differentiates into:

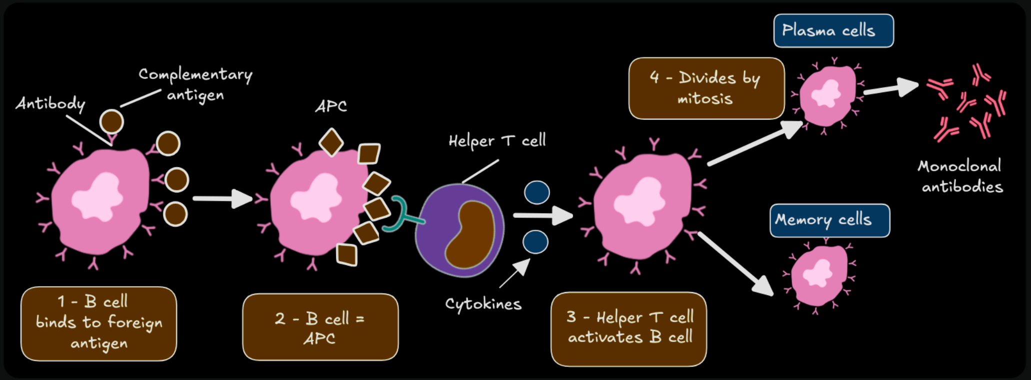
- **Plasma cells** – secrete antibodies
- **Memory cells** – long-term recognition



What is the difference between clonal selection and clonal expansion?

- **Clonal selection** = the antigen **selects** the B (or T) cell with the matching receptor
  - Happens at **binding** (step 1)
- **Clonal expansion** = the selected cell **divides by mitosis** to produce clones
  - Happens at **mitosis** (step 4)

Select first → then expand.



Application — B cell response to vaccination

AQA Paper 3, 2020 · Q3.4 · 3 marks

Ranavirus is a virus that infects frogs.

Describe how the B lymphocytes of a frog would respond to vaccination against Ranavirus.

You can assume that the B lymphocytes of a frog respond in the same way as B lymphocytes of a human.

Do NOT include details of the cellular response in your answer.

Mark scheme — B cell response (3 marks max)

1. B cell (antibody) **binds** to viral specific/complementary receptor/antigen *(1 mark)*
2. B cell **clones**

   OR divides by mitosis *(1 mark)*
3. **Plasma cells** release/produce (monoclonal) **antibodies** (against the virus) *(1 mark)*
4. (B/plasma cells produce/develop) **memory cells** *(1 mark)*

Accept: *B cell forms antigen-antibody complex* · *B cell undergoes clonal selection/expansion*

> **Notice:** the mark scheme skips T helper cells and goes straight from binding to mitosis. That's because the question said *"Do NOT include details of the cellular response"* – so T helper cell activation is excluded. Always read the restrictions!

Application — APC presentation → antibody secretion

AQA Paper 1, 2017 · Q7.3 · 3 marks

Describe how presentation of a virus antigen leads to the secretion of an antibody against this virus antigen.

Hint: this is the **synoptic chain** – start at the APC, end with antibodies. Walk through every cell type in between.

Mark scheme — APC → antibody (3 max)

1. **Helper T cell binds to the antigen** (on the antigen-presenting cell / phagocyte) *(1 mark)*
2. **Helper T cell stimulates a specific B cell** *(1 mark)*
3. **B cell clones**

   OR B cell divides by mitosis *(1 mark)*
4. (Forms) **plasma cells that release antibodies** *(1 mark)*

"Helper" needed once only (in MP1 or MP2). Accept "T cell stimulates competent B cell" or "T cell stimulates B cell to undergo clonal selection" – this single statement covers MP2 + MP3.

> **Compare to the previous question:** the last one told you NOT to include cellular response, so the mark scheme started at B cell binding. This one starts with the APC and explicitly wants the T helper step. **Always read what the question is – and isn't – asking.**

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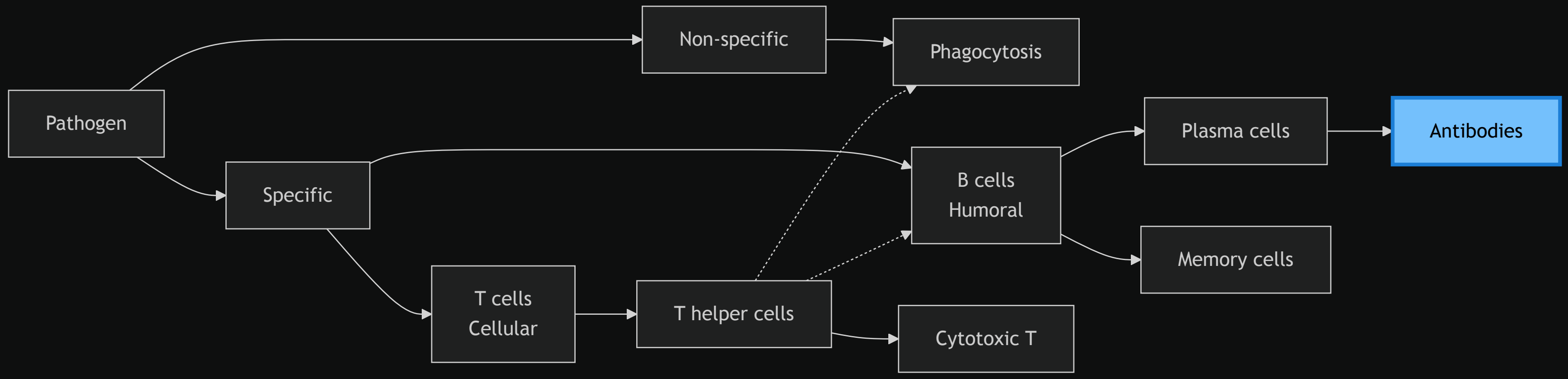
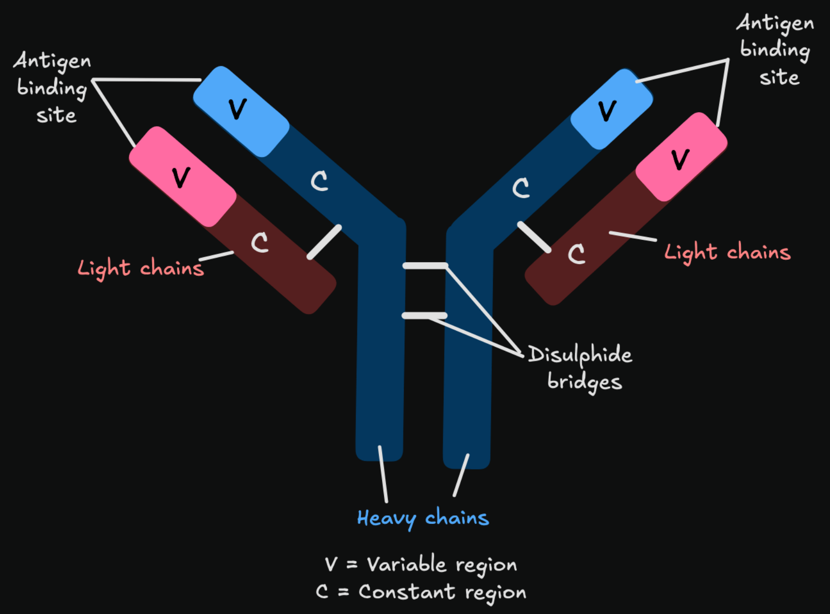
Antibodies



What is an antibody?

- A **protein** (specifically a **quaternary**-structure protein)
- Produced by **plasma cells**
- Binds to a **specific antigen**

Monoclonal = identical antibodies, produced from a single clone of plasma cells → all bind the **same specific antigen**.



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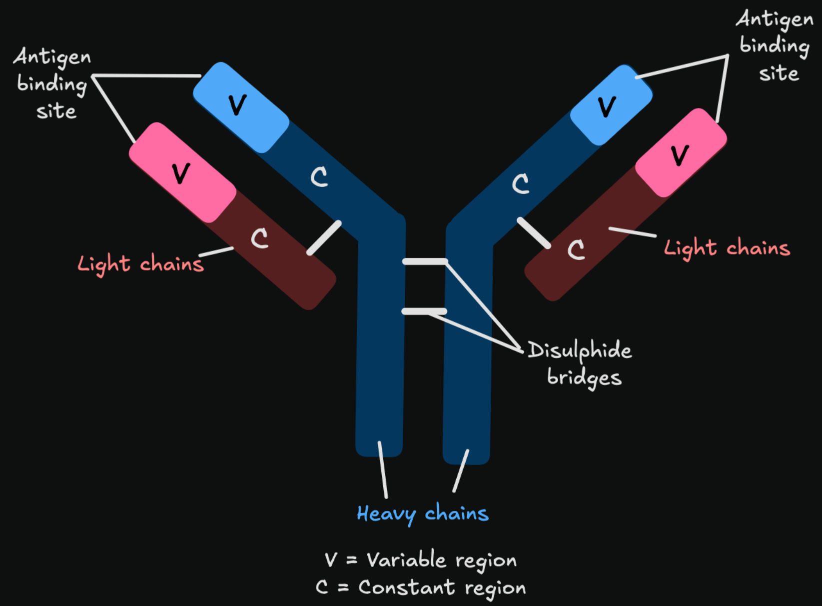
Antibody structure

- **Quaternary protein** – 4 polypeptide chains
- 2 **heavy** + 2 **light** chains
- Held together by **disulfide bridges**
- **2 identical variable regions** – antigen binding sites
- **Constant region** – same in all antibodies



Why are antibodies important?

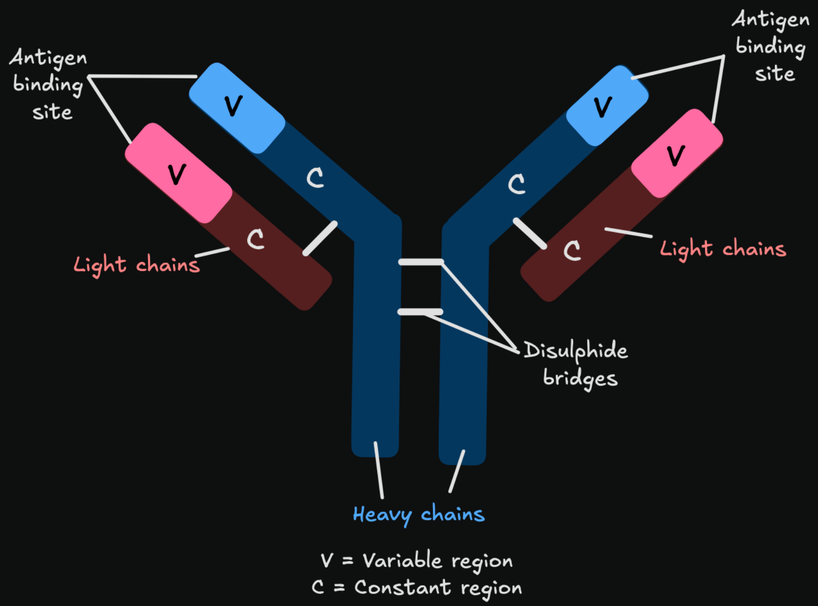
When an antibody binds its specific antigen, they form an **antigen-antibody complex**. The pathogen is destroyed by:

- **Agglutination** – 2 binding sites → links 2 bacteria → clumps them together → easier for phagocytes to engulf
- **Phagocytosis of bacterial cells** – the clumps are engulfed and destroyed
- **Neutralising toxins** – antibodies bind to bacterial toxins, preventing them from affecting cells

Question — antigen-antibody complex

AQA · 1 mark

Label the antibody diagram with an X to show where an antigen-antibody complex forms.



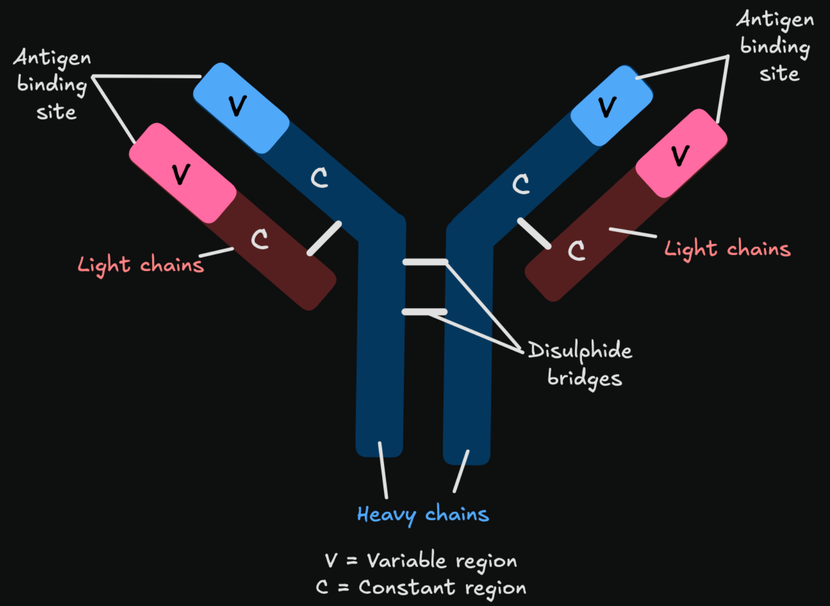
Mark scheme — antigen-antibody complex

- **X written at either or both ends of the Y shape** *(1 mark)*
- The tips of the variable regions are the antigen-binding sites – this is where the complex forms

Question — disulfide bridges

AQA · 1 mark

What is the role of disulfide bridges in forming the quaternary structure of an antibody?



Mark scheme — disulfide bridges

- **Joins two (different) polypeptides** *(1 mark)*

Accept: *holds / attaches*, or *prevents polypeptide chains separating*.

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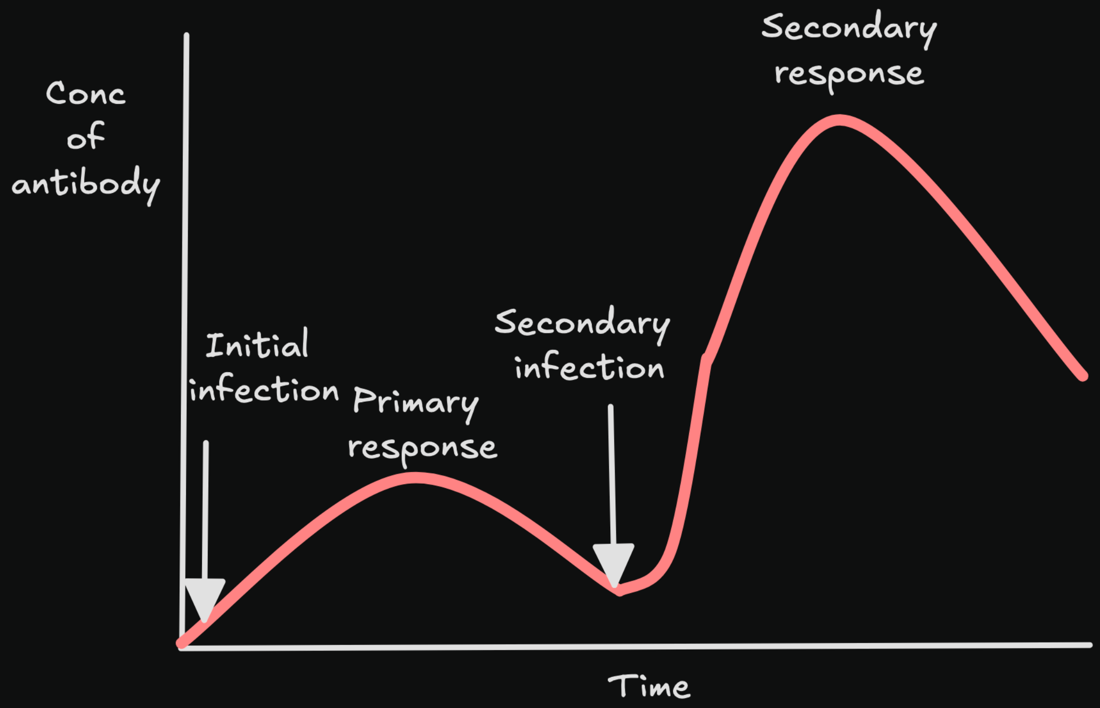
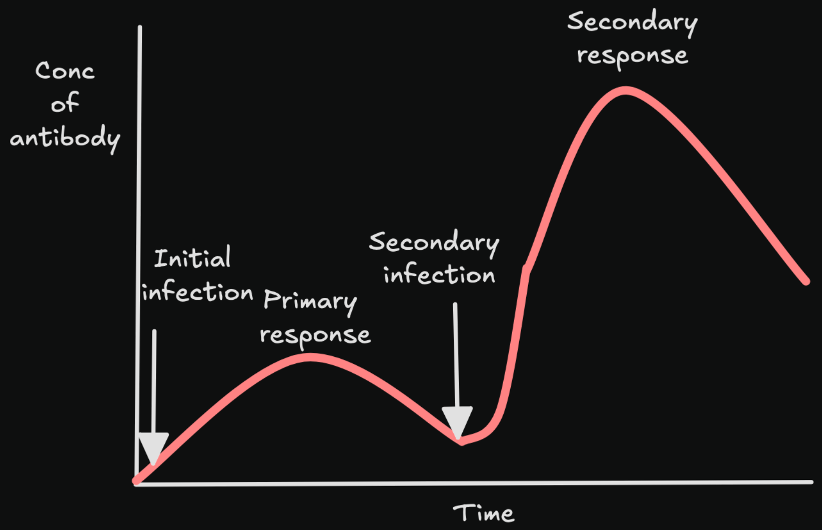
Primary vs Secondary Response



The same pathogen, two encounters. Why is the second one so different?

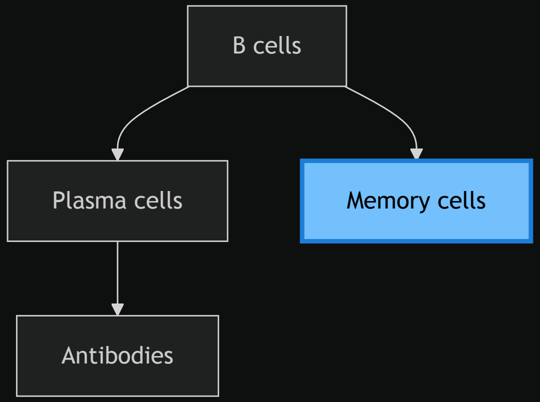
Why is the secondary response faster?

- **Primary** – first exposure
  - Slow (days)
  - Few antibodies
  - Symptoms experienced
  - Memory cells produced
- **Secondary** – re-exposure
  - Fast (hours)
  - Lots of antibodies
  - Usually no symptoms
  - Memory cells **already specific** → clonal selection much faster



Primary vs secondary — at a glance

	Primary	Secondary
Speed	Slow (days)	Fast (hours)
Antibody concentration	Low	Much higher
Symptoms	Often experienced	Usually none
Why	First exposure – no memory cells yet	Memory cells divide rapidly into plasma cells



Memory cells are the reason the secondary response is fast.

Vaccination & Immunity

How does a vaccine work?

A vaccine contains **antigens** from a pathogen in a form that **can't cause disease** – either a **dead** (inactivated) pathogen, an **attenuated** (weakened) pathogen, or just the **antigens** from its surface.

- Triggers a **primary immune response** without illness
- **Memory cells** are produced
- If you later meet the real pathogen → **secondary response** before symptoms appear

What is herd immunity?

- If a large enough proportion of the population is vaccinated, the pathogen **can't spread easily**
- Even unvaccinated people are protected – they're unlikely to come into contact with the pathogen
- Critical for those who **can't be vaccinated**:
  - Infants
  - Immunocompromised
  - People with allergies / contraindications

Active vs passive immunity

	Active	Passive
Antibodies made by	Your own immune system	Given from another source
Memory cells	**Yes**	**No**
Speed	Slow	Fast
Duration	Long-term	Short-term

Active = you do the work → memory → long-term
Passive = you're given antibodies → no memory → short-term

Can you name examples of active and passive immunity?

- **Active immunity**
  - Catching a disease naturally (e.g. chickenpox)
  - **Vaccination**
- **Passive immunity**
  - Antibodies passed from **mother to baby** (placenta, breast milk)
  - **Anti-venom** / **anti-toxin** injections (e.g. snake bites, tetanus)

Describe the difference between active and passive immunity

AQA · 5 marks

Describe the difference between active and passive immunity.

Mark scheme — active vs passive (5 max)

1. **Active** involves **memory cells**; passive does not *(1 mark)*
2. **Active** involves **production of antibody by plasma cells / memory cells** *(1 mark)*
3. **Passive** involves **antibody introduced into the body from outside / named source** *(1 mark)*
4. **Active** is **long term**, because antibody is produced in response to antigen *(1 mark)*
5. **Passive** is **short term**, because antibody (given) is broken down *(1 mark)*
6. **Active** can take time to develop / work; **passive** is fast acting *(1 mark)*

5 max – any 5 of these 6 points gets full marks.

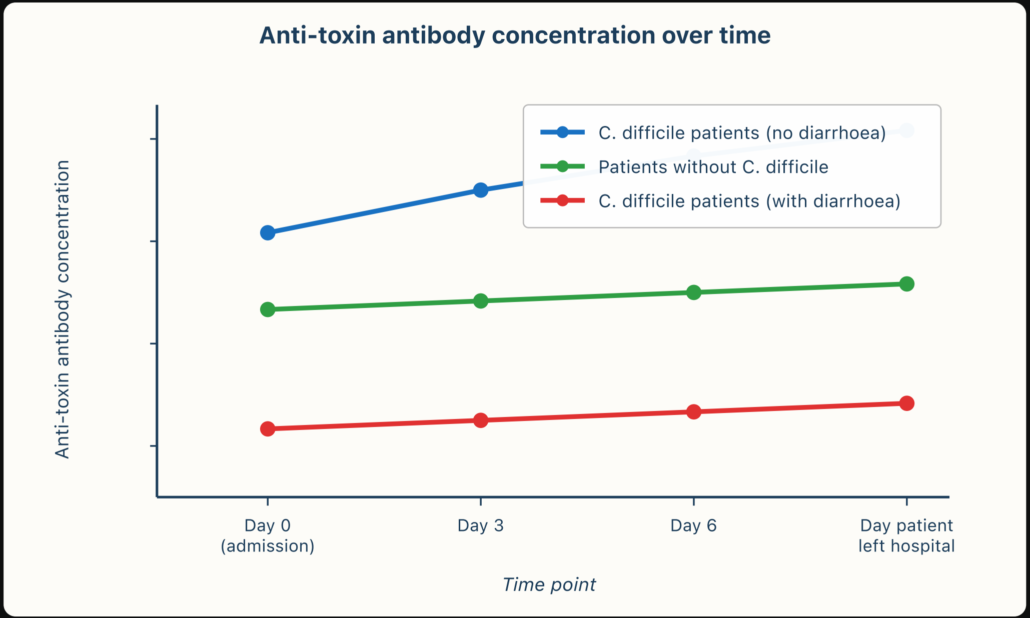
← All slides

Application — anti-toxin antibody and passive immunity

AQA Paper 1, 2023 · Q4.2 · 3 marks

Scientists measured the **anti-toxin antibody concentration** in hospital patients with and without *C. difficile* infection. They suggest the anti-toxin antibody could be given to some patients as a form of **passive immunity**.

Use Figure 4 to suggest how this passive immunity would work AND which patients should be offered this anti-toxin antibody.



Mark scheme — anti-toxin antibody

1. Anti-toxins/antibodies cause **phagocytosis / destruction / agglutination / neutralisation** (of toxin) *(1 mark)*

2. Anti-toxin/antibody **prevents/reduces diarrhoea**
   OR patients with no diarrhoea have **highest** anti-toxin
   OR patients with diarrhoea have **lowest** anti-toxin *(1 mark)*

3. Offered to **patients with diarrhoea**
   OR patients with **low antibody concentrations** *(1 mark)*

For "neutralised": accept idea of preventing toxin binding / damaging cells lining the ileum.

Context for Q — how antivenom is made

- Animals (e.g. horses) are **vaccinated** with snake venom
- Their immune system produces **antibodies** against the venom – this is **active immunity in the animal**
- Animal blood is collected → antibodies are **purified** → this becomes **antivenom**
- Antivenom is then injected into a snake-bite patient → patient gets **passive immunity** (pre-made antibodies)

Application — antivenom & passive immunity

AQA Paper 1, 2018 · Q7.1 · 2 marks

When a person is bitten by a venomous snake, the snake injects a **toxin**. **Antivenom** is injected as treatment – it contains **antibodies against the snake toxin**. This is **passive immunity**.

Explain how the treatment with antivenom works AND why it is essential to use passive immunity, rather than active immunity.

Mark scheme — antivenom

1. (Antivenom) **antibodies bind to the toxin/venom/antigen** and cause its **destruction** *(1 mark)*
2. **Active immunity would be too slow** *(1 mark)*

For "destruction" accept agglutination or phagocytosis. Accept "passive immunity is faster" – but NOT just "passive immunity is fast" (must be comparative).

Application — why mix venoms from several snakes?

AQA Paper 1, 2018 · Q7.2 · 2 marks

In producing antivenom, a **mixture of venoms from several snakes of the same species** is used.

Suggest why.

Hint: think about antigen variability within a single species.

Mark scheme — mixture of venoms

1. May be **different forms of antigen/toxin** within one species

   OR snakes may have **different mutations/alleles** *(1 mark)*

2. **Different antibodies needed** in the antivenom

   OR several antibodies complementary to several antigens *(1 mark)*

> **Why?** Even snakes of the **same species** don't all have identical venom. Because of **genetic variation** (different mutations/alleles), individual snakes produce **slightly different versions** of their venom proteins – so the same protein exists as **different forms of the antigen**. One antibody can't bind all of them, so the antivenom needs to be a mix.

NO marks for "different species of snake" – the question specifies same species.

Application — small dose then larger dose

AQA Paper 1, 2018 · Q7.5 · 3 marks

During vaccination of the **animals** producing antivenom, each animal is initially injected with a **small volume** of venom. Two weeks later, it is injected with a **larger volume** of venom.

Use your knowledge of the humoral immune response to explain this vaccination programme.

Mark scheme — vaccination programme

1. **B cells specific to the venom reproduce by mitosis** *(1 mark)*
2. (B cells produce) **plasma cells and memory cells** *(1 mark)*
3. The **second dose** produces antibodies (in **secondary immune response**) in **higher concentration and quickly**

   OR the first dose must be **small so the animal is not killed** *(1 mark)*

> **Why this programme?** The **first (small) dose** triggers a **primary response**: B cells with complementary receptors are selected and divide by mitosis, producing **plasma cells** (release antibodies) and **memory cells**. The **two-week gap** gives memory cells time to build up. The **second (larger) dose** meets these memory cells → fast, much bigger **secondary response** → far more antibodies produced → more antivenom harvested.
>
> The first dose has to be **small** for safety – a large dose of venom on an unprotected animal could kill it.

Accept "clone" for "reproduce by mitosis". "Clonal selection of B cells" = MP1. Accept "a lot of antibody" for "higher concentration of antibody".

Recap — what you should know

The AQA spec points for this topic – by the end of this lesson you should be able to:

- ✅ Define **antigen** and explain how the body distinguishes self from non-self
- ✅ Explain **antigen variability** and why it matters
- ✅ Describe **phagocytosis** (mark scheme phrasing)
- ✅ Describe the **cellular response** – T lymphocytes
- ✅ Describe the **humoral response** – B lymphocytes (plasma + memory cells)
- ✅ Describe **antibody structure** + how antigen-antibody complexes form
- ✅ Compare **primary and secondary** responses
- ✅ Explain how **vaccines** work + **herd immunity**
- ✅ Distinguish **active vs passive** immunity

Common mark-scheme phrasing

- **"Lysozymes"** (NOT lysosomes) for the digestion step
- **"Complementary in shape"** (NOT "active site") for antibodies
- **"Agglutination"** + **"phagocytosis of bacterial cells"** – exact wording
- **"Memory cells"** drive the secondary response – not "old antibodies"
- **"Antigen variability"** / **"antigens change"** – link to **why** vaccines fail
- **"Both sides"** for ethics questions – concerns + benefits