REVISION GUIDE

How the body *fights back*

Defence against disease

Every day your body meets bacteria, viruses and fungi, and almost always wins without you noticing. Skin keeps most out, a clot seals the cuts, and a team of cells recognizes what is left, destroys it and remembers it for years.

17 chapters · a dry lab

The interactive guide, with every animation:

biologybybradford.com/guides/ib-biology-c3-2-defence-against-disease

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By the end you can

- [ ] Name the four kinds of pathogen and say how careful observation led to the control of infectious disease C3.2.1
- [ ] Explain how skin and mucous membranes act as a physical and chemical barrier C3.2.2
- [ ] Explain how platelets and the clotting cascade seal a cut C3.2.3
- [ ] Compare the innate and the adaptive immune systems C3.2.4
- [ ] Describe how phagocytes move to an infection, engulf pathogens and digest them C3.2.5
- [ ] Explain how lymphocytes cooperate to make antibodies C3.2.6
- [ ] Explain why antigens are recognition molecules that trigger antibody production C3.2.7
- [ ] Explain how helper T cells activate B cells C3.2.8
- [ ] Explain how activated B cells divide to form clones of antibody-secreting plasma cells C3.2.9
- [ ] Explain how memory cells give immunity C3.2.10
- [ ] Describe how HIV is passed on in body fluids C3.2.11
- [ ] Explain how HIV destroys lymphocytes and leads to AIDS C3.2.12
- [ ] Explain why antibiotics block bacteria but not eukaryotic cells, and why they fail against viruses C3.2.13
- [ ] Explain how bacteria evolve resistance to several antibiotics C3.2.14
- [ ] Give examples of zoonoses and the ways they reach humans C3.2.15
- [ ] Explain how vaccines produce immunity without causing the disease C3.2.16
- [ ] Explain how herd immunity prevents epidemics C3.2.17
- [ ] Evaluate data from the COVID-19 pandemic, calculating percentage change and percentage difference C3.2.18

Tick each one when you can do it without the book.

C3.2.1 PATHOGENS AS THE CAUSE OF INFECTIOUS DISEASES

01 Four kinds of *pathogen*

Most microbes do you no harm. A **pathogen** is one that does, and when it passes from person to person the illness is an **infectious disease**.

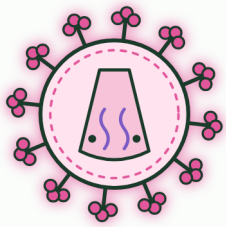
KEY POINTS

- **Viruses** are not cells. They copy themselves only inside a host cell.
- **Bacteria** are prokaryotes. Toxins they make, or damage they do, cause the disease.
- **Fungi and protists** are eukaryotes. Some are parasites that live in or on a host.
- Pathogens are not all alike, so one treatment cannot work on every kind.

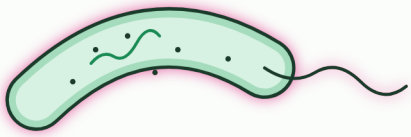
KEY TERMS

pathogen
An organism or virus that causes disease.

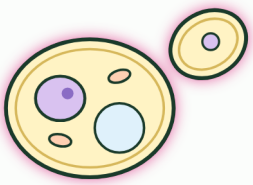
infectious disease
A disease caused by a pathogen that can be passed from one host to another.



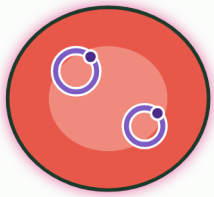
Virus
HIV
about 0.1 µm across



Bacterium
Vibrio cholerae
about 2 µm long



Fungus
Candida albicans
about 5 µm



Protist
Plasmodium falciparum
in a red blood cell

Virus: HIV Far smaller than any cell, and not a cell at all: a protein coat round a little RNA, with spikes that grab on to a host cell. It has no metabolism of its own, so it can only copy itself inside a living cell.

Fungus: Candida albicans A eukaryotic cell with a cell wall, here a yeast budding off a daughter cell. It lives harmlessly on many people and causes thrush when the balance slips. Other fungi grow as threads, like the one that causes athlete's foot.

Bacterium: Vibrio cholerae A single prokaryotic cell with a cell wall and no nucleus. This curved rod lives in dirty water and its toxin makes the lining of the gut lose water, which is cholera. Most bacteria are harmless, and many are useful.

Protist: Plasmodium falciparum A single-celled eukaryote that is a parasite. Here it is the ring stage inside a red blood cell, which it feeds on and bursts. It is passed on by *Anopheles* mosquitoes and causes malaria.

PREDICT

One group of microbes is often listed beside bacteria and fungi, yet no member is known to cause disease in humans. Which?

(A) Protists, because they are too large to enter the body

(B) Viruses, because they are not living cells

(C) Archaea, which are prokaryotes like bacteria

(D) Prions, because they contain no nucleic acid

Decide before you look. Answer on page 53.

HOW WE FOUND OUT

In 1854 John Snow mapped cholera deaths in London's Soho and found them clustered round one water pump; removing the pump handle was followed by fewer cases. In the 1840s Ignaz Semmelweis showed that doctors washing their hands with chlorinated lime cut deaths from childbed fever in his Vienna ward. Both reasoned from careful observation, before anyone had seen the bacteria responsible.

C3.2.2 SKIN AND MUCOUS MEMBRANES AS A PRIMARY DEFENCE

02 The first line is *a wall*

Your best protection is not letting microbes in. The skin is a **physical barrier**: its outer layer is dead cells packed with the protein keratin, tough and waterproof, and sheets of it lap over each other so there are no gaps. It is also a **chemical barrier**: sweat and oils make the surface acidic and sweat carries lysozyme, an enzyme that splits bacterial cell walls.

Where your body opens to the outside, in the nose, windpipe, gut and reproductive tract, there is no thick dry skin. A thin, moist **mucous membrane** lines the surface instead. Mucus is sticky and traps microbes. In the airways, cilia beat to sweep the mucus up towards the throat, where you swallow it, and the acid in the stomach destroys most of what arrives.

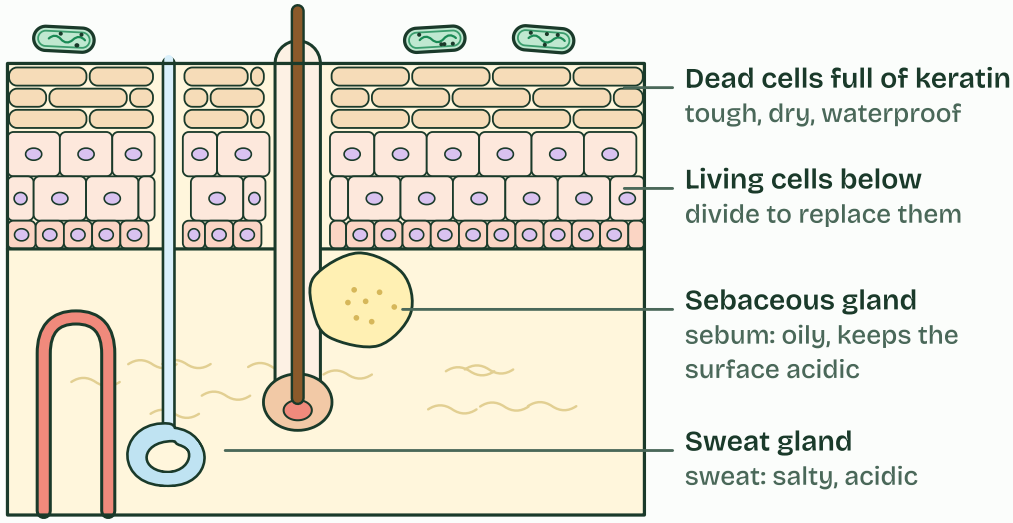
KEY TERMS

physical barrier
A barrier that stops a pathogen getting through by being in the way, such as the tough outer layer of skin.

chemical barrier
A barrier that harms pathogens with chemicals, such as acidic sweat and enzymes.

mucous membrane
The moist lining of body passages. Its cells make mucus, which traps microbes, and in places it carries cilia.

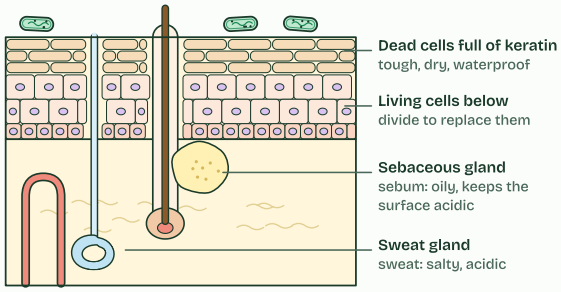
Skin: a tough outer barrier



SWITCH IT 2 SETTINGS

1

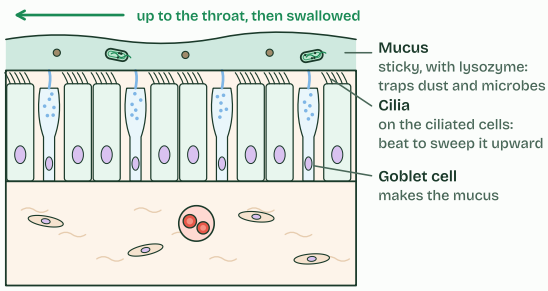
Skin: a tough outer barrier



Skin

2

Airway lining: a mucous membrane



Airway lining

PREDICT

A person with a severe burn is at high risk of infection. The best reason is:

(A) The burnt tissue gives off chemicals that attract bacteria

(B) A burn removes the physical barrier, so microbes can get straight in

(C) Heat destroys every white blood cell in the body

(D) The body stops making mucus after an injury

Decide before you look. Answer on page 53.

WATCH OUT

Mucus is not just a nuisance. A runny nose is the airway's trap filling up, doing its job.

03 A cut seals *itself*

C3.2.3 SEALING OF CUTS IN SKIN BY BLOOD CLOTTING

Break the skin and you have both bleeding and an open door for microbes. The body plugs it in seconds. Blood contains cell fragments called **platelets**. At a cut they stick to the damaged vessel and release **clotting factors**.

These start a **cascade**. The last steps are the ones to know. The enzyme **thrombin** (made from inactive prothrombin) converts soluble fibrinogen, which is in the plasma all the time, into insoluble **fibrin** fibres. They form a mesh that traps red blood cells and makes a clot, which dries into a scab.

KEY TERMS

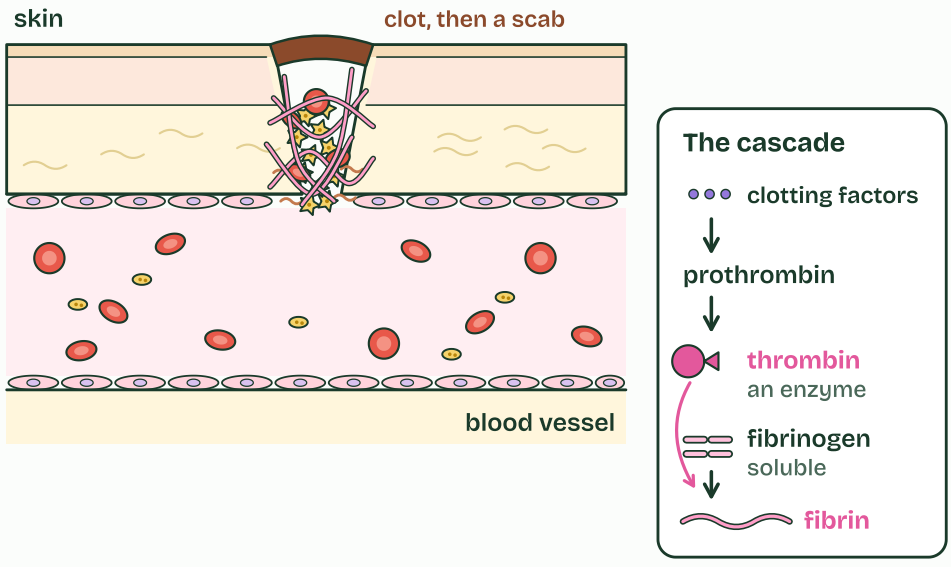
platelets
Small cell fragments in blood that stick to damaged vessels and start clotting.

clotting factors
Chemicals released by platelets and damaged tissue that start the clotting cascade.

cascade
A chain of reactions in which each step triggers the next.

thrombin
The active enzyme made from prothrombin during clotting. It turns fibrinogen into fibrin.

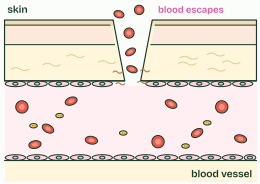
fibrin
Insoluble protein fibres that form a mesh across a wound, trapping blood cells to make a clot.



STEP BY STEP

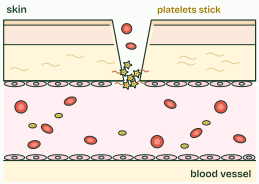
5 STEPS

1



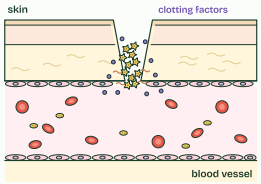
A **cut** breaks the skin and the vessel under it. Blood leaks out and microbes could get in.

2



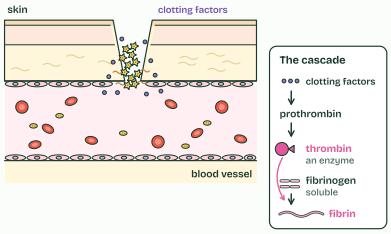
Platelets stick to the damaged vessel and to each other, a first plug.

3



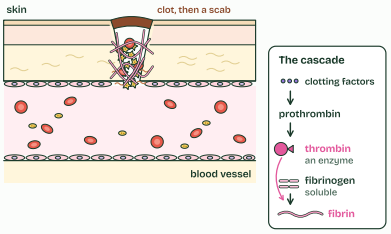
Platelets and damaged cells release **clotting factors**.

4



The factors start a **cascade**: thrombin forms, and thrombin converts soluble fibrinogen to insoluble fibrin.

5

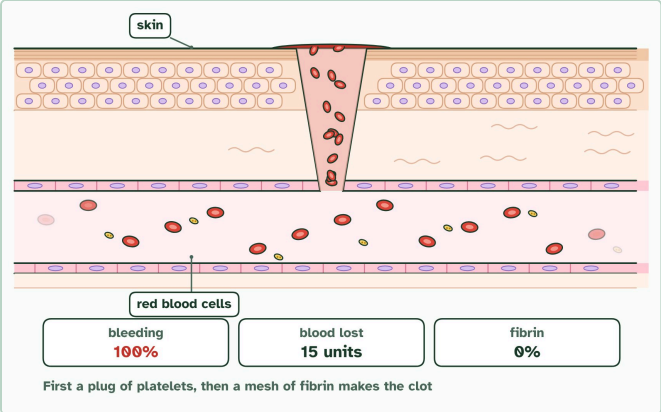


Fibrin fibres make a **mesh** that traps red blood cells: a clot, which dries into a scab.

ANIMATION · A CUT AND THE CLOT

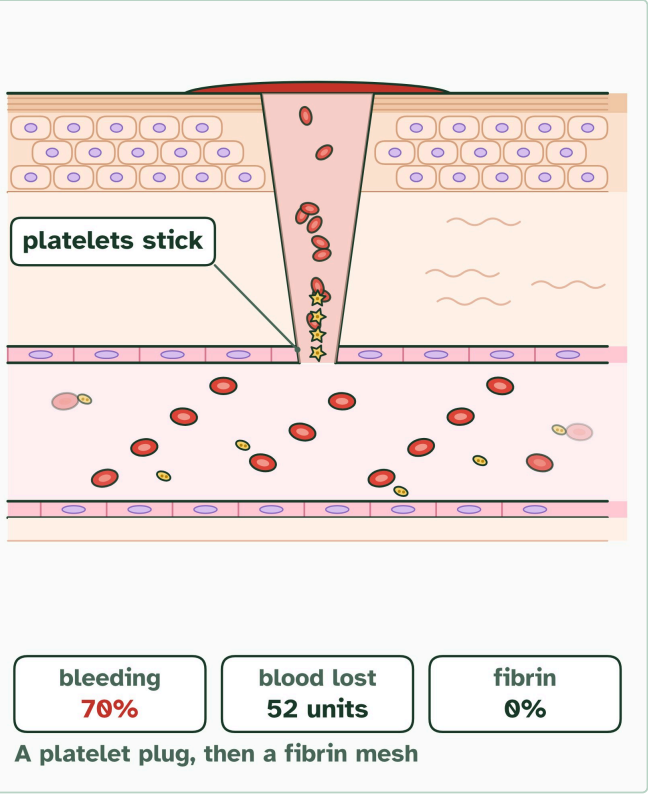
5 MOMENTS

1 CLOT: 0% SEALED



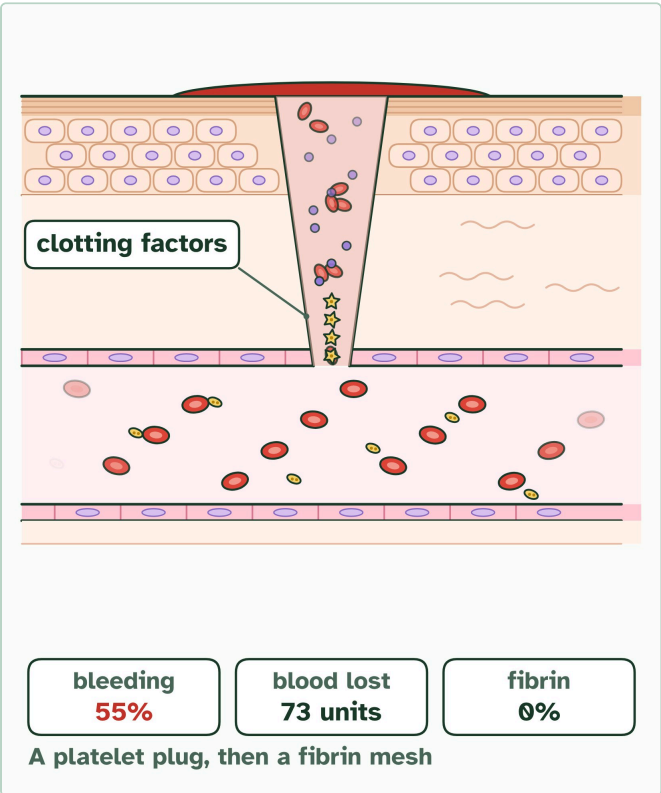
A **cut** breaks the skin and a small blood vessel under it. Blood leaks out, and microbes could get in.

2 CLOT: 30% SEALED



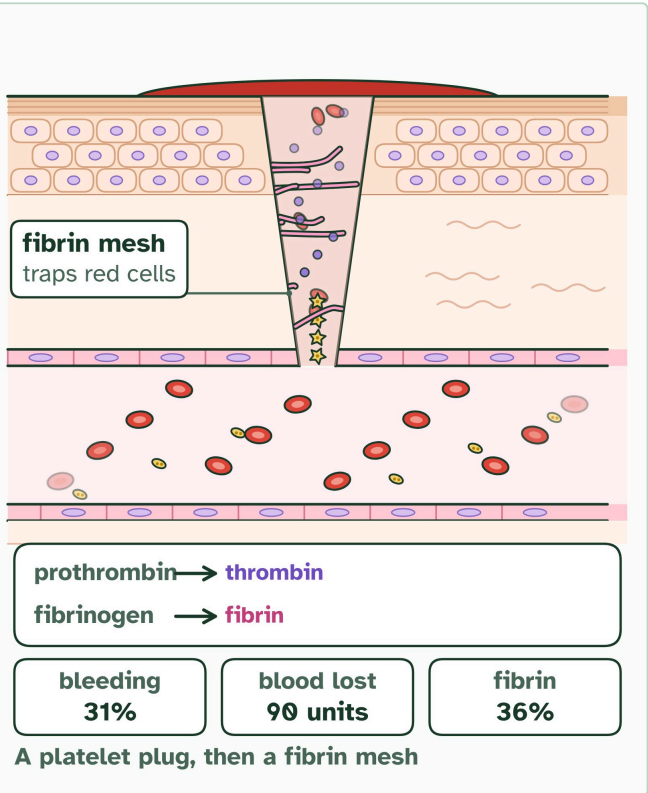
Platelets (cell fragments) stick to the damaged vessel wall and to each other. They make a first plug, and the bleeding slows.

3 CLOT: 45% SEALED



The platelets and the damaged cells release **clotting factors** (the purple dots).

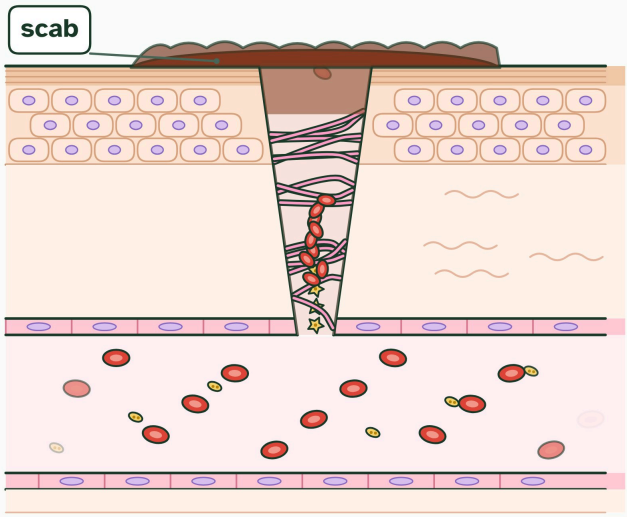
4 CLOT: 69% SEALED



The factors start a **cascade** of reactions. It makes **thrombin**, and thrombin converts soluble **fibrinogen** into insoluble **fibrin**.

5

CLOT: 100% SEALED



bleeding	blood lost	fibrin
0%	94 units	100%

A platelet plug, then a fibrin mesh

Fibrin threads make a **mesh** that traps red blood cells: a **clot**. The bleeding has stopped, and the clot will dry into a scab.

Watch it move, and change it as it runs: biologybybradford.com/guides/ib-biology-c3-2-defence-against-disease#clotting

PREDICT

Fibrinogen is dissolved in your blood plasma all the time. Why does your blood not clot inside healthy vessels?

(A) Blood vessels are too narrow for fibrin fibres to form in them

(B) Thrombin is made only after platelets and damaged tissue release clotting factors

(C) Fibrinogen can only be changed into fibrin in the presence of air

(D) Red blood cells stop fibrinogen reaching the lining of the vessels

Decide before you look. Answer on page 53.

REMEMBER IT

The clotting order

P	F	C	M
Penguins	Frequently	Catch	Minnows
Platelets	**Factors**	**Cascade**	**Mesh**
stick to the cut and plug it	clotting factors are released	prothrombin becomes thrombin	thrombin turns fibrinogen into a fibrin mesh

04 Two systems, *one fixed and one that learns*

C3.2.4 DIFFERENCES BETWEEN THE INNATE IMMUNE SYSTEM AND THE ADAPTIVE IMMUNE SYSTEM

You are born with the **innate immune system**. It responds to broad categories of pathogen, such as bacteria, in the same way however many times you meet them. It acts within minutes or hours. The phagocytes in the next chapter are your innate defenders.

The **adaptive immune system** is different. It responds in a specific way to one particular pathogen, takes days to get going on a first meeting, and builds a memory of every pathogen it meets, so the second response is faster and stronger. Lymphocytes do this work.

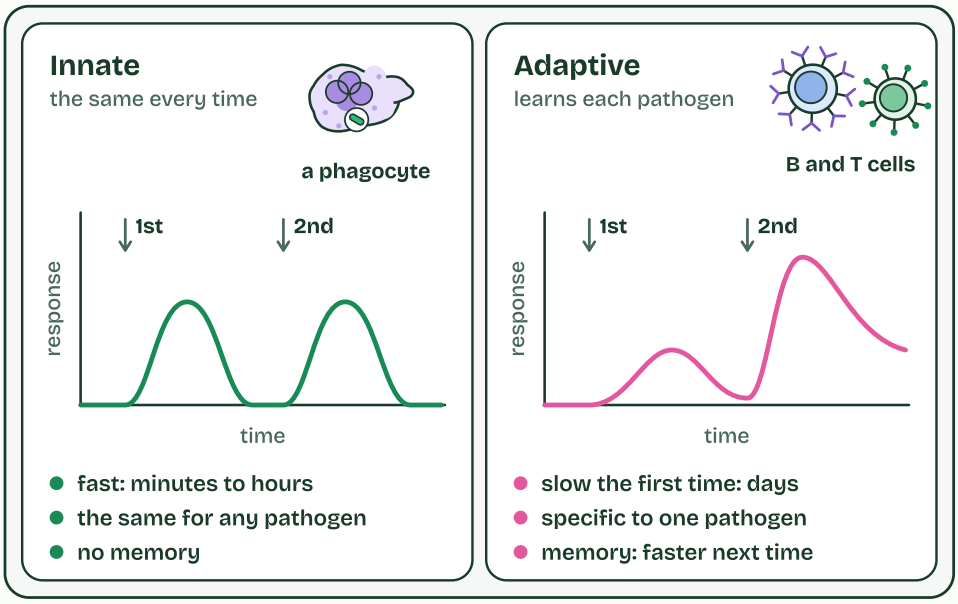
KEY POINTS

- **Innate:** broad categories of pathogen, quick, unchanged by experience.
- **Adaptive:** specific to one pathogen, slower the first time, builds a memory, improves with each meeting.

KEY TERMS

innate immune system
The defences you are born with. They respond to broad categories of pathogen, in the same way every time.

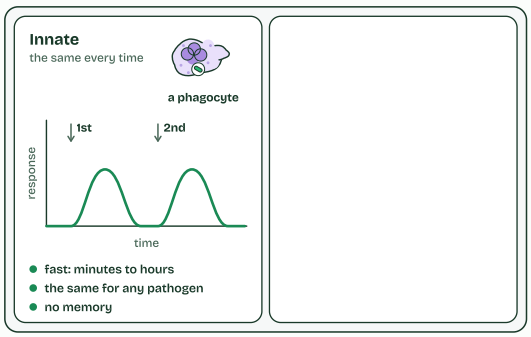
adaptive immune system
The part of the immune system that responds to one particular pathogen and remembers it, so the response improves with each meeting.



STEP BY STEP

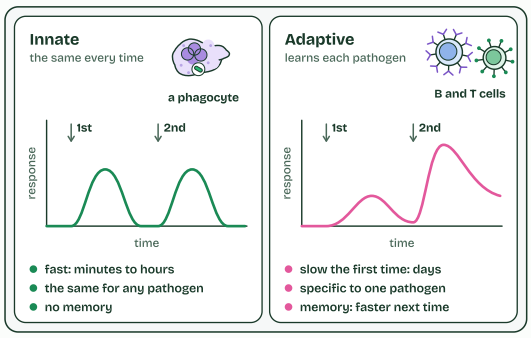
2 STEPS

1



Innate: the response is as strong at the second meeting as at the first. It does not change with experience.

2



Adaptive: the first response is small and slow, the second is much stronger. The system has learned the pathogen.

PREDICT

A child catches chickenpox and recovers. Meeting the virus again years later, she does not get ill. Which system explains that?

(A) Innate, because it responds to the same virus every time

(B) The skin, because the virus cannot get through it again

(C) Adaptive, because it remembers the particular pathogen

(D) Innate, because phagocytes learn from each infection

Decide before you look. Answer on page 53.

05 Cells that *eat* microbes

C3.2.5 INFECTION CONTROL BY PHAGOCYTES

Whatever gets past the barriers meets **phagocytes**, white blood cells that eat pathogens. They circulate in the blood and, at an infection, squeeze out between the cells of the capillary wall and move through the tissue by **amoeboid movement**, following chemicals released by the damaged tissue and the microbes.

A phagocyte recognizes a pathogen by receptors that bind to molecules on its surface and engulfs it by **endocytosis**. The pathogen is inside a vesicle, which fuses with **lysosomes**. Their enzymes digest it. The pus in a boil is largely dead phagocytes and the pathogens they ate.

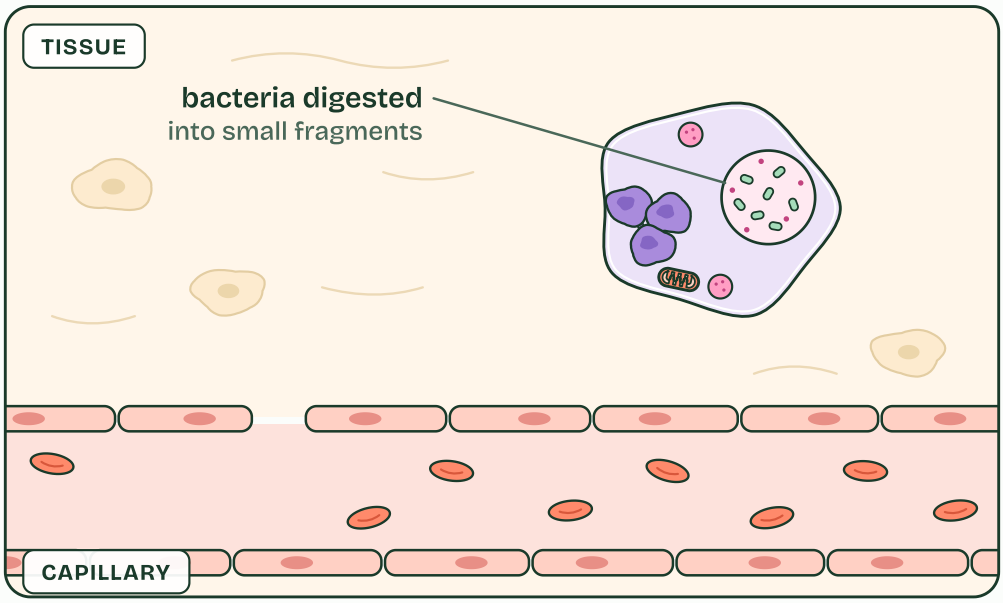
KEY TERMS

phagocytes
White blood cells that engulf pathogens and digest them. Neutrophils and macrophages are phagocytes.

amoeboid movement
Movement by pushing out a part of the cell, a pseudopodium, and flowing into it.

endocytosis
Taking material into a cell by folding the plasma membrane round it to make a vesicle.

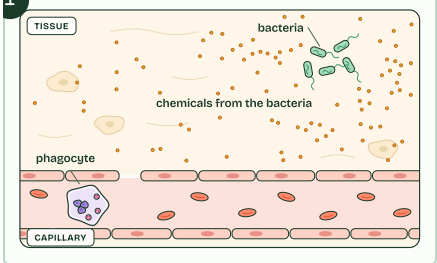
lysosomes
Small organelles that hold digestive enzymes.



STEP BY STEP

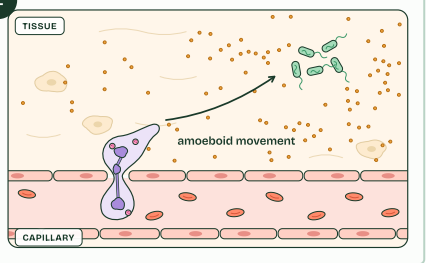
5 STEPS

1



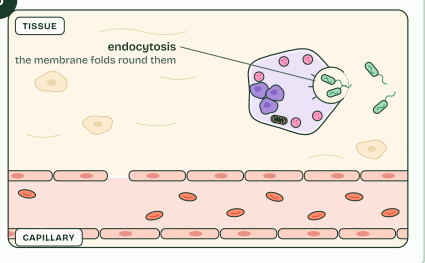
A phagocyte in a blood vessel senses **chemicals** from an infection in the tissue above.

2



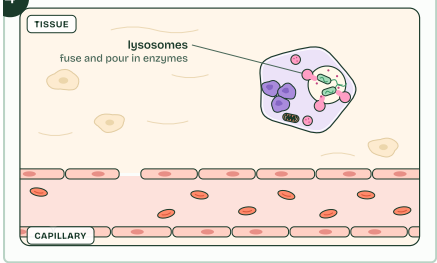
It leaves the vessel and moves through the tissue by **amoeboid movement** towards the bacteria.

3



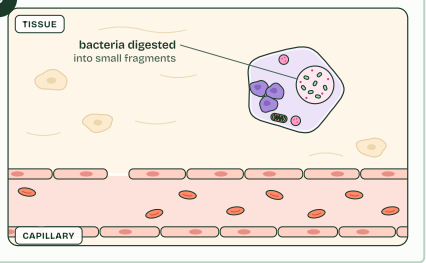
It **recognizes** the bacteria and engulfs them by **endocytosis**: they are now inside a vesicle.

4



Lysosomes fuse with the vesicle and pour in digestive enzymes.

5



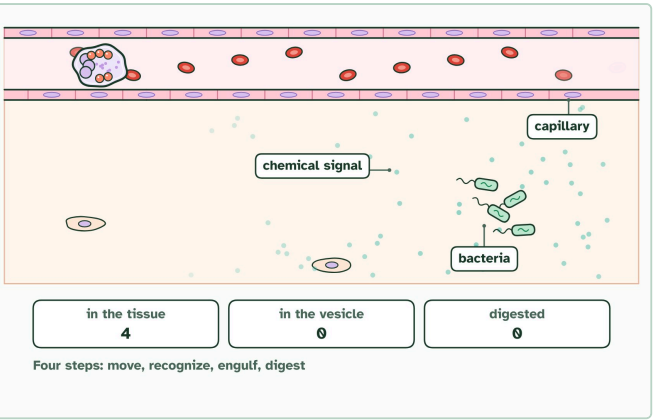
The bacteria are **digested** into fragments.

ANIMATION · A PHAGOCYTE AT WORK

5 MOMENTS

1

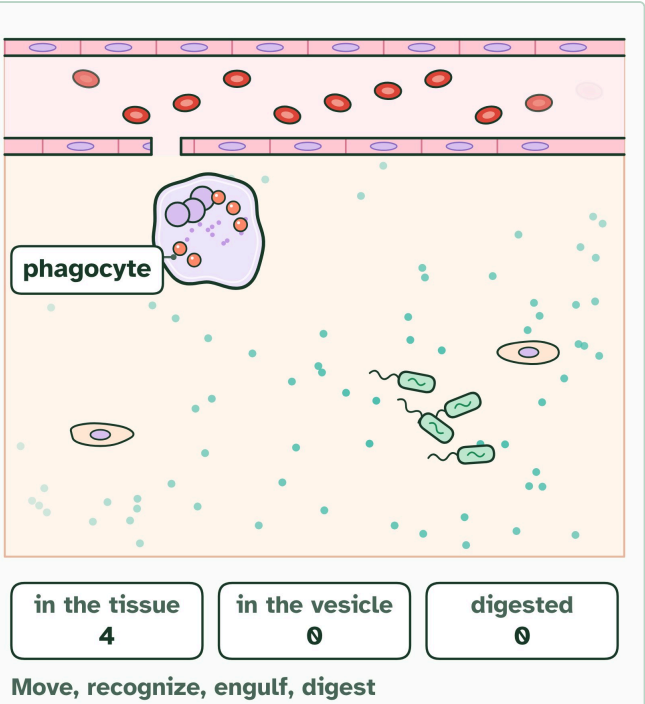
0 OF 4 BACTERIA DIGESTED



Bacteria in a wound give off **chemicals**. They spread out through the tissue, stronger near the bacteria.

2

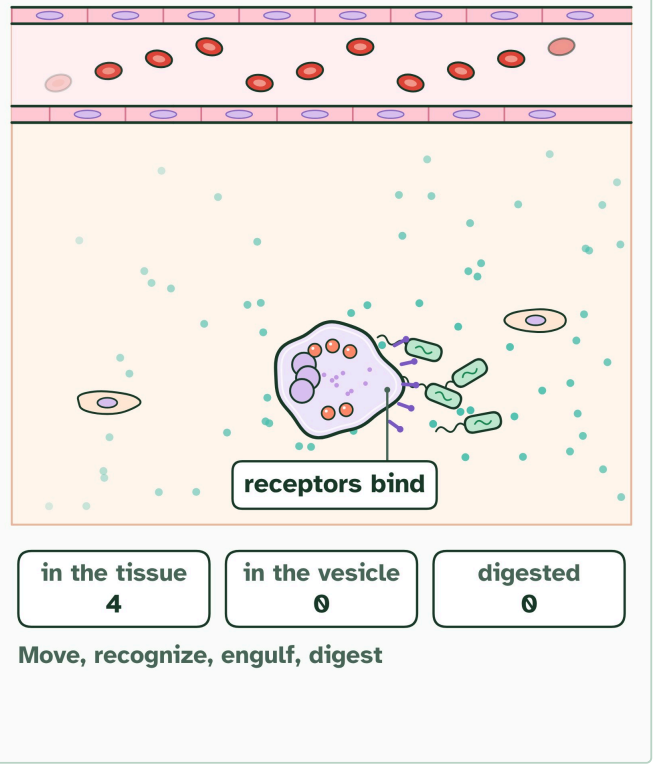
0 OF 4 BACTERIA DIGESTED



A **phagocyte** squeezes out of the blood vessel. It moves by changing its shape, **amoeboid movement**, towards the stronger signal.

3

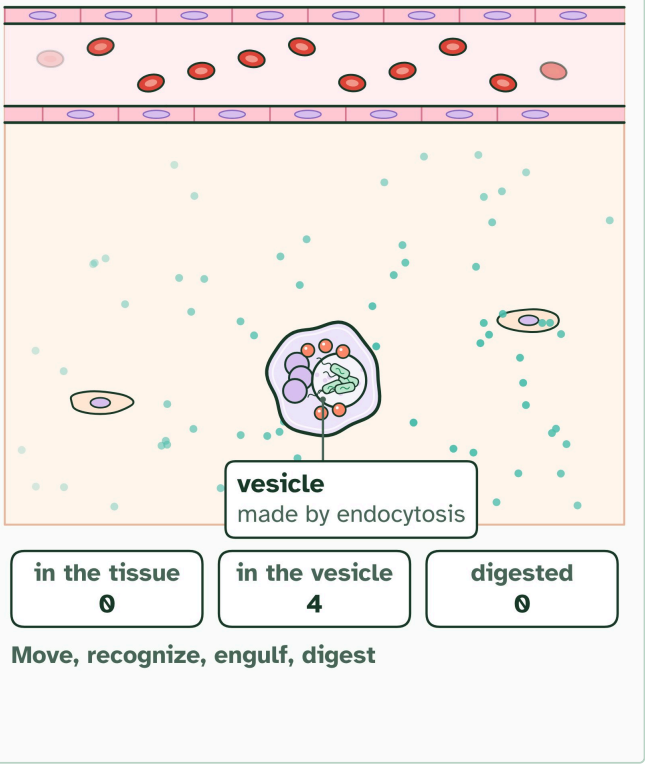
0 OF 4 BACTERIA DIGESTED



Receptors on the phagocyte surface **recognize** and bind to the bacteria.

4

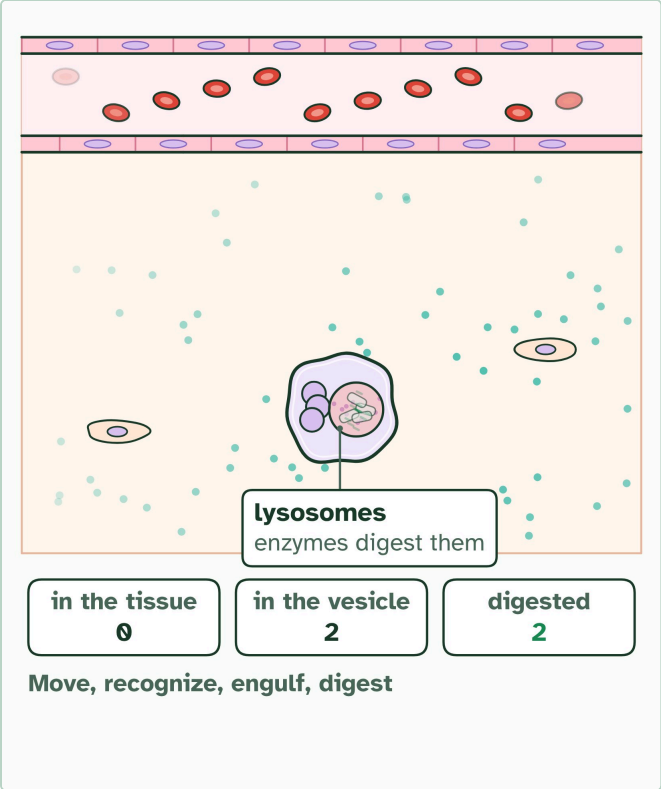
0 OF 4 BACTERIA DIGESTED



The phagocyte **engulfs** the bacteria by **endocytosis**. They are now inside a vesicle.

5

2 OF 4 BACTERIA DIGESTED



Lysosomes fuse with the vesicle and empty their enzymes into it. The enzymes **digest** the bacteria.

Watch it move, and change it as it runs: biologybybradford.com/guides/ib-biology-c3-2-defence-against-disease#phagocytes

PREDICT

Why can a phagocyte digest a bacterium without digesting its own cytoplasm?

(A) It has no digestive enzymes of its own

(B) The enzymes are released into the vesicle that holds the bacterium, not into the cytoplasm

(C) The bacterium makes the enzymes that destroy it

(D) Phagocytes do not digest; they only store bacteria

Decide before you look. Answer on page 53.

REMEMBER IT

The four stages of phagocytosis

M My	R Rhino	E Eats	D Dinner
Move to the infection by amoeboid movement	**Recognize** the pathogen with surface receptors	**Engulf** it by endocytosis into a vesicle	**Digest** it with enzymes from lysosomes

IN THE EXAM

Phagocytosis in four short points:

1. Phagocytes move out of the blood to the infection by amoeboid movement.
2. They recognize the pathogen by receptors binding to its surface.
3. They engulf it by endocytosis, forming a vesicle.
4. Lysosomes fuse with the vesicle and their enzymes digest it.

C3.2.6 C3.2.7 LYMPHOCYTES AS CELLS IN THE ADAPTIVE IMMUNE SYSTEM ANTIGENS AS RECOGNITION MOLECULES THAT TRIGGER ANTIBODY PRODUCTION

06 A *lock* for every key

An **antigen** is a recognition molecule. It is usually a protein or glycoprotein on the outside of a pathogen, which the immune system sees as foreign. Your own cells carry molecules the system learns to leave alone.

Lymphocytes are the cells that do the recognizing, and they circulate in the blood and gather in lymph nodes. Each **B cell** makes one type of **antibody**, with binding sites that fit one antigen. You carry a huge variety of B cells, each with a different fit, so for almost any antigen there is a few that match. Antibodies are made only after a match, by the B cells that match, and with the help of other lymphocytes.

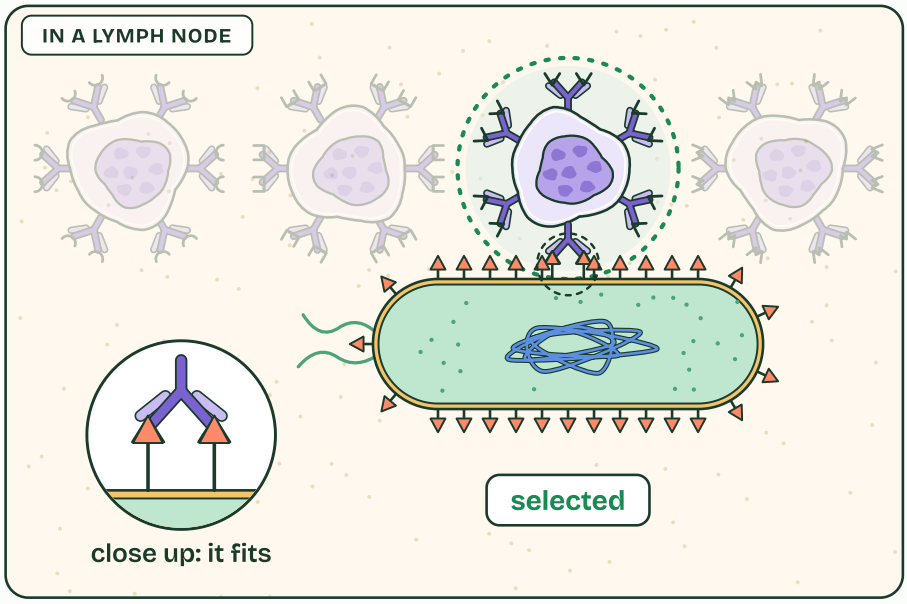
KEY TERMS

antigen
A molecule that is recognized by the immune system and triggers an immune response, usually a protein or glycoprotein on the surface of a pathogen.

lymphocytes
White blood cells of the adaptive immune system: B cells and T cells.

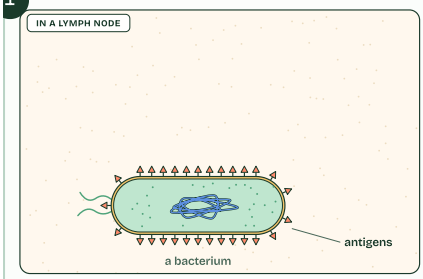
B cell
A lymphocyte that makes antibodies once activated.

antibody
A Y-shaped protein made by plasma cells that binds to one specific antigen.



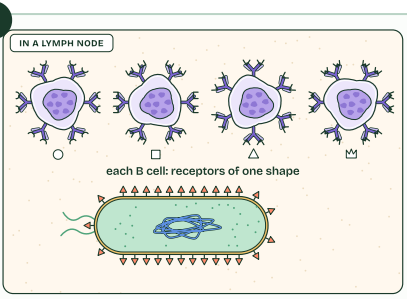
STEP BY STEP
4 STEPS

1



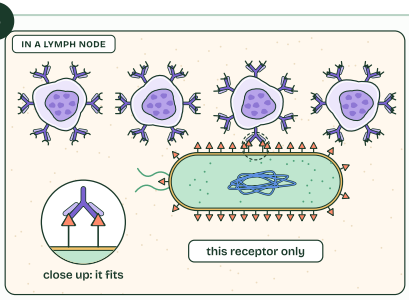
A bacterium carries **antigens** on its surface: here, triangles.

2



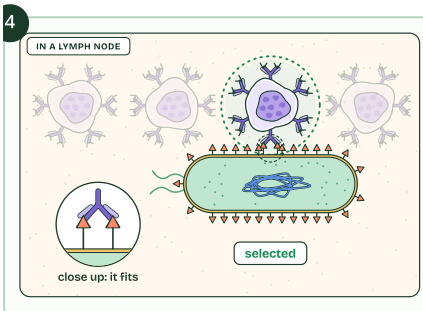
You have many **B cells**, each with receptors of one shape. Between them they fit millions of antigens.

3



An antigen **fits** only the receptor that matches it, like a key in a lock.

4



The B cell that matches is **selected**. The others stay as they are.

PREDICT

A B cell with receptors that fit a measles antigen meets a mumps virus. What happens?

(A) It makes antibodies against mumps anyway, to be safe

(B) Nothing: the receptor does not fit the mumps antigens, so the cell is not selected

(C) It changes its receptor to fit the mumps antigen at once

(D) It is destroyed by the mumps virus

Decide before you look. Answer on page 53.

WATCH OUT

Antibodies are not made in advance for everything, and an antigen does not shape a new antibody. The B cell with the right fit is already there, and it is selected.

07 A B cell *needs a second opinion*

C3.2.8 ACTIVATION OF B-LYMPHOCYTES BY HELPER T-LYMPHOCYTES

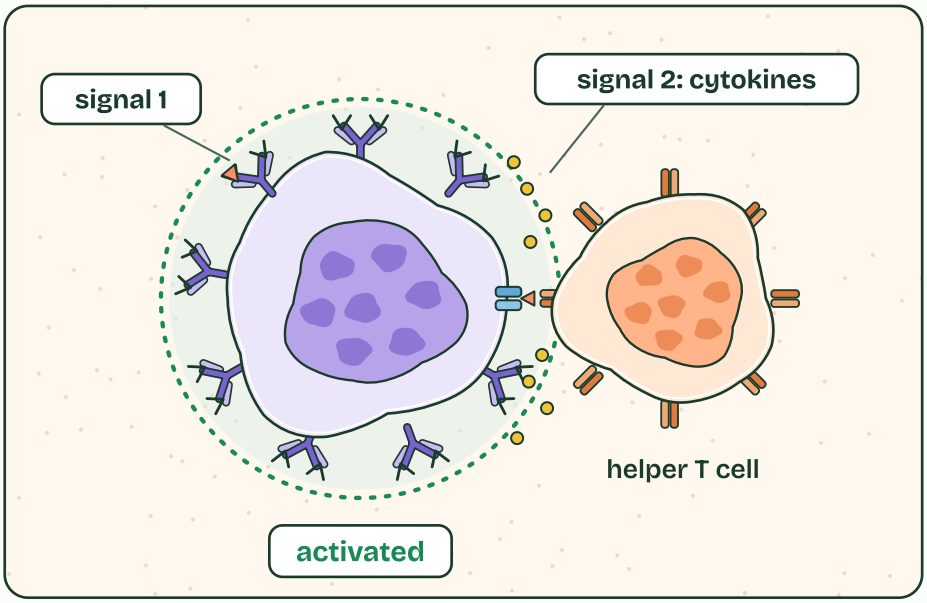
A B cell does not start making antibodies just because an antigen fits. It must be **activated**, and that needs two things. First, its receptors bind their specific antigen. Second, a **helper T cell** that has been activated by the same type of antigen makes contact and releases chemical signals.

Having two signals is a safety check. A single mistaken match is not enough to set off a powerful response against your own tissue. The cells only act together when both lymphocytes have found the same enemy.

KEY TERMS

activated
Triggered to divide and make antibodies. A B cell needs two things to be activated: its specific antigen and contact with a helper T cell.

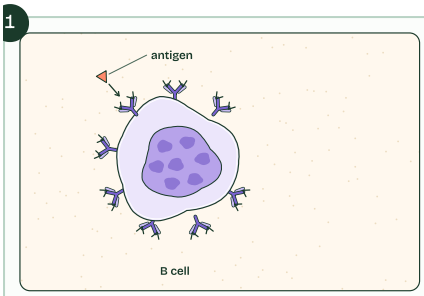
helper T cell
A lymphocyte that has itself been activated by the same antigen and which activates B cells by contact and chemical signals.



STEP BY STEP

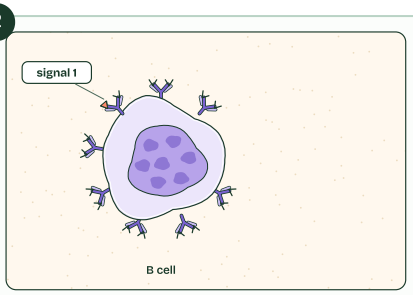
6 STEPS

1



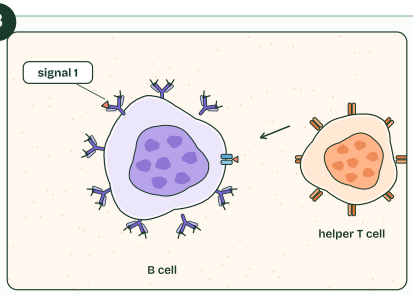
A B cell with a matching **receptor**, and the antigen about to bind.

2



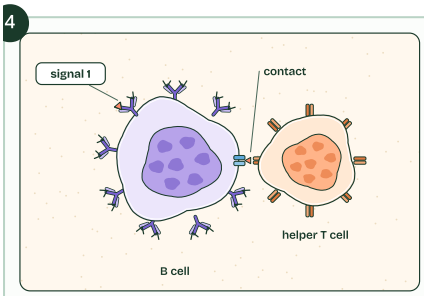
Signal 1: the antigen binds the receptor.

3



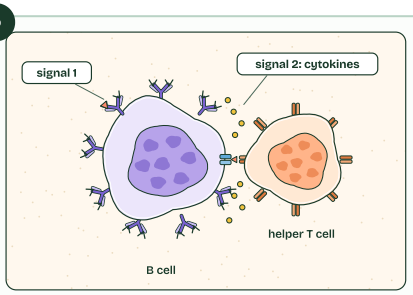
A **helper T cell** that was activated by the same antigen approaches.

4



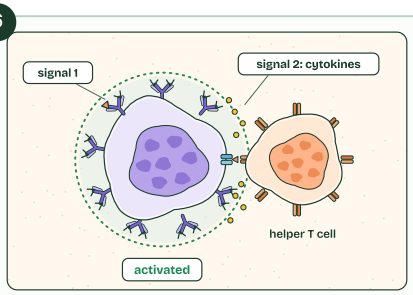
The two cells make **contact**: the T cell's receptor binds the antigen the B cell now shows.

5



Signal 2: the T cell releases chemical signals called **cytokines**.

6



With both signals, the B cell is **activated**.

PREDICT

A B cell binds its antigen but no helper T cell has been activated by that antigen. What does the B cell do?

(A) Divides and makes antibodies at once

(B) Remains inactive, because it has only one of the two signals

(C) Turns into a helper T cell

(D) Is engulfed by a phagocyte

Decide before you look. Answer on page 53.

C3.2.9 MULTIPLICATION OF ACTIVATED B-LYMPHOCYTES TO FORM CLONES OF ANTIBODY-SECRETING PLASMA CELLS

08 One cell becomes *an army*

Very few B cells match any one antigen, far too few to make enough antibody. So an activated B cell first divides by **mitosis**, again and again, to make a **clone** of identical cells. Most become **plasma cells**: big cells full of rough endoplasmic reticulum, built to make one type of antibody, the one the original B cell's receptor fits, and release it in huge quantity. Some of the clone stay behind as memory cells.

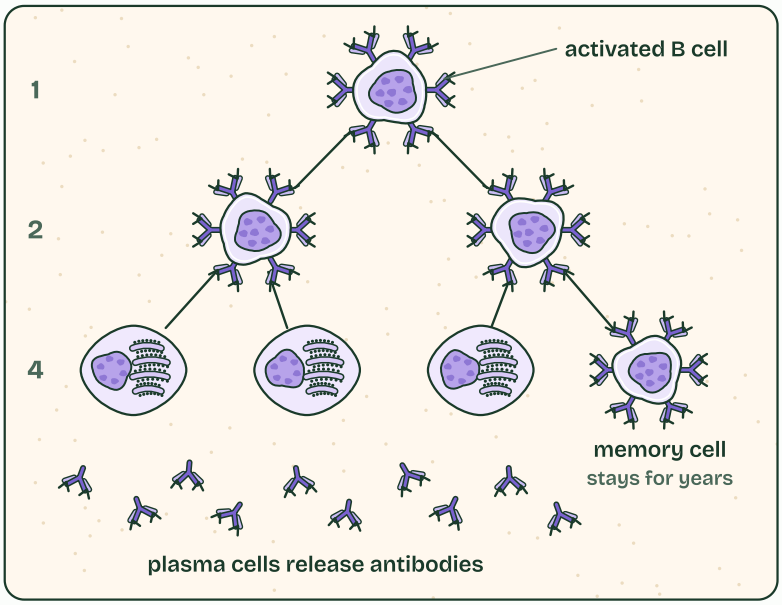
This is called clonal selection. The antigen selects the one B cell that fits, and that cell multiplies.

KEY TERMS

mitosis
Cell division that produces two genetically identical daughter cells.

clone
A group of genetically identical cells all descended from one parent cell.

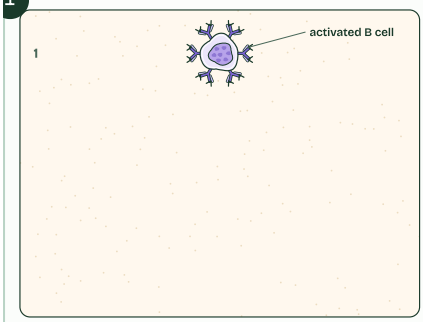
plasma cells
Activated B cells that make and release large amounts of one type of antibody.



STEP BY STEP

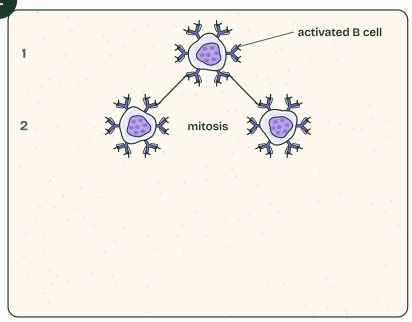
4 STEPS

1



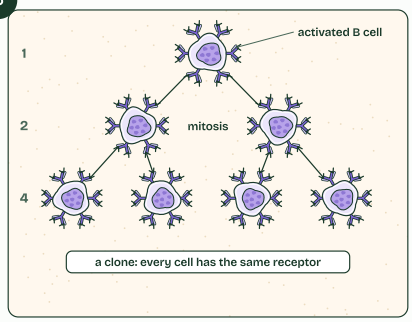
One **activated** B cell.

2



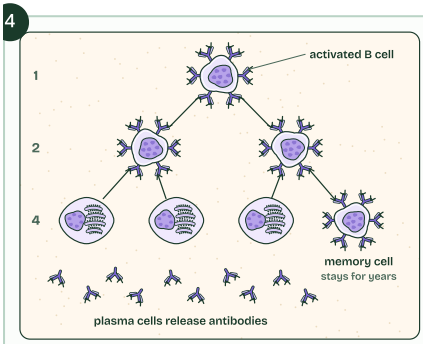
It divides by **mitosis**: two identical cells.

3



They divide again, and again. The **clone** grows quickly, every cell with the same receptor.

4



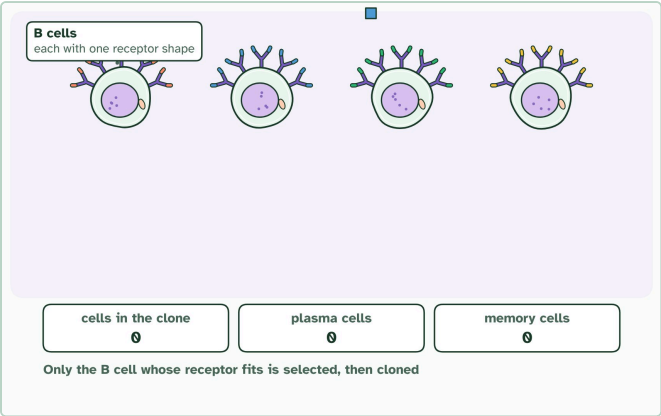
Most of the clone become **plasma cells** that release antibodies. Some stay as **memory cells**.

ANIMATION · CLONAL SELECTION

5 MOMENTS

1

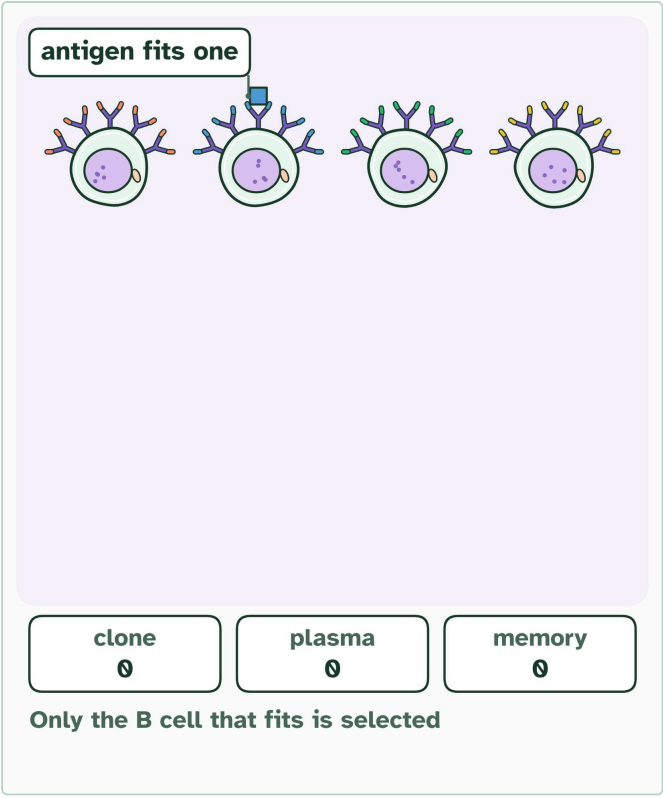
ANTIGEN: SQUARE



You have millions of kinds of **B cell**. Each kind has **receptors of one shape** on its surface, and most never meet the antigen that fits them.

2

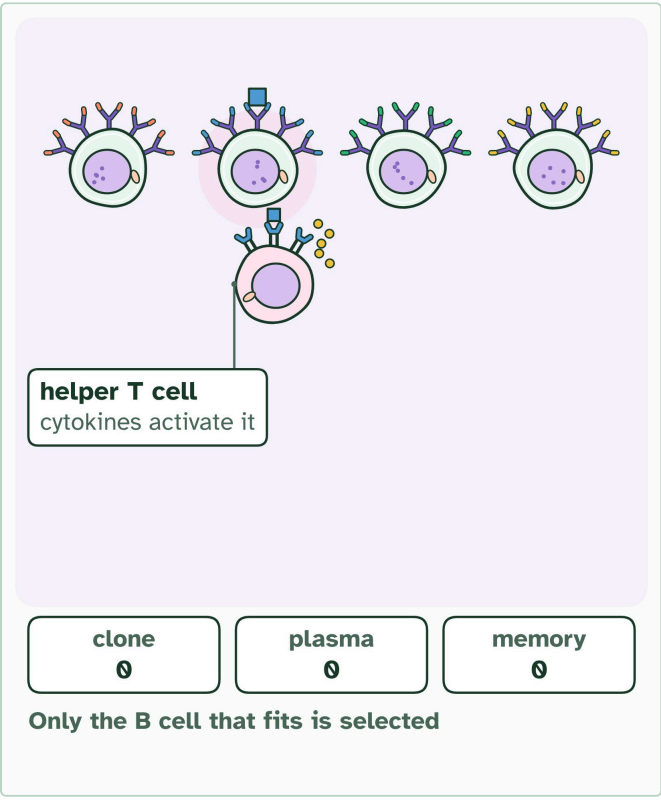
ANTIGEN: SQUARE



An **antigen** arrives. It binds only to the B cell whose receptor **fits** it. The other B cells ignore it. This is **selection**.

3

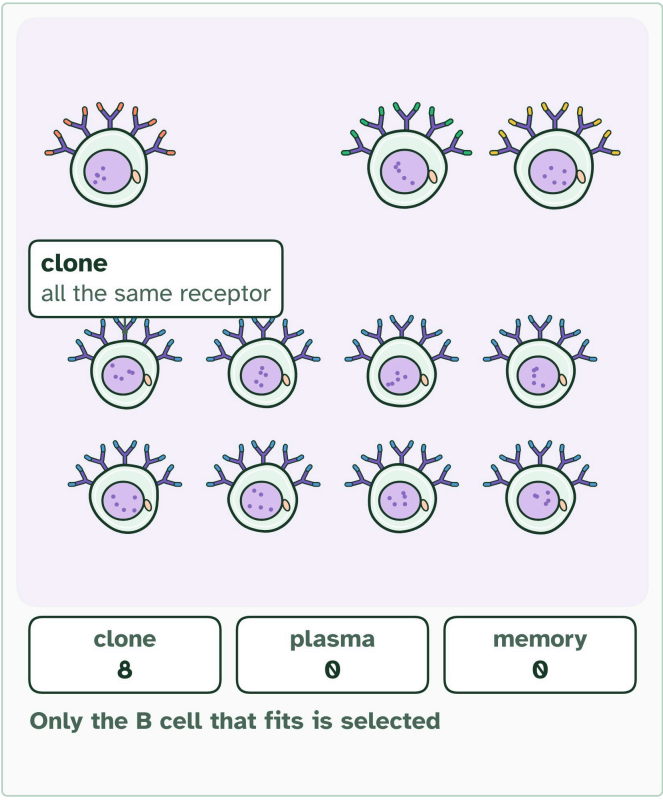
ANTIGEN: SQUARE



A **helper T cell**, activated by the same antigen, touches the B cell and **activates** it.

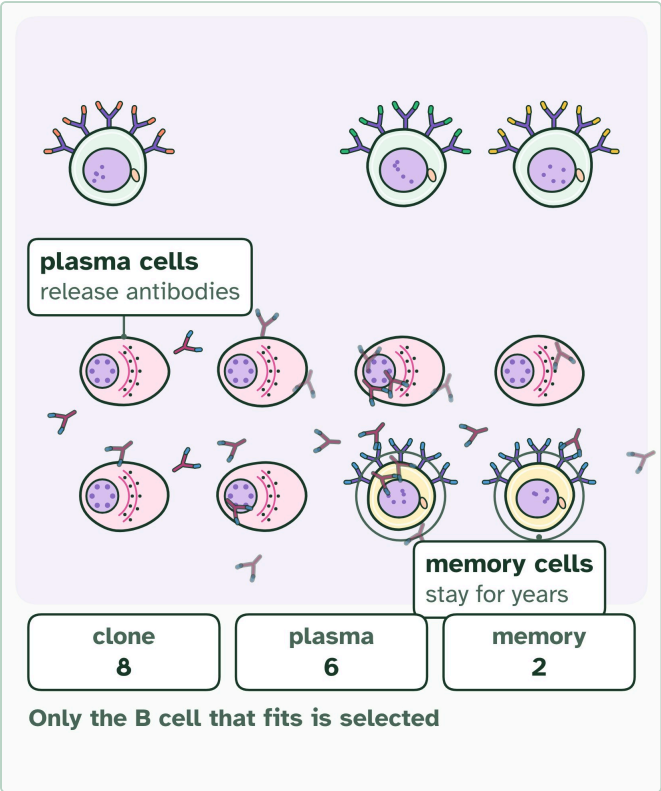
4

8 CELLS IN THE SQUARE CLONE



The activated B cell divides by **mitosis**, again and again, to make a **clone**: every cell has the same receptor.

5 8 CELLS IN THE SQUARE CLONE



Most of the clone becomes **plasma cells** that release **antibodies**. A few become **memory cells**, which stay in the body and respond fast next time.

Watch it move, and change it as it runs: biologybybradford.com/guides/ib-biology-c3-2-defence-against-disease#clones

PREDICT

Why are the plasma cells packed with rough endoplasmic reticulum?

(A) They store the antibody for years until it is needed

(B) They are making large amounts of protein, which antibodies are, to secrete

(C) They need it to divide by mitosis

(D) It is where they recognize antigens

Decide before you look. Answer on page 53.

REMEMBER IT

Clonal selection in five steps

R Rude	A Aliens	M Might	S Steal	R Rockets
Recognize one B cell's receptor fits the antigen	**Activate** a helper T cell gives the second signal	**Multiply** the B cell divides by mitosis into a clone	**Secrete** plasma cells release antibodies	**Remember** memory cells stay in the body

IN THE EXAM

Antibody production in short points:

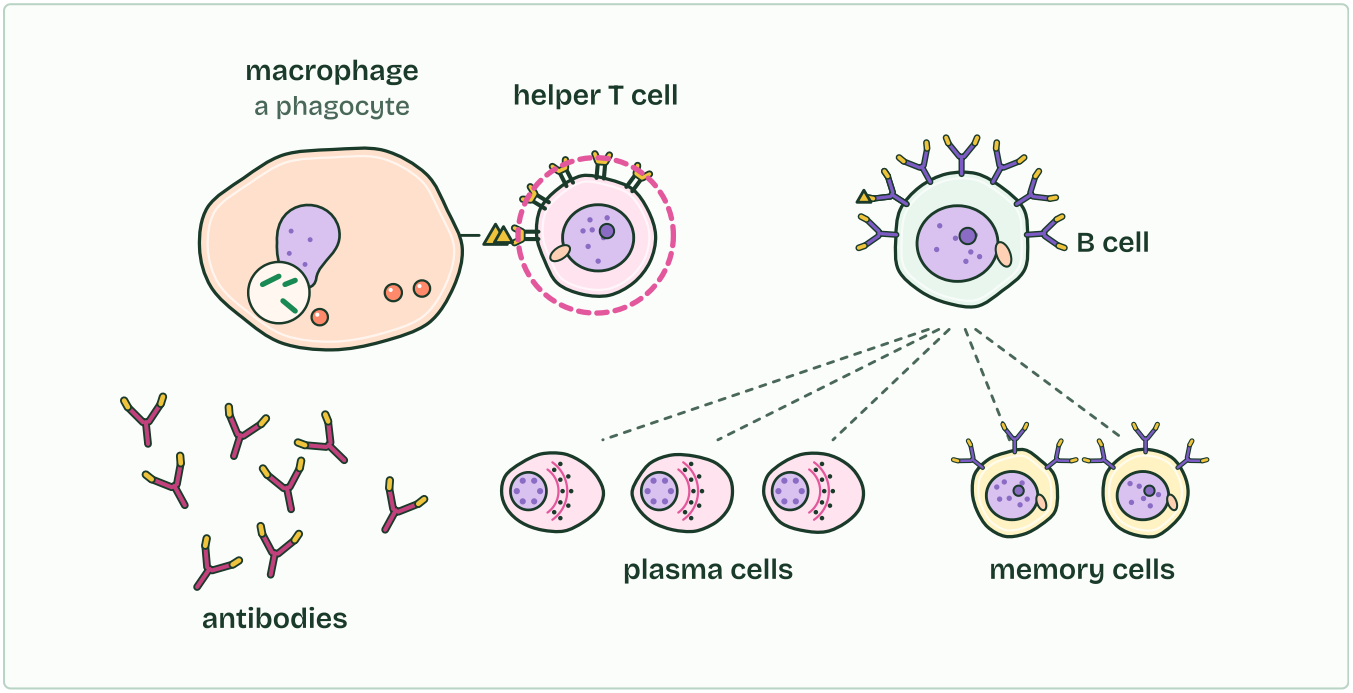
1. An antigen binds to the receptor of the one B cell it fits.
2. A helper T cell activated by the same antigen activates the B cell.
3. The B cell divides by mitosis to form a clone.
4. The clone becomes plasma cells that secrete antibodies, and memory cells.

09

C3.2.6 THE WHOLE FLOW OF ANTIBODY PRODUCTION

From pathogen to *antibodies and memory*

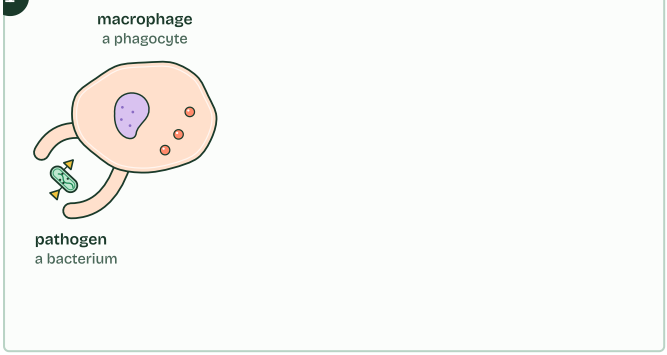
Put the last four chapters together and the response runs as one chain. A phagocyte takes in the pathogen and shows its antigen. A helper T cell that fits the antigen is activated. A B cell that fits the same antigen binds it, and the helper T cell then activates the B cell. The B cell divides into a clone, most of the clone become plasma cells that secrete antibody, and a few become memory cells. Step through the animation one stage at a time, and say each stage aloud before you read its caption.



STEP BY STEP

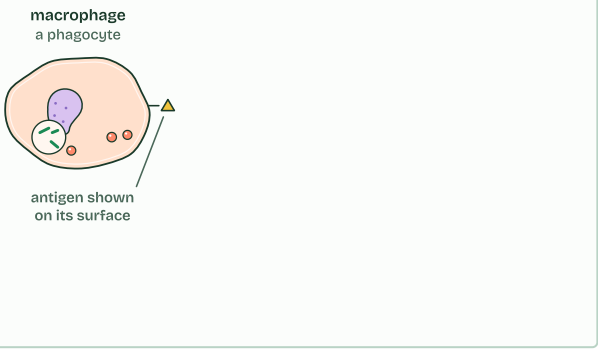
8 STEPS

1



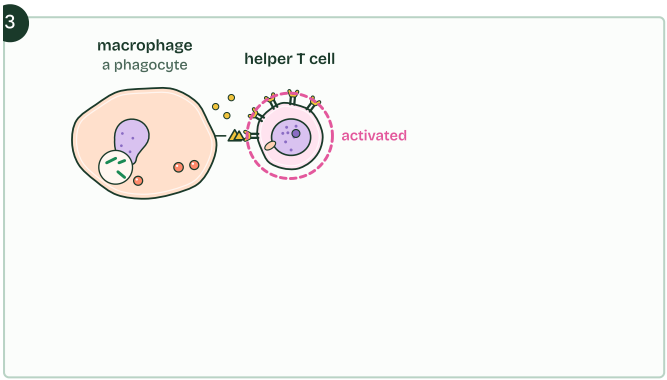
Engulf. A **macrophage**, a kind of phagocyte, reaches round a pathogen and takes it in by endocytosis.

2



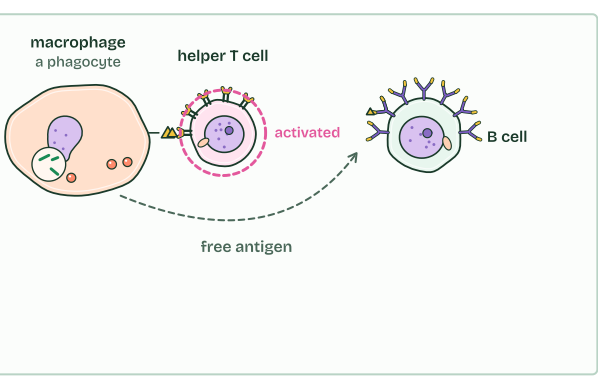
Present. Lysosomes digest the pathogen, but the macrophage keeps pieces of its **antigen** and shows them on its own surface.

3



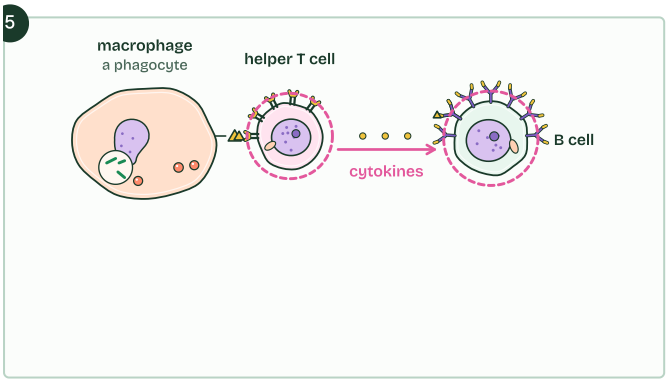
Activate. A **helper T cell** whose receptor fits that antigen binds it, and the macrophage's chemical signals activate the T cell.

4



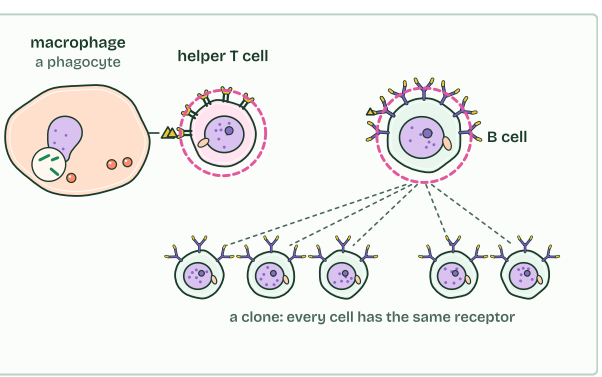
Bind. Free antigen reaches a **B cell** whose receptor fits the same antigen, and binds to it.

5



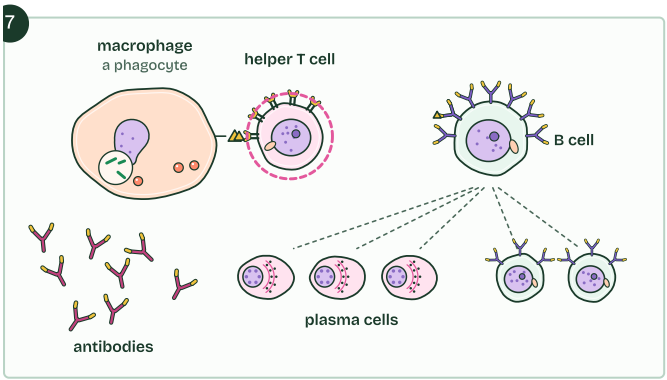
Help. The activated helper T cell binds the same B cell and releases **cytokines**. With both signals the B cell is activated.

6



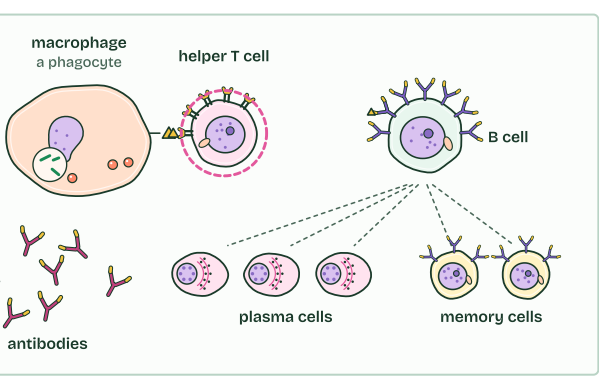
Multiply. The activated B cell divides by **mitosis**, again and again, into a **clone** of identical cells.

7



Secrete. Most of the clone become **plasma cells**, packed with rough ER, which release huge numbers of one **antibody**.

8



Remember. Some of the clone become **memory cells**. They stay for years and divide fast if the same antigen returns.

REMEMBER IT

Antibody production, from pathogen to memory, in eight steps

E	P	A	B	H	M	S	R
Every	Pink	Aardvarks	Bring	Hot	Mango	Smoothies	Regularly
Engulf a macrophage takes in the pathogen	**Present** it shows the antigen on its surface	**Activate** a helper T cell with the matching receptor is activated	**Bind** a B cell with the matching receptor binds the antigen	**Help** the T cell gives the B cell its second signal	**Multiply** the activated B cell divides by mitosis into a clone	**Secrete** plasma cells release antibodies	**Remember** memory cells stay for a faster response next time

IN THE EXAM

A long answer on antibody production earns its marks one step at a time:

1. A phagocyte engulfs the pathogen and presents its antigen on its surface.
2. A helper T cell with a receptor that fits the antigen is activated.
3. A B cell with a receptor that fits the same antigen binds it.
4. The activated helper T cell activates the B cell.
5. The B cell divides by mitosis to form a clone.
6. The clone becomes plasma cells that secrete antibodies, and memory cells.

10 The second time is *faster and bigger*

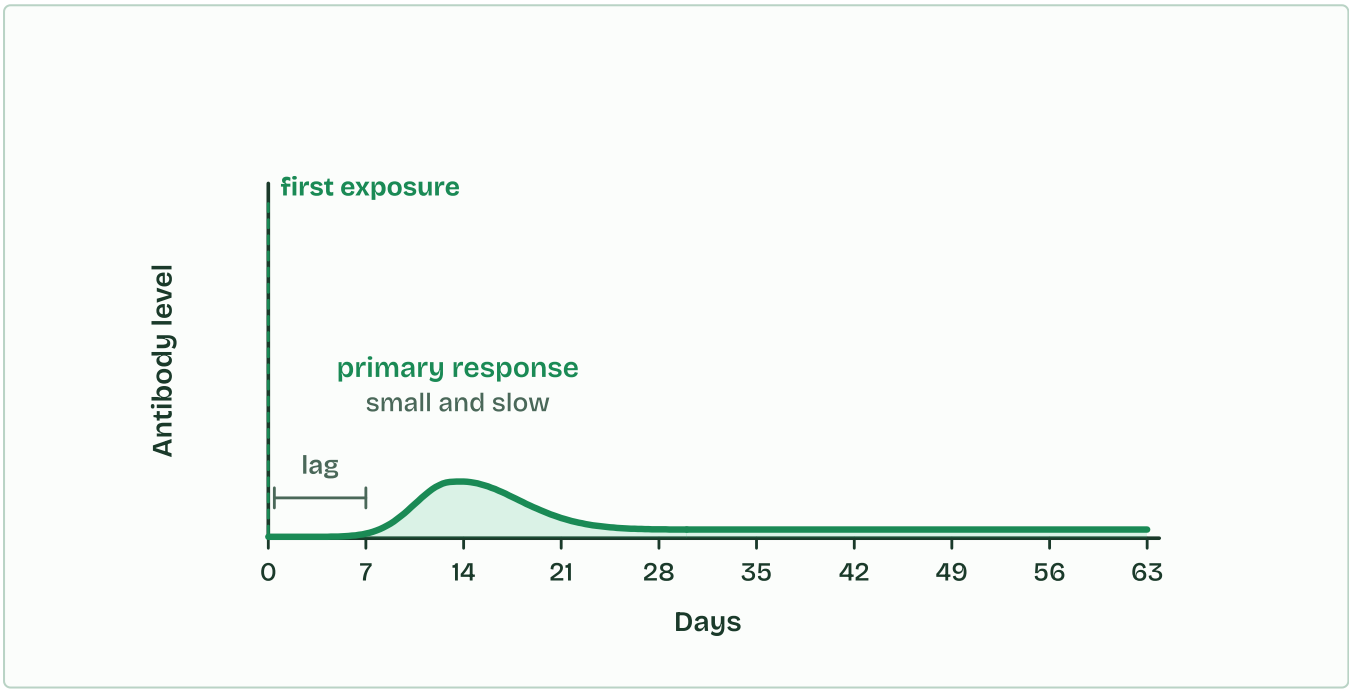
C3.2.10 IMMUNITY AS A CONSEQUENCE OF RETAINING MEMORY CELLS

When the infection is over, the plasma cells die and the antibody level falls, but some of the clone survive for years as **memory cells**. They are lymphocytes that can make the specific antibody for that pathogen. This is **immunity**: if the same pathogen returns, memory cells are already there, so the response starts at once, rises higher and lasts longer.

KEY TERMS

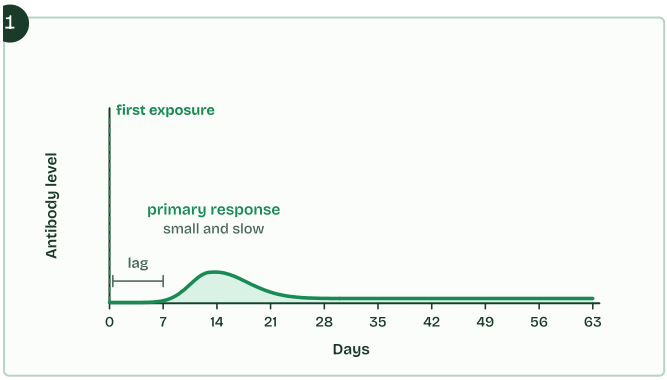
memory cells
Long-lived lymphocytes that remain after an infection and can respond very quickly if the same antigen returns.

immunity
The ability to eliminate an infectious disease from the body, because memory cells for it are already present.



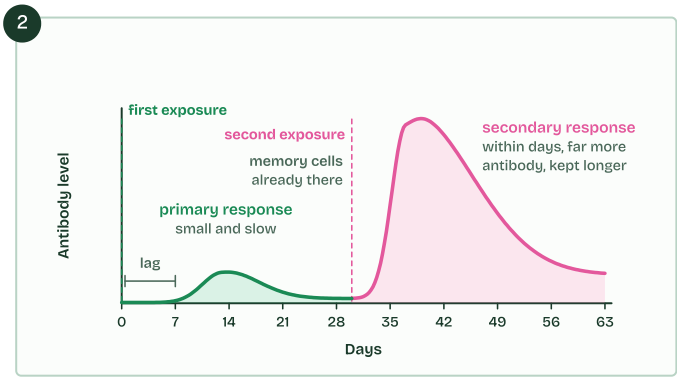
SWITCH IT 2 SETTINGS

1



First exposure

2



Both exposures

PREDICT

On the second exposure to the same pathogen, compared with the first, which description is right?

(A) A longer lag, but a taller peak

(B) The same lag and the same peak, because the antigen is the same

(C) A shorter lag and a taller peak

(D) A shorter lag and a smaller peak, because there is less to fight

Decide before you look. Answer on page 54.

IN THE EXAM

Primary against secondary response, in points:

1. First exposure: a lag of several days while the matching B cell is selected, activated and cloned.
2. Memory cells remain after the infection.
3. Second exposure: memory cells respond quickly, so the lag is short.
4. More antibody, made faster and kept for longer, so the pathogen is destroyed before it causes illness.

C3.2.11 C3.2.12 TRANSMISSION OF HIV IN BODY FLUIDS INFECTION OF LYMPHOCYTES BY HIV WITH AIDS AS A CONSEQUENCE

11 The virus that *takes out the commander*

HIV is carried in body fluids: blood, semen, vaginal fluids and breast milk. It passes between people through those fluids in a few ways, shown in the first figure, and not by everyday contact.

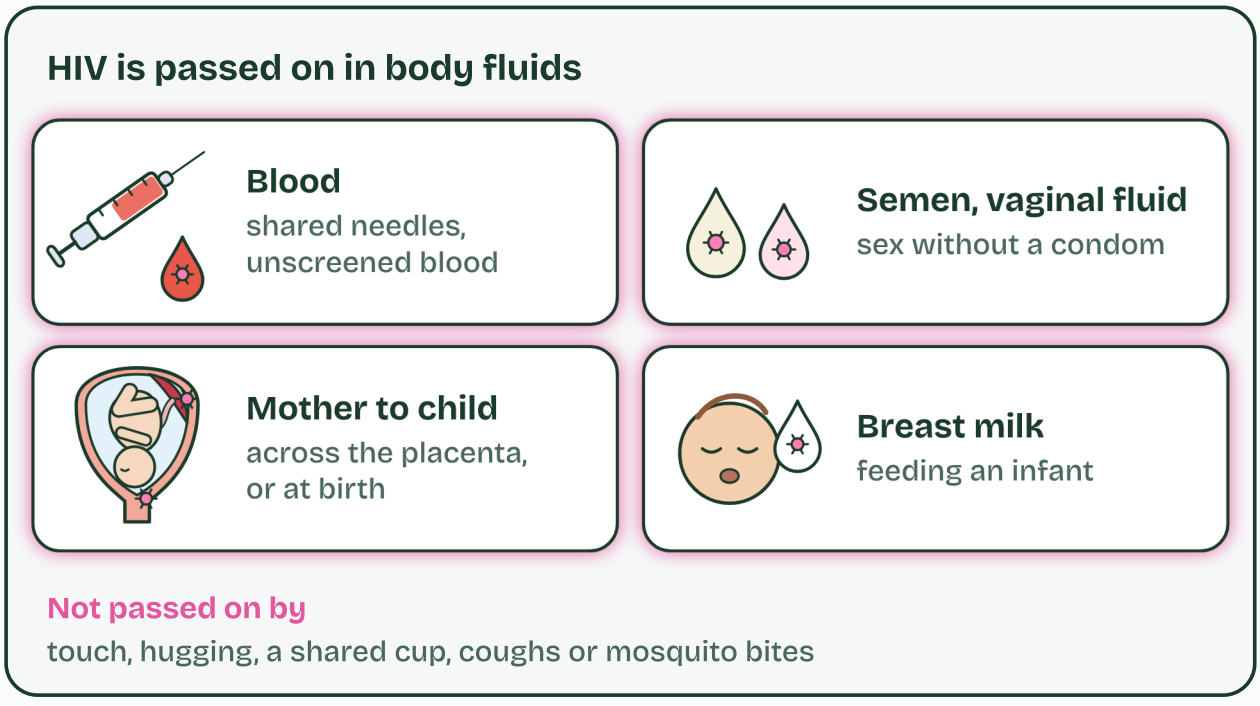
Once inside, HIV infects certain lymphocytes, the helper T cells, and kills them. After a first burst of virus the immune system holds it down for years, but the helper T cell count slowly falls. Without helper T cells, B cells cannot be activated, so fewer antibodies are made and the person cannot fight infections that a healthy immune system would deal with. When the count falls far enough, the person has **AIDS**. They die from **opportunistic infections**, not from HIV itself. Drugs that block the virus's copying keep the count high, and a person on treatment can stay well.

KEY TERMS

HIV
Human immunodeficiency virus, an RNA virus that infects and kills helper T cells.

AIDS
Acquired immunodeficiency syndrome: the stage of HIV infection when too few helper T cells are left to fight off opportunistic infections.

opportunistic infections
Infections by organisms that would not normally harm a person with a healthy immune system.

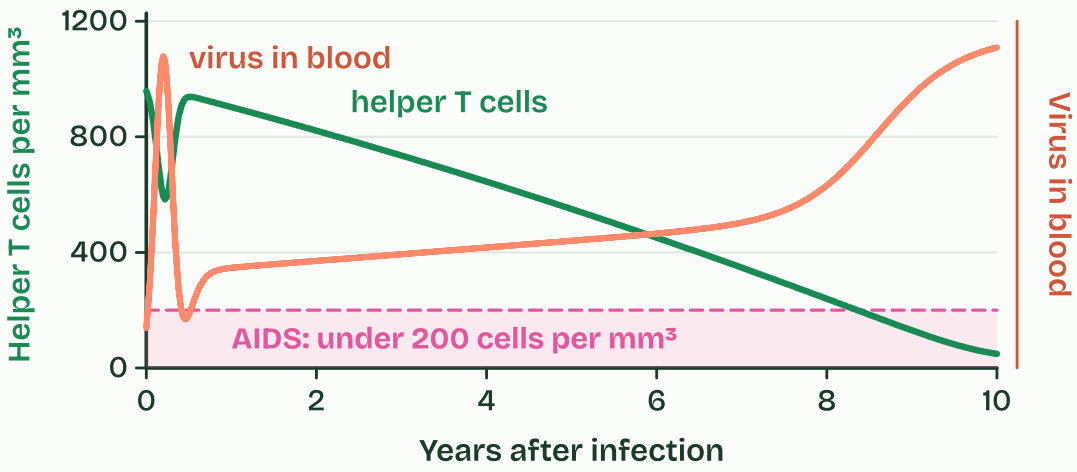


Blood Sharing needles that carry blood, or receiving blood or blood products that were not screened. In places where donated blood is tested, this route is now very rare.

Mother to child The virus can pass through the placenta or at birth. Medicines for the mother lower this risk to very small.

Semen and vaginal fluids HIV passes during sex without a condom, through the lining of the genital tract or the rectum. Condoms greatly reduce the risk.

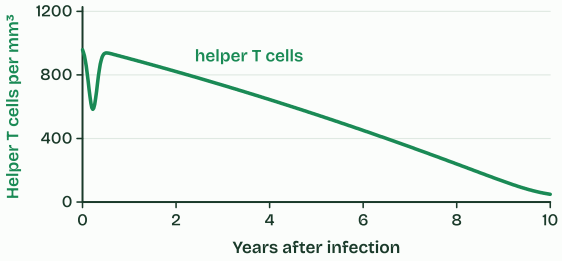
Breast milk The virus is present in breast milk. Where safe alternatives or treatment are available, this route is avoidable.



STEP BY STEP

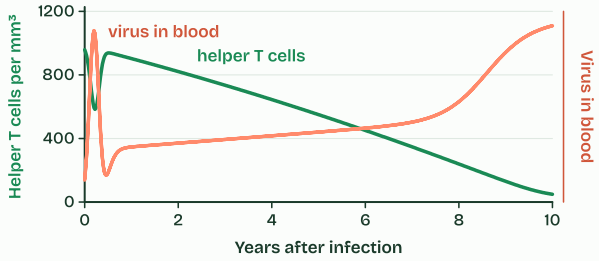
3 STEPS

1



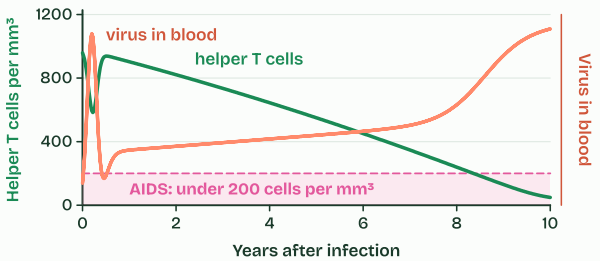
The number of **helper T cells** dips early, then falls slowly over years in an untreated infection.

2



The **virus** in the blood peaks early, is held down for years, then rises as the immune system fails.

3



When helper T cells fall below about 200 per mm³, the person has **AIDS**, and opportunistic infections take hold.

PREDICT

A person with AIDS dies of an ordinary lung infection. What is the best explanation?

(A) HIV is a bacterium that spreads to the lungs

(B) Helper T cells are needed to activate B cells, so with few of them the immune response to ordinary infection fails

(C) HIV destroys the phagocytes that clear the lungs

(D) AIDS is caused by mucus that cannot be cleared

Decide before you look. Answer on page 54.

C3.2.13 ANTIBIOTICS AS CHEMICALS THAT BLOCK PROCESSES OCCURRING IN BACTERIA BUT NOT IN EUKARYOTIC CELLS

12 Poisons that *aim at bacteria*

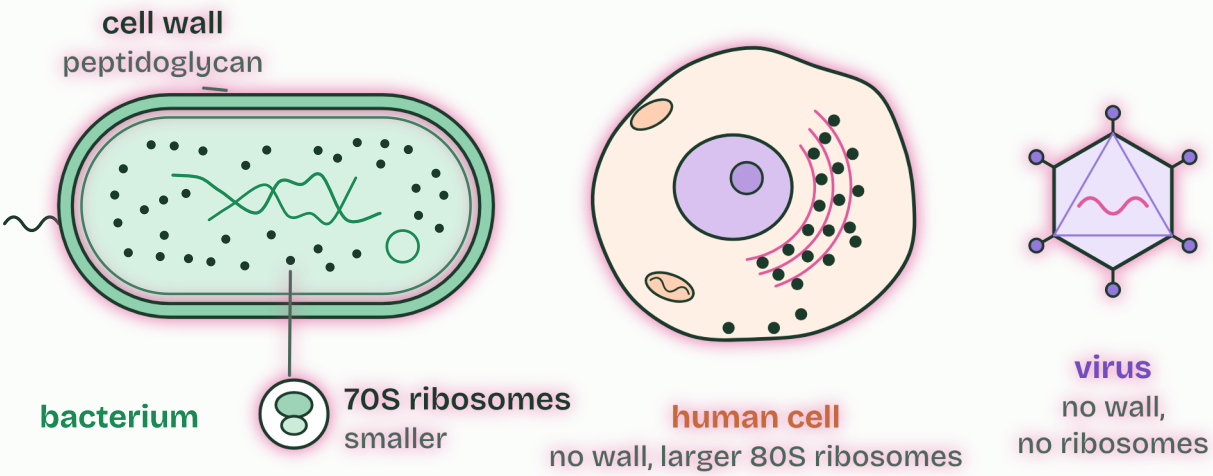
Antibiotics are chemicals that block a process in bacteria that does not occur, or differs, in eukaryotic cells. That is why they harm the bacteria and not the person taking them.

Two targets are the ones to know. Penicillin blocks the building of the bacterial cell wall, which human cells do not have. Tetracyclines block the bacterial ribosome, which differs from ours. Neither does anything useful against a virus, which has no cell wall of its own and uses its host's ribosomes, so antibiotics are no use against colds or flu.

KEY TERMS

antibiotics
Chemicals that kill bacteria or stop them growing by blocking a process that bacteria carry out but eukaryotic cells do not.

What can an antibiotic target?



Bacterial cell wall Bacteria have a cell wall made of peptidoglycan, which human cells do not have. Penicillin stops the wall being built, so the growing cell swells and bursts. Our cells have no wall to damage.

Human cell A eukaryotic cell with no cell wall and with larger 80S ribosomes. The targets antibiotics attack are missing or different here, so an antibiotic harms the bacteria and spares the person.

Bacterial ribosomes Bacteria make proteins on 70S ribosomes, which differ in shape from the 80S ribosomes in the cytoplasm of our cells. Antibiotics such as tetracycline fit the bacterial ribosome and stop protein synthesis, leaving our ribosomes alone.

Virus A virus has no cell wall and no ribosomes. It makes new viruses by borrowing the host cell's own machinery, so an antibiotic has nothing of the virus to attack. It would have to harm the host cell's own processes to affect the virus.

PREDICT

A doctor refuses to give antibiotics for a patient's flu. The best reason is:

(A) Flu is caused by a virus, and viruses have no processes of their own for antibiotics to block

(B) The patient's phagocytes would destroy the antibiotic

(C) Antibiotics only work on infections of the skin

(D) Flu will have been cured already by the time the drug takes effect

Decide before you look. Answer on page 54.

13 Antibiotics *select* for the resistant

C3.2.14 EVOLUTION OF RESISTANCE TO SEVERAL ANTIBIOTICS IN STRAINS OF PATHOGENIC BACTERIA

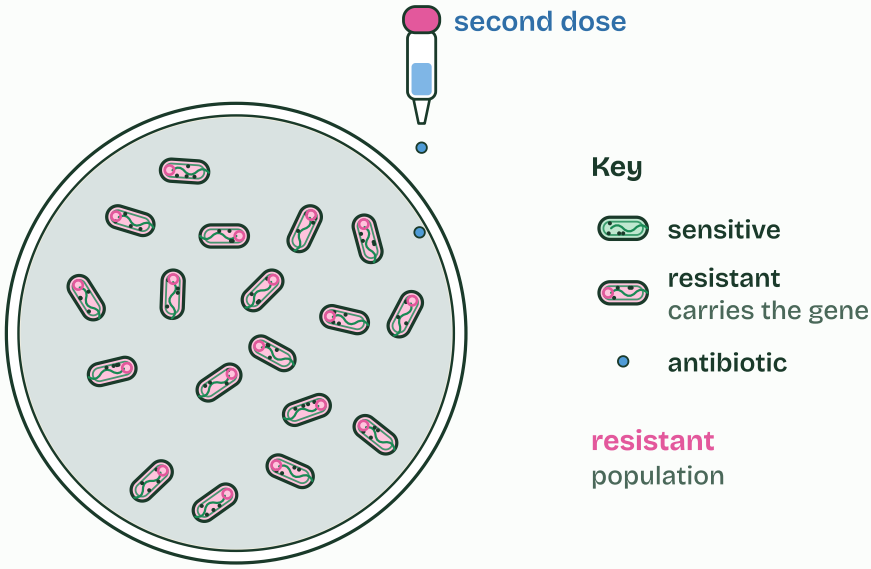
In a huge population of bacteria, a mutation by chance gives one cell a gene that lets it survive an antibiotic. Without the drug the gene gives it no advantage. With the drug, the sensitive cells die and the resistant one survives and divides, and its descendants all carry the gene. The antibiotic acts as a **selection pressure**. This is natural selection, over a few days rather than thousands of years.

Bacteria also pass genes to each other, on small rings of DNA called plasmids, even between species. A strain that picks up resistance genes for several drugs becomes **multiresistant**. MRSA, resistant to many antibiotics, is a well-known example. Using antibiotics only when needed, and finishing the course, slows this.

KEY TERMS

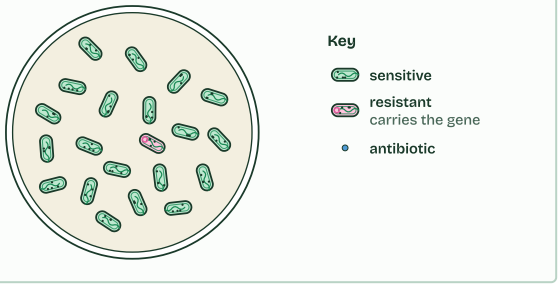
selection pressure
A factor in the environment that makes some individuals more likely to survive and reproduce than others.

multiresistant
Resistant to several different antibiotics.



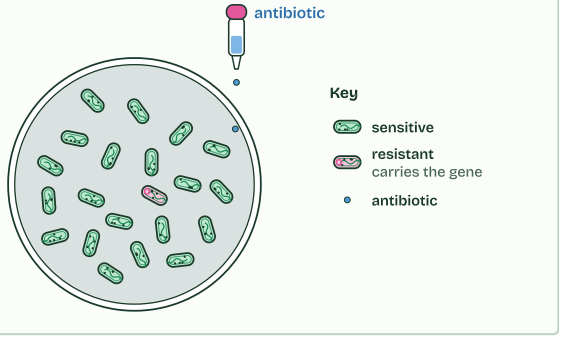
STEP BY STEP
5 STEPS

1



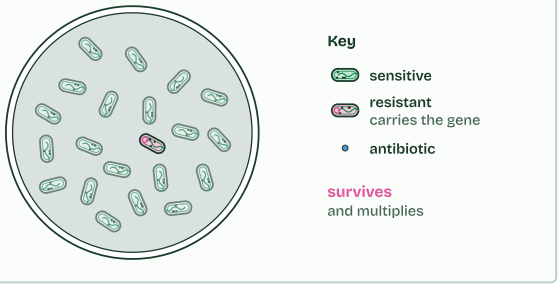
A population of bacteria. By chance one carries a **resistance gene**, here on a plasmid.

2



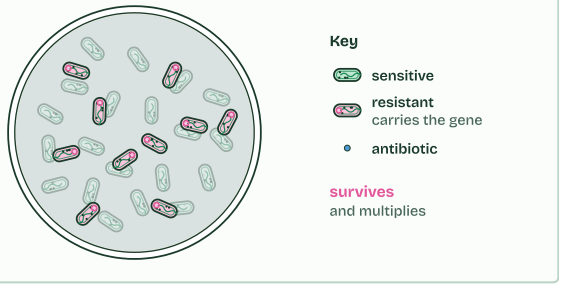
The **antibiotic** is added.

3



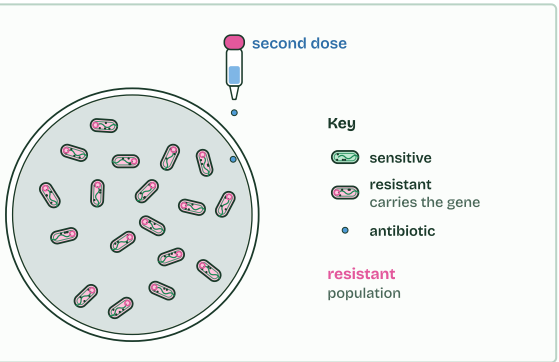
The **sensitive** bacteria die. The resistant one survives.

4



It **divides**, and so do its descendants: all carry the gene.

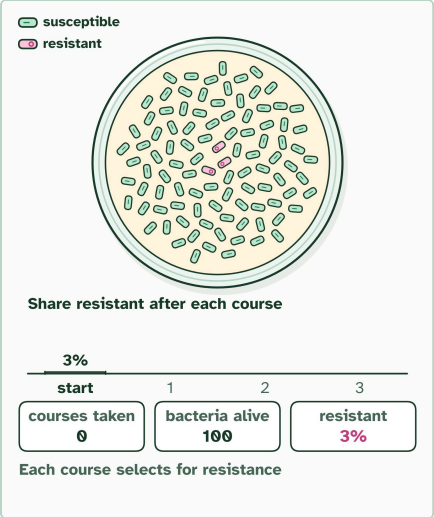
5



A second dose does nothing. The population is now **resistant**.

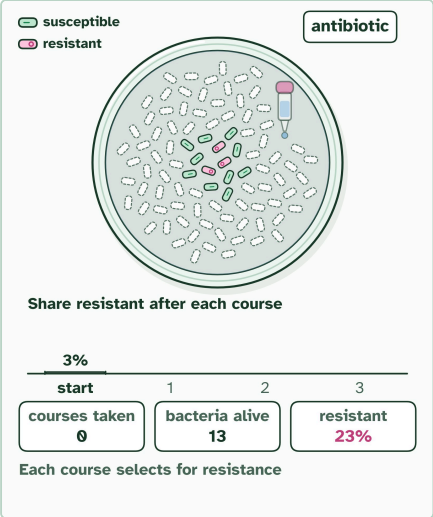
ANIMATION · RESISTANCE ON A PLATE
5 MOMENTS

1 3% OF THE BACTERIA RESISTANT



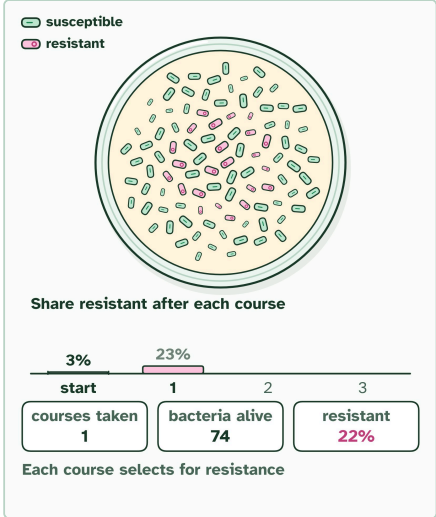
A dish of bacteria. Nearly all are **susceptible** (green). A few have a gene that makes them **resistant** to the antibiotic (pink).

2 23% OF THE BACTERIA RESISTANT



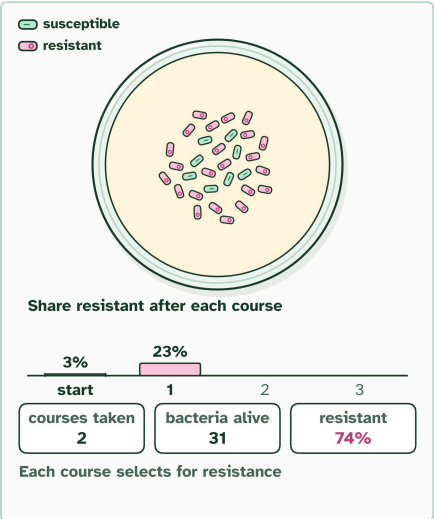
The first **antibiotic course**. It kills most of the susceptible bacteria. The resistant ones are not harmed.

3 22% OF THE BACTERIA RESISTANT



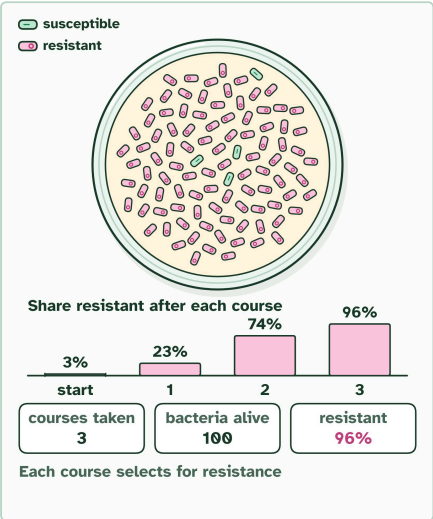
The survivors **divide** and fill the space. They pass on the resistance gene, so a bigger share of the dish is now resistant.

4 74% OF THE BACTERIA RESISTANT



A second course kills the susceptible bacteria that are left. Again the resistant ones survive and divide.

5 96% OF THE BACTERIA RESISTANT



After more courses, most of the dish is resistant. The antibiotic was the **selective pressure**: **natural selection** at work.

Watch it move, and change it as it runs: biologybybradford.com/guides/ib-biology-c3-2-defence-against-disease#resistance

PREDICT

A patient stops taking antibiotics after three days because they feel better. The best description of the risk is:

(A) The bacteria that remain were exposed to a little drug and are now more likely to be resistant

(B) The antibiotic is wasted but no harm is done

(C) The remaining bacteria will have been made resistant by the drug

(D) The patient will be immune to the drug

Decide before you look. Answer on page 54.

NEW LEADS

Because resistance keeps arriving, researchers search chemical libraries for new antibiotics. In 2019 a machine-learning screen of thousands of molecules found halicin, a compound that kills a range of resistant bacteria in the lab.

14 Diseases that *cross species*

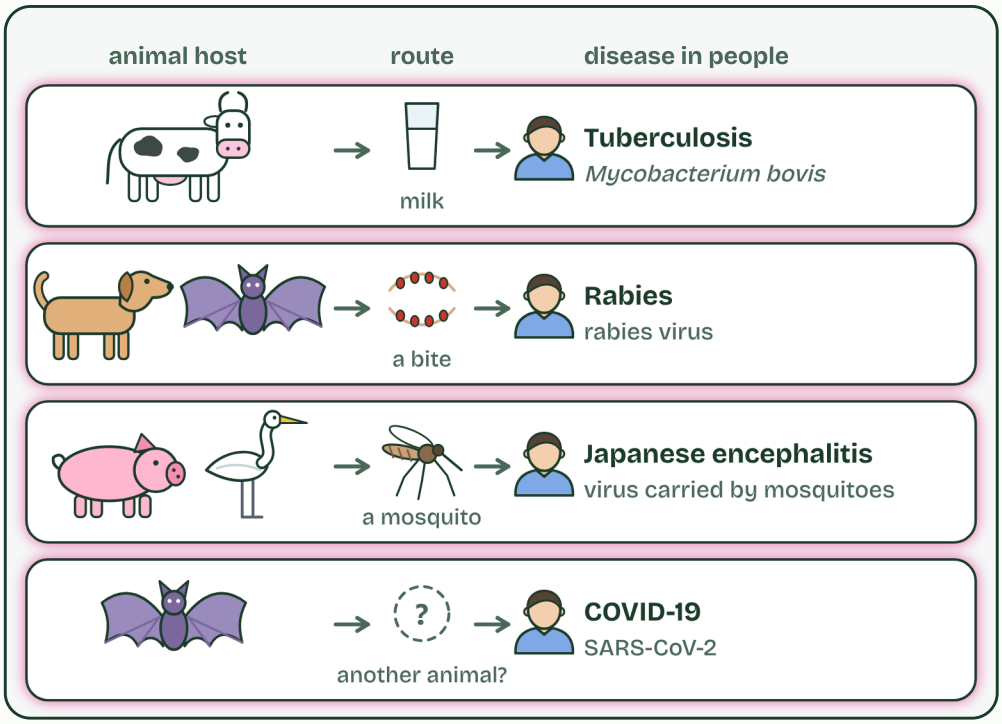
C3.2.15 ZOONOSES AS INFECTIOUS DISEASES THAT CAN TRANSFER FROM OTHER SPECIES TO HUMANS

A **zoonosis** is an infectious disease that can pass from other animals to people. They are common: most new human diseases of recent decades began in animals. The route varies. A pathogen can be eaten in milk, passed in a bite, carried by a mosquito that bites an animal and then a person, or breathed in.

Spread is easier now because people live more closely with livestock and wild animals, clear forest, trade animals and travel the world in hours. COVID-19 is the recent example: a virus that moved to humans and, passing between us, caused a pandemic.

KEY TERMS

zoonosis
An infectious disease that can pass from another species of animal to humans.



Tuberculosis *Mycobacterium bovis* infects cattle and can pass to people in unpasteurized milk or by close contact. (Most human tuberculosis is caused by a related species, *M. tuberculosis*, passed between people.)

Japanese encephalitis The virus lives in pigs and wading birds and is carried by *Culex* mosquitoes. A person is a dead end: it is usually not passed on from them.

Rabies The rabies virus is in the saliva of infected mammals and enters through a bite. Most human cases follow a dog bite. Without treatment before symptoms, it is almost always fatal.

COVID-19 SARS-CoV-2 is a coronavirus closely related to viruses found in bats. It reached people, probably via an animal host, and then spread between people through the air, with profound consequences for human health and economies.

PREDICT

Which of these is a zoonosis?

(A) A cold, which passes between people by droplets

(B) Rabies, which passes to people from the saliva of an infected animal

(C) Cholera, which passes between people through dirty water

(D) HIV in a person who has been infected through a blood transfusion

Decide before you look. Answer on page 54.

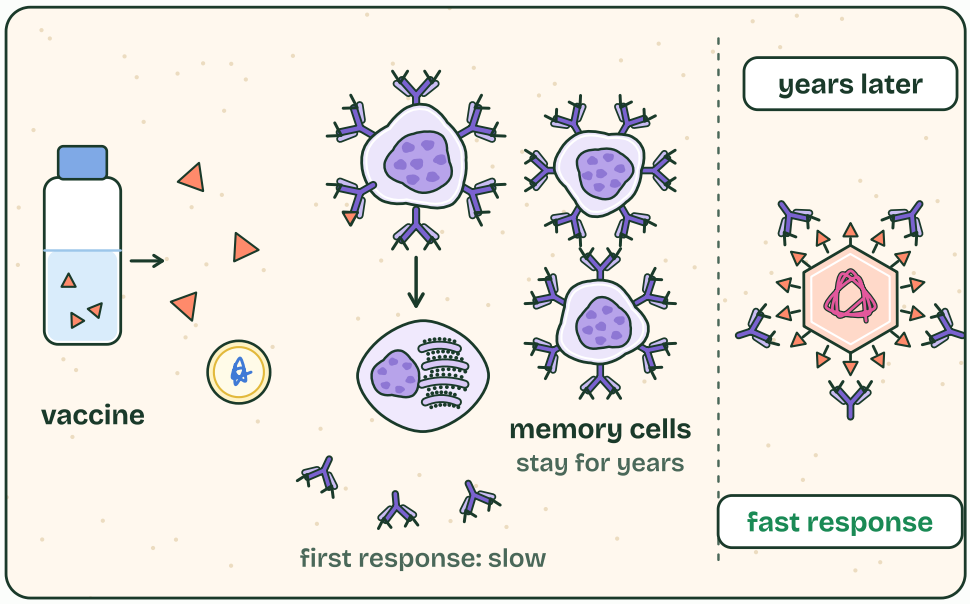
15 A *rehearsal* without the disease

C3.2.16 VACCINES AND IMMUNIZATION

A **vaccine** gives your immune system a look at a pathogen's antigen without the disease. It can contain the antigen itself, for example a harmless part of the pathogen or a weakened or killed whole one, or nucleic acid (DNA or RNA) with a sequence that codes for the antigen, so your own cells make it. The immune system responds as it would to the real pathogen: B cells are selected and activated, antibodies are made and memory cells are left behind. Immunization is giving the vaccine.

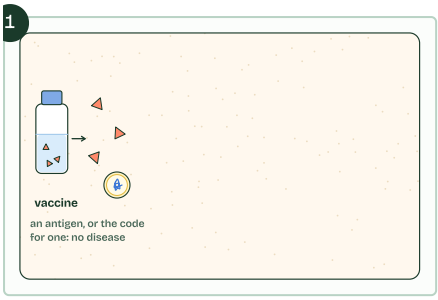
KEY TERMS

vaccine
A preparation that gives the immune system an antigen, or instructions for making one, so it builds memory cells without the person catching the disease.

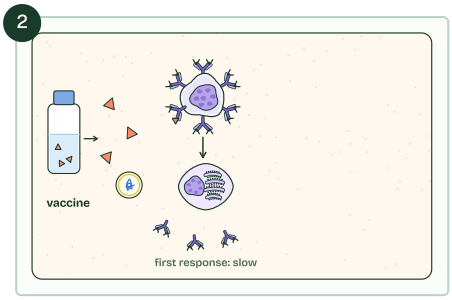


STEP BY STEP

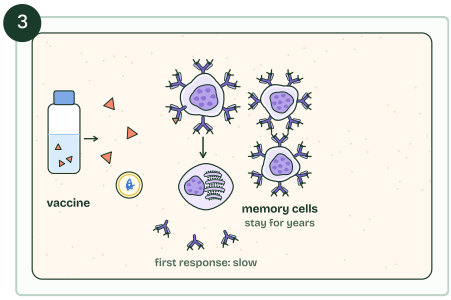
4 STEPS



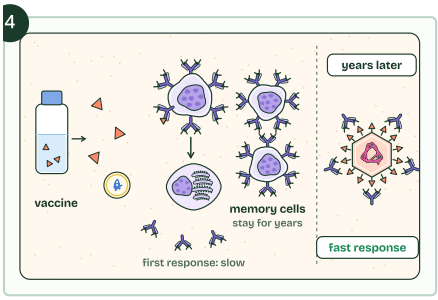
The vaccine carries an **antigen**, or the genetic code for one, but cannot cause the disease.



The immune system **responds** to it: a matching B cell is selected and plasma cells make antibodies.



Memory cells are left behind, and stay for years.



If the real pathogen arrives, it meets memory cells and a fast **secondary response**.

PREDICT

Why can a vaccine not give you the disease it protects against?

(A) The antigen alone, or the code for it, cannot make a whole working pathogen

(B) Vaccines always contain antibiotics that kill any pathogen

(C) The immune system destroys every vaccine before it can do anything

(D) Vaccines work only on pathogens that cannot cause disease

Decide before you look. Answer on page 54.

TWO KINDS

In the mRNA COVID-19 vaccines the injection carries mRNA that codes for the virus's spike protein. Your cells make the spike protein, the immune system responds to it, and the mRNA is soon broken down.

16 Protecting people *who cannot be protected*

C3.2.17 HERD IMMUNITY AND THE PREVENTION OF EPIDEMICS

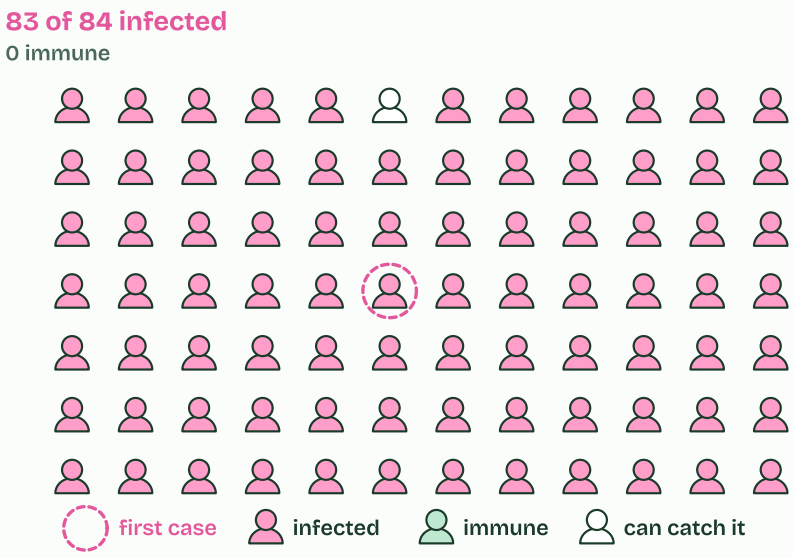
A pathogen needs to find a new host to keep spreading. If enough of a population is immune, most of the people it reaches cannot pass it on, so outbreaks die out. This is **herd immunity**. It protects people who cannot be vaccinated, such as newborn babies or people with a weakened immune system, because the disease seldom reaches them. Everyone in a population depends on everyone else.

How many need to be immune depends on how easily the pathogen spreads, measured by R_0, the number of people one case infects in a population with no immunity. The proportion that must be immune is about $1 - 1/R_0$. Measles spreads very easily (R_0 of about 12 to 18), so about 95% need to be immune. If coverage drops below that, measles comes back.

Try the dry lab to find the point where an outbreak stops taking off.

KEY TERMS

herd immunity
Protection of a population from an infectious disease when enough people are immune that transmission is greatly impeded, which also protects those who are not immune.



SWITCH IT

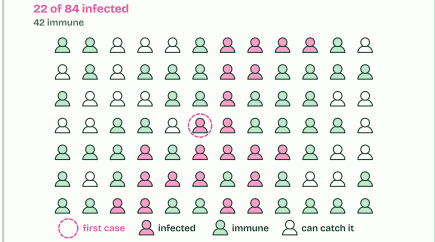
3 SETTINGS

1



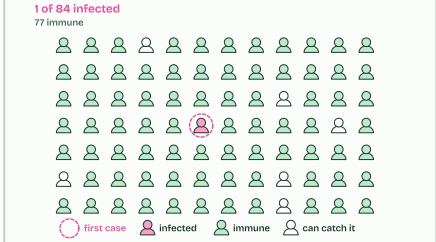
Nobody immune

2



Half immune

3



Nearly all immune

PREDICT

Vaccination coverage for a disease falls from 95% to 85%. The most likely result is:

(A) Nothing, because 85% is still a majority

(B) Outbreaks become possible, because the disease can now find enough susceptible people to keep spreading

(C) The disease disappears faster, because the vaccinated people spread immunity

(D) Only the unvaccinated can be affected, so the vaccinated need not care

Decide before you look. Answer on page 54.

IN THE EXAM

Herd immunity in points:

1. A high proportion of the population is immune, by vaccination or past infection.
2. So a case is likely to meet only immune people and cannot pass the pathogen on.
3. Transmission is greatly impeded and the outbreak dies out.
4. People who are not immune are protected, because the disease rarely reaches them.

17 Reading the numbers *carefully*

C3.2.18 EVALUATION OF DATA RELATED TO THE COVID-19 PANDEMIC

COVID-19 filled the news with graphs, and the exam asks you to work with that kind of data. Two calculations come up. **Percentage change** compares a value with where it started: (new − old) ÷ old × 100. **Percentage difference** compares two values with neither being the starting point: the difference divided by their mean, × 100.

The numbers in the chart below are made up, to practise on. Real data come from published studies and public health reports; check what the figures count, how many people were included and over what time.

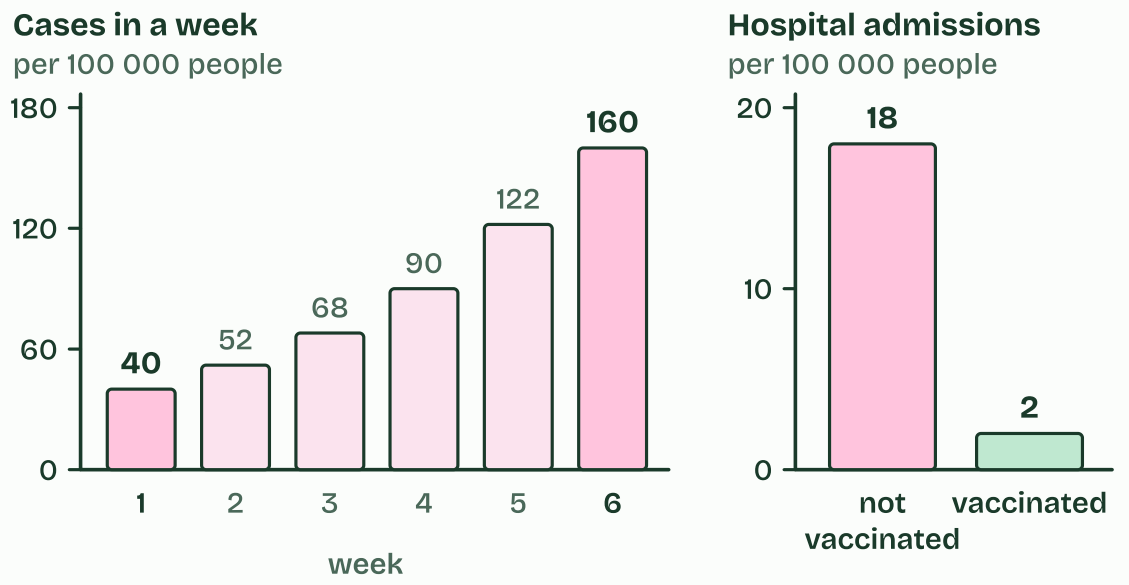
KEY POINTS

- **Correlation is not causation.** A fall in cases after a measure may have other causes, such as a changing variant or behaviour.
- **Compare like with like.** The vaccinated and unvaccinated may differ in age and health.
- **Size, source and time.** A large sample from a reliable source over a long period is stronger evidence.
- **Early reports can change.** Findings reported in the media while still being checked by other scientists may be revised.

KEY TERMS

percentage change
The change in a value as a percentage of its starting value: (new − old) ÷ old × 100.

percentage difference
The difference between two values as a percentage of their mean, used when neither value is the starting point.



PREDICT

Weekly cases rose from 40 to 160 per 100 000. What is the percentage change?

(A) +75%

(B) +120%

(C) +300%

(D) +400%

Decide before you look. Answer on page 54.

PREDICT

Admissions were 18 per 100 000 in unvaccinated people and 2 in vaccinated people. What is the percentage difference?

(A) 89%

(B) 160%

(C) 800%

(D) 16%

Decide before you look. Answer on page 54.

Key terms

Every term this guide defines, A to Z, with the chapter it is in. Cover the right-hand side and test yourself.

activated 07
Triggered to divide and make antibodies. A B cell needs two things to be activated: its specific antigen and contact with a helper T cell.

adaptive immune system 04
The part of the immune system that responds to one particular pathogen and remembers it, so the response improves with each meeting.

AIDS 11
Acquired immunodeficiency syndrome: the stage of HIV infection when too few helper T cells are left to fight off opportunistic infections.

amoeboid movement 05
Movement by pushing out a part of the cell, a pseudopodium, and flowing into it.

antibiotics 12
Chemicals that kill bacteria or stop them growing by blocking a process that bacteria carry out but eukaryotic cells do not.

antibody 06
A Y-shaped protein made by plasma cells that binds to one specific antigen.

antigen 06
A molecule that is recognized by the immune system and triggers an immune response, usually a protein or glycoprotein on the surface of a pathogen.

B cell 06
A lymphocyte that makes antibodies once activated.

cascade 03
A chain of reactions in which each step triggers the next.

chemical barrier 02
A barrier that harms pathogens with chemicals, such as acidic sweat and enzymes.

clone 08
A group of genetically identical cells all descended from one parent cell.

clotting factors 03
Chemicals released by platelets and damaged tissue that start the clotting cascade.

endocytosis 05
Taking material into a cell by folding the plasma membrane round it to make a vesicle.

fibrin 03
Insoluble protein fibres that form a mesh across a wound, trapping blood cells to make a clot.

helper T cell 07
A lymphocyte that has itself been activated by the same antigen and which activates B cells by contact and chemical signals.

herd immunity 16
Protection of a population from an infectious disease when enough people are immune that transmission is greatly impeded, which also protects those who are not immune.

HIV 11
Human immunodeficiency virus, an RNA virus that infects and kills helper T cells.

immunity 10
The ability to eliminate an infectious disease from the body, because memory cells for it are already present.

infectious disease 01
A disease caused by a pathogen that can be passed from one host to another.

innate immune system 04
The defences you are born with. They respond to broad categories of pathogen, in the same way every time.

lymphocytes 06
White blood cells of the adaptive immune system: B cells and T cells.

lysosomes 05
Small organelles that hold digestive enzymes.

memory cells 10
Long-lived lymphocytes that remain after an infection and can respond very quickly if the same antigen returns.

mitosis 08
Cell division that produces two genetically identical daughter cells.

mucous membrane 02
The moist lining of body passages. Its cells make mucus, which traps microbes, and in places it carries cilia.

multiresistant 13
Resistant to several different antibiotics.

opportunistic infections 11
Infections by organisms that would not normally harm a person with a healthy immune system.

pathogen 01
An organism or virus that causes disease.

percentage change 17
The change in a value as a percentage of its starting value: (new − old) ÷ old × 100.

percentage difference 17
The difference between two values as a percentage of their mean, used when neither value is the starting point.

phagocytes 05
White blood cells that engulf pathogens and digest them. Neutrophils and macrophages are phagocytes.

physical barrier 02
A barrier that stops a pathogen getting through by being in the way, such as the tough outer layer of skin.

plasma cells 08
Activated B cells that make and release large amounts of one type of antibody.

platelets 03
Small cell fragments in blood that stick to damaged vessels and start clotting.

selection pressure 13
A factor in the environment that makes some individuals more likely to survive and reproduce than others.

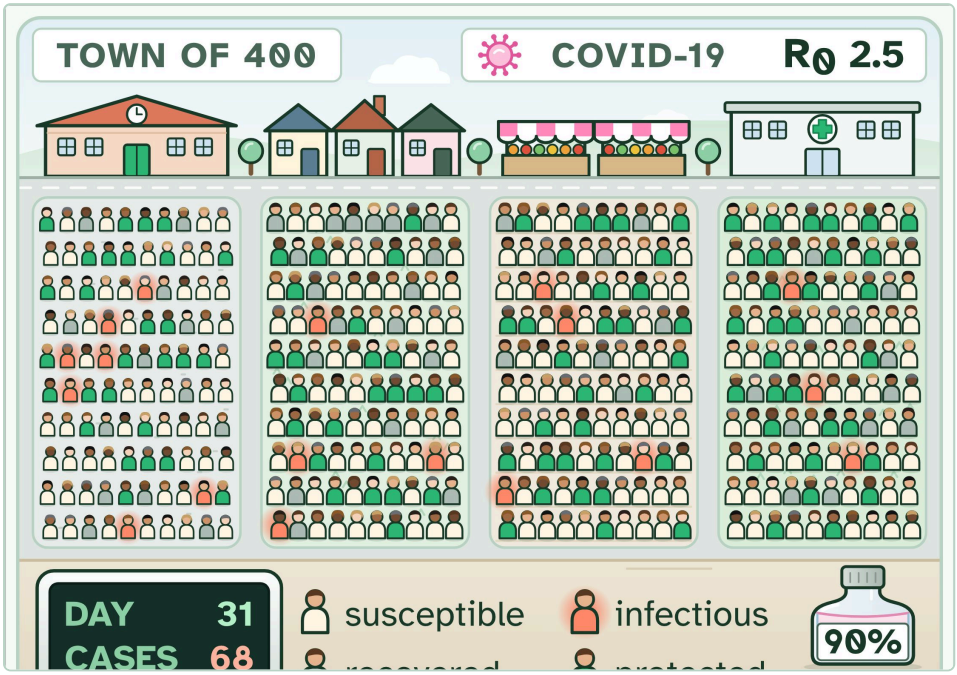
thrombin 03
The active enzyme made from prothrombin during clotting. It turns fibrinogen into fibrin.

vaccine 15
A preparation that gives the immune system an antigen, or instructions for making one, so it builds memory cells without the person catching the disease.

zoonosis 14
An infectious disease that can pass from another species of animal to humans.

Dry lab: *Herd immunity*

A town of 400 people, one disease and one vaccine. Change how many people are vaccinated and see when an outbreak dies out, then find the coverage that protects everyone.



Run it online: change the variables, run the trials, and the table and graph fill in as you go. `biologybybradford.com/guides/ib-biology-c3-2-defence-against-disease#lab`

Your question

What percentage of a population has to be immune before an outbreak fizzles out? The bench starts with COVID-19 as it spread in 2020 and a vaccine that works for 90% of those who have it; measles, which spreads far more easily, is one click away.

What you can change

The disease (how many people one case infects in a fully susceptible town, R0), the percentage of people vaccinated, and how well the vaccine works. Keep the rest the same: 400 people, three people infected on day 0, and the same infectious period.

What to record

For each coverage, the percentage of the town that ends up infected, from at least five runs because every run is random, with the mean. Graph the mean against coverage and mark where the outbreak stops taking off. Download the CSV to plot your own.

Find out

- **How easily does each disease spread?** With nobody vaccinated, run each disease five times and record the mean share of the town infected.
- **Where does measles stop?** Run measles at 0, 50, 80, 90, 95 and 100% vaccinated, five runs at each. Where does the mean fall to a few per cent?
- **Does the vaccine matter?** Repeat the last test with a vaccine that works for 70% and then 97% of people who have it.
- **Is the line right?** The pink dashed line is where the outbreak should stop. How close are your results to it, and why does a run near the line sometimes go either way?

Record

- The disease, the share vaccinated, how well the vaccine works, and the share of the town infected at the end of each run.
- At least five runs at each setting, because every run is random. Work out a mean for each.
- A graph of the mean share infected against the share vaccinated for each disease. The graph under the table does this as you go.
- Download the CSV to plot your own graph.

Your question What percentage of a population has to be immune before an outbreak fizzles out? The bench starts with COVID-19 as it spread in 2020 and a vaccine that works for 90% of those who have it; measles, which spreads far more easily, is one click away.**What you can change**The disease (how many people one case infects in a fully susceptible town, R0), the percentage of people vaccinated, and how well the vaccine works. Keep the rest the same: 400 people, three people infected on day 0, and the same infectious period.**What to record**For each coverage, the percentage of the town that ends up infected, from at least five runs because every run is random, with the mean. Graph the mean against coverage and mark where the outbreak stops taking off. Download the CSV to plot your own.

Back to biology

A newborn is too young for the measles vaccine. Use your results to explain how the town protects the baby, and why measles needs more people vaccinated than influenza.

QUESTIONS ON THE LAB

QUESTION 1

Influenza needs about a third of the town to be immune, and measles about 93%. Why the difference?

(A) Measles is more deadly, so more people have to be protected

(B) Each measles case infects far more people, so many more must be immune to break the chains

(C) The measles vaccine is less effective, so more people must have it

(D) Influenza is spread by a different route, so it cannot be stopped

Answer on page 55.

QUESTION 2

In a run, 90% of the town is vaccinated against measles, with a vaccine that works for 90% of people, and the outbreak still reaches about one person in six. Why?

(A) The vaccine stops working as soon as an outbreak has started

(B) The vaccine fails in one in ten of those given it, so only 81% of the town is immune, below the 93% needed

(C) Vaccinated people still pass the disease to each other as easily as anyone else

(D) Measles outbreaks are not affected by how many people are vaccinated

Answer on page 55.

Check yourself

The check for this guide is online: a fresh paper of past-paper style questions from the exam bank on C3.2 each time, marked as you go, and saved to your Exam practice progress when you are signed in. Scan the code, or type the link.

biologybybradford.com/exam-maker/sit?topics=C3.2&level=SL&paper=quiz&marks=15&src=ep

Answers

PREDICTIONS IN THE CHAPTERS

01 One group of microbes is often listed beside bacteria and fungi, yet no member is known to cause disease in humans. Which?

C **Archaea, which are prokaryotes like bacteria**

Archaea. They are prokaryotes that look much like bacteria, yet none is known to cause a disease in humans. The usual list of human pathogens is viruses, bacteria, fungi and protists. (Protists do cause disease, malaria among them, and viruses cause plenty.)

02 A person with a severe burn is at high risk of infection. The best reason is:

B **A burn removes the physical barrier, so microbes can get straight in**

A burn takes away the skin, the physical and chemical barrier. Microbes that could not have crossed it can now reach the tissue underneath, which is why burn wards guard against infection so carefully.

03 Fibrinogen is dissolved in your blood plasma all the time. Why does your blood not clot inside healthy vessels?

B **Thrombin is made only after platelets and damaged tissue release clotting factors**

The cascade does not start without a trigger. Fibrinogen is always there, but it becomes fibrin only when thrombin is active, and thrombin is made only when platelets and damaged tissue release clotting factors at an injury.

04 A child catches chickenpox and recovers. Meeting the virus again years later, she does not get ill. Which system explains that?

C **Adaptive, because it remembers the particular pathogen**

The adaptive system. Its lymphocytes keep memory cells for the specific pathogen, so the second response is quick and strong enough that she does not fall ill. The innate system responds to the virus no better the second time.

05 Why can a phagocyte digest a bacterium without digesting its own cytoplasm?

B **The enzymes are released into the vesicle that holds the bacterium, not into the cytoplasm**

The lysosome's enzymes empty into the vesicle holding the bacterium, so the digestion happens inside a closed bag of membrane. The rest of the cell is never exposed to them.

06 A B cell with receptors that fit a measles antigen meets a mumps virus. What happens?

B **Nothing: the receptor does not fit the mumps antigens, so the cell is not selected**

Nothing happens. Each B cell can bind only the antigens that fit its receptor, which is why antibody production is specific. The mumps virus will be matched by a different B cell.

07 A B cell binds its antigen but no helper T cell has been activated by that antigen. What does the B cell do?

B **Remains inactive, because it has only one of the two signals**

It remains inactive. B cells make antibodies and become memory cells only when they have been activated, and activation needs both the antigen and contact with a helper T cell activated by the same type of antigen.

08 Why are the plasma cells packed with rough endoplasmic reticulum?

B **They are making large amounts of protein, which antibodies are, to secrete**

Antibodies are proteins made on ribosomes on rough ER, then packaged and secreted. A cell built to secrete antibody in bulk has a great deal of rough ER and Golgi.

10 On the second exposure to the same pathogen, compared with the first, which description is right?

C **A shorter lag and a taller peak**

A shorter lag and a taller peak. Memory cells are already present in numbers, so they divide and make plasma cells straight away and the antibody level climbs higher and stays up longer. The person usually has no symptoms at all.

11 A person with AIDS dies of an ordinary lung infection. What is the best explanation?

B **Helper T cells are needed to activate B cells, so with few of them the immune response to ordinary infection fails**

HIV kills helper T cells. Without them, B cells are not activated, antibody production falls and the body cannot fight infections that would normally be controlled.

12 A doctor refuses to give antibiotics for a patient's flu. The best reason is:

A **Flu is caused by a virus, and viruses have no processes of their own for antibiotics to block**

Flu is a virus. It has no cell wall or ribosomes of its own and makes new virus using the host cell's machinery, so antibiotics have no target. Using them would only encourage resistance.

13 A patient stops taking antibiotics after three days because they feel better. The best description of the risk is:

A **The bacteria that remain were exposed to a little drug and are now more likely to be resistant**

A short course kills the most sensitive bacteria first, leaving the more tolerant ones to recover and multiply. The drug does not make bacteria resistant; it selects for resistance already there, so stopping early can leave behind a more resistant population.

14 Which of these is a zoonosis?

B **Rabies, which passes to people from the saliva of an infected animal**

Rabies. The pathogen lives in animals and reaches a person by a bite. A zoonosis starts in another species, even if it can later spread between people, as COVID-19 did.

15 Why can a vaccine not give you the disease it protects against?

A **The antigen alone, or the code for it, cannot make a whole working pathogen**

A vaccine has only the antigen, or the nucleic acid that codes for it, or a pathogen that is killed or weakened, so it cannot cause the disease. It is enough for the immune system to practise and keep memory cells.

16 Vaccination coverage for a disease falls from 95% to 85%. The most likely result is:

B **Outbreaks become possible, because the disease can now find enough susceptible people to keep spreading**

For a disease as infectious as measles, 95% is about what is needed to block spread. At 85% the pathogen can find enough susceptible people to keep passing on, so outbreaks return, and some of the people it reaches are those who cannot be vaccinated.

17 Weekly cases rose from 40 to 160 per 100 000. What is the percentage change?

C **+300%**

(160 − 40) ÷ 40 × 100 = +300%. The value has quadrupled, which is an increase of 300%, not 400%.

17 Admissions were 18 per 100 000 in unvaccinated people and 2 in vaccinated people. What is the percentage difference?

B **160%**

The difference is 16. Their mean is (18 + 2) ÷ 2 = 10. 16 ÷ 10 × 100 = 160%.

QUESTIONS ON THE DRY LAB

Q1 Influenza needs about a third of the town to be immune, and measles about 93%. Why the difference?

B **Each measles case infects far more people, so many more must be immune to break the chains**

The share that must be immune is about $1 - 1/R_0$, where R_0 is the number of people one case infects in a population with no immunity. Measles has an R_0 of around 15 and influenza around 1.5, so measles needs about 93% and influenza about a third. How dangerous a disease is does not enter into it.

Q2 In a run, 90% of the town is vaccinated against measles, with a vaccine that works for 90% of people, and the outbreak still reaches about one person in six. Why?

B **The vaccine fails in one in ten of those given it, so only 81% of the town is immune, below the 93% needed**

Coverage and immunity are not the same. If the vaccine works for 90% of those who have it, 90% × 90% = 81% of the town is immune, below the 93% measles needs, so the disease can still spread, though slowly because most of the people it meets are immune.

How this was made. The course structure, the activities and the teaching choices come from my own classroom, my presentations and my years of teaching IB and AP Biology. I use Claude, an AI, to help write and build the interactive guides, lesson plans, slides and practice questions, and to draw the diagrams and code the interactives. I direct the work and review it against the IB and AP specifications before it goes live.

Written and drawn for Biology by Bradford, biologybybradford.com. Free to print and share with a class.