Defence against disease

C3.2 · SL and HL

A cut and a new virus require different kinds of protection

Connect entry barriers, removal and specific memory

Full shared C3.2: barriers/clotting, innate and adaptive responses, antibody production/memory, HIV, antibiotics/resistance, zoonoses, vaccination and infection data.

Prerequisites: cells, membrane receptors, proteins and mitosis. There are no additional Higher-Level-only objectives in this topic.

Numerical examples are invented except the explicitly identified early WHO reports. Use supplied data, safe simulations or approved school cultures.

Separate the agent from its effects on the host

Term or group	Meaning and limit
Pathogen	An agent that causes disease; not every microorganism is pathogenic
Bacteria/Archaea	Both prokaryotic groups; some bacteria cause disease, but no established human pathogen is an archaeon
Fungi/protists	Eukaryotic groups containing important pathogens
Virus/infection	Acellular replication depends on host cells; infection can occur without visible disease

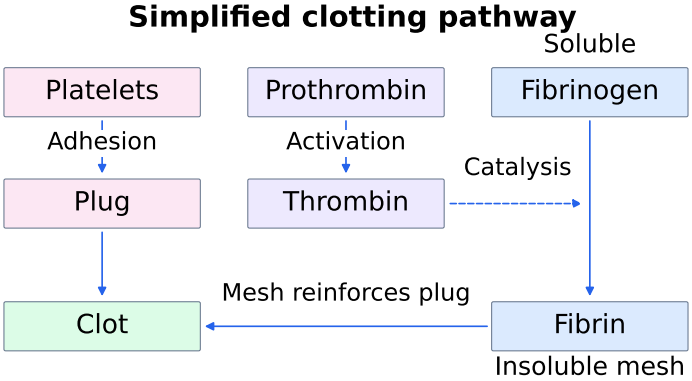
Identify the barrier and explain how it works

Barrier	Mechanism
Intact skin	Closely packed cells and a tough outer layer limit entry
Airway mucus/cilia	Mucus traps material; cilia move it toward removal/swallowing
Tears/saliva	Washing plus antimicrobial components; lysozyme attacks bacterial wall material
Stomach acid	Inhibits many swallowed organisms; not a guarantee against every agent

Use observations to test routes of infection

1. **Childbed fever.** Semmelweis compared deaths in Vienna and introduced hand disinfection: material carried on hands could connect patients with an infection source.
2. **Cholera.** Snow compared mortality among London households supplied by different water companies: water supply could explain exposure differences.
3. **Evidence.** Compare groups and alternative explanations. Observations supported practical controls before every microbial mechanism was understood; a famous name alone is not proof.

Separate the enzyme, its substrate and its product



Thrombin catalyses fibrin formation; platelets form a plug.

Follow the simplified clotting pathway

1. **Platelets.** Adhere at damaged tissue, activate and recruit a plug; provide signals/surfaces supporting clotting reactions.
2. **Cascade.** Clotting factors activate downstream factors; prothrombin becomes active thrombin.
3. **Product.** Thrombin converts soluble fibrinogen to an insoluble fibrin mesh that reinforces the plug and traps blood cells.

The clot closes an entry route as well as limiting leakage

A localized defence needs control

Platelets are cell fragments. They do not become red blood cells or antibodies.

Fibrin is a protein network, not the enzyme digesting a pathogen. Red cells can be trapped within it.

A useful clot at a cut differs in consequence from an obstructing clot in an important vessel; uncontrolled clotting would impair circulation.

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Trace recognition, engulfment and enclosed digestion

1. **Arrival.** A phagocyte can leave small vessels and move through tissues by changing shape.
2. **Engulfment.** Broad target recognition leads membrane extensions to surround and enclose material in a vesicle.
3. **Digestion.** Lysosomal enzymes break down the enclosed material; appropriate **antigen** presentation can help activate adaptive responses.

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Compare recognition without separating cooperating systems

Response	**Mechanism and relationship**
Innate	Available without earlier exposure to the particular **antigen**; broad recognition and rapid response
Adaptive	Selected lymphocyte receptor specificity; expansion and possible long-lived memory
Cooperation	Some phagocytes present antigen-derived material to help activate T cells
Wrong substitution	A plasma cell secretes antibodies; it is not a red cell engulfing a **pathogen**

An antigen is a recognized structure, not always a foreign organism

Self and external molecules can both be antigens

Proteins and glycoprotein components can carry antigenic sites. A whole pathogen can have several different sites.

A particular antibody or immune receptor recognizes a compatible structure; recognition does not automatically mean danger.

Red-cell surface antigens are normal body components but can provoke an incompatible recipient response.

Many receptor specificities exist before the encounter

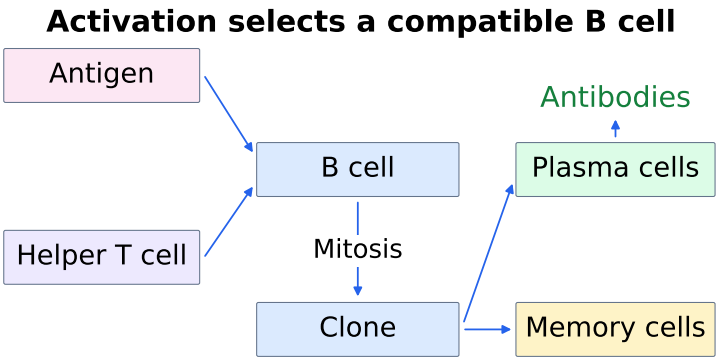
The antigen selects a compatible clone

B and T lymphocytes have different roles. A clone consists of descendants retaining the relevant receptor specificity.

An antigen does not instruct an arbitrary lymphocyte to replace its receptor with any required matching shape.

Cells circulate in blood/lymph; lymph nodes support encounters between antigen and appropriate lymphocytes.

Follow activation, expansion and differentiation



A selected B-cell clone produces plasma and memory descendants.

Explain a typical helper-T-dependent protein-antigen response

1. **Recognition.** A compatible B-cell receptor binds **antigen**; uptake permits display of antigen-derived material.
2. **Cooperation.** An appropriate helper T cell recognizes presented material and provides activating signals; other presenting cells help activate helper T cells.
3. **Expansion.** The activated B cell divides by mitosis. Descendants become antibody-secreting plasma cells and memory B cells.

Specific binding enables removal without conscious choice

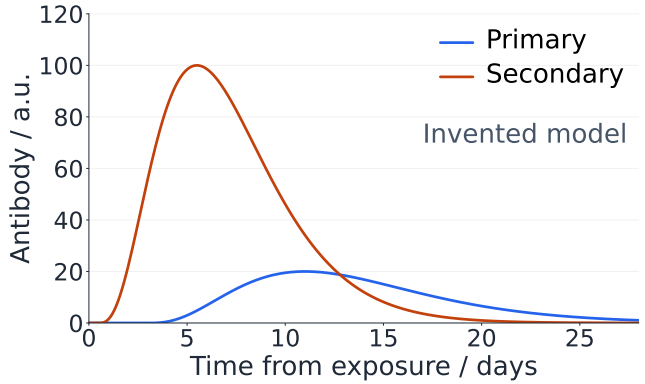
An antibody is a protein product, not a dividing cell

Plasma cells secrete antibodies with compatible antigen-binding regions; an individual antibody does not reproduce by mitosis.

Binding can neutralize toxins, interfere with attachment or assist other removal mechanisms.

Antigen change can reduce binding, but not every small change abolishes every form of protection. B-cell activation routes also vary.

Compare responses using time since each exposure



Memory can support an earlier and larger **antibody** response.

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Quantify the change while keeping delay and peak separate

1. **Invented peaks.** Primary 20 units; secondary 100. Ratio $100/20 =$ five; increase $80/20 \times 100 = 400\%$.
2. **Detection delay.** Six $\to$ two days: four/six $\times$ 100 $\approx$ 66.7% decrease under the same detection rule.
3. **Interpretation.** Selected memory populations support faster expansion. A first visible graph point need not be the instant the first **antibody** molecule was made.

A remembered target does not guarantee lifelong sterilizing immunity

Protection depends on target, time and infection site

Many viruses cause colds; a later cold need not be the same antigenic challenge.

Antigen change, waning responses and infection-site differences can limit protection despite memory.

An antibody assay needs its target, specificity and sampling time stated. Detectable antibody is not a universal guarantee against disease.

Connect the viral target with weakened immune coordination

1. **Target.** HIV infects CD4 helper T cells and some other susceptible cells.
2. **Consequence.** Progressive helper-cell damage can weaken coordination, including effective help for B-cell responses.
3. **Distinction.** AIDS is advanced severe immune damage with characteristic vulnerabilities, not a synonym for every HIV infection. Treatment can suppress replication and preserve function.

Identify a biological transmission route without stigma

Situation	Route or conclusion
Sexual exposure	Infectious semen/pre-seminal, vaginal or rectal fluids can reach susceptible tissue
Contaminated injection equipment	Blood can enter a recipient bloodstream
Perinatal transmission	Can occur during pregnancy, birth or breastfeeding
Ordinary classroom contact	Sitting nearby, touching and shared classroom air are not HIV transmission routes

Name the target before predicting drug action

Feature	Explanation
Bacterial targets	Cell-wall synthesis, bacterial ribosomes or particular metabolic pathways
Selectivity	Differences between bacteria and human cells support selective action
Viruses	Lack their own bacterial wall/ribosomes, so drugs targeting those do not act on a virus by the same mechanism
Complication	Treating a secondary bacterial infection does not mean the drug directly kills the original virus

Selection changes proportions even if resistant counts fall

1. **Invented starting population.** 100 resistant + 9,900 susceptible = 10,000; resistant proportion 1%.
2. **Survivors.** 50 resistant + 99 susceptible = 149; resistant fraction $50/149 \times 100 \approx 33.6\%$.
3. **Explain.** The resistant count halved, but susceptible cells fell much more strongly. Differential survival changes composition; later reproduction can expand survivors.

Heritable variation comes before selection by exposure

Mutation and acquired genes are not conscious responses to need

Variants can arise through mutation; gene transfer can spread resistance genes between bacteria.

Different mechanisms or genes can accumulate, producing resistance to multiple antibiotics.

Appropriate use and infection prevention can reduce selection and spread, while research screens compounds and then tests mechanism, toxicity and effectiveness.

An inhibition zone is not a complete medicine trial

1. **Controlled comparison.** Use only an approved organism and protocol; standardize inoculum, medium, disc dose, incubation and measurement.
2. **Evidence.** Suitable controls and independent repeats help compare growth inhibition under these conditions.
3. **Limit.** A clear zone alone does not establish human safety, effective dosing or clinical benefit. Do not grow unknown pathogens.

Match the animal connection with the actual route

Example	Host and route
Bovine tuberculosis	M. bovis: cattle; contaminated unpasteurized dairy or close exposure; distinct from mainly human-associated M. tuberculosis
Rabies	Infected mammal saliva can enter through a bite
Japanese encephalitis	Culex mosquitoes transmit virus maintained among pigs/water birds; humans usually do not continue that mosquito cycle
COVID-19 emergence	Evidence supports zoonotic spillover; the precise first route remains unresolved

An animal origin does not make every later case an animal exposure

Distinguish reservoir, vector, spillover and later transmission

A reservoir maintains an agent; a vector carries it between hosts. Contact alone does not ensure successful infection in a new species.

A zoonotic origin can be followed by sustained human-to-human transmission. Japanese encephalitis is not spread by ordinary contact with a patient.

For SARS-CoV-2, unresolved details concern the original spillover route; later human-to-human transmission is established. Do not invent a proven first intermediate host.

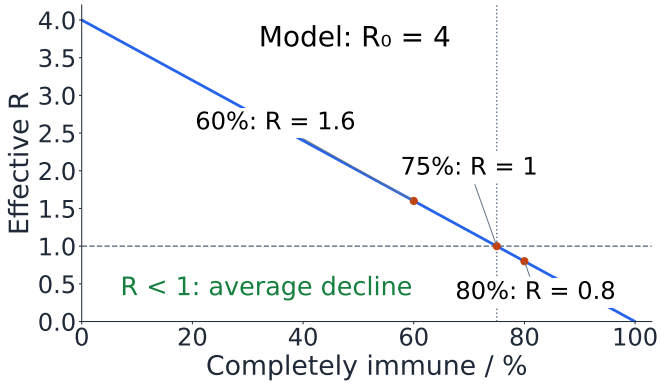
Different delivery methods prepare a specific response

1. **Antigen delivery.** Vaccines can use inactivated/weakened pathogens, purified components or instructions for cells to produce a chosen **antigen**.
2. **Response.** Recognition, activation, clonal expansion and differentiation produce effector activity and memory; boosters can strengthen or renew it.
3. **Limit.** Producing one antigen from vaccine mRNA is not the same as containing a whole reproducing virus. Protection depends on the measured endpoint.

Distinguish relative reduction from percentage-point difference

1. **Invented equal follow-up.** Unvaccinated: 80/1,000 = 8%; vaccinated: 20/1,000 = 2% develop the defined outcome.
2. **Compare.** Risk ratio 2/8 = 0.25; relative reduction 75%; absolute difference six percentage points.
3. **Evaluate.** Trials test benefits and harms; monitoring continues. Low risk is not zero side effects. Early media reports may precede completed scientific evaluation. Check design, endpoint, denominator and uncertainty.

Locate the boundary and then identify average decline



At the boundary $R = 1$; greater immunity gives $R < 1$ in this model.

Solve the uniformly mixing model with complete protection

1. **Relation.** $R = R_0(1 - p)$, where p is completely protected proportion contributing no onward transmission.
2. **Boundary.** For $R_0 = four$, $p = one - one/four = 75\%$ gives $R = one$.
3. **Compare.** At 60%, $R = 1.6$; at 80%, $R = 0.8$. More than the boundary is required for average decline, not a guarantee of zero new cases.

Coverage is not automatically the protected proportion

1. **Additional model.** If **vaccination** coverage is v and protection against transmission is e, approximate $p = v \times e$.
2. **Calculate.** At $R_0 =$ four and $e = 0.90$, boundary coverage $= 0.75/0.90 \approx 83.3\%$; decline requires greater coverage.
3. **Assumptions.** Uniform exposure, otherwise susceptible population and stable protection. Clustering, waning and variants matter; severity is different from infectiousness.

Choose the right interpretation of the count

Measure	What it describes
Prevalence	Existing cases at a stated time or over a defined period
Incidence	New cases over a period, with population-at-risk or person-time denominator
Cumulative reported total	All reports since the stated starting point; not the current number ill
Case definition	Detected infection, symptomatic disease or admission are different outcomes

Read two historical WHO totals with their reporting dates

1. **Report 1.** Published 21 January 2020; data reported by 20 January: 282 globally confirmed cases.
2. **Report 2.** Published 22 January; data reported by 21 January: 314. Difference 32; increase $32/282 \times 100 \approx 11.3\%$.
3. **Limit.** These early reported totals are not present-day pandemic totals. Delay, testing and reporting mean the difference is not exactly the infections acquired during that day.

The denominator determines the meaning of percentage difference

1. **Percentage change.** Use the earlier value: $32/282 \times 100 \approx 11.3\%$.
2. **Symmetric difference convention.** Use the mean, 298: $32/298 \times 100 \approx 10.7\%$.
3. **State the rule.** Percentage difference has different conventions; show the formula requested. Neither calculation identifies a biological cause by itself.

Compare published annual rates per equal population

1. **Historical CDC TB data.** US incidence: 2.5 per 100,000 in 2022; 2.9 per 100,000 in 2023.
2. **Standardize.** Multiply each by ten: 25 versus 29 per million; the displayed difference is four cases per million per year.
3. **Interpretation.** These rounded TB-disease surveillance rates are not latent infections or current prevalence. Match windows, definitions and detection; consider exposure and age before claiming causes.

Attempt the full handout questions before checking

Use mechanisms to support the arithmetic

Explain barriers, clotting, phagocytosis, antigen specificity, B/T cooperation, clonal expansion and memory.

Interpret HIV routes, antibiotic selection, zoonotic host/vector roles and the endpoint measured in a vaccine comparison.

Calculate antibody changes, resistant proportions, risks, transmission boundaries and reported-case changes; state assumptions and safe methods.

Check response and resistance calculations

1. **Antibody response.** $15 \rightarrow 90$ units gives sixfold peak and 500% increase; eight $\rightarrow$ two days gives 75% shorter detection delay.
2. **Resistance.** Initial $200/10{,}000 = 2\%$; survivors $100/198 \approx 50.5\%$ resistant, although resistant cells fell from 200 to 100.
3. **Causal distinction.** Memory supports a later specific response; antibiotic exposure selects heritable variants rather than bacteria choosing resistance.

Check vaccine and herd-model calculations

1. **Vaccine.** 60/1,200 = 5%; 15/1,200 = 1.25%; risk ratio 0.25, relative reduction 75%, difference 3.75 percentage points.
2. **Transmission.** R_0 = five gives boundary 80%; 75% immune gives R = 1.25, while 85% gives R = 0.75.
3. **Imperfect protection.** At e = 90%, **vaccination** boundary 0.80/0.90 $\approx$ 88.9%; greater coverage is needed for average decline under the assumptions.

Check counts, time windows and equal denominators

1. **Reported totals.** $400 \to 500$: 25% increase; symmetric difference $100/450 \approx 22.2\%$. Reports need not equal infections acquired that day.
2. **One-month risks.** $24/800 = 3\% = 30$ per 1,000; $36/1{,}600 = 2.25\% = 22.5$ per 1,000; ratio 1.33 and difference 0.75 percentage points.
3. **Interpretation.** Fewer cases can still mean greater risk. Prevalence concerns existing cases; supplied data or a controlled contact-network simulation avoids unknown-pathogen cultures.

Check yourself

Name the cell, molecule, route and denominator

Platelets recruit, thrombin catalyses, fibrin forms a mesh, plasma cells secrete and memory populations support a later response.

Antigens can be self or external. Specificity belongs to selected clones and compatible binding sites, not every antibody attacking every agent.

Separate HIV infection from AIDS; viruses from antibiotic targets; immunity from vaccine coverage; and a threshold boundary from average decline.