

3 · Infection and response

AQA · GCSE · Biology

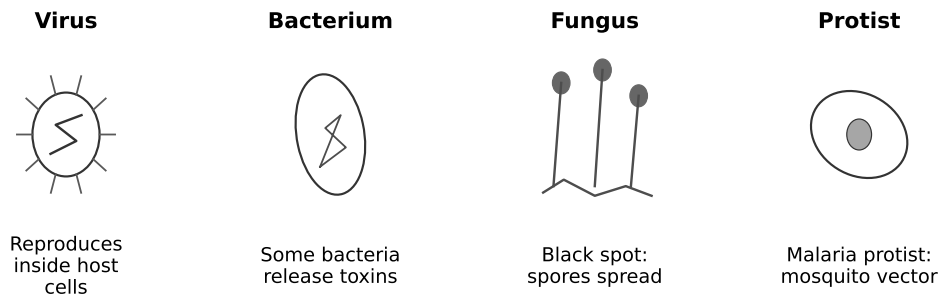
Infection and response: the war inside

Pathogens invade, defences respond, and medicine arms the fight. This reference covers **AQA GCSE Biology 8461, topic 4.3 Infection and response**.

How the exam treats this topic:

- Paper 1 carries this topic; RP2 (antibiotics/antiseptics on agar) links from topic 1.
- **Monoclonal antibodies** (4.3.2) and **plant disease** (4.3.3) are **biology only**, and monoclonals plus plant-disease detection are **HT only**.
- Recall exactly: each named disease → pathogen type, symptoms and spread; the defence systems; the drug-testing stages.

Communicable diseases and pathogens (4.3.1.1)



Pathogens are microorganisms causing infectious disease: **viruses, bacteria, protists, fungi**. They infect plants or animals and spread by **direct contact, water or air**. Bacteria may produce **toxins** that damage tissues; viruses live and reproduce inside cells.

Reducing spread: hygiene (hand washing), sterilising wounds, clean water, sewage treatment, using condoms, vector control (mosquito nets, draining water).

Named diseases (4.3.1.2–4.3.1.5)

Disease	Pathogen	Spread	Symptoms/signs	Control
measles	virus	droplet inhalation (sneezes, coughs)	fever, red skin rash; can be fatal	vaccination of young children
HIV	virus	sexual contact, exchange of body fluids (shared needles)	flu-like illness; attacks immune cells → late-stage AIDS	antiretroviral drugs; safe sex; clean needles
TMV (tobacco mosaic virus)	virus (plants)	contact between plants	mosaic discolouration of leaves; reduced photosynthesis and growth	resistant varieties; hygiene
Salmonella	bacterium	food ingested in unhygienic conditions	fever, cramps, vomiting, diarrhoea (bacteria + toxins)	poultry vaccinated (UK); hygienic food prep
gonorrhoea	bacterium	sexual contact	thick yellow/green discharge, pain urinating	antibiotics (resistance rising); condoms
rose black spot	fungus (plants)	spores in water/air	black spots on leaves; leaves yellow and drop	removing/burning leaves; fungicide
malaria	protist	mosquito vector	recurrent fever; can be fatal	nets; stopping mosquito breeding

From knowledge to exam answers:

Pathogen means a microorganism causing infectious disease: viruses, bacteria, protists or fungi. A **vector** carries a pathogen; malaria's mosquito is the vector and its protist is the pathogen. The teacher drawings above are schematic examples, not to scale. Virus shapes and genetic material vary.

Choose controls by transmission route: reduce inhalation of infected droplets; use hygiene to prevent food contamination; use clean water; reduce exchange of infected body fluids; avoid vector bites and breeding. Bacteria may release toxins that damage tissue. Viruses reproduce inside host cells, causing damage.

Measles precision (2025 Q01.1/.2/.4): virus; inhaled cough/sneeze droplets; fever or red rash. **Malaria terminology (2023 Q05.1):** mosquito = vector, protist =

pathogen. Match each prevention to the transmission route.

Worked symptom mechanism (original 2023 Q03.1 [1]): Salmonella bacteria release toxins. The scheme ignores “toxins” alone: state who produces them.

Worked spread/control (sheet Q3.1 [4], teacher rubric): bacteria are swallowed in contaminated food; poor food hygiene permits contamination; UK poultry are vaccinated; fewer infected animals mean fewer bacteria reach human food. Symptoms alone do not explain spread or control. Original 2023 Q03.2 [4] additionally requires specific antibodies/rapid secondary response in the animal and a link to less contaminated human food; see the immunity section.

Worked growth chain (sheet Q3.2 [3]): TMV reduces functioning chlorophyll-containing leaf area → less photosynthesis → less glucose for respiration and making biomass → less growth. Black spot also reduces photosynthesis when leaves are damaged or lost.

Worked resistance (sheet Q3.3 [4]): random mutation produces resistant variants → penicillin kills susceptible bacteria → survivors reproduce/pass on resistance → repeated selection raises the resistant proportion. Bacteria do not learn resistance or mutate because they need it.

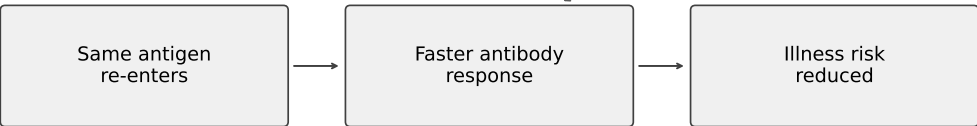
Worked vector control (sheet Q1.3): mosquito is the vector [1]; nets or preventing mosquito breeding is a suitable control [1]. Teacher explanation: nets reduce infectious bites; fewer breeding sites reduce the number of vectors.

Human defence systems and immunity (4.3.1.6–4.3.1.7)

Vaccination



Later re-exposure



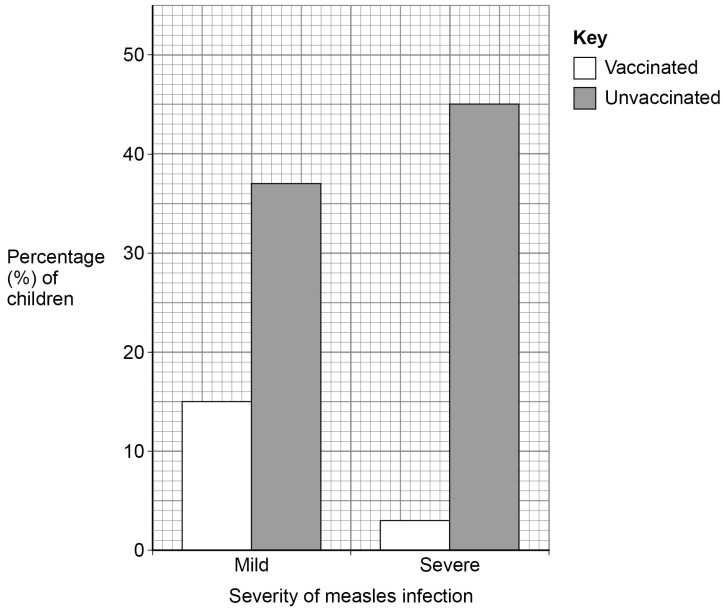
Teacher sequence model, not measured response data.

Non-specific defences: skin forms a physical barrier; scabs seal wounds. Nose hairs/mucus trap inhaled pathogens. Airway mucus traps pathogens; cilia move it towards the throat to be swallowed. Stomach hydrochloric acid kills many swallowed pathogens.

White blood cells defend by **phagocytosis** (engulf/digest), **antibody production** (antibodies complementary to a specific antigen), and **antitoxin production** (neutralising toxins). An antigen is the target recognised by an antibody; antitoxins are not antibiotics.

Worked vaccination (sheet Q3.1 [4]): small amounts of dead/inactive pathogen supply antigens → white cells make specific antibodies → memory cells remain → later exposure produces correct antibodies quickly, reducing illness risk. This is the syllabus vaccine model, not a claim that every vaccine has the same manufacture or guarantees zero infection.

Population protection (original 2025 Q01.5 [2]; sheet Q3.2 [3]): fewer susceptible/infected hosts → fewer opportunities to pass on the pathogen → unvaccinated people are less likely to encounter it. “Herd immunity” alone does not supply the mechanism. Vaccination reduces infection and transmission risk; protection is not absolute.



Original AQA June 2025 Figure 1; percentages of all surveyed children.

Original 2025 Figure 1: percentages refer to the entire surveyed sample, not to the vaccinated or unvaccinated group alone.

Severity	Vaccinated	Unvaccinated
Mild	15%	37%
Severe	3%	45%

Worked number (Q01.6 [3]): 240 surveyed children; severe/unvaccinated bar = 45%.

$$N = \frac{p}{100} \times N_{\text{total}}$$

$$N = \frac{45}{100} \times 240 \text{ children}$$

$$N = 108 \text{ children}$$

Evidence (Q01.7 [2]): severe cases represent 3% vaccinated versus 45% unvaccinated in the whole sample. This supports reduced severity; vaccinated infected children are still present. Do not silently change the denominator or label these raw percentages as vaccine efficacy.

Animal-to-human chain (2023 Q03.2 [4]): specific antibodies in vaccinated animals → rapid response to later Salmonella infection → fewer bacteria in animal products → fewer bacteria ingested by humans, reducing infection/toxin production. The original level-two requirement links the animal immune response to human food poisoning.

Antibiotics, painkillers and drug development (4.3.1.8–4.3.1.9)

Antibiotics kill susceptible infective bacteria; they do not kill viruses. The cold is viral, so antibiotics do not cure it (sheet Q1.3 [2]). **Painkillers** relieve symptoms without killing pathogens. Viruses reproduce in host cells, making antiviral development difficult; this does not mean antiviral treatment is impossible.

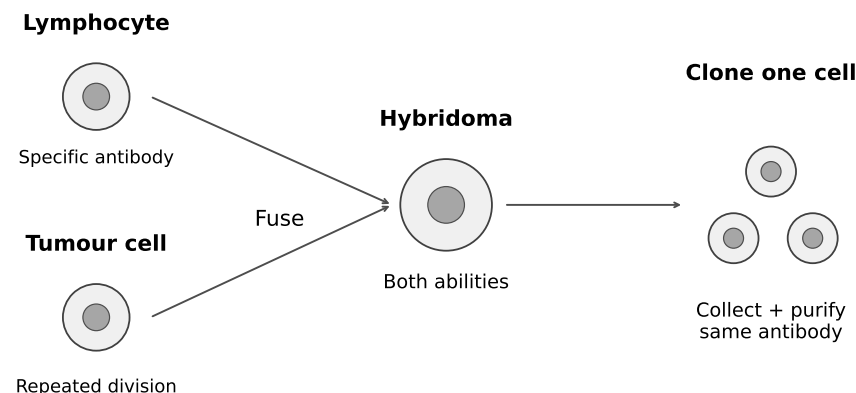
Sources: digitalis–foxglove (heart); aspirin–willow (painkiller; 2025 Q01.3); penicillin–Penicillium mould (Fleming). Modern synthesis can start from plant chemicals.

Testing questions (2023 Q01.7 [2]): toxicity (harm/side effects), efficacy (does it work?), dose (how much?). Choose distinct reasons.

Worked testing sequence (sheet Q3.3 [5]): preclinical cells/tissues/live animals investigate toxicity and potential efficacy → healthy volunteers start at very low dose for safety → patients test efficacy/optimum dose → appropriate controlled double-blind comparison → peer review scrutinises methods/results. Original 2024 Q08.6 [1] identifies testing live tissues in a laboratory as preclinical; volunteers/patients are clinical.

Placebo and blinding: placebo lacks the active drug and helps account for expectation effects. In a double-blind comparison neither participants nor outcome assessors know allocation, reducing bias. Comparator choice depends on the trial; not every trial uses placebo. Peer review is scrutiny, not a guarantee of success. Original 2023 Q01.8 [1]: company financial gain or competitor loss can motivate bias; Q01.9 [1]: peer review scrutinises claims.

Monoclonal antibodies (biology only, HT)



Teacher schematic, not to scale; clone one antibody-producing cell.

Monoclonal antibodies come from one clone and bind one site on one protein antigen. Complementary shape means a binding fit, not identical shape.

Production (sheet Q1.1/Q3.1): stimulate mouse lymphocytes with the relevant antigen → fuse an antibody-producing lymphocyte with a repeatedly dividing tumour cell → **hybridoma** combines both abilities → clone a single suitable hybridoma → collect and purify its identical antibody. Activated lymphocytes can divide; the culture-production issue is sustained division, not “lymphocytes cannot divide”. Original 2025 Q07.4 requires the relevant liver antigen/cells, not an unspecified pathogen.

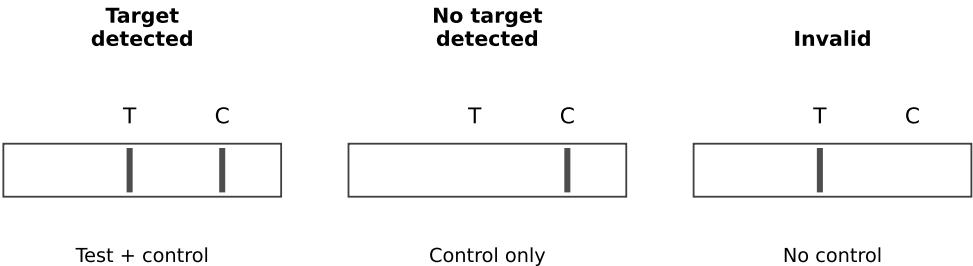
After fusion (original 2023 Q04.5 [3]): clone → many identical cells → same antibody → collect/purify. The original question starts from the hybridoma, so naming it alone is not enough.

Use	How binding is useful
Diagnosis / laboratory	detect hCG, a pathogen antigen or another chemical; measure hormone levels
Research	a fluorescent label locates a specific molecule
Targeted treatment	an attached toxic/radioactive agent or growth-inhibitor is delivered towards target cells

Cancer model (sheet Q3.2 [3]): assume the target antigen is confined to cancer cells. Antibody binds that antigen; attached agent is delivered to the target cells, reducing exposure elsewhere. “Without harming other cells” describes this ideal model, not guaranteed real treatment safety. Side effects can occur and must be evaluated.

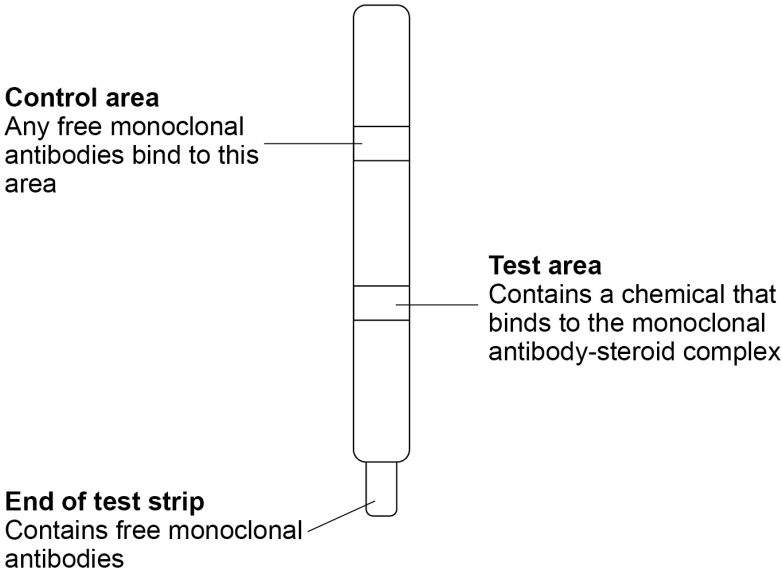
Transplant model (original 2025 Q07.5 [3]): the supplied drug antibody binds

liver antigens because it is complementary in shape → recipient antibodies cannot bind the occupied sites → recognition/attack is reduced. Specificity alone is insufficient for the three-mark explanation; do not substitute the cancer-payload mechanism.

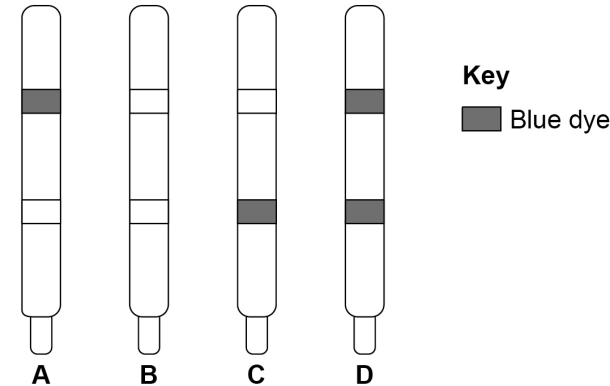


Teacher sandwich-test model: *T* = test; *C* = control. Other assays must be read using their supplied mechanism.

Positive hCG model (sheet Q3.3 [3]): urine hCG binds mobile labelled antibody → complex moves and is captured by fixed antibody at the test zone → accumulated label forms a line. The control shows the process worked. Control only = no target detected under these conditions; no control = invalid, even if the test line appears. A negative-looking invalid result does not prove target absence.



Original AQA 2023 Figure 5: test captures complex; control captures free antibody.



Original Figure 6; the grey key denotes blue dye.

Original 2023 Q04.7–.9: Figure 5 states test area captures antibody–steroid complex; control captures free antibody; binding changes the dye colour. Figure 6: A has control only; B neither line; C test without control; D both. B is invalid because its control has no dye; possible failure is no sample/antibody movement or missing antibody. D is the valid positive. Do not call C positive without checking control.

Plant disease (biology only)

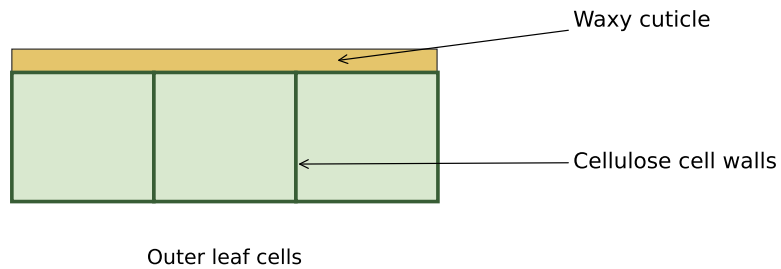
Detection and identification (HT). Signs include stunted growth, spots, decay, growths, malformed stems/leaves, discolouration and visible pests. A sign alerts us to a problem; it does not uniquely identify its cause. Compare the pattern with a **gardening manual/website**, send an affected plant/sample to a **laboratory**, or use a suitable **monoclonal-antibody kit** to detect a specific pathogen antigen. Follow the kit instructions and check its control; failure to detect one target does not exclude every disease.

Named problems: **TMV** (virus, mosaic discolouration), **rose black spot** (fungus, purple/black spots and leaf loss), **aphids** (insect pests).

Deficiency	Mechanism and effect
nitrate ions	less amino-acid/protein synthesis → reduced growth
magnesium ions	less chlorophyll → chlorosis (yellow leaves), less light absorbed → reduced photosynthesis

Worked evidence (2025 Q04.7–.8). Purple spots with leaf loss suggest rose black spot. Yellow leaves without spots **may** result from magnesium shortage: magnesium is needed to make chlorophyll. Lack of light is another accepted explanation. Yellowing alone does not prove a deficiency or rule out infection; 2023 Q01.6 also accepts several plausible causes. Use identification evidence before asserting a diagnosis.

Leaf surface: barriers to pathogen entry



Defences: physical – cellulose cell walls and waxy cuticle resist pathogen entry; bark/dead outer layers protect stems and can fall off. Chemical – antibacterial chemicals act against bacteria; poisons deter herbivores. Mechanical – thorns/hairs deter feeding, touch-induced drooping/curling can discourage herbivores, and mimicry can deter them. Explain the function of the named feature, rather than saying only “protects the plant”.

Growth chain: less chlorophyll → less photosynthesis → less glucose for biomass and respiration; respiration releases energy for growth. Nitrate shortage acts through reduced protein synthesis, not by being a fuel for respiration.

Checklist before you call this topic done

- Four pathogen types; three routes of spread; two ways to reduce each named disease's spread.
- Recite the disease table: pathogen, spread, symptoms, control.
- Four non-specific defences; three white-blood-cell actions.
- Vaccination mechanism + population effect; antibiotic vs painkiller; why antibiotics fail on viruses.
- Drug sources (foxglove, willow, Penicillium) and the preclinical/clinical stages with placebo and double-blind.
- (HT, biology) Hybridoma production sequence and four monoclonal uses.
- (Biology) Plant disease signs, identification methods, nitrate/magnesium deficiencies, three defence classes.