

Cambridge AS & A Level · Biology 9700 · Notes

Infectious diseases

AS

Read online at pastopic.com/cambridge/biology-9700/notes/infectious-diseases

Last checked 30 September 2026

Read first: [Cell structure](#)

© 2026 Pastopic. Free for personal study. Please share the link, not the file.

What you need to know

By the end of this topic you should be able to:

1. **State that infectious diseases are caused by pathogens and are transmissible:** the pathogen can be passed from an infected person to an uninfected one.
2. **Name the pathogen and its type for four diseases:**
 - cholera: the bacterium *Vibrio cholerae*;
 - malaria: the protoctists *Plasmodium falciparum*, *P. malariae*, *P. ovale* and *P. vivax*;
 - tuberculosis (TB): the bacteria *Mycobacterium tuberculosis* and *M. bovis*;
 - HIV/AIDS: the human immunodeficiency virus (HIV).
3. **Explain how cholera, malaria, TB and HIV are transmitted.**
4. **Discuss the biological, social and economic factors** to consider when preventing and controlling these four diseases. The details of the malarial parasite's life cycle are not needed.
5. **Outline how penicillin acts on bacteria**, and explain why antibiotics do not affect viruses.
6. **Discuss the consequences of antibiotic resistance** and the steps that reduce its impact.

Questions often bring in ideas from other topics: prokaryotic and eukaryotic cells (cell structure), vaccines, antibodies and T-helper cells (immunity), and graphs and tables of data. They also introduce unfamiliar diseases (Zika, sleeping sickness, measles) and expect you to compare them with these four.

Understand it

Pathogens, hosts and vectors

A **pathogen** is an organism that causes disease. An **infectious disease** is one caused by a pathogen that can be passed from one host to another: it is **transmissible**. Diseases caused by faulty genes, a poor diet or the environment (such as lung cancer from smoking) are not infectious, because nothing is passed on.

The pathogens in this topic belong to three different groups, and the group decides how you can treat them:

Disease	Pathogen	Type	Cell?
Cholera	<i>Vibrio cholerae</i>	bacterium	prokaryotic cell: peptidoglycan wall, 70S ribosomes, circular DNA
Tuberculosis (TB)	<i>Mycobacterium tuberculosis</i> (and <i>M. bovis</i> , from cattle)	bacterium	prokaryotic cell
Malaria	<i>Plasmodium falciparum</i> , <i>P. malariae</i> , <i>P. ovale</i> , <i>P. vivax</i>	protocist	eukaryotic cell: nucleus, 80S ribosomes, no peptidoglycan
HIV/AIDS	human immunodeficiency virus (HIV)	virus	not a cell: RNA inside a protein coat, no ribosomes, no metabolism of its own

A **vector** is an organism that carries a pathogen from one host to another without being the cause of the disease itself. The female *Anopheles* mosquito is the vector of malaria; the mosquito is not the pathogen and not the parasite. *Plasmodium* is. Cholera, TB and HIV have no vector.

Write the species name as the syllabus does: genus with a capital, species in lower case, *Vibrio cholerae*, *Mycobacterium tuberculosis*. On paper you underline it instead of using italics. After the first use you may shorten the genus: *V. cholerae*, *M. tuberculosis*. Mark schemes want the spelling right.

Cholera

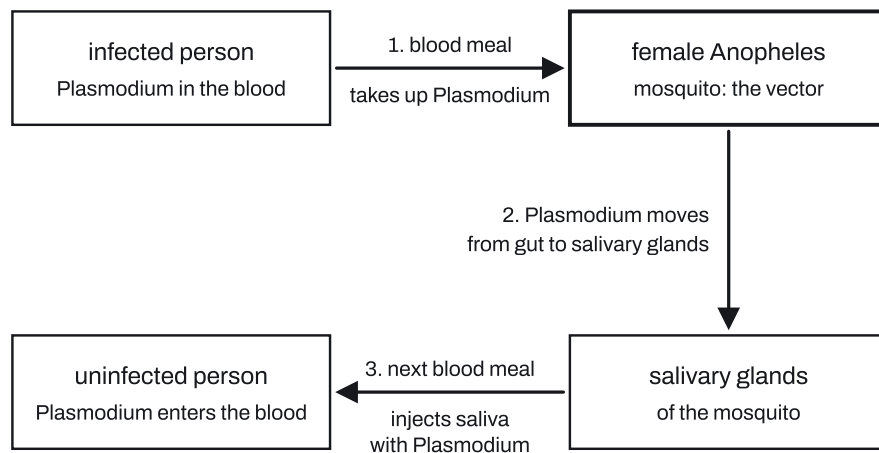
V. cholerae lives in the small intestine. It releases a toxin that makes the intestine lose chloride ions and a lot of water, so the person has severe watery diarrhoea and becomes dehydrated. Untreated, many die within days.

Transmission: the faecal-oral route. The faeces of an infected person contain huge numbers of the bacteria. If they get into drinking water (untreated sewage, no toilets, floods) or onto food (washed or grown in dirty water, handled with unwashed hands, landed on by flies), another person swallows the bacteria. Cholera is **not** spread by airborne droplets, by touch alone or by a vector. Many infected people have no symptoms but still pass the bacteria in their faeces, so they spread it without knowing.

Treatment. Oral rehydration therapy, drinking water with glucose and salts, replaces what is lost and saves most patients. Antibiotics are kept for severe cases. Shorter illness means fewer bacteria released into the environment.

Malaria

Plasmodium spends part of its life in human liver cells and red blood cells and part in the mosquito. Only the **female** *Anopheles* mosquito feeds on blood (she needs it to make eggs), so only females transmit malaria.



Other routes: blood transfusion, shared needles, mother to baby

Transmission. The mosquito takes a blood meal from an infected person and takes up *Plasmodium*. The parasite moves to the mosquito's salivary glands. When she feeds again she injects saliva (which stops the blood clotting) and, with it, *Plasmodium* into the blood of an uninfected person. Less often, *Plasmodium* passes in a blood transfusion, on a shared needle, or from mother to baby across the placenta.

Malaria is found mostly in tropical and subtropical regions, because *Anopheles* needs warmth, humidity and still water to breed, and *Plasmodium* needs warmth to develop inside the mosquito.

Tuberculosis

TB usually infects the lungs. The bacteria are slow-growing, and many people carry them without being ill: in **latent TB** the bacteria are dormant, walled off in the lungs by the immune system and surviving inside macrophages. Latent TB can become active years later if the immune system is weakened, for example by HIV, malnutrition or old age.

Transmission: droplet infection (also called aerosol infection). A person with active TB in the lungs coughs, sneezes or talks, releasing tiny **airborne droplets** containing the bacteria. An uninfected person breathes them in. It spreads most in crowded, poorly ventilated homes, where people spend a long time close together.

M. bovis causes TB in cattle. People catch it by drinking **unpasteurised milk** or eating undercooked meat from infected cattle.

HIV/AIDS

HIV infects **T-helper cells** (a type of lymphocyte) and slowly destroys them. With few T-helper cells the immune system cannot respond properly, and the person eventually develops AIDS: they catch **opportunistic infections**, such as TB and pneumonia, that a healthy immune system would control. These infections, not HIV itself, are what usually kill.

Transmission: body fluids. HIV passes in blood, semen, vaginal fluid and breast milk:

- unprotected sexual intercourse;

- sharing needles (for example, people who inject drugs), or unsterilised medical equipment;
- a transfusion of blood that was not screened;
- from mother to baby across the placenta, during birth, or in breast milk.

It is not spread by droplets, by touch, in food or water, or by insects.

Why HIV makes TB (and other infections) more likely

T-helper cells release **cytokines** that switch on the rest of the immune response. When HIV destroys them:

- fewer B-lymphocytes are activated to divide and become **plasma cells**, so fewer **antibodies** are made;
- fewer T-killer cells are activated to destroy infected cells;
- macrophages are less stimulated, so less phagocytosis;
- fewer memory cells form, and vaccines work less well.

So a person with HIV is far more likely to catch TB, and latent TB is more likely to become active. More cases of active TB then spread it to others. This is why rises in HIV infection have been followed by rises in TB, and why **antiretroviral therapy (ART)**, which keeps the number of T-helper cells up, also cuts deaths from TB and other infections.

Biological, social and economic factors

"Discuss the factors in prevention and control" means thinking in three groups:

- **Biological:** about the pathogen, vector or host, such as how it spreads, whether it mutates, whether there is a vaccine, symptomless carriers, latent infection, drug or insecticide resistance, the climate the vector needs.
- **Social:** about how people live, such as crowding, sanitation, clean water, education, travel and migration, war and natural disasters, willingness to be tested or vaccinated, whether people finish their treatment.
- **Economic:** about money, such as the cost of drugs, vaccines, nets, water treatment and staff, and whether a country can pay for them. Almost every control measure costs money, so almost every measure has an economic side.

The table sums up control and what makes each disease hard to control.

Disease	How it is prevented and controlled	Why it is hard to control
Cholera	Treat sewage; chlorinate, boil or filter drinking water; build toilets and latrines away from water; wash hands with soap after the toilet and before handling food; cook food, peel or wash fruit in clean water, cover food from flies; oral rehydration therapy and isolate cases; oral vaccine in outbreaks	Symptomless carriers; outbreaks after floods, earthquakes and wars, and in crowded camps with broken sanitation; the vaccine protects for only about two years, takes weeks to work and is not always affordable; cases are under-reported; antibiotic resistance

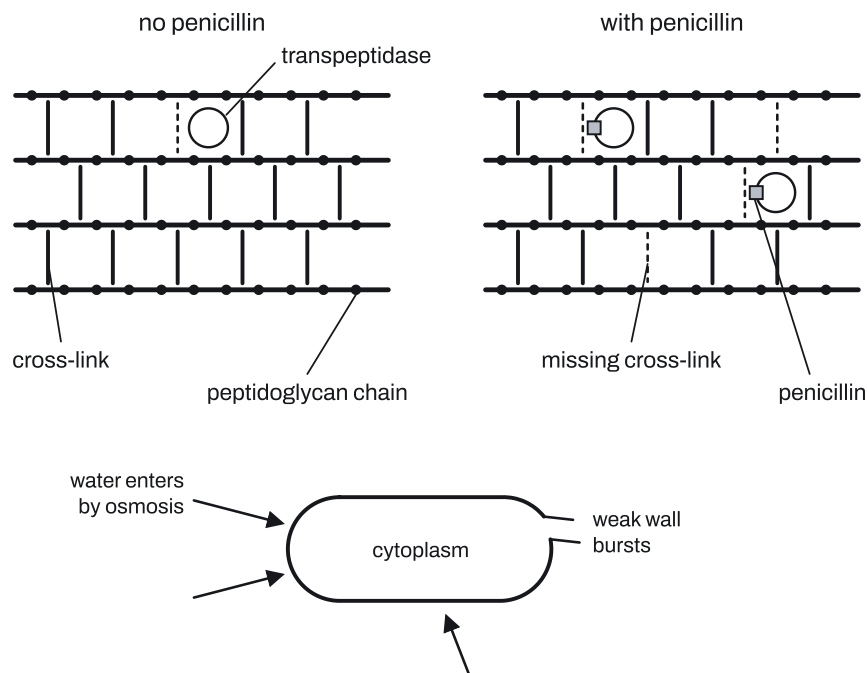
Disease	How it is prevented and controlled	Why it is hard to control
Malaria	Sleep under insecticide-treated nets; insect repellent, clothes covering the skin; spray insecticide inside houses; drain or cover standing water, spread oil on water, stock ponds with fish that eat larvae, release sterile males; antimalarial drugs to treat and to protect travellers, given as a combination of drugs; quick diagnosis with test strips	Mosquitoes resistant to insecticides; <i>Plasmodium</i> resistant to drugs; no vaccine gives full protection (newer ones give partial protection); <i>Plasmodium</i> is a eukaryote hidden inside liver and red blood cells; climate change widens the mosquito's range; travel, migration and war; cost of nets, spraying and drugs
TB	BCG vaccine; find and test contacts of each case (contact tracing); isolate and treat active cases with several antibiotics for at least six months, watched by a health worker (DOTS); treat latent TB; pasteurise milk, test and remove infected cattle; better housing, less overcrowding	Latent TB carriers; long treatment that many do not finish; multi-drug-resistant strains; HIV; poverty, overcrowding and malnutrition; migration from high-TB areas; BCG protects adults less well
HIV/AIDS	Condoms (a barrier during sex); testing, contact tracing and education; needle-exchange schemes and sterile medical equipment; screen or heat-treat donated blood; ART, which lowers the amount of virus and so the chance of passing it on; drugs taken before exposure (PrEP) or after it (PEP); ART in pregnancy and formula milk instead of breast milk	No vaccine and no cure; many people do not know they are infected; long symptomless period during which they infect others; the virus mutates; cost of lifelong ART; stigma

How penicillin works

An **antibiotic** is a substance that kills bacteria or stops them growing, without harming human cells. Penicillin works on the bacterial **cell wall**, which is made of **peptidoglycan**: long chains joined sideways by short **cross-links**. The cross-links make the wall strong enough to stop the cell bursting when water enters it by osmosis.

When a bacterium grows, enzymes called autolysins open small gaps in the wall so new peptidoglycan can be added, and enzymes called **transpeptidases** then form new cross-links to seal the wall again. Penicillin has a shape that fits the active site of the transpeptidases, so it **inhibits** them (it acts like a competitive inhibitor):

1. new cross-links are not formed in a growing wall;
2. the wall is left weak where it was being enlarged;
3. water enters the bacterium by **osmosis** (its cytoplasm has a lower water potential than its surroundings);
4. the weak wall cannot resist the pressure, and the cell **bursts** (osmotic lysis).



Two consequences follow. Penicillin only kills bacteria that are **growing** and making new wall; it does not break down a finished wall or "make holes" in it. And it only works where there is peptidoglycan, so it does nothing to human cells, fungi or protists such as *Plasmodium*.

Why antibiotics don't affect viruses

Antibiotics act on structures and enzymes that bacteria have and human cells don't: the peptidoglycan wall and its transpeptidases (penicillin), 70S ribosomes (for example tetracycline), or bacterial RNA polymerase (rifampicin). A virus has **none** of these. It is not a cell: it has no cell wall, no ribosomes and no metabolism of its own, and it copies itself inside a host cell using the host's enzymes and ribosomes. So there is nothing for the antibiotic to act on. Antibiotics are still given to people with HIV/AIDS, but to treat or prevent the **bacterial** infections they catch, not HIV itself.

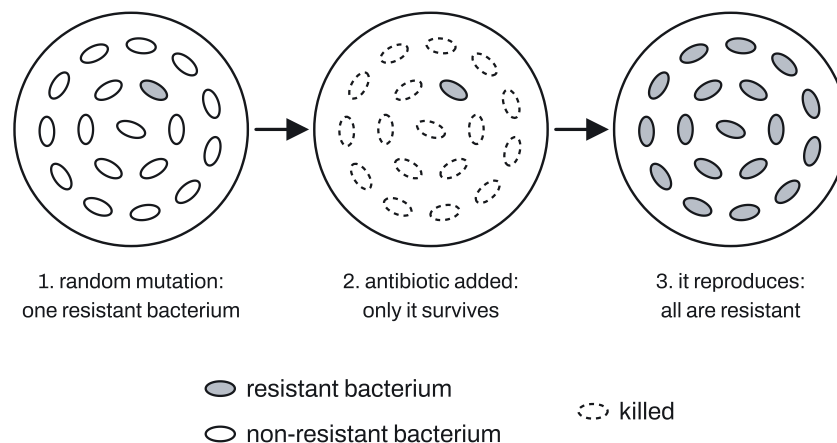
The same reasoning explains why many antibiotics don't harm us: our cytoplasm has 80S ribosomes, which have a different shape, so a drug that fits a 70S ribosome doesn't bind to them, and our cells have no wall.

Antibiotic resistance

A resistant bacterium is one that is not killed by an antibiotic that used to kill it. Resistance evolves by **natural selection**:

1. A **random mutation** in a bacterium's DNA gives an allele for resistance. The antibiotic does not cause the mutation; it happens by chance, whether or not the antibiotic is there.
2. This creates **variation** in the population.
3. The antibiotic acts as a **selection pressure**: non-resistant bacteria are killed, and resistant ones survive.
4. The survivors reproduce (by binary fission) and pass the resistance allele to their offspring. Resistance alleles on plasmids can also pass to other bacteria, even of other species.

5. Over many generations the **frequency of the resistance allele** rises until most of the population is resistant.



How a mutation gives resistance (questions describe the mechanism and ask you to explain it):

- an enzyme that breaks down the antibiotic (for example penicillinase, which breaks down penicillin);
- a change in the shape of the target protein, so the antibiotic binds less well (penicillin binding less to the enzymes that make the wall);
- a carrier protein in the cell surface membrane that **actively transports** the antibiotic out of the cell using ATP (an efflux pump), so it never reaches a toxic concentration.

A bacterium resistant to several different antibiotics is **multi-drug-resistant** (MDR). MDR-TB is a major problem.

The more an antibiotic is used, the stronger and more frequent the selection pressure, so resistance spreads fastest where antibiotics are overused or misused: given for viral infections, given "just in case", given to healthy farm animals, courses stopped early (which leaves the tougher survivors alive), or broad-spectrum drugs used where a specific one would do.

Consequences of resistance:

- infections that used to be easy to cure can no longer be treated with the usual antibiotic;
- people are ill for longer, stay infectious for longer and so infect more people;
- more people die, including from routine operations and minor wounds that become infected;
- second-line drugs cost more and may have worse side effects, and hospital stays are longer;
- resistance alleles pass to other bacteria, so other diseases become harder to treat too;
- new antibiotics must be researched, which is slow and expensive.

Steps that reduce the impact:

- prescribe antibiotics only when they are really needed: never for viral infections, not "just in case";
- identify the pathogen first, then use the right, **narrow-spectrum** antibiotic at the right dose;

- **finish the whole course** (for TB, a health worker watches each dose: DOTS), and don't share or keep leftover antibiotics;
- use **two or more antibiotics with different modes of action** at once, so a bacterium would need several mutations at the same time to survive (the same idea as combination therapy for malaria);
- limit antibiotic use in farming, especially in healthy animals;
- prevent infections so fewer antibiotics are needed: vaccination, hand hygiene in hospitals, isolating patients with resistant infections;
- monitor and report resistance, and educate doctors and the public;
- develop new antibiotics and other antibacterial treatments.

Key facts and formulas

Nothing in this topic is in the syllabus's list of given formulae, so learn all of it.

Formula	Status
Percentage change = $\frac{\text{new} - \text{old}}{\text{old}} \times 100$ (negative means a decrease)	Learn it
Case-fatality rate = $\frac{\text{number of deaths}}{\text{number of cases}} \times 100$	Learn it
Rate per 100 000 = $\frac{\text{number of cases}}{\text{population}} \times 100\,000$	Learn it
Cholera: <i>Vibrio cholerae</i> , bacterium, faecal-oral (water and food)	Learn it
Malaria: <i>Plasmodium</i> (four species), protoctist, female <i>Anopheles</i> mosquito vector	Learn it
TB: <i>Mycobacterium tuberculosis</i> and <i>M. bovis</i> , bacteria, airborne droplets (and unpasteurised milk for <i>M. bovis</i>)	Learn it
HIV/AIDS: human immunodeficiency virus, body fluids (sex, needles, blood, mother to baby)	Learn it
Penicillin inhibits transpeptidases, so no cross-links in a growing peptidoglycan wall, then osmotic lysis	Learn it

How to do it

We have tagged 118 questions on this topic from 2021–2025 (79 multiple-choice, 39 structured). 32 of them mix more than one type, so the counts add up to more than 118.

Question type	Questions
Name the pathogen, its type, or define infectious	36
Describe how a disease is transmitted	26
Suggest and explain prevention and control measures	24
Interpret disease or antibiotic data	20
Explain how antibiotics act, and why not on viruses or human cells	18
Explain why a disease is hard to control, why outbreaks happen, or its distribution	17
Explain how antibiotic resistance arises	11
Steps to reduce antibiotic resistance, or its consequences	11
Explain why HIV/AIDS raises the risk of TB and other infections	7

1. Name the pathogen, its type, or define infectious (36 questions)

- Species name:** give the full binomial, spelled exactly: *Vibrio cholerae*, *Mycobacterium tuberculosis* (*M. bovis* if the question is about cattle), *Plasmodium falciparum* (or *malariae*, *ovale*, *vivax*). For HIV write out "human immunodeficiency virus".
- Type:** bacterium (prokaryote) for cholera and TB, protoctist (eukaryote) for malaria, virus for HIV. If asked for the type, "protoctist" is the answer for malaria; "eukaryote" alone is not enough.
- Definitions:** infectious = caused by a pathogen **and** transmissible (two marks, two ideas). Transmissible = the pathogen can be passed from an infected host to an uninfected one. Pathogen = an organism that causes disease.

Variations you will meet:

- Which disease is caused by a eukaryote?** Malaria. Cholera and TB are prokaryotes; HIV (and measles, smallpox) are viruses.
- What do the pathogens share?** All have genes (nucleic acid). Only the bacteria and *Plasmodium* are cells with a cell surface membrane and ribosomes; HIV has neither (its outer envelope is taken from the host cell, and it is not a cell). Only *V. cholerae* and *M. tuberculosis* have peptidoglycan, so only they stain with a stain for peptidoglycan (**9700/12/O/N/24 Q40**).
- The mosquito:** a vector, never "the pathogen" or "the parasite" (**9700/13/M/J/22 Q37**).
- Unfamiliar diseases** (measles is caused by *Morbillivirus*, smallpox by *Variola*): use the information given.

2. Describe how a disease is transmitted (26 questions)

Give **both ends** of the transmission: how the pathogen leaves the infected person and how it gets into the uninfected one. Use the correct term.

- Cholera:** faeces of an infected person contaminate drinking water or food; an uninfected person drinks the water or eats the food. The term is **faecal-oral**. Never "infected water", never airborne.

- **TB:** an infected person coughs or sneezes, releasing airborne droplets containing *M. tuberculosis*; an uninfected person inhales them. The term is **droplet infection** or **aerosol infection**. Write "droplets containing the bacteria", not "infected droplets" or "air droplets". For *M. bovis*: drinking unpasteurised milk (or eating meat) from infected cattle.
- **Malaria** (3 marks): vector, the female *Anopheles* mosquito; takes a blood meal from an infected person, with *Plasmodium* in the blood; injects *Plasmodium* with saliva (anticoagulant) into an uninfected person at the next meal. Also transfusion, shared needles, placenta.
- **HIV:** exchange of body fluids: unprotected sex, shared needles, unscreened blood, mother to baby (placenta, birth, breast milk).

Variations you will meet:

- **Compare with an unfamiliar disease.** Zika and sleeping sickness are also carried by insects that feed on blood. Look for the differences in the stem: the insect (*Aedes* mosquito, tsetse fly, not *Anopheles*), the type of pathogen (virus, protoctist), and which sex feeds. For malaria only the female mosquito feeds on blood; for sleeping sickness both sexes of tsetse fly do. A mosquito **is** an insect, so "an insect versus a mosquito" is not a difference.
- **Tick-table MCQs** matching pathogen to route: droplets is *Mycobacterium*, insect vector is *Plasmodium*, water is *Vibrio* ([9700/11/M/J/21 Q35](#)).
- **An air filter or water filter** with a size limit: work out which pathogens are larger than the pores (viruses such as HIV are about 0.1 μm , bacteria about 1–5 μm), then ask whether that disease is carried by that route at all. A filter that traps larger particles can still help if the bacteria are attached to something bigger, such as the small water animals *V. cholerae* lives on, but free bacteria pass through. A filter in an air system can only matter for an airborne disease (TB), not for malaria or cholera.
- **Likely source of an outbreak:** untreated sewage, contaminated drinking water, food washed in dirty water, infected people arriving from elsewhere.

3. Suggest and explain prevention and control measures (24 questions)

1. **State the measure** precisely (not "be hygienic" but "wash hands with soap after using the toilet").
2. **Explain how it breaks transmission:** link it to the route. For example, a latrine keeps faeces out of drinking water, so *V. cholerae* is not swallowed.
3. **Give as many different measures as there are marks**, from different groups: stop the pathogen reaching people, protect people (vaccines, nets), and treat cases quickly so they stop spreading it.

Use the table in "Understand it" for the measures.

Variations you will meet:

- **Mosquito control** ([9700/24/M/J/25 Q4](#)): stop females feeding (nets, especially insecticide-treated ones, repellents, covering skin), kill adults (spraying insecticide inside homes), stop breeding (drain or cover standing water, oil on the surface, fish that eat larvae), sterile males.

- **Why treatment also controls spread:** rehydration and isolation in treatment centres shorten the time a cholera patient passes bacteria, and keep contaminated faeces out of the water; treating TB quickly means fewer people inhale the bacteria.
- **Match the measure to the disease** in MCQs: chlorinated water for cholera, destroying the vector for malaria, contact tracing (and vaccination) for TB ([9700/12/O/N/24 Q39](#)). Boiling water does nothing for TB; removing stagnant water does nothing for TB or cholera.
- **Which factors are economic?** Anything that needs money: chlorine, drugs, treatment centres, health workers, records. In [9700/14/M/J/25 Q37](#) every one of the five measures counts.
- **Vaccination programmes:** success needs enough people vaccinated for **herd immunity**, a pathogen whose antigens don't change, a vaccine that is cheap, heat-stable and needs one dose, trained staff, and trust. Smallpox was eradicated because its virus did not mutate and a stable live vaccine was easy to give.
- **Rapid test strips** (monoclonal antibodies): quick, cheap, easy to use without training or equipment, so treatment starts sooner and drugs are not used "just in case".

4. Interpret disease or antibiotic data (20 questions)

1. **Describe a trend:** overall direction first, then the changes within it (rise, peak, fall, plateau) with the **years** and **figures with units** read from the graph.
2. **Calculations:** write the formula, then the numbers, then the answer to the precision asked.
 - Percentage change = $\frac{\text{new} - \text{old}}{\text{old}} \times 100$. A rise from 0.2% to 1.5% is a $\frac{1.3}{0.2} \times 100 = 650\%$ increase, not 1.3% or 87% ([9700/11/M/J/24 Q36](#)). A fall from 5.3 to 3.2 days is a 39.6% decrease ([9700/11/M/J/24 Q34](#)).
 - Case-fatality rate = $\frac{\text{deaths}}{\text{cases}} \times 100$.
 - A percentage of a number: 18% of 462 000 deaths = $0.18 \times 462\,000 = 83\,160$ ([9700/13/O/N/24 Q38](#)).
3. **Conclusions:** say only what the data show. Correlation is not cause, so "overuse caused the rise" cannot be concluded from a graph of resistance alone. Check each option against the actual numbers.
4. **Evaluate a claim:** give points that support it and points that don't, each with data.

Antibiotic test data:

- **Zone of inhibition:** the clear ring around an antibiotic disc where bacteria cannot grow. A **larger zone** means a more effective antibiotic against that bacterium. A zone that **shrinks** day after day means resistant bacteria are growing into it.
- **Counts of colonies** killed, stopped or still growing: the best antibiotic kills the most and leaves the fewest still growing ([9700/14/O/N/25 Q37](#)).
- **Rates per 100 000** let you compare years or countries with different population sizes.

5. Explain how antibiotics act, and why not on viruses or human cells (18 questions)

1. **Penicillin, in order:** inhibits transpeptidase enzymes, so cross-links between peptidoglycan chains are not formed in a **growing** wall; the wall is weak; water enters by osmosis; the cell bursts.

2. **Why not viruses:** no cell wall or peptidoglycan, no transpeptidase, no ribosomes, no metabolism of their own; they are not cells and reproduce inside host cells.
3. **Why not human cells:** no cell wall or peptidoglycan (penicillin); 80S, not 70S, ribosomes in the cytoplasm (drugs that bind 70S ribosomes).

Variations you will meet:

- **Other antibiotics, described in the stem.** Use the information and the complementary-shape idea:
 - a drug that binds to 70S ribosomes (tetracycline, erythromycin, doxycycline) stops translation, so the bacterium makes no proteins or enzymes and cannot grow; it does not bind to human 80S ribosomes because its binding site has a different shape;
 - rifampicin binds to bacterial RNA polymerase (in its active site, or elsewhere so the active site changes shape), so no mRNA is made;
 - vancomycin, like penicillin, stops cross-links forming, but it binds to the peptidoglycan building blocks rather than to the enzyme.
- **Lysozyme versus penicillin:** both weaken the wall so the cell bursts; lysozyme is an enzyme that **hydrolyses** peptidoglycan, while penicillin **inhibits** the enzyme that builds it and so only acts on growing cells.
- **Gram-positive bacteria** are killed more easily by penicillin because their thick peptidoglycan layer is on the outside, exposed; in Gram-negative bacteria an outer membrane covers it ([9700/13/M/J/24 Q37](#)).
- **Drugs against HIV** (tenofovir, stem given): a molecule shaped like a nucleotide competes for the active site of reverse transcriptase, and once added it stops the DNA chain growing.

6. Explain why a disease is hard to control, why outbreaks happen, or its distribution (17 questions)

1. Decide which disease and **which route**. Every factor you give should connect to that route or to the host's immunity.
2. Give factors from all three groups (biological, social, economic) using the table in "Understand it", each with a short reason.

Variations you will meet:

- **Outbreaks after a disaster** (flood, cyclone, earthquake, war, camps): cholera, because sewage mixes with drinking water, water treatment stops, people cannot wash hands, there are no toilets, and people are crowded together ([9700/12/F/M/23 Q38](#)). Cholera spreads within days; HIV and TB do not respond so fast.
- **Global distribution of malaria:** warm, humid tropical areas with rainfall and still water where *Anopheles* breeds, often at low altitude; spreading with climate change, travel and new wetlands. Not because of a vaccine ([9700/12/O/N/25 Q38](#)).
- **Malaria returning** where it had gone: mosquitoes resistant to insecticide, *Plasmodium* resistant to drugs, infected people arriving (migration, war).

- **TB hard to eliminate:** latent carriers, long courses not completed, MDR strains, HIV, overcrowding, poverty, migration. Antigenic variability is **not** a reason for TB (9700/12/M/J/22 Q37).
- **Why a TB course is long:** the bacteria reproduce slowly and are not very sensitive to antibiotics (9700/12/M/J/23 Q38).
- **Features that increase or decrease spread:** symptomless carriers and short-lived vaccine immunity increase it; a pathogen that kills its host quickly decreases it, because a dead host stops spreading it (9700/12/F/M/22 Q37).
- **Measles** (older papers): very infectious, not everyone vaccinated so no herd immunity, babies too young, boosters missed, vaccination disrupted by war.

7. Explain how antibiotic resistance arises (11 questions)

1. **Mutation first:** a random mutation in the bacterial DNA gives a resistance allele. It is not caused by the antibiotic.
2. **Selection:** the antibiotic kills non-resistant bacteria; resistant ones survive.
3. **Reproduction:** the survivors divide and pass on the allele.
4. **Result:** the frequency of the resistance allele in the population increases.

The order in 9700/11/M/J/23 Q37 is mutation → more variation → change in reproductive success → rise in allele frequency.

Variations you will meet:

- **A mechanism is described:** an extra membrane protein that uses ATP is a pump that actively transports the antibiotic out (9700/21/O/N/25 Q4); an enzyme that breaks the antibiotic down; a changed target protein that binds the antibiotic less well (9700/13/M/J/25 Q37). To pump the drug out, the bacterium needs the gene for the carrier protein and a supply of ATP; it has no mitochondria.
- **Why a drug with two targets is less likely to meet resistance** (teixobactin): a single mutation can stop penicillin binding its one protein, but at least two mutations would be needed at once, and targets that are the same in all species are probably essential, so they cannot change.
- **Why resistance to antibiotics evolves more often than escape from vaccines:** antibiotics are used on a huge population of bacteria already in the body, often target a single site (one mutation is enough) and are often misused; a vaccine presents many antigens and memory cells destroy the pathogen before its numbers build up.
- **Data on resistant populations:** at higher antibiotic concentrations, fewer non-resistant bacteria survive, and the resistant fraction of the population is larger.
- **Words:** bacteria become **resistant**, never "immune". The **bacteria** are resistant, not the person.

8. Steps to reduce antibiotic resistance, or its consequences (11 questions)

1. **Steps:** give distinct ones, each a clear action (see the list in "Understand it"): only when needed, identify the pathogen and use a specific antibiotic, finish the course (DOTS), combinations of drugs with different modes of action, less use in farming, prevent infection (vaccines, hygiene, isolation), monitor, develop new

drugs.

2. **Consequences:** can't treat the infection, longer illness, more spread, more deaths, costlier drugs, resistance passed to other bacteria, need for new antibiotics.

Variations you will meet:

- **MCQs on good practice:** sharing antibiotics, treating viral infections and giving antibiotics to healthy animals all make resistance **worse**; vaccination programmes make it better (**9700/11/M/J/25 Q38**). A broad-spectrum antibiotic given for a virus, a broad-spectrum antibiotic for TB and a narrow-spectrum antibiotic for sick cattle can all select for resistance (**9700/12/F/M/21 Q38**).
- **Research data** (for example a second drug that stops TB bacteria pumping rifampicin out): the antibiotic stays in the cell and kills faster, so the course is shorter, more people finish it, and there is less time for resistance to arise.
- **Preventive antibiotics** for people with HIV and latent TB cut TB cases, but the disadvantage is a greater risk of selecting resistant bacteria.
- **Words:** "stronger" or "weaker" doses earn nothing. Say "the correct dose".

9. Explain why HIV/AIDS raises the risk of TB and other infections (7 questions)

1. HIV destroys **T-helper cells**.
2. So less cytokine is released, and fewer B-lymphocytes become plasma cells, so fewer antibodies; fewer T-killer cells are activated; macrophages are less active; fewer memory cells.
3. So the person cannot fight off *M. tuberculosis*: TB is an **opportunistic infection**, and latent TB becomes active.
4. More active TB cases spread TB to others, so a rise in HIV is followed by a rise in TB cases.

Variations you will meet: why ART reduces deaths from cholera, TB and malaria (it keeps T-helper cell numbers up, so the immune system still works); why antibiotics are prescribed to someone with HIV/AIDS (for bacterial infections they are prone to, not for HIV); in MCQs, HIV followed by TB is the classic comorbidity (**9700/12/O/N/21 Q38**).

Worked examples

The bold codes show where marks are earned, as in Cambridge mark schemes. The number is how many marks.

- **M1** method mark: for a correct method, even if the numbers slip.
 - **A1** accuracy mark: for a correct answer; it needs the M mark before it.
 - **B1** mark for a correct result on its own, with no method needed.
-

Example 1

Tuberculosis and cholera are both caused by bacteria.

- (a) Name the species of bacterium that causes cholera. **[1]**
 - (b) Explain how tuberculosis is transmitted from an infected person to an uninfected person. **[2]**
 - (c) Explain why a person who has cholera for a shorter time is less likely to pass it on. **[2]**
- (a)** *Vibrio cholerae*, spelled correctly. **B1**
- (b)** The infected person coughs or sneezes, releasing airborne droplets that contain *Mycobacterium tuberculosis*. **B1** The uninfected person breathes in the droplets; this is droplet (aerosol) infection. **B1**
- (c)** The person's faeces contain *V. cholerae* for as long as they are ill. **B1** A shorter illness means fewer bacteria released into sewage or water, so there is less chance that water or food is contaminated and swallowed by someone else. **B1**

Example 2

A child with a sore throat caused by a virus asks her doctor for penicillin.

- (a) Outline how penicillin kills bacteria. **[3]**
 - (b) Explain why penicillin would not help the child. **[2]**
 - (c) Suggest why the doctor should not prescribe it anyway, "just in case". **[2]**
- (a)** Penicillin inhibits the transpeptidase enzymes that form cross-links between peptidoglycan chains in a growing bacterial cell wall. **B1** The wall is weakened, **B1** so water enters the bacterium by osmosis and the cell bursts. **B1**
- (b)** A virus has no cell wall and no peptidoglycan, **B1** and no transpeptidase or metabolism of its own: it reproduces inside the host's cells, so there is nothing for penicillin to act on. **B1**
- (c)** Using an antibiotic when it is not needed puts a selection pressure on the bacteria living in the child's body. **B1** Any bacterium with a resistance mutation survives and multiplies, so resistant bacteria become more common and the antibiotic is less effective when it is really needed. **B1**

Example 3

After a flood, a camp of 12 000 people had 1 240 cases of cholera and 42 deaths in its first month. In the second month, after chlorinated water and latrines were provided, there were 310 cases and 4 deaths.

- (a) Calculate the percentage change in the number of cases from the first month to the second. **[2]**
- (b) The case-fatality rate is the number of deaths as a percentage of the number of cases. Calculate it for each month, to 1 decimal place. **[2]**

(c) Explain why chlorinated water and latrines reduced the number of cases. [3]

(a)

$$\frac{310 - 1240}{1240} \times 100 = -75.0\%$$

M1 for the formula with the right numbers, **A1** for -75% (a decrease of 75%).

(b) First month: $\frac{42}{1240} \times 100 = 3.4\%$. Second month: $\frac{4}{310} \times 100 = 1.3\%$. **M1 A1**

(c) Cholera is spread by the faecal-oral route: *V. cholerae* in faeces contaminates water that others drink. **B1** Chlorine kills the bacteria in the drinking water. **B1** Latrines keep faeces away from the water supply, breaking the transmission cycle. **B1**

Example 4

In one hospital, 4% of samples of a species of bacterium were resistant to an antibiotic in 2014 and 18% in 2024. The antibiotic was widely prescribed over this period.

(a) Calculate the percentage increase in the proportion of resistant samples. [2]

(b) Explain how the proportion of resistant bacteria increased. [4]

(c) Suggest two steps the hospital could take to slow the rise. [2]

(a)

$$\frac{18 - 4}{4} \times 100 = 350\%$$

M1 A1. The answer is not 14% : that is the difference in percentage points.

(b) A random mutation in the DNA of a bacterium produced an allele for resistance. **B1** When the antibiotic was used, non-resistant bacteria were killed but resistant ones survived: the antibiotic was a selection pressure. **B1** The survivors reproduced and passed the resistance allele to their offspring. **B1** So over many generations the frequency of the allele in the population increased; widespread prescribing made the selection happen more often. **B1**

(c) Any two, for example: identify the pathogen and prescribe a specific antibiotic only when it is needed; make sure patients complete the course; use two antibiotics with different modes of action together; isolate patients with resistant infections and improve hand hygiene so fewer infections need treating. **B1 B1**

Common mistakes

- **Misspelling the pathogens.** Examiners see *Myobacterium*, *Microbacterium*, *Vibro* and *cholera* (for *cholerae*) every year, and a misspelled name gets no mark. Learn the four binomials letter by letter.

- **Calling the wrong thing the pathogen.** Malaria is the disease and *Plasmodium* the pathogen; TB is the disease and *M. tuberculosis* the bacterium; the mosquito is the vector. *V. cholerae* is a bacterium, not a virus: read the stem, which often says so.
- **Wrong transmission route.** Cholera is not caught from airborne droplets or by touching someone. TB is not caught from water. "Infected droplets" or "air droplets" is too vague: say droplets **containing** the bacteria, breathed out and breathed in.
- **Forgetting "female" and the vector** in malaria. Only the female *Anopheles* feeds on blood; say she takes blood from an infected person and injects *Plasmodium* with saliva into an uninfected one.
- **Antibody versus antibiotic.** Antibodies are proteins made by plasma cells; antibiotics are drugs that act on bacteria. Examiners report this mix-up every session.
- **"The bacteria become immune."** Bacteria become **resistant**; immunity is what a person has. And it is the bacteria that are resistant, not the patient or the disease.
- **Saying the antibiotic causes the mutation.** Mutations are random and happen anyway; the antibiotic only selects the resistant bacteria already there.
- **Saying penicillin "makes holes" in the wall or digests it.** It stops the enzyme that forms cross-links, and only in growing bacteria.
- **Vague control measures.** "Better hygiene" or "cleaner water" earns little. Name the action (wash hands with soap after using the toilet) and link it to the route it breaks.
- **Percentage change errors.** Divide by the **original** value, and don't confuse a change in percentage points with a percentage change.

How to get the marks

- **"State" or "name":** one precise word or phrase, spelled right. A binomial needs both names.
- **"Describe" transmission:** both ends of the route, how the pathogen leaves one person and enters the next, with the proper term (faecal-oral, droplet infection, vector).
- **"Explain":** every point needs a "so" or "because" that links it to the biology. For control measures, say which step of transmission each one breaks.
- **"Suggest":** the context is unfamiliar. Apply what you know about the route or the pathogen type; any sensible, linked idea can score.
- **"Discuss" factors:** give several different factors, from biological, social and economic, each explained. Marks are for range, so don't write three sentences about the same idea.
- **Data:** quote figures with units and years; show working for every calculation, because a correct method earns a mark even if the answer slips.
- **Order of events** (penicillin, resistance): write the steps in the right order; a mark scheme often needs the sequence (mutation before selection; no cross-links before osmosis before bursting).
- **Tick tables** for pathogens and transmission: fill every box, as the question says.

Quick check

1. Give the name and type of the pathogen that causes malaria, and explain why the mosquito is called a vector.
2. Explain why antibiotics that bind to 70S ribosomes can treat cholera but not HIV/AIDS.
3. Cases of TB in a region fell from 8 400 in one year to 5 250 ten years later. Calculate the percentage change.
4. (a) In an outbreak, 18 of 1 500 people with cholera died. Calculate the case-fatality rate. (b) A health programme predicts 3 600 new TB cases next year. Giving ART to people with HIV is expected to prevent 25% of them. How many cases would still be expected?
5. Explain why a rise in the number of people with HIV can lead to a rise in cases of TB.

Answers

1. *Plasmodium* (for example *P. falciparum*), a protoctist. The female *Anopheles* mosquito carries *Plasmodium* from an infected person to an uninfected person when it feeds on blood, but it does not cause the disease itself, so it is a vector.
2. *V. cholerae* is a bacterium with 70S ribosomes, so the antibiotic binds and stops translation; the bacterium cannot make proteins and cannot grow. HIV is a virus: it has no ribosomes of its own and is made by the host cell's 80S ribosomes, which the antibiotic does not bind.
3. $\frac{5250-8400}{8400} \times 100 = -37.5\%$, a decrease of 37.5%.
4. (a) $\frac{18}{1500} \times 100 = 1.2\%$. (b) 25% of 3 600 is 900 cases prevented, so $3\,600 - 900 = 2\,700$ cases would still be expected.
5. HIV destroys T-helper cells, so less cytokine is released, fewer B-lymphocytes become plasma cells making antibodies, and T-killer cells and macrophages are less active. So the person cannot fight off *M. tuberculosis* (an opportunistic infection) and latent TB becomes active. Each new active case spreads TB to others by droplet infection.

Past questions to try

- [9700/21/M/J/24 Q3](#): a flow chart of pathogens and transmission, a drug against HIV, and HIV control.
- [9700/21/M/J/21 Q5](#): malaria transmission, test strips and the global distribution of malaria.
- [9700/21/O/N/21 Q3](#): cholera transmission and the consequences of antibiotic resistance.
- [9700/23/O/N/22 Q2](#): TB data, a percentage change and why TB is hard to control.