Biology Units 3 & 4

Chapter 9 — Genetic changes in populations over time

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Chapter 9 Genetic changes in populations over time — 9A Mutations

Theory

A **population** is a group of individuals of the same species living in the same place at the same time, and its **gene pool** is the total collection of alleles carried by all of its members. Chapter 9 is about how that gene pool changes over time. Before any of those changes can happen, though, the alleles have to come from somewhere — and there is only one way a genuinely new one can appear. This subtopic is about that one way: **mutation**.

What a mutation is. A **mutation** is a permanent change in the base sequence of an organism's DNA, or a change in the structure or number of its chromosomes. Cells copy DNA with extraordinary accuracy, but not perfectly, and DNA can also be damaged. When a change is not corrected before the cell divides, it is copied into the daughter cells and becomes permanent.

It matters that you use the right word for the result. A **gene** is a length of DNA at a particular position (locus) on a chromosome that codes for a polypeptide. An **allele** is one of the alternative versions of that gene. A mutation in a gene therefore produces a **new allele** — a new version of a gene that already existed — not a brand-new gene.

Mutation is the ultimate source of new alleles. Every allele in every gene pool on Earth traces back to a mutation at some point in the past. Nothing else can make one:

- **Meiosis and fertilisation** (crossing over, independent assortment, and which gamete meets which) produce enormous variation, but only by shuffling **existing** alleles into new **combinations**. A new combination is not a new allele.
- **Natural selection, genetic drift and gene flow** (9B) change how **common** existing alleles are in a gene pool. They can make an allele rarer or more common, or move it from one population to another, but they cannot invent one.

For that reason mutation is described as the **ultimate source of all genetic variation** — the raw material that every other process works on. If mutation stopped, no new allele would ever appear again.

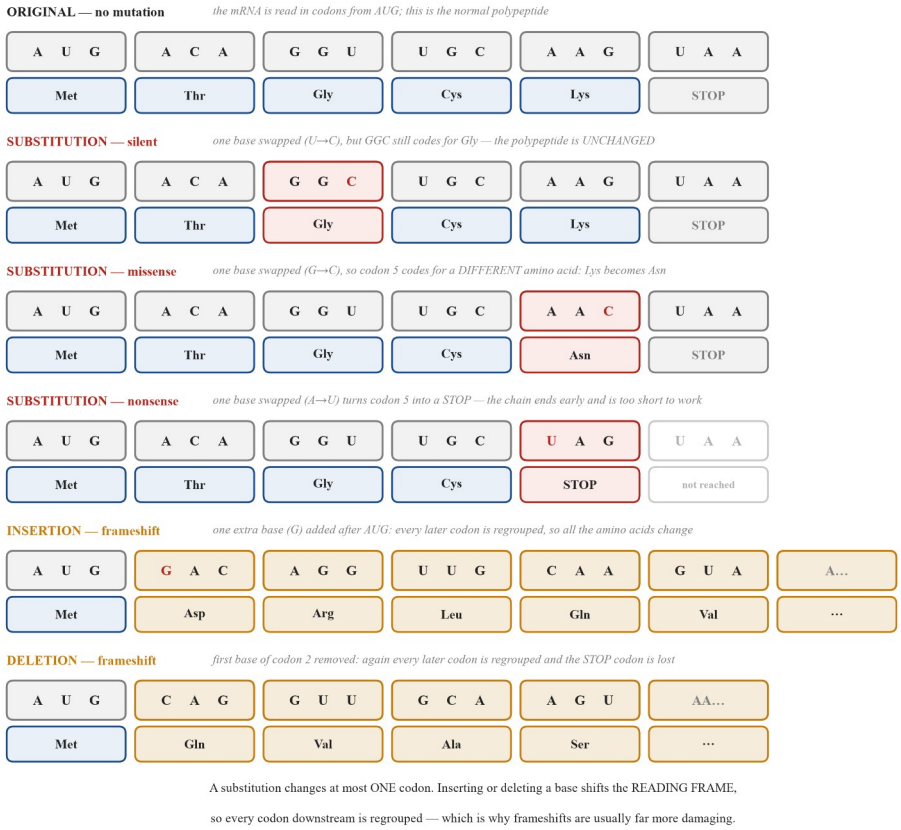
Point mutations. A **point mutation** is a change involving a single nucleotide. The most common kind is a **substitution**, in which one base is replaced by a different base. If the substitution falls inside the coding part of a gene, there are three possible outcomes for the polypeptide:

- **Silent** — the new codon still specifies the **same amino acid**, so the polypeptide is completely unchanged. This is possible because the genetic code is degenerate (2B): most amino acids have more than one codon, and those codons usually differ only in the third base.
- **Missense** — the new codon specifies a **different amino acid**, so one amino acid in the polypeptide is replaced. How much this matters depends on **where** the change falls. A missense change in the active site of an enzyme, or at a position that holds the molecule in shape, can destroy the protein's function; the same kind of change elsewhere in the chain may make almost no difference.
- **Nonsense** — the new codon is a **stop codon**. Translation ends early and a **truncated (shortened)** polypeptide is released. Because a large part of the protein is simply missing, a nonsense mutation almost always produces a non-functional product.

Insertions, deletions and the reading frame. An **insertion** adds one or more nucleotides to the sequence; a **deletion** removes one or more. The consequences depend entirely on **how many**.

The ribosome reads the mRNA in non-overlapping groups of three, starting at AUG — this grouping is the **reading frame**. Add or remove a number of bases that is **not a multiple of three** and every base after the change is pushed into a different group. This is a **frameshift**: **every codon downstream of the mutation is regrouped**, so nearly all of the amino acids after that point are different, and stop codons are commonly lost or created in the wrong place. The protein made is usually the wrong length and completely non-functional. This is why a frameshift is normally far more damaging than a substitution, which can change **at most one codon**.

If the number of bases inserted or deleted **is** a multiple of three, the reading frame is preserved. Whole codons are gained or lost, so the effect is limited to adding or removing whole amino acids — the rest of the polypeptide is read normally.



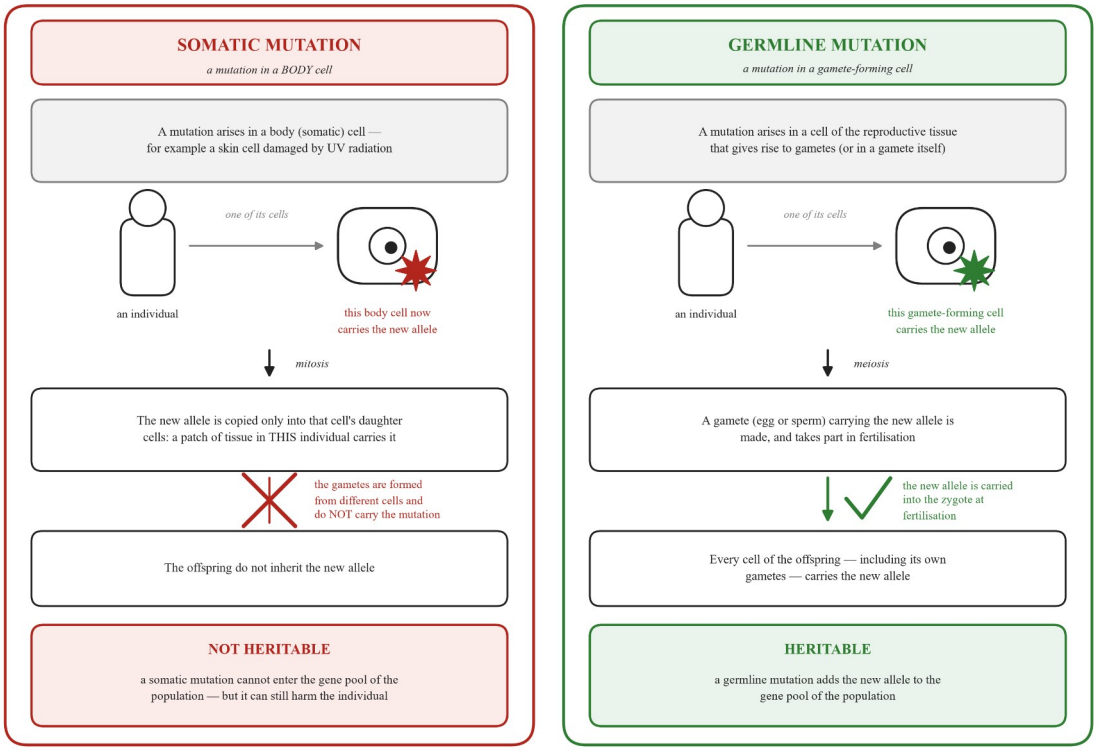
Block (chromosomal) mutations. Not all mutations are small. A **block mutation** changes a whole segment of a chromosome, and so can affect **many genes at once**:

- **Duplication** — a segment is repeated, so the genes on it are present in extra copies.
- **Deletion** — a segment is lost, taking every gene on it with it.
- **Inversion** — a segment breaks out, turns around, and rejoins in the reverse orientation, so the genes on it are in the opposite order.
- **Translocation** — a segment breaks off one chromosome and joins a different, non-homologous chromosome.

Because a block mutation moves, loses or duplicates a stretch of DNA carrying many genes, its effects are usually much larger than those of a point mutation.

Somatic and germline mutations. *Where* in the body a mutation happens decides whether it can ever be inherited — and therefore whether it can enter the gene pool at all.

- A **somatic mutation** occurs in a **body cell**. Mitosis copies it into that cell's daughter cells, so a patch of tissue in that one individual carries the new allele. It **cannot be passed to offspring** and **cannot enter the population's gene pool**. That does not make it unimportant: a somatic mutation in a gene that controls cell division can lead to a tumour, and a somatic mutation can affect the individual's health for life.
- A **germline mutation** occurs in a **gamete**, or in a cell of the reproductive tissue that gives rise to gametes. If that gamete takes part in fertilisation, the new allele is present in the zygote and therefore in **every cell of the offspring**, including that offspring's own gametes. Germline mutations are **heritable**, and they are the **only** mutations that can add a new allele to a population's gene pool.



Causes of mutation. Mutations arise in two ways.

- **Spontaneous mutations** are errors made during **DNA replication**. The enzymes that copy DNA very occasionally pair the wrong base, or slip and add or omit one, and not every error is repaired before the next division. Spontaneous mutations happen at a low rate in **every** organism, even one that has never met a mutagen. The rate per gene per generation is tiny, but genomes contain many thousands of genes and populations contain many individuals, so new alleles are appearing all the time.
- **Induced mutations** are caused by a **mutagen** — any agent that increases the rate at which mutations occur. Mutagens include:
    - **Ionising radiation** (X-rays, gamma rays), which carries enough energy to break DNA strands and alter bases;
    - **Ultraviolet (UV) radiation**, which causes adjacent bases in the same strand to bond abnormally to each other, distorting the molecule so that it is copied incorrectly;
    - **Chemical mutagens** — for example reactive compounds in tobacco smoke and certain industrial chemicals, which react with bases so that they pair wrongly, or slip in between the bases so that extra bases are added or lost at the next replication.

A mutagen raises the **rate** of mutation, but it does not choose which gene is hit or what the new allele will do. Mutations are **random** with respect to the needs of the organism: an organism under stress does not somehow produce the mutation that would help it.

Effects of a new allele. Once a new allele exists, its effect on the organism can be:

- **Harmful** — it lowers the organism's chance of surviving and reproducing (for example a nonsense mutation that destroys an essential enzyme).
- **Neutral** — it has no measurable effect on the phenotype. Most mutations are neutral, because most of a eukaryotic genome does **not** code for protein, and because degeneracy makes many substitutions in coding DNA silent.
- **Beneficial** — it raises the organism's chance of surviving and reproducing.

The critical point is that **an allele is not harmful or beneficial in itself — it is harmful or beneficial in a particular environment**. The same new allele can improve survival in one place and reduce it in another, and if the environment

changes, an allele that was a disadvantage can become an advantage (or the reverse). Describing a mutation as "good" or "bad" without naming the environment is meaningless.

Why this matters for evolution. A population can only change over time if there is variation to change. Mutation is the only process that puts genuinely new variation into a gene pool; everything else in Chapter 9 redistributes what mutation has already supplied. A population that carries a large store of variation is therefore far better placed to cope when its environment changes.

Codon extract (for reference). The full genetic code was covered in 2B. The codons used in this subtopic are listed here so you can check any sequence quickly.

Codon	Amino acid	Codon	Amino acid	Codon	Amino acid
AAA	Lys	CAG	Gln	GUU	Val
AAC	Asn	CAU	His	UCA	Ser
AAG	Lys	CCA	Pro	UGC	Cys
AAU	Asn	CGU	Arg	UGG	Trp
ACA	Thr	CUA	Leu	UUA	Leu
ACU	Thr	GAA	Glu	UUC	Phe
AGA	Arg	GAC	Asp	UUG	Leu
AGG	Arg	GAU	Asp	UUU	Phe
AGU	Ser	GCA	Ala	UAA	STOP
AUG	Met (start)	GGA	Gly	UAG	STOP
AUU	Ile	GGC	Gly	UGA	STOP
CAA	Gln	GGU	Gly		
CAC	His	GUA	Val		

Worked examples

Example 1 — scaffolded

Part of an mRNA reads **5′-AUG-UCA-CAU-GGA-UUC-UAA-3′**. A single base substitution changes the fourth codon from **GGA** to **UGA**. Name the type of mutation and describe its effect on the polypeptide.

Working	Comment
Read the original codons: AUG = Met, UCA = Ser, CAU = His, GGA = Gly, UUC = Phe, UAA = stop. The normal polypeptide is **Met – Ser – His – Gly – Phe** (5 amino acids).	Always establish the normal product first — the effect of a mutation is a comparison.
Only **one base** has changed (the first base of codon 4, G → U), so this is a **point mutation**, specifically a **substitution**.	Name the change at the DNA/mRNA level before naming its consequence.
The new codon **UGA is a stop codon**, so it codes for no amino acid and translation ends there. This makes it a **nonsense** mutation.	Silent / missense / nonsense describe the *consequence* of a substitution, not the change itself.
The polypeptide released is **Met – Ser – His** — only 3 amino acids instead of 5. It is **truncated**.	State the actual new product, not just "it is shorter".
Because a large part of the chain is missing, the protein will not fold into its normal shape and is very likely to be **non-functional**.	Link back to the organism: a missing protein region usually means lost function.

Example 2 — scaffolded

Two mutations occur in the same female mouse. Mutation P arises in a cell of her liver. Mutation Q arises in a cell of her ovary that later gives rise to an egg. For each mutation, state whether her offspring could inherit it and whether it could enter the gene pool of the mouse population.

Working	Comment
Mutation P is in a **liver cell**, which is a body cell, so P is a **somatic** mutation.	Classify by the *type of cell*, not by how serious the effect is.
Mitosis copies P only into the daughter cells of that liver cell, so it is confined to a **patch of liver tissue in this mouse**. Her gametes are produced by different cells and do not carry it.	The mechanism (mitosis only) is what limits a somatic mutation to one individual.
∴ P **cannot be inherited** by her offspring and **cannot enter the gene pool**.	Answer both halves of the question for each mutation.
Mutation Q is in an **ovary cell that gives rise to an egg**, so Q is a **germline** mutation.	Reproductive tissue that forms gametes = germline.
If that egg is fertilised, Q is present in the **zygote** and therefore in **every cell of the offspring**, including its own gametes.	This is why germline mutations are heritable — the zygote is the ancestor of every cell.
∴ Q **can be inherited**, and because it is now carried by a member of the population it **has entered the gene pool**.	Only germline mutations add new alleles to a gene pool.

Example 3 — unscaffolded

Explain why the insertion of a single nucleotide into the coding sequence of a gene usually damages the protein far more than the substitution of a single nucleotide.

The mRNA is read by the ribosome in **non-overlapping groups of three bases**, starting from the start codon AUG; this grouping is the **reading frame**. A **substitution** replaces one base with another without changing the number of bases, so the grouping is untouched and **only the one codon containing the substituted base is altered**. At worst, one amino acid changes (missense) or one premature stop appears (nonsense) — and if the code's degeneracy means the new codon specifies the same amino acid, the protein is not changed at all (silent).

An **insertion of one nucleotide** adds a base to the sequence, so every base after the insertion is pushed one place along and falls into a **different codon**. This is a **frameshift**: **every codon downstream of the insertion is regrouped**, so nearly all of the amino acids from that point on are wrong, and the original stop codon is usually destroyed (or a new stop created too early). The result is a polypeptide of the wrong length with a largely incorrect amino acid sequence, which cannot fold into the normal shape. ∴ a single insertion typically ruins the whole of the protein after the mutation site, whereas a substitution changes at most one codon.

Example 4 — unscaffolded

A mutation in a species of freshwater snail produces a new allele that makes the shell noticeably thicker. Building the thicker shell uses more of the calcium and energy the snail takes in, and snails with the allele grow more slowly. Explain why this new allele cannot simply be described as "beneficial" or "harmful".

Whether a new allele helps or harms depends on the **environment the organism lives in**, because the same feature has different consequences in different conditions. In a pond containing predators that crush snail shells, the thicker shell makes the snail much harder to eat, so snails carrying the allele are more likely to survive and reproduce — here the allele is **beneficial**, and the cost of slower growth is outweighed. In a calcium-poor pond with no shell-crushing predators, the same allele brings no protective advantage but still imposes its costs: those snails grow more slowly and struggle to obtain enough calcium, so they are **less** likely to survive and reproduce — here the allele is **harmful**. In a third pond, with plentiful calcium and no such predators, the extra shell may cost the snail little and gain it little, so the allele is effectively **neutral**.

∴ the allele itself has no fixed value; it is beneficial, neutral or harmful only in relation to a particular environment, and a change in that environment can change which of the three it is.

Practice questions

Scaffolded

Q1. Mutation is described as the ultimate source of all genetic variation. (a) Define the term *mutation*. (b) Explain what is meant by calling mutation the *ultimate source of new alleles*. (c) Meiosis and fertilisation generate huge variation among offspring. Explain why they nevertheless cannot produce a new allele.

Q2. A single base substitution occurs in the coding sequence of a gene. (a) Name the three possible consequences of a substitution for the polypeptide produced. (b) One codon changes from **UUA** to **UUG**. Using the codon extract, state the effect on the polypeptide and name this type of mutation. (c) Explain why a substitution in the **third** base of a codon is often silent.

Q3. Mutations may be spontaneous or induced. (a) Define the term *mutagen*. (b) Name one physical and one chemical mutagen. (c) Explain why mutations still occur in an organism that has never been exposed to a mutagen.

Q4. Part of an mRNA reads **5′-AUG-CCA-GAU-UGG-AAA-UAG-3′**. A mutation **deletes the first base of the third codon** (the G of GAU). (a) Write the sequence of codons that would now be read from AUG. (b) Using the codon extract, give the new sequence of amino acids. (c) Name this type of mutation and state one reason its effect is likely to be severe.

Q5. Some mutations change a whole block of a chromosome. (a) Name the four types of block (chromosomal) mutation. (b) Describe what happens to the chromosome in an **inversion**. (c) Explain why a block mutation usually has a larger effect than a point mutation.

Q6. A new allele may be harmful, neutral or beneficial. (a) State what is meant by describing a new allele as *neutral*. (b) Explain why a silent mutation is usually neutral. (c) Explain why a mutation that does change an amino acid in an enzyme is more likely to be harmful than beneficial.

Unscaffolded

Q7. A student writes: “A mutation creates a new gene.” Explain why this statement is not accurate, making clear the difference between a gene and an allele. *(2 marks)*

Q8. Distinguish between a **missense** mutation and a **nonsense** mutation, with reference to the polypeptide produced in each case. *(2 marks)*

Q9. Compare the likely effect on a polypeptide of inserting **three** nucleotides into a coding sequence with the effect of inserting **one** nucleotide at the same position. *(3 marks)*

Q10. After two unusually dry summers, a gardener notices that some plants in a bed of a native shrub have survived the drought far better than the rest. She concludes that “the drought made those plants produce a mutation for drought tolerance”. Explain why this is not how mutations arise. *(3 marks)*

Q11. A somatic mutation cannot be inherited. Explain why a somatic mutation can nevertheless have serious consequences for the individual in which it occurs. *(2 marks)*

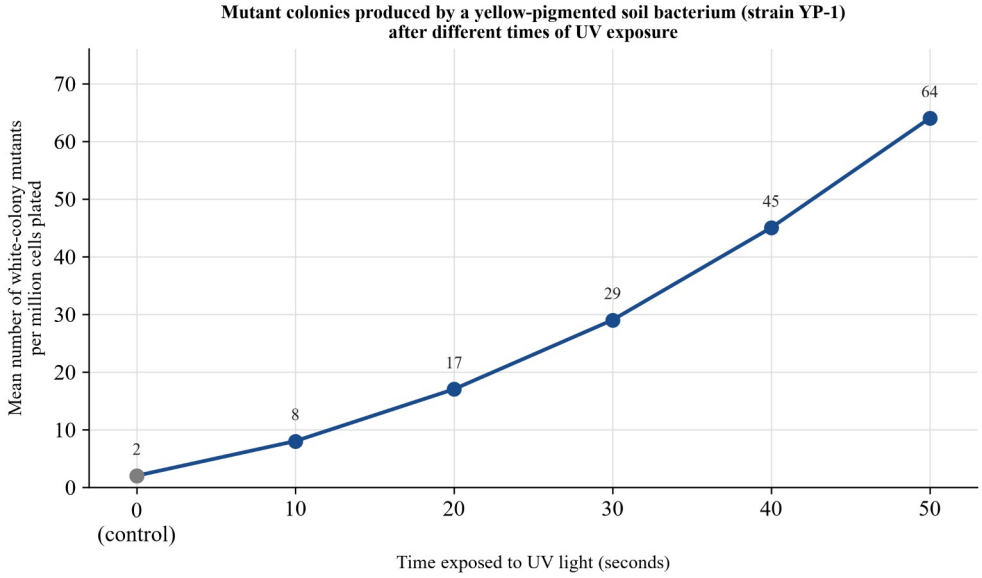
Q12. In an **inversion** no genetic material is lost: every gene that was on the inverted segment is still present on the chromosome. Explain why an inversion can nevertheless leave a cell unable to make one functional protein, and why a block **deletion** of the same segment would usually be far more damaging. *(3 marks)*

Q13. A mutation in an alpine grass produces a new allele that makes plants flower about two weeks earlier than usual. In the population’s present habitat, late spring frosts are common and this allele is harmful. Over the following decades the climate shifts: the late frosts stop, and autumn snow arrives earlier each year, shortening the growing season. Explain what this example shows about describing an allele as “harmful”. *(3 marks)*

Q14. Most of the DNA in a eukaryotic genome does not code for a polypeptide. Suggest why many base substitutions have no effect at all on an organism's phenotype. *(2 marks)*

Q15. Natural selection, genetic drift and gene flow can change how common an allele is in a gene pool, but none of them can produce a new allele. Explain why a population therefore depends on mutation if it is to change over long periods of time. *(3 marks)*

Q16. *(Data interpretation.)* A researcher spread cells of a yellow-pigmented soil bacterium (strain YP-1) onto agar plates and exposed them to a UV lamp for different lengths of time. Cells carrying a mutation in the pigment gene form **white** colonies instead of yellow ones. The mean number of white colonies per million cells plated is shown below. All plates were prepared from the same culture and incubated at 30 °C for 48 hours.



(a) Identify the independent variable.
(b) Identify the dependent variable.
(c) State one variable that must be controlled in this experiment.
(d) Describe the relationship shown by the data.
(e) Explain what the result at 0 seconds shows about the origin of mutations. *(5 marks)*

Q17. *(Stimulus.)* The mRNA transcribed from the normal allele of gene *LUM-1* in a small marine invertebrate begins:

5′-AUG-GAA-UCA-CGU-UUA-GGC-AAA-UGA-3′

Four mutant alleles were sequenced. Each differs from the normal allele by a single change:

Allele	Change to the normal sequence
W	the fifth codon UUA becomes CUA
X	the fourth codon CGU becomes CAU
Y	the third codon UCA becomes UGA
Z	the G at the start of the second codon (GAA) is deleted

(a) Using the codon extract, name the type of mutation in each of alleles W, X, Y and Z.
(b) State which allele produces a polypeptide identical to the normal one, and explain why. *(6 marks)*

Q18. *(Stimulus.)* A child is born with a condition caused by a faulty version of the gene *NRX-4*. Neither parent has the condition. The coding sequence of the gene was determined for all three people.

Person	Coding sequence (mRNA equivalent)
Mother (both alleles)	5′-AUG-CAU-GGA-ACU-UUG-UAA-3′
Father (both alleles)	5′-AUG-CAU-GGA-ACU-UUG-UAA-3′

Person	Coding sequence (mRNA equivalent)
Child (one allele)	5′-AUGCAUGGCAACUUUGUAA-3′

(a) Identify the type of mutation carried by the child's altered allele.

(b) State in which cell this mutation must have arisen for the child to carry it in every cell of the body.

(c) Explain why neither parent has the condition, and state whether the child could pass the new allele on to their own children. *(4 marks)*

Q19. A student writes: "Mutations are always harmful, so a species would be better off if mutations never happened." Evaluate this statement. *(4 marks)*

Q20. *(Synthesis.)* A population of small fish lives in a sunlit surface stream. The enzyme that makes their dark body pigment is coded for by the gene *pig-1*, whose normal mRNA reads:

5′-AUG-UGG-CCA-AGA-CAU-GGU-UAA-3′

In one fish, a mutation occurs in a cell of the ovary that later forms an egg. In the mutated allele the **first base of the fourth codon changes from A to U**. Some descendants of this fish are later washed into a completely dark limestone cave, where a separate population becomes established.

(a) Using the codon extract, give the amino acid sequence coded for by the normal allele and by the mutated allele, and name the type of mutation.

(b) State whether this mutation can enter the gene pool of the fish population, and justify your answer.

(c) Explain why the new allele would be expected to be harmful in the sunlit surface stream but not in the dark cave. *(8 marks)*

Answers

Q1. (a) A permanent change in the base sequence of DNA (or in chromosome structure/number). (b) All new alleles arise by mutation; no other process can create one. (c) They only shuffle existing alleles into new combinations. **Q2.** (a) Silent, missense, nonsense. (b) No change — UUA and UUG both code for leucine; a silent mutation. (c) Because the code is degenerate, codons for the same amino acid usually differ only in the third base. **Q3.** (a) An agent that increases the rate at which mutations occur. (b) Physical: UV or ionising (X-ray/gamma) radiation. Chemical: reactive compounds in tobacco smoke or certain industrial chemicals. (c) Spontaneous errors occur during DNA replication. **Q4.** (a) AUG-CCA-AUU-GGA-AAU-AG… (b) Met – Pro – Ile – Gly – Asn – … (c) A deletion causing a frameshift; every codon after the deletion is regrouped, so nearly all the remaining amino acids are wrong and the stop codon is lost. **Q5.** (a) Duplication, deletion, inversion, translocation. (b) A segment breaks out, is reversed, and rejoins the same chromosome, so the genes on it are in the opposite order. (c) A block mutation alters a whole segment carrying many genes, whereas a point mutation affects one nucleotide in one gene. **Q6.** (a) It has no measurable effect on the phenotype / on survival and reproduction. (b) The new codon codes for the same amino acid, so the polypeptide is unchanged. (c) The existing amino acid sequence has been shaped over a long time to work; a random change is far more likely to disrupt the shape/active site than to improve it. **Q7.** A mutation produces a new **allele** (a new version of an existing gene), not a new gene: a gene is the length of DNA at a locus coding for a polypeptide; an allele is one of its alternative forms. **Q8.** Missense: the changed codon codes for a different amino acid, so one amino acid in the chain is replaced. Nonsense: the changed codon becomes a stop codon, so translation ends early and a truncated polypeptide is made. **Q9.** Three nucleotides = one whole extra codon, so the reading frame is kept and only one extra amino acid is added; one nucleotide shifts the reading frame, so every downstream codon is regrouped and most of the following amino acids are wrong — far more damaging. **Q10.** Mutations arise randomly, from replication errors or mutagens; drought is not a mutagen and no environmental condition can direct which gene mutates or produce the allele an organism "needs". The drought-tolerance allele must already have been present, having arisen earlier by chance. **Q11.** Mitosis copies it into a whole patch of tissue in that individual, and if the affected gene controls an essential protein or cell division the individual can be seriously harmed (e.g. a tumour). **Q12.** The chromosome must break in two places for the segment to be inverted, and a break falling inside a gene interrupts that gene's base sequence, so it no longer codes for a functional polypeptide. A deletion removes the segment altogether, so every gene on it is lost, not merely reordered. **Q13.** The allele has not changed, but the environment has: while late frosts occurred it was harmful (flowers killed before pollination); once frosts stop and the season shortens, early flowering lets seed be set before the snow, so the same allele is now beneficial. An allele is therefore only harmful or beneficial in relation to a particular environment. **Q14.** A substitution in non-coding DNA changes no polypeptide, and in coding DNA degeneracy means many substitutions are silent. **Q15.** Those processes only alter the frequencies of alleles already present; mutation is the only source of new alleles, so without it a gene pool could only lose variation and could not produce new adaptations. **Q16.** (a) Time exposed to UV light. (b) Number of white-colony mutants per million cells plated. (c) Any one of: incubation temperature, incubation time, the bacterial strain/culture, number of cells plated, agar composition. (d) As UV exposure time increases, the number of mutant colonies increases. (e) Mutants appear with no UV at all, showing that mutations also arise spontaneously. **Q17.** (a) W = silent substitution; X = missense substitution; Y = nonsense substitution; Z = deletion causing a frameshift. (b) W — CUA and UUA both code for leucine, so the amino acid sequence is unchanged. **Q18.** (a) A single base insertion causing a frameshift. (b) In a gamete (or a gamete-forming cell) of one parent. (c) Each parent carries only normal alleles, so neither is affected; the child can pass the new allele on because it is present in every cell, including the gametes. **Q19.** Partly true but overstated: many mutations are harmful and some are lethal, but most are neutral and a few are beneficial, and mutation is the only source of new alleles — without it a species could not adapt to a changing environment. Judgement: the statement should be rejected. **Q20.** (a) Normal: Met – Trp – Pro – Arg – His – Gly; mutated: Met – Trp – Pro (AGA becomes UGA, a stop codon); nonsense (point substitution). (b) Yes — it is a germline mutation in a cell forming an egg, so it passes into the zygote and into the population's gene pool. (c) In the sunlit stream the loss of pigment removes camouflage and protection from UV, so it is harmful; in the dark cave pigment gives no advantage and the resources saved may even help, so it is neutral or beneficial.

Fully-worked solutions

Q1. (a) A **mutation** is a **permanent change in the base sequence of an organism's DNA**, or a change in the structure or number of its chromosomes. (b) Calling mutation the **ultimate source of new alleles** means that **every allele that exists arose, at some point, by mutation**. No other process can create a new version of a gene; mutation

supplies the raw material that all the other processes in Chapter 9 then act on. (c) **Crossing over, independent assortment and random fertilisation** produce enormous variation among offspring, but they do so only by **combining alleles that already exist** in new ways. A new **combination** of existing alleles is not a **new allele**: the base sequences themselves are unaltered. Only a change to the base sequence — a mutation — makes a version of the gene that was not there before.

Q2. (a) A substitution may be **silent** (the new codon codes for the same amino acid), **missense** (the new codon codes for a different amino acid) or **nonsense** (the new codon is a stop codon). (b) From the codon extract, **UUA = leucine (Leu)** and **UUG = leucine (Leu)**. The amino acid at that position is therefore **unchanged** and the whole polypeptide is identical — this is a **silent** mutation. (c) The genetic code is **degenerate**: most amino acids are specified by more than one codon, and those codons very often **differ only in the third base**. A substitution in the third base therefore has a good chance of producing another codon for the **same** amino acid, leaving the polypeptide unchanged.

Q3. (a) A **mutagen** is any **agent that increases the rate at which mutations occur** in an organism's DNA. (b) A **physical** mutagen: **ultraviolet (UV) radiation** (or **ionising radiation** such as X-rays or gamma rays). A **chemical** mutagen: reactive compounds in **tobacco smoke** (or certain industrial chemicals). (c) Mutations also arise **spontaneously**, as **errors during DNA replication**. The enzymes that copy DNA occasionally pair the wrong base or slip and add or omit one, and not every error is corrected before the cell divides. These spontaneous mutations occur at a low rate in every organism, so mutations still accumulate with no mutagen present at all.

Q4. (a) The original bases are AUG CCA GAU UGG AAA UAG. Removing the **G** at the start of the third codon leaves AUGCCAAUUGGAAAUAG, which is regrouped from AUG as **AUG-CCA-AUU-GGA-AAU-AG…** (b) From the codon extract: AUG = Met, CCA = Pro, AUU = Ile, GGA = Gly, AAU = Asn. The new sequence is **Met – Pro – Ile – Gly – Asn – …** (compared with the original Met – Pro – Asp – Trp – Lys). (c) This is a **deletion** causing a **frameshift**. It is likely to be severe because removing one base shifts the **reading frame**, so **every codon after the deletion is regrouped**; nearly all of the remaining amino acids are therefore wrong, and the original stop codon UAG has been destroyed, so the chain will also be the wrong length. The protein cannot fold correctly and will almost certainly not function.

Q5. (a) **Duplication, deletion, inversion** and **translocation**. (b) In an **inversion**, a **segment of the chromosome breaks out, turns through 180°, and rejoins the same chromosome in the reverse orientation**, so the genes on that segment are now in the opposite order. (c) A **point mutation** changes a single nucleotide within a single gene, so at most one gene product is affected. A **block mutation** alters a **whole segment of a chromosome**, and such a segment carries **many genes** — so a block mutation can duplicate, lose, reverse or relocate many genes at once, and its effects on the phenotype are correspondingly larger.

Q6. (a) A **neutral** allele is one that has **no measurable effect on the phenotype**, and therefore no effect on the organism's chance of surviving and reproducing. (b) A **silent** mutation produces a codon that specifies the **same amino acid** as the original. The amino acid sequence of the polypeptide is therefore unchanged, so the protein has the same shape and the same function, and the phenotype is unaffected. (c) The amino acid sequence of a working enzyme has been shaped over a very long time so that the molecule folds into a precise shape with a functioning active site. A mutation changes an amino acid at **random**, so it is far more likely to **disrupt** that fit — altering the folding or the active site — than to happen upon a change that improves it. Beneficial changes do occur, but they are much rarer than damaging ones.

Q7. The statement is not accurate because a mutation produces a **new allele**, not a new gene. A **gene** is a length of DNA at a particular locus that codes for a polypeptide; an **allele** is one of the **alternative versions** of that gene. When a mutation alters the base sequence of an existing gene it creates a different version of **that same gene** at the **same locus** — a new allele — rather than adding a new gene to the genome. *(1 mark: mutation produces a new allele, not a new gene; 1 mark: correct distinction between gene and allele.)*

Q8. In a **missense** mutation the substituted base changes a codon into one that codes for a **different amino acid**, so **one amino acid in the polypeptide is replaced** by another; the chain is still full length, and how much function is lost depends on where the change falls. In a **nonsense** mutation the substituted base changes a codon into a **stop codon**, so translation **ends early** and a **truncated (shortened) polypeptide** is released, which is almost always non-functional because a large part of the molecule is missing. *(1 mark for each side of the comparison, both referring to the polypeptide.)*

Q9. The mRNA is read in groups of three from AUG, so the effect of an insertion depends on whether it disturbs that grouping. Inserting **three** nucleotides adds **one whole codon**, so the **reading frame is preserved**: every codon before and after the insertion is grouped exactly as before, and the only change to the polypeptide is that **one extra amino acid is added** at that point. The rest of the sequence is normal, so the protein often still works, at least partly. Inserting **one** nucleotide instead causes a **frameshift**: every base after the insertion falls into a different codon, so **every codon downstream is regrouped**, nearly all the following amino acids are wrong, and the original stop codon is usually lost. ∴ the single insertion is expected to be far more damaging than the three-base insertion. *(1 mark: three bases = one codon, reading frame preserved; 1 mark: only one extra amino acid added; 1 mark: one base shifts the reading frame so all downstream codons are altered.)*

Q10. Mutations arise in only two ways: as **spontaneous errors during DNA replication**, and as **induced** changes caused by a **mutagen** such as ionising or UV radiation or a reactive chemical. Drought is neither — it is not a mutagen, so it does not even raise the mutation rate. More importantly, mutations are **random with respect to the needs of the organism**: even a genuine mutagen only increases how **often** mutations occur; it cannot choose **which** gene is altered or what the new allele will do. There is no mechanism by which an environmental condition can call into existence the particular allele that would be useful in it. The correct interpretation is therefore that the drought-tolerance allele **already existed** in those plants, having arisen at some earlier time by a **random mutation**, long before the dry summers revealed the difference between them. *(1 mark: mutations arise from spontaneous replication errors or mutagens, and drought is not a mutagen; 1 mark: mutations are random with respect to need — no environmental factor directs which mutation occurs; 1 mark: the allele must already have been present, having arisen earlier by chance.)*

Q11. A somatic mutation is copied by **mitosis** into all the daughter cells of the mutated cell, so although it never reaches the gametes it can come to be carried by a **whole patch of tissue** within that individual. If the affected gene codes for an essential protein, or is one that **controls cell division**, those cells may function abnormally or divide uncontrollably, producing a tumour. The consequences for that individual's health can therefore be severe and lifelong, even though the new allele will disappear when the individual dies. *(1 mark: mitosis spreads it through a patch of tissue in the individual; 1 mark: consequence for the individual, e.g. loss of an essential protein or uncontrolled cell division/tumour.)*

Q12. For a segment to be inverted the chromosome must **break in two places**, and there is nothing to stop a break falling **inside a gene**. Where it does, that gene's base sequence is **interrupted**: part of it is now separated from the rest, or joined to sequence it does not belong with, so the gene can no longer be read to make a **functional polypeptide**. The cell has therefore lost a protein even though every gene is still physically present on the chromosome. A **block deletion** of the same segment is usually far more damaging because the segment — and every gene carried on it — is **lost altogether**, so none of those gene products can be made from that chromosome at all; an inversion, by contrast, typically disturbs only the gene or genes at the two **breakpoints**, and leaves all the rest intact, merely in the reverse order. *(1 mark: an inversion requires breaks, and a break inside a gene interrupts its sequence so it no longer codes for a functional polypeptide; 1 mark: a deletion removes the segment, so every gene on it is lost and none of their products can be made; 1 mark: the comparison — a deletion loses many genes at once, whereas an inversion usually affects only those at the breakpoints.)*

Q13. In the **present habitat**, late spring frosts are common, so plants carrying the allele open their flowers while frosts are still occurring; the flowers are killed before they can be pollinated and little seed is set, which is why the allele is **harmful** there. **After the climate shifts**, the frosts no longer occur and the growing season is shorter because autumn snow arrives earlier. Now flowering two weeks early means these plants are pollinated and set seed **before** the snow returns, while normal plants may be caught still flowering — so the very same allele has become **beneficial**. Nothing about the allele itself has changed; only the environment has. ∴ an allele cannot be labelled "harmful" (or "beneficial") in isolation — it is harmful or beneficial only **in relation to a particular environment**, and a change in that environment can reverse its effect. *(1 mark: why it is harmful in the present habitat, referring to the frosts; 1 mark: why it becomes beneficial after the change, referring to the shorter season; 1 mark: the conclusion that the allele is unchanged and its effect depends on the environment.)*

Q14. Most of a eukaryotic genome does **not code for a polypeptide**, so a substitution falling in a non-coding region generally changes **no protein at all** and therefore cannot change the phenotype. In addition, even a substitution that does fall in a coding sequence is often **silent**, because the genetic code is **degenerate** and the new codon frequently specifies the **same amino acid**. Between them, these two effects mean a large proportion of substitutions are **neutral**.

(1 mark: a substitution in non-coding DNA alters no polypeptide; 1 mark: degeneracy makes many coding substitutions silent.)

Q15. Natural selection, genetic drift and gene flow all act on the alleles a population **already has**: they make some alleles more common and others rarer, or move existing alleles between populations. None of them can produce a **base sequence that did not previously exist**, so none of them adds a new allele to the gene pool. **Mutation is the only process that does**, which is why it is called the ultimate source of genetic variation. Over long periods a gene pool acted on only by selection and drift would steadily **lose** variation as alleles were fixed or lost, leaving the population with no new variation to draw on if its environment changed. Continuing mutation replenishes the gene pool and is therefore essential if a population is to keep changing over time. *(1 mark: the other processes only alter the frequencies of existing alleles; 1 mark: mutation is the only source of new alleles; 1 mark: without new alleles a gene pool loses variation and cannot produce new adaptations.)*

Q16. (a) The **independent variable** is the **time for which the cells were exposed to UV light** (the variable the researcher deliberately manipulated — not the individual times chosen). (b) The **dependent variable** is the **mean number of white-colony mutants per million cells plated**. (c) Any one **controlled** variable, for example: the **incubation temperature (30 °C)**, the incubation time (48 hours), the bacterial strain and culture used, the number of cells plated on each plate, or the composition of the agar. (d) **As the time of UV exposure increases, the number of white-colony mutants increases** — from 2 per million with no UV to 64 per million after 50 seconds. (e) At **0 seconds** no UV was applied, yet **2 mutant colonies per million cells** still appeared. This shows that mutations also arise **spontaneously**, from errors in DNA replication, and do not require a mutagen; the UV acts by **increasing the rate** at which they occur, not by being the only cause. *(1 mark each for (a), (b), (c), (d) and (e).)*

Q17. (a) Reading the normal allele: AUG = Met, GAA = Glu, UCA = Ser, CGU = Arg, UUA = Leu, GGC = Gly, AAA = Lys, UGA = stop, giving **Met – Glu – Ser – Arg – Leu – Gly – Lys**.

- **Allele W** — UUA → CUA. Both codons code for **leucine**, so the polypeptide is unchanged: a **silent substitution**.
- **Allele X** — CGU → CAU. Arginine is replaced by **histidine**, so one amino acid changes: a **missense substitution**.
- **Allele Y** — UCA → UGA. UGA is a **stop codon**, so translation ends after only Met – Glu: a **nonsense substitution**.
- **Allele Z** — the G at the start of GAA is **deleted**, giving AUG-AAU-CAC-GUU-UAG…, that is Met – Asn – His – Val followed by a stop. Every codon after the deletion has been regrouped, so this is a **deletion causing a frameshift**.

(b) **Allele W** produces a polypeptide identical to the normal one. The substitution changes the codon from UUA to CUA, but the genetic code is **degenerate** and both of these codons specify **leucine**, so the amino acid at that position — and therefore the entire amino acid sequence — is unchanged. *(a: 4 marks — 1 for each allele correctly named; b: 2 marks — 1 for identifying W, 1 for explaining that both codons code for leucine because the code is degenerate.)*

Q18. (a) Both parents' sequence contains 18 bases; the child's altered allele contains **19**, with an extra **C** after the eighth base. This is a **single base insertion**, which causes a **frameshift**. Regrouping the child's sequence from AUG gives **AUG-CAU-GGC-AAC-UUU-GUA-A…**, that is Met – His – Gly – Asn – Phe – Val – …, compared with the normal Met – His – Gly – Thr – Leu; from the fourth codon onwards every amino acid is different and the stop codon UAA has been lost. (b) For the child to carry the mutation in **every** cell of the body, it must have been present in the **zygote**, so it must have arisen in a **gamete (or in a gamete-forming cell) of one of the parents** — it is a **germline** mutation. (c) Neither parent has the condition because the sequencing shows **both** of each parent's alleles are normal: the new allele was not present in their body cells, only in the single gamete in which it arose, so neither parent's own proteins are affected. The **child**, however, carries the new allele in **every cell, including the cells that will form their gametes**, so the child **can** pass it on to their own children. *(a: 1 mark — single base insertion / frameshift; b: 1 mark — in a gamete or gamete-forming cell of a parent; c: 2 marks — 1 for both parental alleles being normal so neither is affected, 1 for the child carrying it in every cell including the gametes and so being able to pass it on.)*

Q19. The statement contains some truth but is wrong overall. **In its favour:** mutations do occur at random, and because a functioning protein depends on a precise amino acid sequence, a random change is more likely to disrupt it than improve it — nonsense and frameshift mutations in particular almost always destroy the protein, and some

mutations are lethal or cause serious disease. **Against it:** "always harmful" is false. **Most** mutations are in fact **neutral**, because most of the genome does not code for protein and because degeneracy makes many substitutions silent; and a **small proportion are beneficial**, improving an organism's chance of surviving and reproducing in its environment. Whether a given allele is harmful or beneficial also **depends on the environment**, so an allele that is a disadvantage now may be an advantage if conditions change. Most importantly, mutation is the **only** source of new alleles: a species with no mutation would have no new variation entering its gene pool at all, and so could not adapt to any environmental change. ∴ the statement should be **rejected**: mutation carries real costs to individuals, but it is indispensable to a species. *(1 mark: a supported point in favour — many mutations are harmful and why; 1 mark: most mutations are neutral; 1 mark: some are beneficial / the effect depends on the environment; 1 mark: judgement, tied to mutation being the only source of new alleles.)*

Q20. (a) Reading the normal allele from the codon extract: AUG = Met, UGG = Trp, CCA = Pro, AGA = Arg, CAU = His, GGU = Gly, UAA = stop, giving **Met – Trp – Pro – Arg – His – Gly**. Changing the first base of the fourth codon from A to U turns **AGA into UGA**, which is a **stop codon**, so translation ends there and the polypeptide is **Met – Trp – Pro**. This is a **point mutation (substitution)** whose consequence is a **nonsense** mutation, producing a truncated enzyme that will not function, so no dark pigment is made.

(b) **Yes.** The mutation arose in a **cell of the ovary that later forms an egg**, so it is a **germline** mutation. When that egg is fertilised the new allele is present in the zygote and therefore in every cell of the offspring, including their gametes; the allele is now carried by members of the population and has **entered the gene pool**, where it can be passed on down the generations. (A mutation in a body cell of the same fish could not do this.)

(c) The value of the allele depends on the **environment**. In the **sunlit surface stream**, dark pigment gives camouflage against predators and shields the fish from ultraviolet light. A fish carrying two copies of the new allele makes no functional pigment enzyme, so it is pale, far more conspicuous to predators and less protected from UV damage — the allele **reduces its chance of surviving and reproducing**, and so is **harmful** there. In the **completely dark cave** there is no light, so pigment provides **no camouflage advantage and no UV protection**; losing it costs the fish nothing, and the resources that would have gone into making pigment can be used elsewhere. In that environment the same allele is **neutral, or even mildly beneficial**. ∴ whether this new allele is harmful or beneficial is determined not by the allele itself but by the environment the population lives in. *(a: 3 marks — 1 normal sequence, 1 mutated sequence, 1 named as a nonsense point substitution; b: 2 marks — 1 germline / arose in a gamete-forming cell, 1 present in every cell of the offspring so it enters the gene pool; c: 3 marks — 1 harmful in the stream with a reason, 1 not harmful/neutral or beneficial in the cave with a reason, 1 conclusion that the effect of an allele depends on the environment.)*

Chapter 9 Genetic changes in populations over time — 9B Evolving and non-evolving populations

Theory

A **population** is a group of individuals of the same species living in the same area and able to interbreed. The **gene pool** of a population is **all of the alleles carried by all of the individuals in that population**, at all of their genes. The gene pool is the pool of genetic material available to the next generation, and it is the gene pool — not any single individual — that changes as a population evolves.

Allele frequency is the proportion of all the copies of a gene in the gene pool that are one particular allele. It is written as a decimal between 0 and 1 (or as a percentage). Because every individual in a diploid species carries **two** copies of each gene, a population of 100 individuals holds 200 copies of that gene in its gene pool. The frequencies of all the alleles of one gene must add to 1.

Calculating an allele frequency. A population of 100 flowering plants is sampled for a gene with two alleles, R and r. The sample contains 45 RR plants, 40 Rr plants and 15 rr plants.

- Total copies of the gene = 2 × 100 = **200**.
- Copies of R = (2 × 45) + 40 = 130, so the frequency of R = 130 ÷ 200 = **0.65**.
- Copies of r = (2 × 15) + 40 = 70, so the frequency of r = 70 ÷ 200 = **0.35**.
- Check: 0.65 + 0.35 = 1.00.

Evolving and non-evolving populations. A population is **evolving** when the allele frequencies in its gene pool **change from generation to generation**. This gradual change in a gene pool over generations is what biologists mean by evolution at the population level. A population is **non-evolving** when its allele frequencies **stay stable over many generations** — the same alleles are present in the same proportions in each new generation.

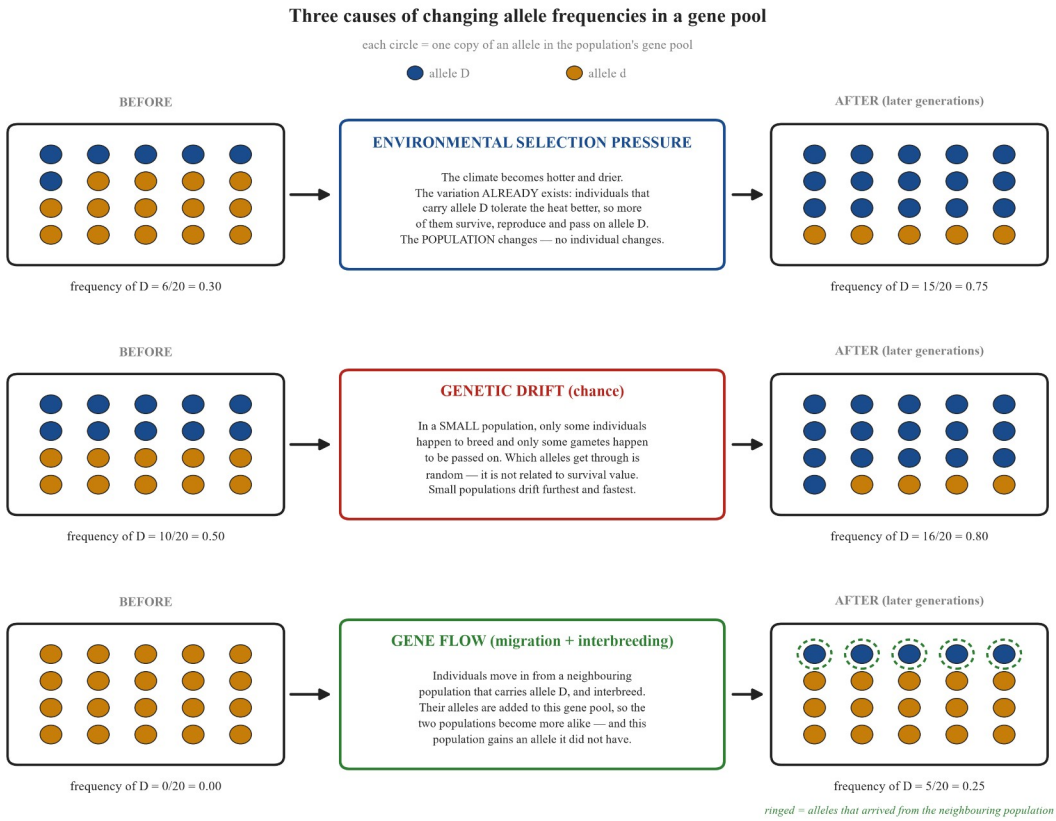
For a population to be non-evolving with respect to a gene, all of the following must apply:

- the population is **very large**, so chance has almost no effect on which alleles are passed on;
- there is **no gene flow** — no individuals (or gametes) move into or out of the population;
- there are **no new mutations** at that gene;
- there is **no selection** — every genotype is equally likely to survive and reproduce.

Real populations almost never meet all four conditions, so allele frequencies usually change at least a little. The conditions matter because each one, when broken, names a **cause of evolution**.

Note what is **not** on the list: **who mates with whom**. Non-random mating — inbreeding, or individuals choosing mates that resemble themselves — changes the way the existing alleles are **combined into genotypes**, producing more homozygotes and fewer heterozygotes. It does not, on its own, change the **frequencies of the alleles** in the gene pool: the same alleles are still there in the same proportions. That is why it is not one of the causes of changing allele frequencies, even though the extra homozygosity it produces matters a great deal for genetic diversity and for the expression of harmful recessive alleles.

The four causes of changing allele frequencies are **mutation** (which supplies new alleles), **environmental selection pressures**, **genetic drift** and **gene flow**. The figure below contrasts the three processes that redistribute alleles already present in a gene pool.



Mutation — the source of new alleles. A mutation is a random change to the DNA base sequence (covered in 9A). A mutation in a gamete-producing cell can be passed to offspring, creating an allele that did not exist in the population before. Mutation is the **only** process that produces genuinely new alleles — selection, drift and gene flow can only change the proportions of alleles that already exist somewhere. Mutation therefore **increases genetic diversity**, but because mutations are rare it changes allele frequencies very slowly on its own. What matters is that mutation supplies the **variation** on which the other processes act.

Environmental selection pressures (natural selection). A **selection pressure** is any environmental factor that affects an organism's chance of surviving and reproducing — a predator, a disease, a drought, a change in temperature, competition for food or mates. Natural selection works like this:

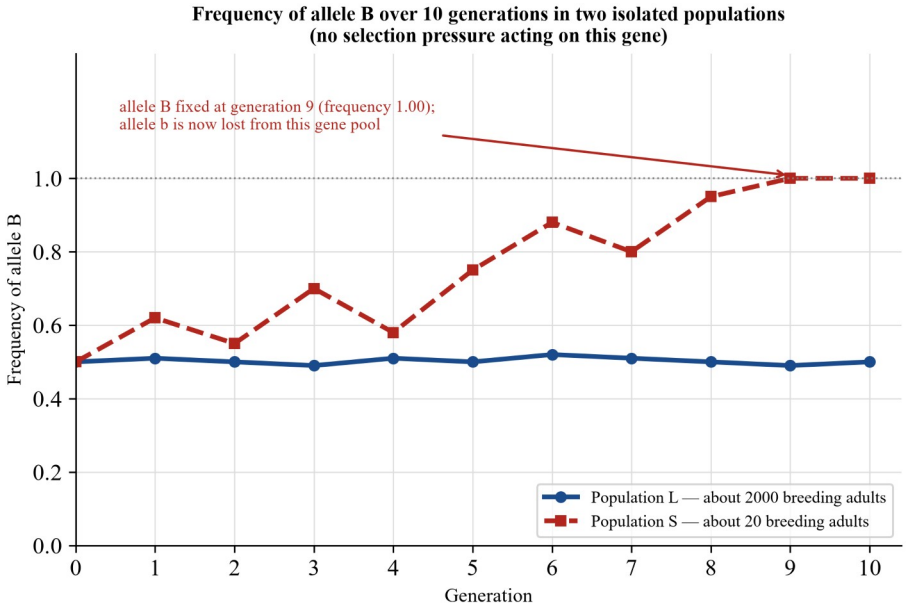
1. **Variation already exists** in the population, produced earlier by mutation and shuffled by sexual reproduction. Different individuals carry different alleles and so have different phenotypes.
2. A **selection pressure** acts on the population. Individuals whose phenotype suits the new conditions are **more likely to survive to reproductive age and to reproduce** (they have higher fitness); individuals with a less suitable phenotype leave fewer offspring.
3. The survivors **pass their alleles** to their offspring, so the alleles for the advantageous phenotype make up a larger share of the next generation's gene pool.
4. Repeated **over many generations**, the frequency of the advantageous allele rises and the frequency of the disadvantageous allele falls. The **population** has changed.

The error examiners see most often. Individuals do **not** adapt, change, develop or acquire a feature because they need it, and a selection pressure does **not** create the variation it acts on. The variation is already there; the environment simply determines which of the existing variants reproduce most. Write about a **change in the proportion of individuals** in the population over generations, never about individuals "becoming" better suited during their lifetimes. Avoid wording such as "the beetles developed a darker colour so they could hide" — write "beetles that already carried the dark allele were less likely to be eaten, so they survived, reproduced and passed the allele on, and the frequency of the dark allele increased over generations".

Genetic drift. Genetic drift is a **random (chance) change in allele frequencies** from one generation to the next. It happens because only some individuals in a population happen to breed, and only some of their gametes happen to be

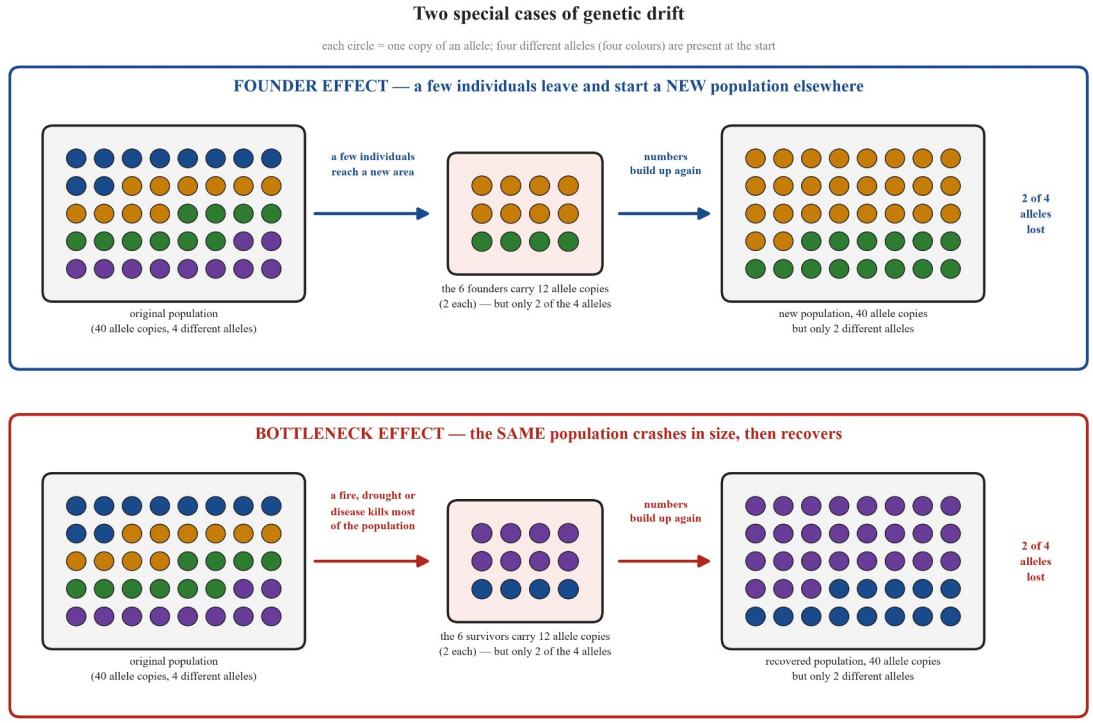
used — so the alleles that get into the next generation are a **sample** of the parents' gene pool, and a sample is rarely a perfect copy. Drift has nothing to do with whether an allele is useful; a beneficial allele can be lost by chance and a neutral or even harmful allele can become common.

Drift matters far more in small populations. In a large population, the chance losses and gains average out, so frequencies stay close to what they were. In a small population, a few deaths or a few lucky breeders change the proportions dramatically — the smaller the sample, the further it can stray. If drift continues, an allele may reach a frequency of 1.00 (it is **fixed**, the only allele left) while its alternative reaches 0 (it is **lost**). Once an allele is lost, only mutation or gene flow can bring it back.



Two situations make drift especially powerful because both suddenly reduce a population to a **small, unrepresentative sample** of the original gene pool:

- **Founder effect** — a **small number of individuals leave and start a new population** in a new place (blown offshore by a storm, rafted to an island, transported by people). The founders carry only some of the alleles of the source population, and in different proportions, so the new population begins with a narrower and different gene pool. The original population continues unchanged.
- **Bottleneck effect** — a **population crashes** (fire, drought, flood, disease, over-harvesting) and only a few individuals survive **in the same place**. Survival is largely a matter of chance, so the survivors are an unrepresentative sample. Even when numbers recover, the recovered population is built from the survivors' alleles only, so the lost alleles do not return.



In BOTH cases a small, unrepresentative sample of the original gene pool founds the next generation, so allele frequencies shift by chance and genetic diversity falls — and stays low even after numbers recover.

Gene flow. Gene flow is the **movement of alleles between populations**. It requires two steps: individuals (or gametes such as pollen and sperm) **move from one population to another**, and they then **interbreed** with the residents so that their alleles enter the new gene pool. Migration without breeding is not gene flow.

Gene flow:

- **changes the allele frequencies of both populations**, and can introduce an allele the receiving population did not have at all;
- **increases the genetic diversity** of the receiving population, and **decreases** the genetic diversity of the population the migrants left if they do not return, because their allele copies leave that gene pool with them;
- makes the two populations **more genetically similar** to each other over time.

If gene flow is **blocked** — by a mountain range, a cleared paddock, a highway or a dam — the two populations no longer exchange alleles. Each is now smaller and isolated, so drift and different local selection pressures push their gene pools further apart.

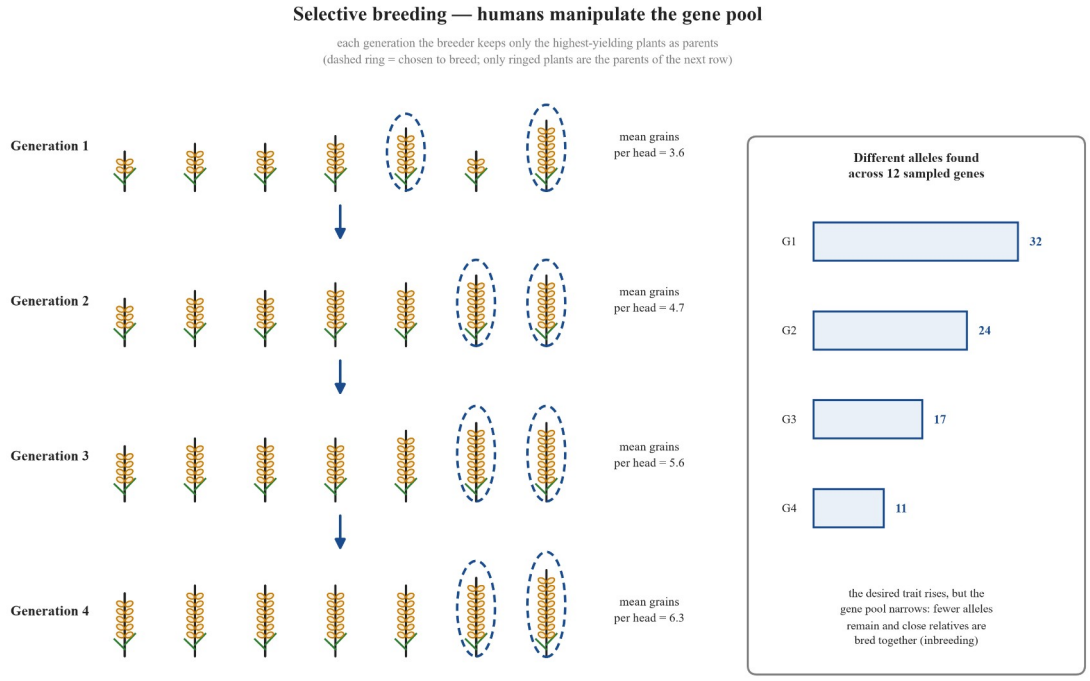
Consequences: increased and decreased genetic diversity. Genetic diversity is the amount of genetic variation in a gene pool — how many different alleles are present and how evenly they occur.

A population with **high genetic diversity** has a better chance of surviving a change in its environment: if a new disease, predator or climate stress appears, it is more likely that **some individuals already carry alleles** that let them survive and reproduce, so the population can change over generations rather than die out. A population with **low genetic diversity** is fragile: most individuals respond to a new selection pressure in the same way, so a single new disease can kill a very large proportion of them; there is little variation for selection to act on; and close relatives are forced to breed together (**inbreeding**), which raises the number of individuals homozygous for harmful recessive alleles. Low diversity is therefore linked to a **higher risk of extinction**.

Process	Effect on the genetic diversity of the population	Why
Mutation	Increases	Creates alleles that did not exist before
Gene flow into a population	Increases	Adds alleles from another gene pool
Genetic drift (including founder and bottleneck effects)	Decreases	Alleles are lost by chance; alleles may become fixed

Process	Effect on the genetic diversity of the population	Why
Strong selection in one direction	Decreases	The alleles for the disfavoured phenotype become rare or are lost
Inbreeding	Decreases (raises homozygosity)	Relatives share alleles, so fewer combinations occur
Gene flow out of a population, with no return	Decreases	Alleles leave the gene pool with the emigrants and are not replaced; rare alleles can be lost altogether

Manipulation of gene pools: selective breeding. In **selective breeding (artificial selection)**, **humans** — not the environment — decide which individuals become parents. A breeder chooses the individuals with the most desirable phenotype (highest milk yield, largest grain, disease-resistant fruit, a particular coat colour), breeds only from them, then repeats the process each generation. The alleles of the chosen parents make up a growing share of the gene pool, so the frequency of the desired alleles rises and the trait becomes more extreme far faster than natural selection would achieve.



The **cost of selective breeding is genetic diversity**. Because only a few individuals are used as parents each generation, many alleles are never passed on and are lost from the gene pool; the breeding population is also small, so drift acts strongly. Repeatedly breeding closely related individuals (inbreeding) increases homozygosity, so harmful recessive alleles are more often expressed — the inherited disorders and reduced fertility seen in some highly bred lines. A crop or breed with a narrow gene pool is also very vulnerable to a **new disease or a change in climate**, because the alleles that might have conferred resistance may no longer be present. Breeders manage this by keeping seed banks and studbooks, and by occasionally out-crossing to unrelated stock — deliberately restoring gene flow.

	Natural selection	Selective breeding (artificial selection)
Who or what selects	An environmental selection pressure	Humans
Which individuals reproduce	Those best suited to the environment	Those with the trait humans want
Speed of change	Usually slow, over many generations	Fast — large changes in few generations
Effect on the gene pool	Frequencies change; diversity may	Frequencies change; diversity

	Natural selection	Selective breeding (artificial selection)
	fall	usually falls sharply

Worked examples

Example 1 — scaffolded

A population of 250 marsh snails is sampled for a shell-thickness gene with two alleles, S and s. The sample contains 90 SS snails, 120 Ss snails and 40 ss snails. Calculate the frequency of each allele in this gene pool.

Working	Comment
Each snail carries **two** copies of the gene, so the gene pool holds 2 × 250 = **500 copies**.	Always start from the total number of allele copies, not the number of individuals.
Copies of S = (2 × 90) + 120 = 180 + 120 = **300**.	Each SS snail contributes two S alleles; each Ss snail contributes one.
Frequency of S = 300 ÷ 500 = **0.60**.	Give the frequency as a decimal (0.60) or 60%.
Copies of s = (2 × 40) + 120 = 80 + 120 = **200**, so frequency of s = 200 ÷ 500 = **0.40**.	Count the homozygous recessive snails twice and the heterozygotes once.
Check: 0.60 + 0.40 = 1.00.	The frequencies of all alleles of one gene must add to 1 — a fast way to catch an arithmetic slip.

Example 2 — scaffolded

In a shallow inland lake, a population of small native fish contains variation in the age at which individuals first breed: some carry allele E and begin breeding early at a small body size, while others carry allele e and breed later at a larger size. A large introduced predator that hunts only the larger fish becomes established in the lake. Over 25 generations the frequency of allele E rises from 0.18 to 0.74. Explain this change.

Working	Comment
Variation in breeding age **already existed** in the population before the predator arrived: both alleles E and e were present, having arisen earlier by mutation.	Establish the pre-existing variation first — this is the step students most often leave out.
The introduced predator is an **environmental selection pressure**. It hunts larger fish, so fish carrying allele e are more likely to be eaten before they breed.	Name the selection pressure explicitly and state which phenotype it acts against.
Fish carrying allele E breed early, at a small size, so more of them **survive to reproduce** and they produce more offspring.	Fitness is about surviving *and* reproducing, not just surviving.
Those offspring **inherit allele E**, so E makes up a larger share of the next generation's gene pool.	The link from "survives" to "allele frequency changes" must be the passing on of alleles.
Repeated **over 25 generations**, the frequency of E rises from 0.18 to 0.74 and e falls from 0.82 to 0.26. The **population** has changed — no individual fish changed its breeding age.	Finish with the population-level statement and the directional change in the data.

Example 3 — unscaffolded

The graph in the theory section shows the frequency of allele B over ten generations in two isolated populations of the same species, L (about 2000 breeding adults) and S (about 20 breeding adults). No selection pressure acts on this gene. Account for the difference between the two populations, and state the consequence for population S.

Both populations start with a frequency of allele B of 0.50, and the changes seen are caused by **genetic drift** — the random sampling of alleles that occurs when only some individuals breed and only some gametes are passed on. In **population L**, the sample of alleles passed to each new generation is very large, so chance gains and losses cancel out

and the frequency stays close to 0.50 (it varies only between 0.49 and 0.52 across the ten generations). In **population S**, only about 20 adults contribute alleles, so each generation's sample is small and can differ greatly from the one before; the frequency swings up and down and drifts steadily upward, reaching **1.00 at generation 9**.

At that point allele B is **fixed** — it is the only allele of this gene left in population S — and allele **b has been lost** from the gene pool. The consequence is a **decrease in genetic diversity**: every individual is now homozygous for B, no variation remains at this gene, and the allele can only be restored by a new mutation or by gene flow from another population. ∴ the same random process produces a trivial change in the large population and complete loss of an allele in the small one.

Example 4 — unscaffolded

Two populations of a native bush-rat live in patches of woodland separated by 3 km of cleared farmland that the rats will not cross. Population W has been isolated for about 60 years and now numbers roughly 40 individuals; population V is much larger and carries several alleles that population W lacks. A revegetated corridor of shrubs is planted to connect the two patches, and rats begin to move along it. Predict the effects on the gene pool of population W, and explain your prediction.

Planting the corridor allows rats to move between the patches and **interbreed with the residents**, which re-establishes **gene flow** between the two populations. Alleles carried by immigrants from population V are added to population W's gene pool, so the **allele frequencies in population W will change**, and alleles that population W did not have at all will now be present. This **increases the genetic diversity** of population W.

Two consequences follow. First, because there is now more variation for selection to act on, population W has a **greater capacity to respond to a future selection pressure** such as a new disease or a hotter, drier climate — it is more likely that some individuals carry alleles that allow them to survive and reproduce. Second, individuals are less likely to breed with close relatives, so **inbreeding is reduced** and fewer offspring will be homozygous for harmful recessive alleles. Over time the two gene pools will also become **more similar to each other**, because gene flow works in both directions. ∴ restoring gene flow raises the genetic diversity of the small population and improves its long-term chance of survival.

Practice questions

Scaffolded

Q1. Define each of the following terms: (a) gene pool; (b) allele frequency; (c) an evolving population.

Q2. In a population of native bees, the frequency of allele T was 0.32 in generation 1 and 0.71 in generation 40. No other allele of this gene exists. (a) State the frequency of the alternative allele t in each of the two generations. (b) State, with justification, whether this population is evolving with respect to this gene. (c) Name two processes that could have produced this change.

Q3. A coastal grassland experiences a run of unusually dry summers. Individuals of one grass species vary in root depth: plants carrying allele F grow deeper roots and reach water further down, while plants carrying allele f grow shallow roots. Over 30 generations the frequency of allele F rises. (a) State the selection pressure acting on this population. (b) Describe what happens to plants that carry allele F when the dry summers begin. (c) Explain why an individual shallow-rooted plant does **not** grow deeper roots in response to the drought, even though deeper roots would help it survive.

Q4. Genetic drift is one cause of changing allele frequencies. (a) Define genetic drift. (b) State two chance events that can cause allele frequencies to drift. (c) Explain what it means for an allele to become fixed, and state the effect of fixation on the genetic diversity of the population.

Q5. A cyclone strikes an island and reduces a possum population from about 900 individuals to 14 survivors. Over the next 40 years the population builds back up to about 800 individuals. (a) Name the process that has acted on this population's gene pool. (b) Explain why the allele frequencies in the 14 survivors are unlikely to match those of the original population of 900. (c) State and explain the effect on genetic diversity once the population has recovered to 800 individuals.

Q6. A plant breeder grows a paddock of an oat variety and each year keeps seed only from the plants with the heaviest grain heads, sowing the next crop from that seed. (a) Name the type of selection the breeder is carrying out. (b) Describe what happens to the frequency of the alleles for heavy grain heads over several generations, and why. (c) State one way this process differs from natural selection.

Unscaffolded

Q7. Every autumn a flock of about 200 lorikeets from a large population in a river red gum forest flies 50 km to an isolated patch of remnant woodland, where a small population of the same species lives. The visitors feed in the remnant patch for three months, then fly back to the forest, where they nest and raise their young. State, with justification, whether gene flow is occurring between the two populations, and explain what effect the annual visits are having on the allele frequencies of the remnant population. *(3 marks)*

Q8. Distinguish between the founder effect and the bottleneck effect, and state one outcome they share. *(3 marks)*

Q9. *(Stimulus.)* For each of the three scenarios below, identify the process that is changing the allele frequencies, and justify your choice with reference to the scenario.

A. A population of native beetles has been surveyed every summer for 40 years. In the most recent survey, one beetle carries a form of a wing-pattern gene that has never been recorded in this species anywhere. The beetle's offspring inherit it, and it is now present in a small number of individuals.

B. Over 30 years the average winter minimum temperature in an alpine valley falls. Shrubs carrying an allele for a thicker leaf cuticle survive frost better and set more seed, and the frequency of that allele rises from 0.20 to 0.61.

C. Each spring, bees carry pollen from an inland population of a wattle to a coastal population 4 km away, and seed is set. Over many years the allele frequencies of the two populations become more similar. *(3 marks)*

Q10. A student wrote: "When the creek became saltier, the small fish living in it developed a tolerance to salt so they could survive. Over time, all the fish became salt-tolerant." Identify **two** biological errors in this statement, and rewrite it as a correct account of natural selection. *(3 marks)*

Q11. A new highway is built through a large woodland, splitting a single population of gliders into two halves that can no longer cross to each other. Predict the genetic consequences for the two populations over many generations, and explain your reasoning. *(4 marks)*

Q12. State what is meant by a non-evolving population, and outline **three** conditions that must apply for a population's allele frequencies to stay stable over many generations. *(4 marks)*

Q13. *(Data interpretation.)* Grasshoppers in a heathland vary in body colour: individuals with at least one copy of allele G are dark, and gg individuals are pale. The grasshoppers are hunted by birds that locate them by sight. A change in fire management leaves the ground blackened for much of the year. Samples of 400 grasshoppers were taken before the change and again 15 generations later.

Genotype	Number before the change	Number 15 generations later
GG	36	144
Gg	168	208
gg	196	48
Total	**400**	**400**

Calculate the frequency of allele G at each sampling, describe the change using the data, and explain the process responsible. *(5 marks)*

Q14. Explain how a selective breeding program increases the frequency of a desired allele in a herd, and explain why the genetic diversity of that herd falls as a result. *(4 marks)*

Q15. Explain why mutation is described as the source of new alleles, and state its effect on the genetic diversity of a gene pool. *(2 marks)*

Q16. *(Data interpretation.)* A population of 30 freshwater crayfish lives in an isolated waterhole. For a gene with two alleles, N and n, the population consists of 24 NN individuals and 6 Nn individuals. A flood then connects the waterhole to a nearby creek, and 20 crayfish from the creek population move into the waterhole, where they stay and breed with the residents. All 20 of the newcomers are nn. (a) Calculate the frequency of allele n in the waterhole population before the flood. (b) Calculate the frequency of allele n in the combined population of 50 crayfish. (c) Explain why the same 20 newcomers would have changed the waterhole's allele frequencies far less if the resident population had numbered 3000 rather than 30, with the same allele frequencies as before. *(4 marks)*

Q17. For each of the following, predict whether the genetic diversity of the population increases or decreases, and give a reason. (a) A mutation occurs in a gamete-producing cell and is passed to offspring. (b) A drought reduces a population from 5000 individuals to 30 individuals. (c) Ten individuals from a genetically different population migrate in and breed with the residents. (d) A strong selection pressure acts in one direction for many generations. *(4 marks)*

Q18. *(Method evaluation.)* To model genetic drift, a class uses two containers of beads, where each bead represents one allele copy. Container X holds 200 beads (100 red, 100 blue) and container Y holds 10 beads (5 red, 5 blue). A student draws one bead at random, records its colour, returns it, and repeats until the number of draws equals the number of beads in that container. The recorded proportions are then used to refill the container, creating the "next generation". This is repeated for ten generations for each container.

Evaluate this activity as a model of genetic drift in a population. *(4 marks)*

Q19. *(Data interpretation.)* A dairy goat herd has been closed to outside animals for many years, with most kids fathered by a small number of prize bucks. The table shows two measures recorded over seven generations.

Generation	Mean number of different alleles per marker gene (10 genes)	Kids born with an inherited recessive disorder (per 100 births)
1	4.6	0.5
3	3.8	1.9
5	3.1	4.2
7	2.4	7.6

Describe the two trends shown, and explain how the breeding practice accounts for both. *(4 marks)*

Q20. *(Synthesis.)* A skink population on a small offshore island was established about 200 years ago when a few individuals rafted across on flood debris from the mainland. The island population has remained isolated and now numbers about 120 adults. Researchers sampled both populations.

Measure	Island population	Mainland population
Frequency of allele M	0.95	0.55
Frequency of allele m	0.05	0.45
Total number of different alleles found across 8 other genes	12	37

(a) Identify the process most likely responsible for the island population's allele frequencies when it was established, and justify your answer using the data.

(b) Explain why the island population is at greater risk than the mainland population if a new fungal disease reaches both.

(c) Predict what is likely to happen to allele m in the island population over the next 50 generations if the population stays small and isolated, and explain your prediction. *(8 marks)*

Answers

Q1. (a) All the alleles carried by all the individuals of a population. (b) The proportion of all copies of a gene in the gene pool that are one particular allele. (c) One whose allele frequencies change from generation to generation. **Q2.** (a) t = 0.68 in generation 1 and 0.29 in generation 40. (b) Yes — the allele frequencies have changed over generations. (c) Any two of: natural selection (an environmental selection pressure), genetic drift, gene flow. **Q3.** (a) The run of dry summers (drought / reduced water availability). (b) They reach deeper water, so more of them survive and reproduce, passing allele F to their offspring. (c) Root depth is determined by the alleles the plant inherited; an individual cannot change its alleles or acquire a new trait because it needs one — only the proportions of individuals in the population change over generations. **Q4.** (a) A random change in allele frequencies from one generation to the next, caused by chance in which alleles are passed on. (b) Any two of: chance deaths of individuals before breeding; only some individuals happening to breed; which gametes happen to be used at fertilisation; a chance event such as a fire or storm. (c) Fixation = the allele reaches a frequency of 1.00 and is the only allele of that gene left; genetic diversity at that gene is lost (the alternative allele is gone). **Q5.** (a) Genetic drift, specifically the bottleneck effect. (b) The 14 survivors are a small sample that survived largely by chance, so their alleles are unlikely to be representative; rare alleles are likely to be missing. (c) Genetic diversity stays low, because the recovered population was rebuilt from only the survivors' alleles — numbers recover but lost alleles do not return. **Q6.** (a) Artificial selection (selective breeding). (b) They increase, because only plants with heavy heads are used as parents, so their alleles make up a growing share of the gene pool each generation. (c) The selecting agent is a human choosing the parents, not an environmental selection pressure (also: it is much faster and aimed at a trait people want, not one that necessarily suits the environment). **Q7.** No — gene flow is not occurring. Movement between populations is not enough; the visitors must also interbreed with the residents so that their alleles enter the remnant population's gene pool, and instead they return to the forest to nest. The visits therefore have no effect on the remnant population's allele frequencies, which continue to change only through drift and local selection. **Q8.** Founder effect = a few individuals leave and start a new population elsewhere; bottleneck effect = the same population crashes in size in place and later recovers; both leave a small, unrepresentative sample of the original gene pool, so allele frequencies shift by chance and genetic diversity falls. **Q9.** A = mutation — an allele that did not exist in the gene pool before has appeared and been inherited, and only mutation can produce a new allele. B = natural selection / an environmental selection pressure — colder winters mean thicker-cuticle plants survive and reproduce more, so the allele increases. C = gene flow — pollen (gametes) moves between populations and fertilisation occurs, so alleles are exchanged and the populations become more similar. **Q10.** Errors: individuals cannot develop a new tolerance because they need it; and the change in salinity did not create the variation — it acted on variation already present (also "all the fish became tolerant" describes individuals changing, not a change in frequency). Correct version: some fish already carried alleles for salt tolerance; when the creek became saltier these fish were more likely to survive and reproduce, passing those alleles to their offspring, so the frequency of the tolerance alleles rose over generations and a greater proportion of the population is now salt-tolerant. **Q11.** Gene flow between the two halves stops; each population is smaller, so genetic drift has a greater effect and alleles are lost or fixed by chance; genetic diversity within each falls (and inbreeding increases); the two gene pools become increasingly different from each other. **Q12.** A non-evolving population is one whose allele frequencies stay the same over generations. Any three of: a very large population; no gene flow; no new mutations at that gene; no selection (all genotypes equally likely to survive and reproduce). **Q13.** Before: G = 0.30. After: G = 0.62 (g falls from 0.70 to 0.38). G increased by 0.32 over 15 generations. Natural selection: on blackened ground the dark (G) grasshoppers are less visible to predators, so more survive and reproduce and pass on G. **Q14.** Only animals with the desired phenotype are bred, so their alleles make up a growing share of the gene pool and the frequency of the desired allele rises each generation; diversity falls because few individuals contribute alleles (many alleles are never passed on) and repeated breeding among relatives increases homozygosity. **Q15.** Mutation is a random change to DNA that produces an allele that did not exist before (the other processes only redistribute existing alleles); it increases the genetic diversity of the gene pool. **Q16.** (a) 6 ÷ 60 = 0.10. (b) 46 ÷ 100 = 0.46. (c) The newcomers' 40 copies of n would be a small part of a combined gene pool of 6040 copies instead of 40 copies out of 100, so n would rise only from 0.10 to about 0.11 — the effect of gene flow depends on the number of migrants relative to the size of the receiving population. **Q17.** (a) Increases — a new allele is added to the gene pool. (b) Decreases — a bottleneck; alleles carried by the individuals that died are lost by chance. (c) Increases — gene flow adds alleles from another gene pool. (d) Decreases — the alleles for the disfavoured phenotype become rare or are lost. **Q18.** Strengths: drawing is random and ignores any survival value, as drift is; comparing the small container with the large one shows that frequencies change far more in the small "population"; repeating over generations shows cumulative change and possible fixation. Limitations: no mutation, gene flow or selection is modelled; the population size never changes; beads are single alleles, so diploid individuals and their pairs of alleles are not represented. Judgement: a valid model

of the random sampling aspect of drift only, and it should not be used to represent evolution as a whole. **Q19.** The mean number of different alleles per gene fell from 4.6 to 2.4, while the incidence of the disorder rose from 0.5 to 7.6 per 100 births. Using few sires in a closed herd means few individuals contribute alleles, so alleles are lost from the gene pool (small population, strong drift and artificial selection); the resulting inbreeding raises homozygosity, so more kids inherit two copies of the harmful recessive allele and show the disorder. **Q20.** (a) The founder effect (genetic drift) — a few individuals founded the population, and their unrepresentative sample of alleles explains M at 0.95 versus 0.55 and only 12 alleles versus 37. (b) Its low diversity means few individuals are likely to carry alleles conferring resistance, so a large proportion could die and the population may not recover, whereas the diverse mainland population is more likely to contain resistant individuals that survive and reproduce. (c) Allele m (frequency 0.05) is likely to be lost and M fixed at 1.00, because in a small isolated population drift causes large random changes and rare alleles are easily lost; with no gene flow, only a new mutation could restore m.

Fully-worked solutions

Q1. (a) The **gene pool** is **all of the alleles present in all of the individuals of a population** at a given time — the genetic material available to the next generation. (b) **Allele frequency** is the **proportion of all copies of a particular gene in the gene pool that are one specific allele**, written as a decimal from 0 to 1 (the frequencies of all alleles of that gene add to 1). (c) An **evolving population** is one in which the **allele frequencies of the gene pool change from generation to generation**; if the frequencies stay stable, the population is non-evolving.

Q2. (a) There are only two alleles, so their frequencies must add to 1. In generation 1, t = 1 − 0.32 = **0.68**; in generation 40, t = 1 − 0.71 = **0.29**. (b) The population **is evolving** with respect to this gene: the frequency of T has risen from 0.32 to 0.71 (and t has fallen correspondingly) **over generations**, and a change in allele frequencies over generations is what evolution of a population means. (c) Any **two** of: **natural selection** — a selection pressure favouring the phenotype produced by T, so those bees survive and reproduce more; **genetic drift** — chance changes in which alleles are passed on, especially if the colony is small; **gene flow** — bees carrying T moving in from another population and interbreeding.

Q3. (a) The selection pressure is the **run of dry summers** — reduced water availability in the soil. (b) When the dry summers begin, plants carrying allele F **already have deeper roots**, so they can still reach water. They are **more likely to survive and to set seed**, and they **pass allele F to their offspring**, so F becomes more common in the next generation's gene pool. (c) A plant's root depth is determined by the **alleles it inherited**; an individual **cannot acquire a new allele or a new trait because it needs one**, and a selection pressure does not create the variation it acts on. The drought simply determines **which of the existing plants reproduce**. Change happens to the **population** across generations (a rising proportion of deep-rooted plants), not to the individual.

Q4. (a) **Genetic drift** is a **random change in the allele frequencies** of a population from one generation to the next, caused by chance in which alleles happen to be passed on rather than by any survival advantage. (b) Any **two**: individuals dying by chance before they breed (a storm, fire or flood); only some individuals happening to find a mate and breed; which gametes happen to be used at fertilisation; a small number of individuals happening to found or survive in a location. (c) An allele is **fixed** when its frequency reaches **1.00** — it is the **only allele of that gene left** in the population, and every individual is homozygous for it. All other alleles of that gene have been **lost** (frequency 0), so the **genetic diversity at that gene is gone** and can be restored only by mutation or gene flow.

Q5. (a) **Genetic drift**, specifically the **bottleneck effect**. (b) The 14 survivors are a very **small sample** of the original 900, and they survived largely **by chance** rather than because of their alleles. A small sample is unlikely to contain the alleles of the original population in the same proportions, and **rare alleles are likely to be missing altogether**, so the survivors' allele frequencies differ from those of the original population. (c) Genetic diversity **remains low**. The recovered population of 800 has been built entirely from the **alleles of the 14 survivors**, so although the **number of individuals** has recovered, the **number of different alleles** has not — alleles lost in the crash do not come back unless a new mutation occurs or individuals migrate in.

Q6. (a) **Artificial selection**, carried out through a **selective breeding** program. (b) The frequency of the alleles for heavy grain heads **increases** over the generations. Only plants with the heaviest heads are allowed to be parents, so **only their alleles are passed on**; those alleles therefore make up a larger share of the gene pool in each successive crop, and the mean head weight of the crop rises. (c) In selective breeding the **selecting agent is a human**, who

decides which individuals reproduce on the basis of a trait people want; in natural selection the selecting agent is an **environmental selection pressure**, and the favoured phenotype is the one best suited to survival and reproduction in that environment. (Selective breeding is also much faster and far more directed.)

Q7. Gene flow is not occurring. Gene flow takes two steps: individuals (or their gametes) must **move from one population to another**, *and* they must then **interbreed with the residents** so that their alleles are passed to offspring and enter that population's gene pool. The lorikeets complete only the first step — they feed in the remnant patch but **return to the forest to nest**, so **none of their alleles is passed to a chick hatched in the remnant patch**. Movement without breeding is not gene flow. The visits therefore have **no effect on the allele frequencies of the remnant population**: its gene pool still contains only the alleles of the resident birds, and because that population stays small and effectively isolated, its frequencies will continue to change through **genetic drift** and local selection instead. *(1 mark: states that gene flow is not occurring; 1 mark: gene flow requires interbreeding, not merely movement, applied to the birds nesting elsewhere; 1 mark: no change to the remnant population's allele frequencies / it remains effectively isolated.)*

Q8. In the **founder effect**, a **small number of individuals leave the original population and start a new population somewhere else**; the new gene pool contains only the alleles those founders happened to carry, while the original population continues. In the **bottleneck effect**, the **same population crashes in size in place** — because of a fire, drought, disease or over-harvesting — and the few survivors rebuild the population. The **shared outcome** is that in both cases the next generation is founded on a **small, unrepresentative sample** of the original gene pool, so allele frequencies shift by chance and **genetic diversity is reduced** (and stays reduced even after numbers recover). *(1 mark: founder effect; 1 mark: bottleneck effect; 1 mark: shared outcome of reduced/unrepresentative gene pool.)*

Q9. A — mutation. A form of the wing-pattern gene that has **never been recorded in the species** has appeared, so this is a **genuinely new allele**, not a redistribution of alleles that already existed. Only **mutation** — a random change to the DNA base sequence — can create a new allele; selection, genetic drift and gene flow can change only the proportions of alleles already present somewhere. Because the beetle's offspring inherit it, the mutation must have occurred in a **gamete-producing cell**, so the new allele has entered the gene pool and its frequency has risen from zero. **B — natural selection (an environmental selection pressure).** The falling winter temperature is an environmental factor that acts on pre-existing variation: shrubs with the thicker cuticle survive frost and set more seed, so they pass that allele on and its frequency rises from 0.20 to 0.61. **C — gene flow.** Pollen (male gametes) is carried from one population to the other and fertilisation occurs, so alleles are physically transferred between the gene pools and the two populations become genetically more similar. *(1 mark per scenario: correct process with a justification drawn from the scenario.)*

Q10. Error 1: individuals **cannot develop a new trait because they need it** — a fish cannot acquire salt tolerance during its lifetime and pass it on; tolerance depends on the alleles it inherited. **Error 2:** the rise in salinity **did not create the variation**; the variation in salt tolerance already existed in the population (from earlier mutation), and the salinity acted only as a selection pressure on it. (A third error: "all the fish became salt-tolerant" describes individuals changing rather than a change in the **proportion** of tolerant individuals.) **Correct version:** Some fish in the population **already carried alleles for salt tolerance**. When the creek became saltier, those fish were **more likely to survive and reproduce** than fish without those alleles, so they **passed the tolerance alleles to their offspring**. Over many generations the **frequency of the tolerance alleles increased**, so a greater proportion of the population is now salt-tolerant — the **population** changed, not the individuals. *(1 mark per error identified; 1 mark for a correct rewritten account.)*

Q11. The highway acts as a barrier, so **gene flow between the two halves stops** — individuals can no longer move across and interbreed, and no alleles are exchanged. Each half is now a **much smaller population**, so **genetic drift has a far greater effect**: allele frequencies in each will change randomly and independently, and alleles may be **lost or fixed** by chance. Because alleles are lost and close relatives are more likely to breed together (**inbreeding**), the **genetic diversity within each population falls**, reducing its capacity to respond to a future selection pressure. Because drift acts independently in each half — and local selection pressures may also differ — the **two gene pools become increasingly different from each other** over many generations. *(1 mark: gene flow stops; 1 mark: smaller populations so drift has a greater effect / alleles lost or fixed; 1 mark: diversity within each population falls, inbreeding rises; 1 mark: the two populations diverge genetically.)*

Q12. A **non-evolving population** is one in which the **allele frequencies of the gene pool remain stable from generation to generation** — the same alleles occur in the same proportions in each new generation. Three conditions

(any three of the four): the population is **very large**, so chance sampling (genetic drift) has a negligible effect; there is **no gene flow** — no individuals or gametes move into or out of the population; there are **no new mutations** at that gene; there is **no selection**, so every genotype is equally likely to survive and reproduce. Each condition is simply one of the four causes of changing allele frequencies switched off. ("Random mating" is **not** creditable here: non-random mating changes how alleles are **combined into genotypes**, raising homozygosity, but it does not by itself change the **allele frequencies** of the gene pool.) *(1 mark: definition; 1 mark each for three valid conditions = 3 marks.)*

Q13. Before the change: total allele copies = 2 × 400 = 800. Copies of G = (2 × 36) + 168 = 240, so **frequency of G = 240 ÷ 800 = 0.30** (and g = 560 ÷ 800 = 0.70). **Fifteen generations later:** copies of G = (2 × 144) + 208 = 496, so **frequency of G = 496 ÷ 800 = 0.62** (and g = 304 ÷ 800 = 0.38). **Description:** the frequency of G **increased** from 0.30 to 0.62 (a rise of 0.32) while g **decreased** from 0.70 to 0.38 over the 15 generations. **Explanation:** the blackened ground is an **environmental selection pressure** acting on variation that already existed. Dark grasshoppers (GG and Gg) are **less visible to predators** on the burnt ground, so a greater proportion of them **survive to reproduce** and **pass allele G to their offspring**, while pale (gg) grasshoppers are eaten more often and leave fewer offspring. Repeated over 15 generations this **natural selection** raised the frequency of G in the gene pool — the population changed, not the individual grasshoppers. *(1 mark: G = 0.30 before; 1 mark: G = 0.62 after; 1 mark: directional description using the data; 1 mark: names natural selection / selection pressure; 1 mark: survival and reproduction of dark individuals passes G on.)*

Q14. Each generation the breeder **chooses only the animals with the desired phenotype as parents** and breeds from them (artificial selection). Because **only those animals pass on their alleles**, the **desired allele makes up a larger share of the gene pool** in the next generation, and repeating this raises its frequency generation after generation until the trait is close to uniform in the herd. Genetic diversity falls for two reasons. First, **only a few individuals contribute alleles**, so the many alleles carried by the animals that were not bred from are **never passed on and are lost** from the herd's gene pool (and the small breeding group also drifts strongly). Second, the chosen parents are often **closely related**, so continued **inbreeding raises homozygosity** — fewer allele combinations occur and harmful recessive alleles are more often expressed. *(1 mark: breeder selects the parents; 1 mark: only their alleles are passed on so the frequency rises over generations; 1 mark: few parents means alleles are lost from the gene pool; 1 mark: inbreeding raises homozygosity / diversity falls.)*

Q15. A **mutation** is a **random change in the DNA base sequence**; if it occurs in a gamete-producing cell it can be inherited, producing an **allele that did not exist in the population before**. It is the only such source, because selection, genetic drift and gene flow can only alter the proportions of alleles that **already exist** — they cannot create a new one. Mutation therefore **increases the genetic diversity** of the gene pool by adding new variation. *(1 mark: mutation creates a new allele / other processes only redistribute existing alleles; 1 mark: increases genetic diversity.)*

Q16. (a) The 30 residents carry 2 × 30 = **60 copies** of the gene. The 24 NN crayfish carry no n allele and each of the 6 Nn crayfish carries **one**, so the waterhole holds **6 copies of n**. Frequency of n = 6 ÷ 60 = **0.10** (and N = 54 ÷ 60 = 0.90). (b) The 20 newcomers are all nn, so they bring 2 × 20 = **40 copies of n**. The combined population of 50 crayfish holds 2 × 50 = **100 copies** of the gene, of which 6 + 40 = **46 are n**, so the frequency of n = 46 ÷ 100 = **0.46** (and N = 54 ÷ 100 = 0.54; check: 0.46 + 0.54 = 1.00). This jump is **gene flow** — alleles have entered the waterhole gene pool from another population. (c) What decides the size of the effect is the number of migrant allele copies **relative to the size of the receiving gene pool**. Here the newcomers' 40 copies are **40 out of 100** — close to half of the combined gene pool — so they dominate the new frequency. A resident population of 3000 crayfish would hold 6000 copies, 600 of them n; adding the same 40 copies of n gives 640 out of 6040, so the frequency of n would rise only from 0.10 to about **0.11**. The **same amount of gene flow therefore shifts a small population's frequencies far more than a large one's**. *(a: 1 mark — 0.10, from 6 copies of n in 60; b: 1 mark — 0.46, from 46 copies of n in 100; c: 2 marks — the migrants are about 40% of the small gene pool but a tiny fraction of the large one; the frequency would change only slightly, from 0.10 to about 0.11.)*

Q17. (a) **Increases** — a mutation produces an allele that did not previously exist, adding new variation to the gene pool. (b) **Decreases** — this is a bottleneck: alleles carried only by the individuals that died are **lost by chance**, and the surviving 30 individuals hold only a small sample of the original gene pool. (c) **Increases** — this is **gene flow**: the migrants interbreed and add alleles from a different gene pool, including alleles the residents may not have had. (d) **Decreases** — sustained directional selection makes the alleles for the disfavoured phenotype **rare or lost**, and the favoured allele may become fixed. *(1 mark each, requiring both the correct direction and a valid reason.)*

Q18. Strengths. The draw is **random and takes no account of any survival advantage**, which is exactly how genetic drift works — it correctly represents allele frequencies changing by **chance sampling** rather than by selection. Running the two containers side by side models the key idea that the **small "population" (Y) shows much larger swings** in frequency, and can reach fixation or loss, while the **large one (X) stays close to 50:50**; repeating for ten generations also shows that the changes **accumulate**. **Limitations.** The model includes **no mutation, gene flow or selection**, so it cannot show how new alleles arise or how a useful allele spreads; the population size is **fixed**, so it cannot model a bottleneck or a growing population; and each bead is a **single allele**, so diploid individuals carrying two alleles, and the way alleles are combined at fertilisation, are not represented. **Judgement.** It is a **valid model of the random-sampling component of drift, and of the effect of population size**, but it is not a model of evolution as a whole and its conclusions should be limited to drift. *(2 marks: any two valid strengths; 1 mark: a valid limitation; 1 mark: a judgement about how far the model can be relied on.)*

Q19. Trend 1: the mean number of different alleles per marker gene **decreased steadily**, from 4.6 in generation 1 to 2.4 in generation 7. **Trend 2:** over the same period the incidence of the inherited disorder **increased**, from 0.5 to 7.6 kids per 100 births. **Explanation:** because the herd is **closed** (no animals brought in) there is **no gene flow**, and because most kids are fathered by a **small number of bucks**, only a few individuals contribute alleles to each generation. Alleles carried by the animals not used as parents are therefore **not passed on and are lost** from the gene pool — and with so few breeding animals, **genetic drift** removes further alleles by chance — which is why the number of different alleles falls. The same practice means kids are increasingly **closely related**, so **inbreeding** rises: two carriers of the same harmful recessive allele are far more likely to mate, and so **more kids inherit two copies** of that allele and show the disorder. *(1 mark: allele number falls, with data; 1 mark: disorder incidence rises, with data; 1 mark: few parents / closed herd means alleles are lost, aided by drift; 1 mark: inbreeding raises homozygosity for the recessive allele.)*

Q20. (a) The process is the **founder effect** (a form of **genetic drift**). The island population was started by a **few individuals** that rafted across, so its gene pool contains only the alleles those founders happened to carry, in whatever proportions they happened to have. The data support this: the island has only **12 different alleles across the 8 other genes compared with 37** on the mainland — most mainland alleles never reached the island — and the frequency of M is **0.95 on the island against 0.55** on the mainland, a large shift with no selective explanation given. (b) The island population has **very low genetic diversity**, so its individuals are **genetically very similar** to one another. If a new fungal disease arrives, it is **unlikely that any individuals carry alleles conferring resistance**, so a very large proportion of them may be affected and die, and with only about 120 adults the population could be **wiped out**. There is also **little variation for selection to act on**, so the population cannot change over generations in response to the disease. The mainland population, with far more alleles, is **more likely to contain resistant individuals** that survive, reproduce and pass resistance alleles on, so it can recover. (c) Allele **m is likely to be lost** (frequency 0) and allele **M fixed** at 1.00. The population is **small (about 120 adults) and isolated**, so **genetic drift** causes **large random changes** in allele frequencies each generation; m is already **rare (0.05)**, so by chance a generation may occur in which no m allele is passed on at all. With **no gene flow** from the mainland, the only way m could reappear is a **new mutation**. ∴ the island population is expected to lose still more of its genetic diversity over time. *(a: 2 marks — founder effect named; justified with both data comparisons. b: 3 marks — low diversity, so few or no resistant individuals; large proportion die / extinction risk; comparison with the diverse mainland population. c: 3 marks — m lost and M fixed; drift is strong in a small population and m is rare; no gene flow, so only mutation could restore it.)*

Chapter 9 Genetic changes in populations over time — 9C Approaches to pathogenic evolution

Theory

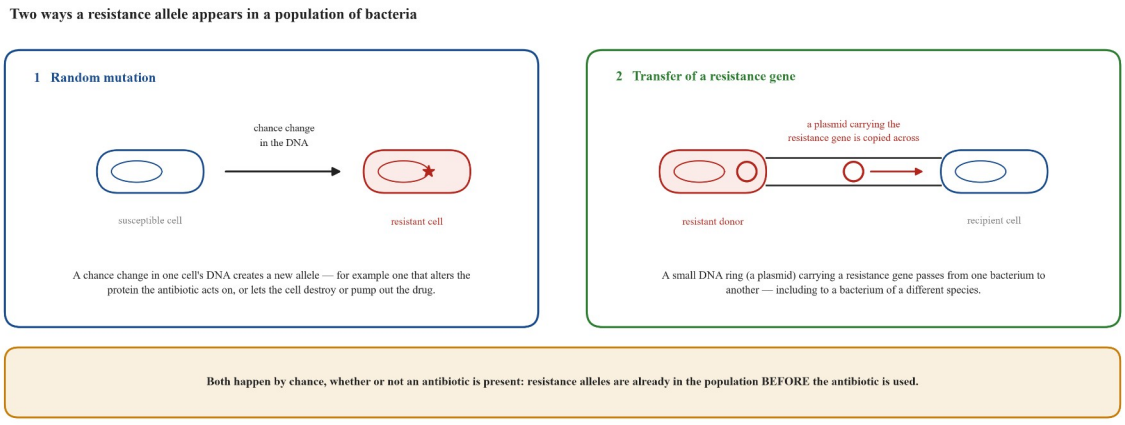
Pathogens are populations of living things (or, for viruses, of replicating particles), so the same processes that change any population over time also change them. Two of those changes matter enormously for human health, and they are the subject of this section:

- **bacterial resistance** to antibiotics, which creates ongoing challenges for **treatment strategies**; and
- **antigenic drift** and **antigenic shift** in viruses, which create ongoing challenges for **vaccination**.

Mutation as the source of new alleles was covered in **9A**, and the way selection pressures, genetic drift and gene flow change allele frequencies in a population was covered in **9B**. Here you **apply** that logic to pathogens. How antibodies and memory cells recognise an antigen belongs to **7D** and **7E**; all you need here is that the recognition is **specific to the shape of the antigen**, so any change to that shape matters.

Antibiotic resistance — what it is. An **antibiotic** is a chemical that kills bacteria or stops them reproducing. A bacterium is **resistant** to an antibiotic if it survives and reproduces at a concentration of that drug which kills other members of its species. Resistance is a **phenotype produced by an allele** — for example an allele coding for an altered version of the protein the drug acts on (so the drug no longer binds), an enzyme that breaks the drug down, or a pump that removes the drug from the cell.

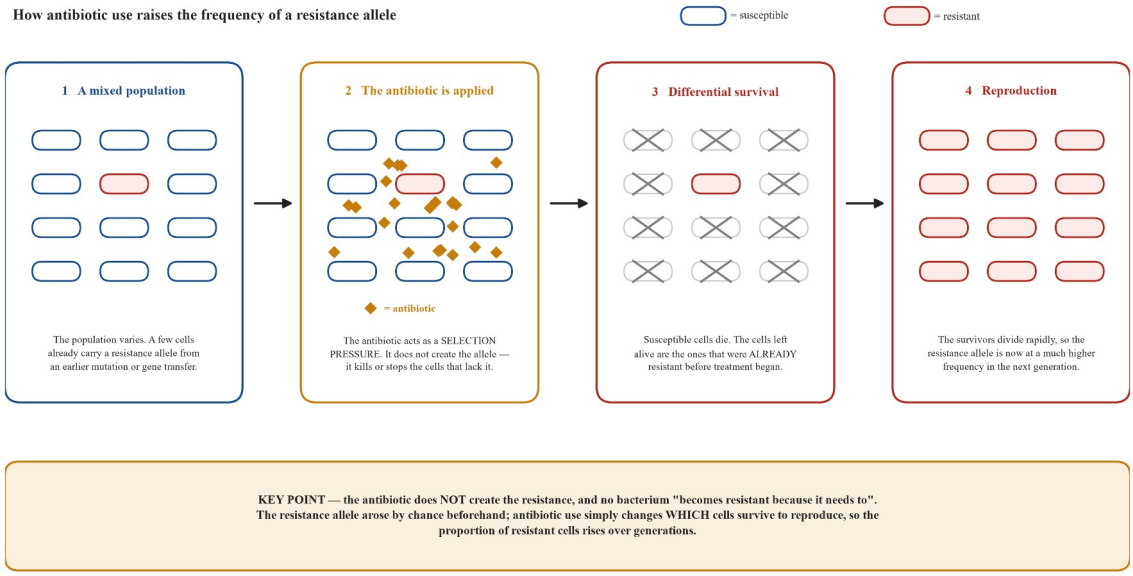
Where a resistance allele comes from. A resistance allele reaches a bacterial population in one of two ways, shown below.



- **Random mutation.** A chance change in the DNA of one cell produces a new allele. Like all mutations (9A), it is **random with respect to what the organism needs** — it is not triggered by the drug, and it can occur in a population that has never met an antibiotic.
- **Transfer of a resistance gene between bacteria.** Bacteria can pass DNA to one another directly, most importantly as a **plasmid** (a small circle of DNA separate from the chromosome) carrying one or more resistance genes. Because this transfer does not require reproduction, a resistance gene can move **sideways** through a population within a single generation — and even into bacteria of a **different species**.

Both events happen **whether or not an antibiotic is present**. In any large bacterial population, resistance alleles are already there, at low frequency, **before** the antibiotic is ever used.

How resistance spreads — selection. Using an antibiotic does not create a resistance allele; it changes **which cells survive to reproduce**. The antibiotic is a **selection pressure**.



The chain of reasoning you must be able to write is:

1. The bacterial population **varies** — a few cells already carry a resistance allele (from an earlier mutation or from gene transfer).
2. The antibiotic is applied and acts as a **selection pressure**.
3. Cells **without** the allele are killed or stopped from reproducing; the cells **with** the allele **survive** — this is **differential survival**.
4. The survivors **reproduce** and pass the allele to their offspring. Bacteria have a very short generation time and enormous population sizes, so many generations pass quickly.
5. The **frequency of the resistance allele rises** over successive generations, until most of the population is resistant.

The error examiners see most often. Antibiotics do **not** cause the mutation, and a bacterium does **not** "become resistant" because it needs to or because it has "sensed" the drug. An individual bacterium does not change during its lifetime in response to the antibiotic. The allele arises by chance beforehand; the antibiotic merely **selects** the cells that already carry it. Write about the **population** changing, not about individuals adapting on demand.

Notice also that a resistance allele is only an advantage **in an environment containing the antibiotic**. Where no antibiotic is present it may give no benefit at all, and may even carry a small cost, which is why resistant cells usually stay rare until the drug is used.

Practices that speed resistance up. The more often bacterial populations meet an antibiotic — and the more often that exposure kills only the most susceptible cells — the stronger the selection pressure favouring resistant cells:

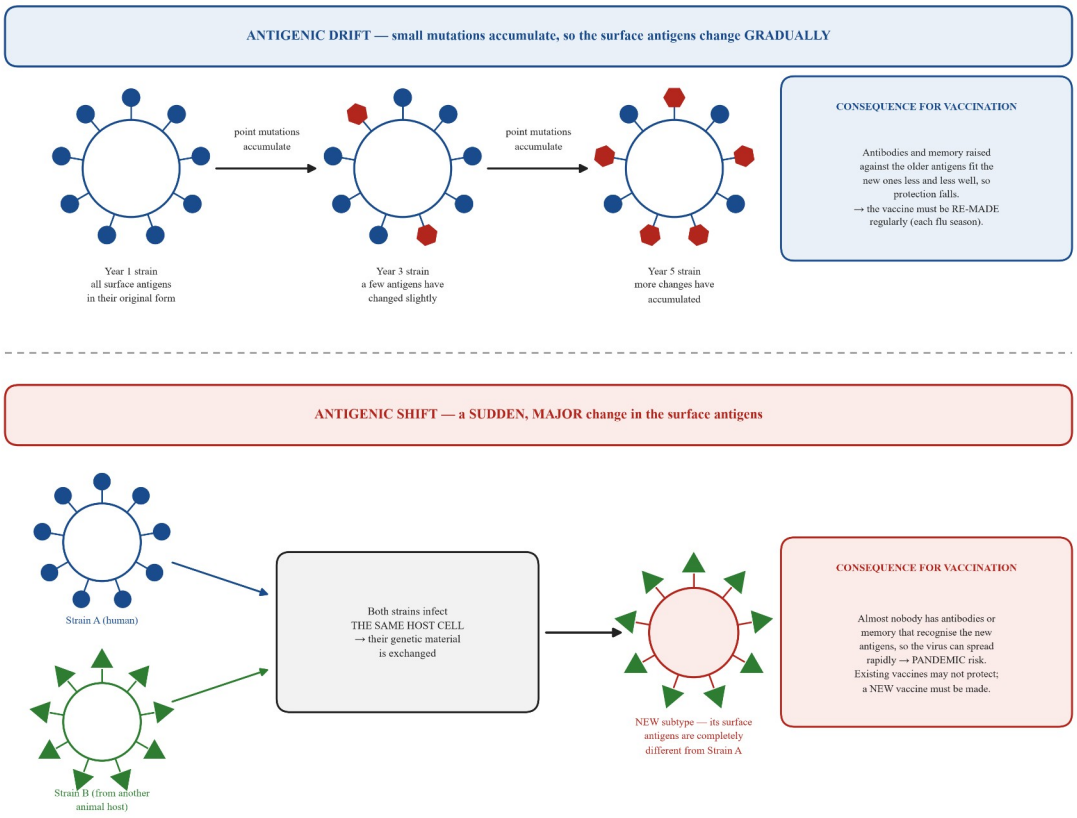
- **Over-prescription** — prescribing antibiotics when they are not needed, including for illnesses caused by **viruses** (antibiotics have no effect on viruses at all), exposes the body's bacteria to the drug for no benefit.
- **Not completing a prescribed course** — the least resistant cells die first, so stopping early can leave behind the cells that were hardest to kill; these survive to reproduce and to be passed on.
- **Routine agricultural use** — giving antibiotics continuously, often at low doses, to healthy livestock maintains a constant selection pressure on the bacteria in those animals, and resistant bacteria (or resistance plasmids) can then reach people.

Poor infection control also speeds resistance up, but by a **different route**, and it is worth being precise about which. Failing to wash hands or to isolate infected patients does not change how much antibiotic the bacteria meet, so it does not by itself increase the selection pressure; what it does is allow strains that are **already resistant** to move from patient to patient, so a resistant strain **spreads** through a hospital or community. It adds to the selection pressure only **indirectly**: more infections mean more courses of antibiotics prescribed. If a question asks how a measure changes the **selection pressure**, an answer about spread alone will not earn the mark.

Consequences for treatment strategies. Resistance is a problem precisely because it changes what doctors can do:

- Infections **no longer respond** to the usual first-line antibiotic, so illness lasts longer and can become more severe or fatal.
- **Multi-drug resistance** — a strain resistant to several unrelated antibiotics — leaves only "last line" drugs, which are often more expensive, more toxic, or must be given in hospital by injection.
- Procedures that depend on controlling infection, such as joint-replacement surgery, transplants and cancer chemotherapy, become **riskier**.
- Health services must therefore change strategy: **test the isolate first** (find out which antibiotics the patient's bacterium is still sensitive to before prescribing), use **combination therapy** (two or more drugs at once, because a cell resistant to both is far rarer), practise **antibiotic stewardship** (prescribe only when genuinely needed, at the right dose and for the full course, and reserve last-line drugs), improve **infection control**, restrict **agricultural use**, and invest in **developing new antibiotics** — which is slow and expensive, and buys only limited time before resistance to the new drug is selected in turn.

Viral surface antigens. A virus carries **antigens on its surface**. These are the molecules the immune system recognises, and they are what a vaccine trains the body against. Because recognition depends on the antigen's **shape**, a virus whose surface antigens change can escape antibodies and memory raised against the earlier form. Two very different kinds of change do this.



Antigenic drift is the **gradual accumulation of small mutations** in the genes coding for the surface antigens. Each mutation changes the antigen only slightly, but the changes build up from one season to the next. The mutations themselves are random; in a population where many people are already immune to the older form, variants whose antigens are **less well recognised** by existing antibodies survive and spread best, so the changed forms become common. Consequences for **vaccination**:

- Antibodies and memory cells raised against last season's antigens **bind the new antigens less well**, so protection falls and a person can catch the same disease again in a later season.
- The vaccine must be **reformulated regularly** — for influenza, viruses circulating around the world are monitored and the vaccine is re-made and re-administered **each year** to match the strains now in circulation. If the match is poor, vaccine effectiveness for that season drops.

Not every virus drifts to this extent. The surface antigens of the **measles** virus, for example, change very little over time, so memory cells formed in childhood still recognise them decades later and a single course of vaccine usually protects for life. It is the **rate at which a virus's antigens change** — not vaccination itself — that decides whether a vaccine must be re-made and re-given.

Antigenic shift is a **sudden, major change** in the surface antigens. It happens when **two different strains of a virus infect the same host cell at the same time** and their genetic material is mixed — in influenza, whose genome is carried in several separate pieces, whole pieces can be swapped between the two strains. The result is a virus displaying a **combination of surface antigens the human population has never met** (a shift can also follow a virus from another animal species acquiring the ability to spread between people). Consequences for **vaccination**:

- Almost **nobody has antibodies or memory cells** that recognise the new antigens, so the virus can spread rapidly through the population — this is how a **pandemic** can begin.
- **Existing vaccines may give little or no protection**, so an entirely **new vaccine** must be designed, tested and manufactured, which takes months during which the virus continues to spread.

Feature	Antigenic drift	Antigenic shift
Mechanism	Small mutations accumulate in the surface-antigen genes	Two strains infect one host cell and exchange genetic material
Size of change	Small; the antigens are altered slightly	Large; the antigens are largely or entirely new
Speed	Gradual, over seasons and years	Sudden, in a single event
Existing immunity	Partly effective, and falling	Little or none
Main consequence	Vaccine must be reformulated regularly (seasonal epidemics)	Pandemic risk; a new vaccine is needed

> **Do not confuse two different "drifts". Genetic drift** (9B) is the change in **allele frequencies** of a population from one generation to the next **by chance alone**, and it has its biggest effect in **small** populations. **Antigenic drift** is the change in a **virus's surface antigens** caused by the **accumulation of mutations**. They share a word and nothing else — genetic drift is a random process acting on allele frequencies in any population; antigenic drift is a specific change to the antigens a virus displays.

The common thread. In both cases **mutation supplies the variation** and **selection decides which variants become common** — antibiotics select resistant bacteria, and the immunity of the host population selects viral variants that are no longer recognised. Because pathogen populations are enormous and their generation times are short, these changes happen fast enough to matter within a human lifetime, which is why treatment and vaccination strategies must keep changing too.

Worked examples

Example 1 — scaffolded

In one hospital ward, antibiotic K was used routinely to treat infections caused by a particular bacterium. When sampling began, 3% of the bacteria isolated from the ward were resistant to antibiotic K. Two years later, 46% were resistant. Explain how the proportion of resistant bacteria increased.

Working	Comment
Before antibiotic K was used, the bacterial population already **varied**: a small number of cells carried a **resistance allele** produced earlier by a random mutation, or received from another bacterium on a plasmid.	Start with the source of the variation. The allele exists **before** the drug is used — never write that the antibiotic created it.
Using antibiotic K in the ward acted as a **selection pressure** on that population.	Name the selection pressure explicitly; this is the marking point that links the scenario to selection.
Cells **without** the resistance allele were killed or stopped from reproducing. The few cells **with** the allele	Differential survival — say what happens to **both** groups, not just the survivors.

Working	Comment
survived the treatment.	
The survivors **reproduced**, passing the resistance allele to their offspring. Bacteria divide rapidly, so many generations passed within the two years.	Selection changes a population only through reproduction, and the short generation time explains why the change was so fast.
Over successive generations the **frequency of the resistance allele increased**, so the proportion of resistant bacteria in the ward rose from **3% to 46%**.	Finish by answering the question in the terms it was asked — quote the data you were given.

Example 2 — scaffolded

Explain why the influenza vaccine offered to the public is re-made every year, whereas many other vaccines are not.

Working	Comment
The **surface antigens** of the influenza virus are the molecules that antibodies and memory cells recognise, and recognition depends on their **shape**.	Set up why a change to the antigen matters. Do not describe how a vaccine works — that is assumed knowledge here.
The genes coding for those surface antigens **mutate frequently**, and small changes **accumulate** from one season to the next. This is **antigenic drift**.	Name the process and define it in the same breath: gradual accumulation of small mutations.
Because much of the population is already immune to the older form, variants whose antigens are **less well recognised** by existing antibodies survive and spread best, so the changed antigens become common.	The mutations are random; selection by host immunity is what makes the changed forms widespread.
Antibodies and memory produced against last season's antigens therefore **bind this season's antigens less well**, and protection falls.	Link the antigen change to falling protection — this is the consequence for vaccination.
The vaccine must therefore be **reformulated** each year so that it contains antigens matching the strains now circulating, and people must be **re-vaccinated** each season.	Answer the question that was asked: the consequence for the vaccination program.

Example 3 — unscaffolded

A student writes: "When a patient takes an antibiotic, the bacteria in their body sense the drug and become resistant to it. That is why the infection comes back." Identify the errors in this statement and write a biologically correct explanation.

There are two errors. First, bacteria do **not sense** an antibiotic and change in response to it: the mutations that produce resistance alleles are **random** and occur **whether or not** the antibiotic is present. Second, an **individual** bacterium does not "become resistant" during its lifetime; it either already carries a resistance allele or it does not.

The correct explanation is that the population **already varied** before treatment: a few cells carried a resistance allele produced by an earlier random mutation, or received on a plasmid from another bacterium. Taking the antibiotic acted as a **selection pressure**. The susceptible cells were killed, while the cells that were **already resistant survived** and **reproduced**, passing the allele to their offspring. Over successive generations the **frequency of the resistance allele rose**, so the population regrowing in the patient was largely resistant and the infection returned. ∴ the antibiotic did not create the resistance — it selected for resistance that was already present.

Example 4 — unscaffolded

A pig on a farm is infected at the same time by an influenza strain circulating in humans and a strain circulating in wild birds. Genetic material from the two strains is mixed inside the pig's cells, and a virus carrying a surface antigen never before seen in people appears and begins to spread from person to person. Name the process that has occurred, and explain why this virus poses a far greater risk to the human population than the small changes influenza viruses undergo every year.

The process is **antigenic shift**: two different strains infected the **same host cell**, their genetic material was **exchanged**, and the resulting virus displays a **combination of surface antigens that is new to humans**. This is a **sudden and major** change, unlike **antigenic drift**, in which small mutations accumulate **gradually** so that each season's antigens are only slightly different from the last.

The risk is greater because the size of the change determines how much existing immunity is left. After drift, antibodies and memory raised against earlier strains still recognise the new antigens **partly**, so many people have some protection and the current vaccine still gives some benefit. After a shift, **almost nobody in the population has antibodies or memory cells that recognise the new antigens**, so the virus can spread rapidly and widely — the conditions for a **pandemic**. The existing vaccine is unlikely to protect, and an entirely **new vaccine** must be developed, tested and manufactured, which takes months while the virus continues to spread. ∴ antigenic shift is far more dangerous because it removes existing immunity all at once, whereas drift erodes it gradually.

Practice questions

Scaffolded

Q1. Define each of the following terms: (a) antibiotic resistance; (b) antigenic drift; (c) antigenic shift.

Q2. A resistance allele must already exist in a bacterial population before an antibiotic can select for it. (a) Name **two** ways a resistance allele can first appear in a population of bacteria. (b) State whether either of these events requires the antibiotic to be present. (c) Explain why a resistance allele may give a bacterium no advantage at all until an antibiotic is used.

Q3. Some human practices speed up the rise of antibiotic resistance. (a) Name **two** such practices. (b) Explain why prescribing an antibiotic for an illness caused by a virus adds to the problem. (c) Explain why adding low doses of antibiotics routinely to the feed of healthy livestock is a concern.

Q4. Antibiotic resistance creates ongoing challenges for treatment. (a) State what is meant by a multi-drug-resistant bacterium. (b) State **two** consequences for a patient infected with a multi-drug-resistant bacterium. (c) Explain why routine operations such as joint replacements become riskier as resistance spreads.

Q5. Influenza viruses undergo antigenic drift. (a) State what causes antigenic drift. (b) Describe the effect of antigenic drift on the virus's surface antigens. (c) Explain why an influenza vaccine made three years ago is likely to give less protection this year than it did when it was made.

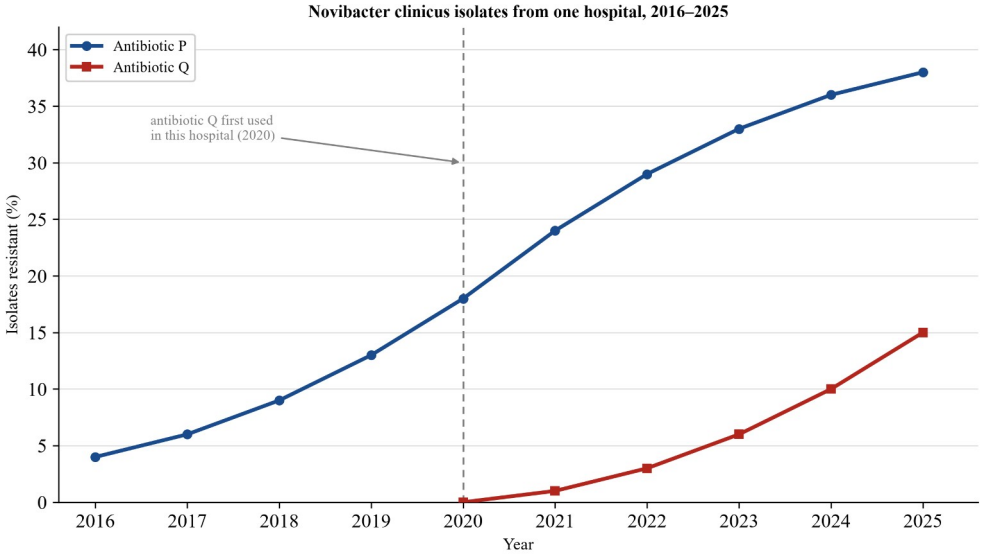
Q6. Influenza viruses can also undergo antigenic shift. (a) Describe how an antigenic shift can arise. (b) Compare the **size** and the **speed** of the change in surface antigens produced by a shift with that produced by drift. (c) Explain why a virus produced by antigenic shift can cause a pandemic.

Unscaffolded

Q7. *(Explain.)* Bacteria were collected from soil at a remote site that has never been treated with antibiotics. A small number of the cells were found to be resistant to antibiotic M. Explain how this is possible. *(2 marks)*

Q8. *(Explain.)* Explain how a resistance gene carried on a **plasmid** can spread through a bacterial population much faster than a resistance allele that arises by mutation, and why it can end up in bacteria of a **different species**. *(3 marks)*

Q9. *(Data interpretation.)* A hospital laboratory tested every isolate of the bacterium *Novibacter clinicus* it received between 2016 and 2025. Antibiotic P had been in use at the hospital for many years and every isolate was tested against it; antibiotic Q was introduced there in 2020, and isolates were tested against Q from that year onwards. The graph shows the percentage of isolates found to be resistant to each drug.



(a) Describe the trend in resistance to antibiotic P over the period shown, quoting data.

(b) Explain this trend in terms of selection acting on the bacterial population.

(c) Account for the pattern shown by antibiotic Q. *(6 marks)*

Q10. *(Explain.)* A patient is prescribed a seven-day course of an antibiotic but stops taking it after three days, as soon as the symptoms disappear. Explain how this can contribute to the rise of antibiotic resistance. *(3 marks)*

Q11. *(Data — table.)* An isolate of *Novibacter clinicus* taken from a patient's wound was spread evenly over an agar plate. Four paper discs, each soaked in a different antibiotic, were placed on the plate, which was then incubated for 24 hours. The diameter of the clear zone (the zone in which no bacteria grew) around each disc was measured.

Antibiotic disc	Diameter of clear zone (mm)
P	0
Q	8
R	21
S	14

(a) Identify the antibiotic that should be chosen to treat this patient, and justify your choice using the data.

(b) State what the result for antibiotic P shows about this isolate.

(c) Explain why testing a patient's isolate in this way before prescribing is a useful part of a strategy to slow the rise of resistance. *(5 marks)*

Q12. *(Evaluate.)* A commentator argues: "Resistance to any new antibiotic will appear eventually, so there is no point spending money developing new ones." Evaluate this argument. *(4 marks)*

Q13. *(Distinguish.)* Distinguish between antigenic drift and antigenic shift, with reference to the **mechanism**, the **size of the change**, and the **consequence for vaccination**. *(4 marks)*

Q14. *(Explain.)* A person caught influenza last winter and recovered. The following winter they catch influenza again. Explain how antigenic drift accounts for this. *(3 marks)*

Q15. *(Data — table.)* Each winter a national laboratory compares the influenza viruses collected from patients with the strain used in that year's vaccine. The table shows the percentage of collected viruses whose surface antigens closely matched the vaccine strain, over four consecutive seasons in which the **same** vaccine formulation was used.

Season	Collected viruses closely matching the vaccine strain (%)
1	91
2	74
3	52

Season	Collected viruses closely matching the vaccine strain (%)
4	30

(a) Describe the trend shown in the data.

(b) Name and describe the process in the virus population that best accounts for this trend.

(c) Using the data, justify the decision of health authorities to change the vaccine formulation before Season 5. *(5 marks)*

Q16. *(Explain.)* Some serious bacterial infections are treated with **two** different antibiotics at the same time rather than with one. Explain why resistance is far less likely to arise in the bacterial population when two drugs are given together. *(3 marks)*

Q17. *(Identify the error.)* A student writes: "Antigenic drift is the change in allele frequencies that happens purely by chance when a population is small." Identify the confusion in this statement, and give a correct account of **each** of the two terms the student has mixed up. *(3 marks)*

Q18. *(Propose.)* A state health service wants to slow the rise of antibiotic resistance. Propose **two** measures it could take, and explain in each case how the measure reduces the selection pressure favouring resistant bacteria. *(4 marks)*

Q19. *(Data — table.)* A virus with two surface antigens was isolated from patients each year for six years. Each antigen is described by a **type** number (antigens of different types have very different structures) and a **variant** letter (variants of the same type differ only slightly).

Year	Surface antigen 1	Surface antigen 2
1	type 2, variant a	type 5, variant a
2	type 2, variant b	type 5, variant a
3	type 2, variant c	type 5, variant b
4	type 2, variant d	type 5, variant c
5	type 7, variant a	type 9, variant a
6	type 7, variant b	type 9, variant a

(a) Identify the years over which antigenic **drift** occurred, and justify your answer using the table.

(b) Identify the year in which an antigenic **shift** occurred, justify your answer using the table, and state what this would mean for people vaccinated using the Year 4 virus. *(4 marks)*

Q20. *(Synthesis.)* A clinic in a remote community faces two separate problems in the same year.

- Skin infections caused by the bacterium *Novibacter clinicus* no longer clear up when treated with antibiotic P, which the clinic has prescribed routinely for many years.
- Each April the clinic runs an influenza vaccination session, and staff are told that people vaccinated last April must be vaccinated again this April. Later in the year, public-health authorities warn that a new influenza subtype has appeared after two different influenza strains infected the same host.

(a) Explain how the population of *Novibacter clinicus* at the clinic came to be resistant to antibiotic P, and state **one** consequence of this resistance for treating the patients.

(b) Explain why a person vaccinated against influenza last April must be vaccinated again this April.

(c) Name the process that produced the new influenza subtype, and explain why it presents a greater public-health threat than the yearly changes referred to in part (b). *(8 marks)*

Answers

Q1. (a) The ability of a bacterium to survive and reproduce at a concentration of an antibiotic that kills or stops other members of its species. (b) The gradual accumulation of small mutations in the genes for a virus's surface antigens, so those antigens change slightly over time. (c) A sudden, major change in a virus's surface antigens, produced when two different strains infect the same host cell and exchange genetic material. **Q2.** (a) A random mutation in a bacterium's DNA; transfer of a resistance gene from another bacterium (often on a plasmid). (b) No — both occur by chance whether or not an antibiotic is present. (c) The allele only gives an advantage in an environment containing the antibiotic; without the drug it gives no benefit (and may carry a small cost), so resistant cells stay rare. **Q3.** (a) Any two of: over-prescription of antibiotics; not completing a prescribed course; routine or low-dose use in healthy livestock; poor infection control. (b) Antibiotics have no effect on viruses, so the patient gains nothing, but the bacteria in and on the patient are still exposed to the drug and are still selected. (c) It maintains a constant selection pressure on the bacteria of those animals, so resistant bacteria (and resistance plasmids) build up and can then reach people. **Q4.** (a) One that is resistant to several different (unrelated) antibiotics. (b) Any two of: the usual antibiotics no longer work; longer, more severe or fatal illness; only last-line drugs remain, which are often more expensive, more toxic or must be given in hospital. (c) Surgery relies on being able to treat any infection that follows; if the bacteria involved are resistant, a post-operative infection may not be treatable, so the risk of the operation rises. **Q5.** (a) The accumulation of small, random mutations in the genes coding for the surface antigens. (b) They change slightly, a little more with each season. (c) The antigens of the circulating viruses have drifted away from those in the old vaccine, so antibodies and memory produced by that vaccine bind them less well and give less protection. **Q6.** (a) Two different strains infect the same host cell at the same time and their genetic material is mixed (pieces of genome are exchanged), producing a virus with surface antigens new to the population. (b) The change is much larger (the antigens are largely new, not slightly altered) and much faster (a single sudden event, rather than gradual change over seasons). (c) Almost nobody has antibodies or memory cells that recognise the new antigens, so the virus can spread rapidly through the population. **Q7.** The resistance allele arose by a random mutation (or was received from another bacterium on a plasmid); such events occur by chance and do not require the antibiotic to be present. **Q8.** A plasmid can be passed directly from cell to cell without reproduction, so the gene moves sideways through the population within a generation, whereas a mutation spreads only as its owner reproduces; and because the transfer does not depend on the two cells being the same species, the plasmid can enter bacteria of a different species. **Q9.** (a) Resistance to P rose steadily, from 4% in 2016 to 38% in 2025, with the steepest rise between 2019 and 2022 and some levelling off after 2023. (b) Resistant cells were already present; using P selected them, susceptible cells died, survivors reproduced, so the resistance allele's frequency rose over generations. (c) Resistance to Q was 0% when it was introduced in 2020 and then rose to 15% by 2025: rare cells resistant to Q (from mutation or gene transfer) were selected once Q began to be used. **Q10.** The least resistant cells die first, so stopping early leaves alive the cells that are hardest to kill; these survive, reproduce and pass on their alleles, raising the frequency of resistance (and the infection may return in a less treatable form). **Q11.** (a) Antibiotic R — it produced the largest clear zone (21 mm), so it inhibited this isolate most effectively. (b) The isolate is resistant to antibiotic P (no clear zone at all — growth right up to the disc). (c) It allows the doctor to prescribe the drug the isolate is most strongly inhibited by, avoiding a useless course of P — which cannot clear this infection but would still select for resistance — and avoiding a weakly inhibiting drug such as Q. **Q12.** Partly valid — resistance to a new antibiotic will be selected in time, so new drugs are not a permanent fix and must be paired with stewardship. But the conclusion is wrong: without new antibiotics, multi-drug-resistant infections become untreatable now, and each new drug buys time and restores treatment options. Judgement: new antibiotics are necessary but not sufficient. **Q13.** Drift = small mutations accumulating in the surface-antigen genes; a small, gradual change; existing immunity still partly works, so the vaccine is reformulated regularly. Shift = two strains infecting one host cell and exchanging genetic material; a large, sudden change; little or no existing immunity, so a completely new vaccine is needed and a pandemic may follow. **Q14.** Antigenic drift has changed this year's virus's surface antigens, so the antibodies and memory produced last winter recognise them less well and give less protection, allowing infection again. **Q15.** (a) The percentage matching the vaccine strain fell steadily, from 91% in Season 1 to 30% in Season 4 (a fall of 61 percentage points). (b) Antigenic drift — small mutations accumulating in the genes for the surface antigens, so fewer and fewer circulating viruses still match the vaccine strain. (c) By Season 4 only 30% of circulating viruses still matched, so the vaccine would protect against a minority of infections; reformulating restores the match and the protection. **Q16.** Resistance to each drug is a separate, rare allele. A cell resistant to only one of the two drugs is still killed by the other, so it does not survive to reproduce; only a cell that already carries resistance alleles to **both** drugs survives, and such cells are far rarer than cells resistant to one. There is therefore almost no resistant variation for the treatment to select. **Q17.** The student has defined **genetic** drift, not antigenic drift. Genetic drift = the change in allele frequencies of a population from one generation to the next by chance alone, greatest in

small populations. Antigenic drift = the gradual change in a virus's surface antigens caused by accumulated mutations in the genes coding for them. **Q18.** Any two, each with a mechanism that reduces **antibiotic exposure**, e.g. antibiotic stewardship (prescribe only when needed, at the correct dose and for the full course) reduces how often bacterial populations meet the drug; restricting routine antibiotic use in healthy livestock removes a constant selection pressure on farm bacteria; testing isolates before prescribing avoids courses of a drug that cannot cure but still selects. Improved infection control alone does not answer this question, because it limits the **spread** of already-resistant strains rather than the exposure that selects them. **Q19.** (a) Years 1 to 4 (and Years 5 to 6): the antigen types stayed the same and only the variant letters changed, which is a small change — antigenic drift. (b) Year 5: both antigens changed type (2→7 and 5→9), a large sudden change — antigenic shift; people vaccinated with the Year 4 virus would have little or no protection against the Year 5 virus. **Q20.** (a) A few cells already carried a resistance allele (mutation or gene transfer); routine use of P selected them; susceptible cells died, resistant cells survived and reproduced, so the frequency of resistance rose. Consequence: infections no longer respond to P, so another (often more expensive or more toxic) antibiotic must be used. (b) Antigenic drift changes the surface antigens between seasons, so last April's vaccine is poorly matched to this year's circulating strains and gives less protection. (c) Antigenic shift — the antigens are entirely new, so almost nobody has existing immunity, the virus can spread rapidly (pandemic risk) and a completely new vaccine must be developed.

Fully-worked solutions

Q1. (a) **Antibiotic resistance** is the ability of a bacterium to **survive and reproduce** at a concentration of an antibiotic that **kills or stops the growth** of other members of its species. It is the phenotype produced by a **resistance allele**. (b) **Antigenic drift** is the **gradual accumulation of small mutations** in the genes coding for a virus's **surface antigens**, so those antigens change **slightly** over time. (c) **Antigenic shift** is a **sudden, major change** in a virus's surface antigens, produced when **two different strains infect the same host cell** and their genetic material is **exchanged**, giving a virus with surface antigens new to the population.

Q2. (a) A **random mutation** in the DNA of a bacterium, producing a new allele; and **transfer of a resistance gene from another bacterium**, most often on a **plasmid**. (b) **Neither.** Both events occur **by chance**, whether or not an antibiotic is present — the antibiotic does not trigger them. (c) A resistance allele is only advantageous **in an environment that contains the antibiotic**. Where no antibiotic is present the altered protein, enzyme or pump gives the cell **no benefit**, and making it may even carry a small **cost**, so resistant cells are not favoured and remain at a **low frequency** until the drug is used.

Q3. (a) Any **two** of: **over-prescription** of antibiotics; **not completing** a prescribed course; **routine (often low-dose) use in healthy livestock**; **poor infection control** in hospitals. (b) Antibiotics act on bacteria and have **no effect on viruses**, so the patient gains **no benefit** at all. However, the bacteria living in and on that patient are still **exposed to the drug**, so resistant cells among them are still **selected** — the population's resistance rises for nothing in return. (c) Continuous low-dose treatment keeps a **constant selection pressure** on the bacteria of those animals, so **resistant bacteria and resistance plasmids build up** in the herd or flock. These bacteria, or the plasmids they carry, can then reach **people** through food, water or direct contact, spreading resistance genes into human infections.

Q4. (a) A **multi-drug-resistant** bacterium is one that is **resistant to several different (unrelated) antibiotics** at the same time. (b) Any **two** of: the usual first-line antibiotics **no longer work**; the illness lasts **longer** and may be **more severe or fatal**; only **last-line drugs** remain, which are often **more expensive, more toxic**, or must be given in hospital by injection. (c) A joint replacement relies on being able to **treat any infection that follows the surgery**. If the bacteria likely to cause such an infection are resistant, a post-operative infection **may not be treatable**, so the **risk of the operation itself rises** and some patients may not be able to have it.

Q5. (a) Antigenic drift is caused by the **accumulation of small, random mutations** in the **genes coding for the surface antigens**. (b) The surface antigens change **slightly**, and a little more with each season, so their shape gradually differs more and more from the original form. (c) Over three years, drift has changed the surface antigens of the circulating viruses so that they **no longer match the antigens in the old vaccine**. Antibodies and memory cells produced in response to that vaccine therefore **bind the current antigens less well**, so the vaccine gives **less protection** than it did when the circulating strains matched it.

Q6. (a) **Two different strains** of the virus infect the **same host cell at the same time**, and their **genetic material is mixed** — in influenza, whose genome is in separate pieces, whole pieces can be **exchanged** between the strains. The virus produced displays a **combination of surface antigens that the population has not met**. (b) The change is **much larger**: shift produces antigens that are **largely or entirely new**, whereas drift alters existing antigens only **slightly**. It is also **much faster**: shift happens in a **single sudden event**, whereas drift accumulates **gradually over seasons and years**. (c) Because the antigens are new, **almost nobody in the population has antibodies or memory cells that recognise them**. With little or no existing immunity, the virus can **spread rapidly and widely** between people, which is how a **pandemic** begins.

Q7. Resistance alleles arise **independently of the antibiotic**. A **random mutation** in the DNA of one soil bacterium could have produced an allele that happens to confer resistance to antibiotic M (for example an altered target protein or an enzyme that destroys the drug), or the cell could have **received a resistance gene from another bacterium on a plasmid**. Because these events are **random with respect to need**, they occur whether or not the antibiotic is ever present, so a small number of resistant cells can exist at a site that has never been treated. *(1 mark: mutation and/or gene transfer as the source; 1 mark: these occur by chance and do not require exposure to the antibiotic.)*

Q8. A plasmid is a **small circle of DNA that can be passed directly from one bacterium to another**. Because this transfer does **not require the cell to reproduce**, the resistance gene can move **sideways through the population within a single generation**, potentially reaching very many cells quickly. A resistance allele that arises by **mutation**, by contrast, is confined to one cell and its descendants, so it can only spread as fast as that cell **reproduces**. In addition, plasmid transfer does **not depend on the donor and recipient being the same species**, so a resistance gene can be passed into a **different species** of bacterium, which then becomes resistant without ever having mutated. *(1 mark: plasmid passed directly, no reproduction needed; 1 mark: mutation spreads only through the descendants of one cell; 1 mark: transfer can cross between species.)*

Q9. (a) Resistance to antibiotic P **increased steadily throughout the period**, rising from **4% of isolates in 2016 to 38% in 2025** — an increase of **34 percentage points**. The rise was **steepest between 2019 and 2022** (13% to 29%) and began to **level off after 2023**. (b) A small proportion of the *Novibacter clinicus* population **already carried a resistance allele** to P, from an earlier mutation or from gene transfer. Continued use of antibiotic P in the hospital acted as a **selection pressure**: susceptible cells were **killed**, while resistant cells **survived and reproduced**, passing on the allele. Over many bacterial generations the **frequency of the resistance allele rose**, which is measured here as the rising percentage of resistant isolates. (c) No isolates were resistant to Q in **2020**, the year it was introduced, but resistance then **rose to 15% by 2025**. Rare cells carrying a Q-resistance allele (arising by **mutation or gene transfer**) existed at very low frequency; once Q began to be used it became a **new selection pressure**, and those cells survived and reproduced, so resistance to a newly introduced drug appeared within a few years. *(a: 2 marks — directional trend, data quoted; b: 2 marks — pre-existing variation selected, survivors reproduce so allele frequency rises; c: 2 marks — rise from 0% to 15% after introduction, explained by selection of rare pre-existing resistant cells.)*

Q10. An antibiotic course is long enough to kill **all** the bacteria causing the infection. The **least resistant cells are killed first**, and symptoms disappear once numbers fall — but cells that are **harder to kill** (those carrying alleles giving partial or full resistance) may still be alive at day three. Stopping the course leaves these cells in the patient with **no further selection pressure to kill them** and **less competition**, so they **survive, reproduce and pass on their resistance alleles**. The bacterial population that regrows therefore has a **higher frequency of resistance alleles**, and those bacteria can also be **passed to other people**. *(1 mark: the hardest-to-kill cells are the ones still alive when symptoms stop; 1 mark: they survive and reproduce; 1 mark: the frequency of resistance in the population rises / resistant bacteria may spread.)*

Q11. (a) **Antibiotic R.** A clear zone forms where the antibiotic has **inhibited bacterial growth**, so a **larger zone means the isolate is more strongly inhibited**. R produced the **largest zone at 21 mm**, considerably larger than S (14 mm) and Q (8 mm), so R is the drug most likely to clear this infection. (b) The zone for P was **0 mm** — the bacteria grew right up to the disc, showing that this isolate is **resistant to antibiotic P** (P has no effect on it). (c) Testing first means the doctor can prescribe the drug the isolate is **most strongly inhibited by**. Prescribing **P** instead would **fail to cure the patient**, because this isolate grows right up to the P disc, while still **exposing all of that patient's bacteria to an antibiotic** — selecting for resistance for no clinical benefit. Choosing a drug that inhibits the isolate only weakly, such as **Q** (8 mm), risks the same outcome: the infection may not be fully cleared, and the surviving bacteria are exactly those least affected by that drug. Targeted prescribing therefore **reduces unnecessary selection pressure**, which slows the rise of resistance. *(a: 1 mark R; 1 mark justified by the largest zone / 21 mm; b: 1 mark resistant to P;*

c: 2 marks — the most effective drug is chosen from the data, and a course that cannot cure the patient but still selects for resistance is avoided.)

Q12. The argument has a **valid element**: because resistance alleles arise by **random mutation and gene transfer** in enormous bacterial populations, any new antibiotic will eventually act as a selection pressure and **resistance to it will be selected**, so a new drug is **not a permanent solution** and is of limited value unless it is paired with **stewardship** and infection control. However, the **conclusion does not follow**. Without new antibiotics, infections caused by existing **multi-drug-resistant** strains become **untreatable now**, and procedures such as surgery and chemotherapy become far riskier. Each new antibiotic **restores treatment options** and **buys time**, and the fact that a benefit is temporary does not make it worthless — the same argument would rule out treating any evolving disease. **Judgement:** developing new antibiotics is **necessary but not sufficient**; it should continue **alongside** measures that reduce the selection pressure. *(1 mark: the valid point that resistance to any new drug will be selected; 2 marks: limitations of the argument — untreatable infections now, new drugs restore options and buy time; 1 mark: a clear overall judgement.)*

Q13. Mechanism. In **antigenic drift**, **small mutations accumulate** in the genes coding for the virus's surface antigens. In **antigenic shift**, **two different strains infect the same host cell** and **exchange genetic material**, producing a new combination of antigens. **Size and speed of change.** Drift alters the antigens **slightly and gradually**, over seasons and years; shift changes them **largely or entirely, in a single sudden event**. **Consequence for vaccination.** After drift, existing antibodies and memory still recognise the antigens **partly**, so protection falls gradually and the vaccine must be **reformulated regularly** (for example each influenza season). After shift, there is **little or no existing immunity**, so an existing vaccine may give **no protection**, an **entirely new vaccine** must be developed, and a **pandemic** may occur in the meantime. *(1 mark: drift mechanism; 1 mark: shift mechanism; 1 mark: congruent contrast of size/speed; 1 mark: congruent contrast of the consequence for vaccination.)*

Q14. Recovering from influenza last winter left the person with **antibodies and memory cells that recognise the surface antigens of last winter's virus**. During the year, **antigenic drift** occurred: **small mutations accumulated** in the genes coding for those surface antigens, so this winter's viruses display **slightly different antigens**. Because recognition depends on the **shape** of the antigen, the person's existing antibodies and memory cells **bind the changed antigens less well**, so the immune response is slower and weaker than it would be against an identical virus. The virus can therefore establish an infection and the person catches influenza again. *(1 mark: drift = accumulated mutations changing the surface antigens; 1 mark: existing antibodies/memory recognise the changed antigens less well; 1 mark: so the person can be infected again.)*

Q15. (a) The percentage of circulating viruses closely matching the vaccine strain **fell progressively across all four seasons**, from **91% in Season 1 to 30% in Season 4** — a fall of **61 percentage points**, dropping by 17, 22 and 22 percentage points between successive seasons. (b) The process is **antigenic drift**: **small mutations accumulate** in the genes coding for the virus's surface antigens, so each season the circulating viruses' antigens differ a little more from those of the unchanged vaccine strain, and a smaller proportion still count as a close match. (c) By **Season 4 only 30%** of circulating viruses still matched the vaccine strain, meaning the vaccine was well matched to **fewer than one in three** of the viruses people were actually meeting — so most vaccinated people would be poorly protected. **Reformulating** the vaccine so that it contains antigens matching the strains **now circulating** restores the match, and therefore the protection, for Season 5. *(a: 2 marks — directional trend, data quoted; b: 1 mark — antigenic drift named and described; c: 2 marks — the low Season 4 match means poor protection, so reformulating restores the match.)*

Q16. Resistance to each antibiotic is produced by a **different allele**, and each arises **independently** — by random mutation or by gene transfer — so each is present in only a **very small proportion** of the population. If a **single** drug is given, every cell already carrying that one allele survives, reproduces and passes it on, so the resistant type is selected quickly. If **both** drugs are given at once, a cell resistant to only the first is still **killed by the second**, and a cell resistant to only the second is still **killed by the first** — neither survives to reproduce. Only a cell carrying resistance alleles to **both** drugs survives the treatment, and because two rare alleles must occur **together in the same cell**, such cells are **very much rarer**. Selection therefore has almost no surviving variant to act on, and the frequency of resistance rises far more slowly. *(1 mark: resistance to each drug is a separate, independently arising and rare allele; 1 mark: a cell resistant to only one drug is still killed by the other, so it does not survive and reproduce; 1 mark: only doubly resistant cells survive and these are far rarer, so there is little or no resistant variation for the treatment to select.)*

Q17. The student has given the definition of **genetic drift**, not antigenic drift; the two share the word "drift" but are **unrelated concepts**. **Genetic drift** is the change in the **allele frequencies** of a population from one generation to the next **by chance alone** (not because some alleles are favoured); it has its greatest effect in **small populations**. **Antigenic drift** is the **gradual change in a virus's surface antigens** brought about by the **accumulation of small mutations** in the genes coding for them, which is why immunity and vaccines directed at the older antigens become less effective. *(1 mark: identifies that the definition given is of genetic drift; 1 mark: correct account of genetic drift; 1 mark: correct account of antigenic drift.)*

Q18. Measure 1 — antibiotic stewardship. Require that antibiotics are prescribed **only when a bacterial infection is confirmed or strongly suspected**, at the **correct dose** and for the **full course**, and reserve last-line drugs. This reduces the **number of occasions on which bacterial populations are exposed to antibiotics**, so there are far fewer episodes in which resistant cells are favoured over susceptible ones, and the frequency of resistance alleles rises more slowly. **Measure 2 — restrict routine antibiotic use in healthy livestock.** Ban continuous low-dose antibiotics in animal feed and permit antibiotics only to treat diagnosed illness. This removes a **constant selection pressure** acting on the very large bacterial populations in farm animals, so resistant bacteria and resistance plasmids no longer accumulate there and pass to people. *(Other measures that reduce antibiotic exposure are equally acceptable — for example public and prescriber education so that antibiotics are not given for viral illnesses, or requiring a patient's isolate to be tested so that only a drug it is sensitive to is prescribed. Improved hospital infection control, offered on its own, does **not** earn the explanation mark as this question is worded: it limits the spread of strains that are already resistant, which does not change how much antibiotic the bacteria meet. It earns the mark only if the explanation is taken through exposure — preventing infections means fewer courses of antibiotics are prescribed, so the selection pressure falls.) (1 mark each for two appropriate measures; 1 mark each for explaining how the measure reduces the selection pressure.)*

Q19. (a) Antigenic **drift** occurred **from Year 1 to Year 4**, and again **from Year 5 to Year 6**. Across Years 1–4 both antigens kept the **same type** (type 2 and type 5) and only the **variant letters changed** (antigen 1: a→b→c→d; antigen 2: a→a→b→c), which the key tells us is a **small change** — the signature of small mutations accumulating gradually. The same pattern appears between Years 5 and 6 (type 7 variant a → type 7 variant b). (b) Antigenic **shift** occurred in **Year 5**. **Both** antigens changed **type** at once — antigen 1 from **type 2 to type 7** and antigen 2 from **type 5 to type 9** — so the surface antigens became **very different in a single step**, which is a large, sudden change. A person vaccinated using the Year 4 virus would have antibodies and memory against **type 2 and type 5** antigens, which would **not recognise** the type 7 and type 9 antigens, so they would have **little or no protection** against the Year 5 virus. *(a: 1 mark — Years 1–4 (and 5–6); 1 mark — justified by type staying the same while only the variant changes; b: 1 mark — Year 5 justified by both antigen types changing at once; 1 mark — little or no protection from a Year 4 vaccine.)*

Q20. (a) Before antibiotic P was ever used at the clinic, the *Novibacter clinicus* population **already varied**: a few cells carried a **resistance allele** produced by an earlier **random mutation** or received from another bacterium **on a plasmid**. Routine prescribing of P acted as a **selection pressure**: susceptible cells were **killed**, while the already-resistant cells **survived** and **reproduced**, passing the allele to their offspring. Over many bacterial generations the **frequency of the resistance allele rose**, so most of the population is now resistant. (The antibiotic did not create the resistance; it selected for it.) **Consequence:** infections no longer respond to antibiotic P, so the clinic must use a **different antibiotic**, which is likely to be more expensive, more toxic or harder to give in a remote setting, and patients stay ill for longer. (b) Between the two Aprils, **antigenic drift** has occurred: **small mutations have accumulated** in the genes coding for the virus's **surface antigens**, so this year's circulating strains display **slightly different antigens** from those in last year's vaccine. Antibodies and memory produced last April therefore **recognise the current antigens less well**, so the vaccine must be **reformulated to match the current strains** and given again. (c) The process is **antigenic shift**: two different strains infected the **same host cell** and their **genetic material was exchanged**, producing a subtype whose **surface antigens are entirely new to the human population**. It is a greater threat than drift because the change is **sudden and complete rather than gradual**, so **almost nobody has antibodies or memory cells that recognise the new antigens**. With essentially no existing immunity the virus can spread rapidly worldwide — a **pandemic** — and the current vaccine is unlikely to protect, so an **entirely new vaccine** must be developed, tested and manufactured, taking months during which the virus keeps spreading. ∴ resistance is being driven by selection at the clinic, while the two viral changes differ in scale: drift erodes protection each year, whereas shift can remove it all at once. *(a: 4 marks — pre-existing resistance allele from mutation/gene transfer; antibiotic as selection pressure killing susceptible cells; survivors reproduce so allele frequency rises; one consequence for treatment. b: 2 marks — antigenic drift defined as accumulated mutations changing surface antigens; existing*

immunity/vaccine less well matched so reformulation and re-vaccination are needed. c: 2 marks — antigenic shift named and mechanism given; little or no existing immunity → rapid spread/pandemic risk and a new vaccine required.)

Chapter 9 Genetic changes in populations over time — 9D Emergence of a new species

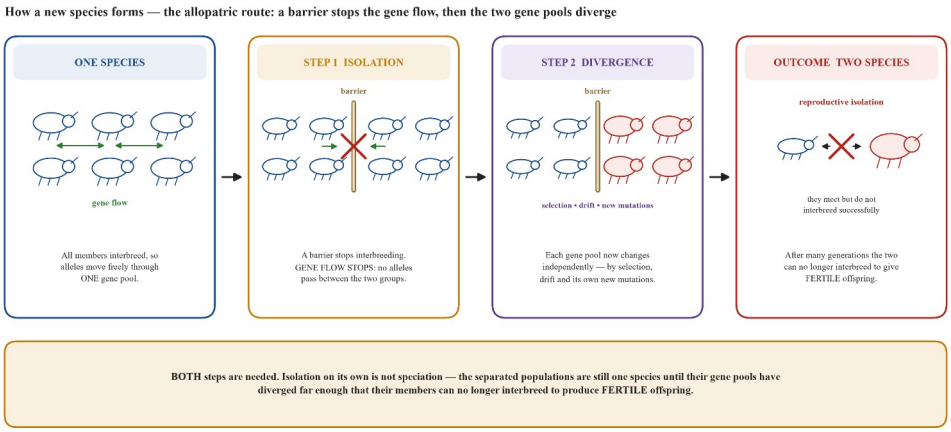
Theory

A **species** is a group of organisms whose members can **interbreed under natural conditions to produce fertile offspring**. The word *fertile* is the part that does the work: two populations that mate and produce healthy young are still one species only if those young can go on to breed themselves. If the offspring are sterile, the two parent populations belong to **different species**.

Speciation is the process by which one species gives rise to two or more new species. It is not a single event; it is a slow change in the make-up of gene pools over many generations. Two things are required.

- **1 Isolation.** Something stops members of the two groups interbreeding, so **gene flow stops** — no alleles pass from one group to the other any more. The one gene pool becomes two.
- **2 Genetic divergence.** The two gene pools change **independently** of each other. Each accumulates its own new **mutations**, **natural selection** acts on each according to its own conditions, and **genetic drift** changes allele frequencies at random in each. (These processes are covered in 9B; here you apply them.) The two gene pools drift further and further apart.

If divergence continues far enough, the two groups become **reproductively isolated**: even if they meet again they can no longer interbreed to produce fertile offspring. At that point they are **two species**.



Why both requirements are needed. Isolation on its own is not speciation. Two populations separated by a barrier are still one species for as long as they could still interbreed successfully if they met. Divergence on its own is not enough either: while gene flow continues, alleles keep being shuffled between the groups, and that mixing pulls the two gene pools back towards each other, so the differences can never build up as far as reproductive isolation. **Isolation is what lets divergence run to completion; divergence is what actually produces the new species.**

Which of the two comes first depends on the pattern. In **allopatric** speciation the order is the obvious one: a barrier appears, gene flow stops, and only then do the separated gene pools begin to diverge. In **sympatric** speciation it is usually the other way round. The two groups are still in the same place and still exchanging gametes when **natural selection begins to favour different alleles in each of them**, because each group is using a different soil, host plant, water depth or breeding site. That divergence is what **produces the isolating mechanism itself** — the shift in flowering time, breeding time or mating signal that finally stops the gene flow. So when you write about a sympatric example, set the chain out in this order: different conditions → **selection favours different alleles in each group** → the difference this creates (for example a different flowering time) → **gene flow stops** → the two gene pools then diverge the rest of the way to **reproductive isolation**.

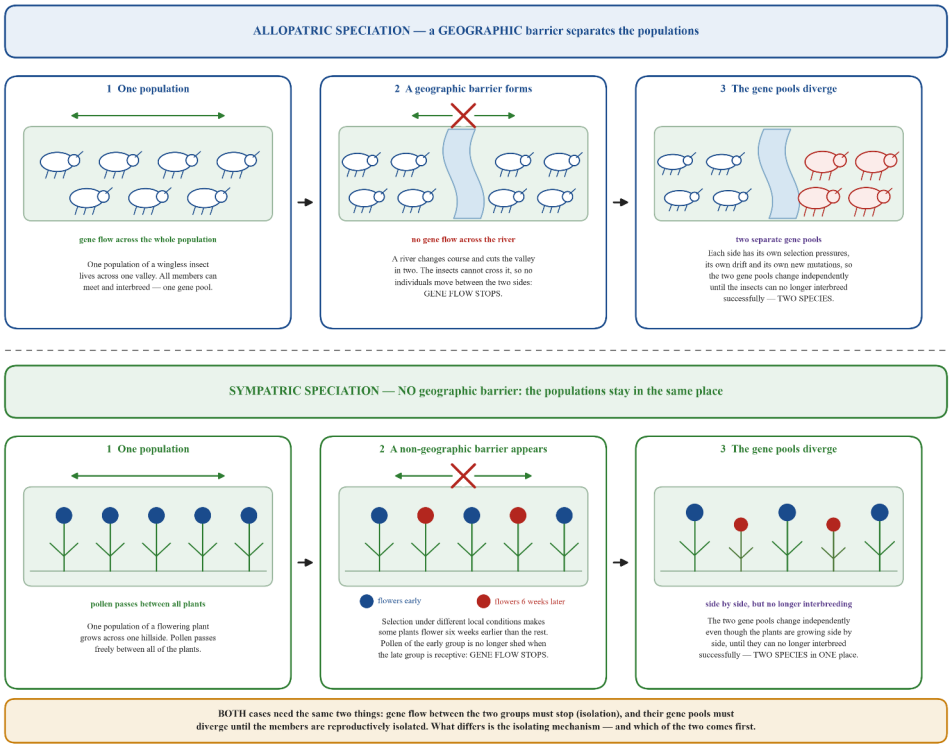
Evidence of speciation. Because speciation takes many generations, biologists look for evidence of the two requirements rather than watching the whole process:

- **Evidence of isolation** — a barrier or other mechanism can be identified, and few or no individuals (or pollen, or gametes) are observed moving between the two groups.

- **Evidence of genetic divergence** — the percentage of DNA sites at which the two populations differ increases with time since they were separated; populations that are still exchanging individuals stay genetically similar, while separated populations keep diverging. Differences in structure, physiology or behaviour also accumulate.
- **Evidence of reproductive isolation** — when members of the two groups are brought together they do not mate, or they mate but produce no offspring, or they produce offspring that are **sterile**. Any one of these outcomes means the two groups are separate species.

Two patterns of speciation. Speciation is classified by *what kind of thing* stopped the gene flow.

- **Allopatric speciation** (*allo-* = other, *patria* = homeland) — a **geographic barrier** physically separates the populations, so they are no longer in the same place.
- **Sympatric speciation** (*sym-* = together) — the populations stay **in the same location, with no geographic barrier**. Gene flow is stopped by some other isolating mechanism instead.



Allopatric speciation in more detail. The barrier is a physical feature of the landscape: a stretch of ocean, a mountain range, a river, a new lava flow, a gorge, or a band of habitat the organism cannot survive in. Whether a feature is a barrier **depends on the organism**. A wide river separates a population of flightless beetles but is no barrier at all to a bird; a treeless plain divides a forest-dwelling possum but not a grassland kangaroo. Once the barrier is in place, gene flow stops, each side experiences its own conditions, and the gene pools diverge.

Sympatric speciation in more detail. Here the two groups live side by side, so a map of where they grow or live shows no gap between them. Something other than distance stops them interbreeding — for example:

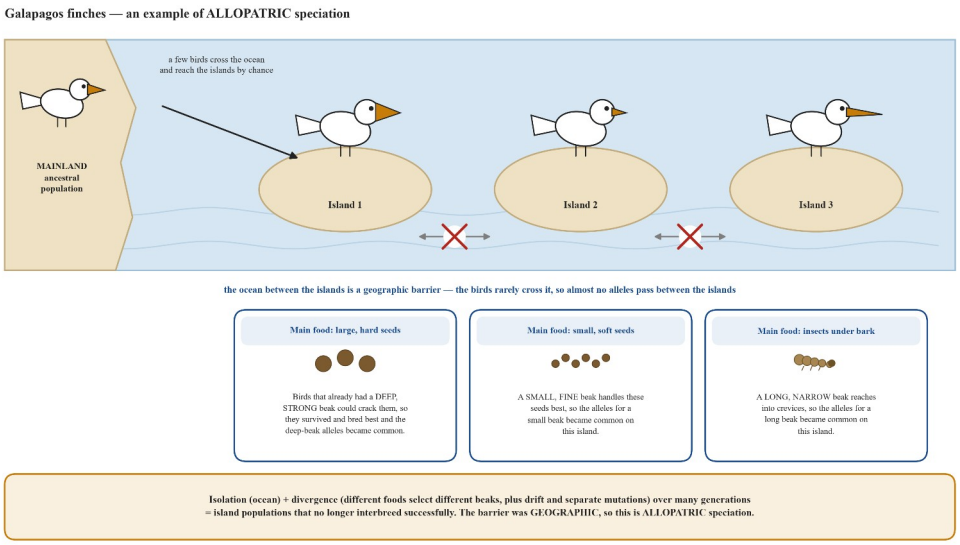
- they **breed or flower at different times of the year**, so the gametes of one group are never available when the other group is receptive;
- they use a **different habitat or host within the same area** (for example a different soil type, a different depth of the same lake, or a different food plant on which they also mate), so members of the two groups seldom meet at breeding time;
- their **mating behaviour or signals** differ, so members of one group do not respond to members of the other.

Each of these mechanisms usually arises **because the two groups are already being selected in different directions**: the group on one soil, host plant or water depth is favoured by different alleles from the group on the other, and a shift in flowering time, breeding time or mating signal is one of the differences that selection produces. Once gene flow has stopped for one of these reasons, the two gene pools diverge the rest of the way in exactly the same manner as in

allopatric speciation, and the outcome is exactly the same: reproductive isolation, and two species — this time occupying the same place.

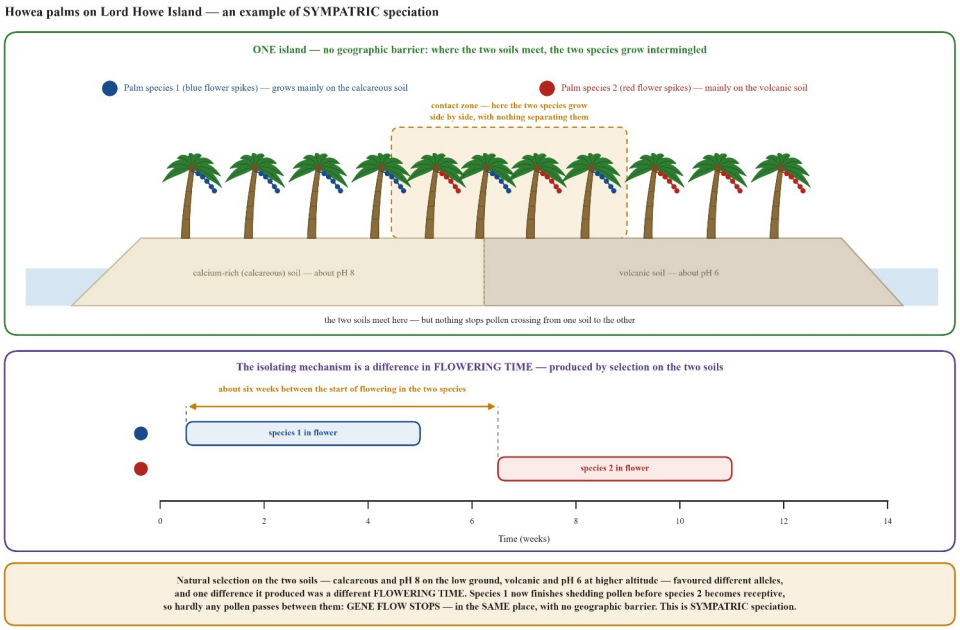
Feature	Allopatric speciation	Sympatric speciation
Where the two populations are	Separated in space	In the same location
What stops the gene flow	A **geographic** barrier	A **non-geographic** isolating mechanism (timing, habitat or host, behaviour)
Study-design example	**Galapagos finches**	**Howea palms, Lord Howe Island**
The other requirement	Genetic divergence of the two gene pools	Genetic divergence of the two gene pools
Which usually comes first	The barrier, and only then divergence	Divergence under different local conditions, which then creates the isolating mechanism
Final outcome	Reproductive isolation — two species	Reproductive isolation — two species

Galapagos finches — an example of allopatric speciation. The Galapagos are a group of volcanic islands in the Pacific Ocean, roughly a thousand kilometres from the coast of South America. A small number of finches from the mainland reached the islands by chance and established populations there.



- **Isolation.** The **ocean between the islands is a geographic barrier**. These small birds rarely fly from one island to another, so almost no alleles pass between the island populations: **gene flow stops**. Each island now holds its own gene pool.
- **Genetic divergence.** The main food available differs from island to island — large, hard seeds on one; small, soft seeds on another; insects living under bark on another. On each island **natural selection favours a different beak**: a deep, strong beak crushes hard seeds; a small, fine beak handles small soft seeds efficiently; a long, narrow beak reaches insects in crevices. Birds with the favoured beak on their own island survive and breed best, so the alleles for that beak become common **on that island only**. Genetic drift and each island's own new mutations add further independent change.
- **Outcome.** After many generations the island populations differ so much in beak shape, body size and song that birds from different islands no longer interbreed successfully. They are now **separate species**. Because the isolating agent was a **geographic barrier (the ocean)**, this is **allopatric speciation**.

Howea palms on Lord Howe Island — an example of sympatric speciation. Lord Howe Island is a small volcanic island in the Tasman Sea, several hundred kilometres east of the Australian mainland. Two species of *Howea* palm grow there and occur naturally nowhere else on Earth.



- **No geographic barrier.** The island is only a few kilometres across and both palms grow on it. One species grows mainly on the **calcium-rich (calcareous) soils of the low ground**, which are basic (about **pH 8**), and the other mainly on the more acidic **volcanic soils** higher up the island (about **pH 6**); but the two soils meet, and where they meet the two kinds of palm grow **side by side**. Nothing separates them in space, and the wind that carries their pollen crosses freely from one soil to the other.
- **Divergence begins — selection on two soils.** The palms of the low, calcareous ground and the palms of the higher, volcanic ground face **different conditions**: a different soil pH, different nutrients and a different altitude. **Natural selection favoured different alleles in each group**, and one of the differences it produced was a change in **when each group flowers**. All of this happened while the two groups were still growing together and pollen was still crossing between them.
- **Isolation.** The palms are **wind-pollinated**, and that shift has left the two kinds **flowering about six weeks apart**. By the time the second species becomes receptive, the first has finished shedding its pollen, so hardly any pollen is exchanged between them. **Gene flow stops** — without any geographic barrier. The difference in flowering time is therefore both a *product* of the early divergence and the *cause* of the isolation.
- **Further genetic divergence.** With gene flow now stopped, the two gene pools change completely independently. Each stays under selection suited to its own soil, and each accumulates its own drift and its own mutations, so the two diverge further in root chemistry, leaf form and seed size.
- **Outcome.** The two gene pools diverged until the palms became **reproductively isolated** — two species, in the **same location**. Because **no geographic barrier** was involved, this is **sympatric speciation**.

Watch out — the classic exam error. *Allopatric* and *sympatric* are told apart by **one thing only**: whether a **geographic barrier** separated the populations. Allopatric = a geographic barrier (finches, separated by ocean). Sympatric = **no** geographic barrier; the populations stay in the same place (Howea palms, separated only by flowering time). Do not decide from how far apart the organisms are on a map, or from how many islands are involved — the Galapagos finches are allopatric even though all the islands are in one group, and the Howea palms are sympatric even though they favour different soils. Both processes still require **both isolation and genetic divergence**, and both end in **reproductive isolation**; what differs is the isolating mechanism — and which of the two requirements comes first.

Worked examples

Example 1 — scaffolded

A population of flightless beetles lives on the slopes of a volcano. An eruption lays down a wide lava flow of bare, hot rock right across the slope. The beetles cannot cross it. Two thousand generations later, beetles from the two sides are placed together and will no longer mate with each other.

Identify the type of speciation that has occurred and explain how it happened.

Working	Comment
The lava flow is a **physical feature of the landscape** that the beetles cannot cross, so the two groups are **separated in space**. This is **allopatric speciation**.	Classify from the *kind* of barrier, not the distance. A landscape feature = a geographic barrier = allopatric.
Isolation: because no beetle can cross the lava, no individuals move between the two sides, so **gene flow stops** and the single gene pool becomes two separate gene pools.	Always name the barrier *and* state its consequence — that gene flow stops. Marks are lost for naming the barrier alone.
Genetic divergence: each gene pool now changes independently — different new mutations arise on each side, natural selection acts on each according to its own conditions, and genetic drift alters allele frequencies at random in each.	This is the second requirement. Name the processes; you are applying 9B here, not re-explaining it.
Over 2000 generations the two gene pools diverged so far that the beetles are now **reproductively isolated** — they no longer mate with each other, so they can no longer produce fertile offspring together.	Reproductive isolation is the *end point*, and it is what makes them two species rather than two separated populations.
∴ a geographic barrier caused isolation, the separated gene pools diverged, and the beetles are now **two species** — an example of allopatric speciation.	Finish with the conclusion the question asked for.

Example 2 — scaffolded

In one lake, a population of a small native fish spawns over the shallow rocky margins. Some individuals begin instead to spawn over the deep muddy bottom of the same lake, and the fish court and pair only at their own spawning site. After many generations the shallow-spawning and deep-spawning fish differ in body shape and no longer produce fertile offspring together.

Identify the type of speciation and explain how gene flow was stopped without any geographic barrier.

Working	Comment
Both groups live in the **same lake**; there is no ocean, mountain, river or other landscape feature between them. With **no geographic barrier**, this is **sympatric speciation**.	State explicitly that there is no geographic barrier — that is the whole basis of the classification.
Isolation: the fish court and pair **only at their own spawning site**, so shallow-spawners breed with shallow-spawners and deep-spawners with deep-spawners. Almost no gametes are exchanged between the two groups, so **gene flow stops**.	The isolating mechanism here is a **difference in breeding habitat within one location**. Explain how it prevents interbreeding — do not simply name it.
Genetic divergence: the two gene pools now change independently. Selection over the rocky shallows favours different body shapes from selection over deep mud, and each gene pool also accumulates its own drift and its own new mutations.	The same two requirements apply as in allopatric speciation. But note the order: in a sympatric case the difference that stops the gene flow — here, where each group spawns — is itself produced by selection acting while the two groups were still interbreeding. Divergence begins first and *creates* the isolation.
Divergence continued until the two groups became **reproductively isolated** — they no longer produce fertile offspring together — so they are now two species living in the **same lake**.	Two species in one location is the signature outcome of sympatric speciation.
∴ this is **sympatric speciation**: gene flow was stopped by a difference in **spawning habitat**, not by a	Name the isolating mechanism in the conclusion.

geographic barrier.

Example 3 — unscaffolded

A student writes: "A new mountain range rose across the range of a native mouse 500 generations ago. The mice on the two sides can no longer meet, so they are now two species."

Explain why this conclusion is not justified.

The student has described only the **first** requirement for speciation. The mountain range is a **geographic barrier**, so the two populations are **isolated** and **gene flow between them has stopped** — but isolation on its own is **not** speciation. While the two groups are apart, their separated gene pools must also undergo **genetic divergence**: each accumulates its own new **mutations**, is shaped by its own **natural selection** pressures, and is altered independently by **genetic drift**. Only when that divergence has gone far enough that the two groups are **reproductively isolated** — so that they could no longer interbreed to produce **fertile offspring** even if they were brought back together — are they two species. Until then they remain **one species in two separated populations**. Nothing in the student's statement shows that this test has been met; after only 500 generations the mice may well still interbreed successfully if reunited. ∴ the conclusion is not justified, because isolation has been demonstrated but genetic divergence to the point of reproductive isolation has not.

Example 4 — unscaffolded

Two populations of a native lizard live on either side of a broad desert. Researchers bring 30 individuals from each population into a large shared enclosure. The lizards court and mate readily with each other and produce healthy hybrid offspring. When those hybrid offspring are kept together for two breeding seasons, none of them produces any young.

State whether the two populations belong to the same species or to different species, and justify your answer.

The two populations belong to **different species**. A species is defined as a group whose members can interbreed to produce **fertile** offspring — offspring that can themselves reproduce. In this trial the lizards passed the first two tests: they **mated** with each other and they **produced offspring**. However, the hybrid offspring produced **no young at all** over two breeding seasons, which indicates that they are **sterile**. Because the hybrids cannot reproduce, no alleles can pass from one population's gene pool into the other's through them, so the two gene pools remain permanently separate. This is **reproductive isolation**, and it shows that the two gene pools have already **diverged** far enough during their separation by the desert for speciation to be complete. ∴ producing healthy offspring is not enough — because the hybrids are sterile, the two populations are separate species.

Practice questions

Scaffolded

Q1. Define each of the following terms: (a) species; (b) speciation; (c) reproductive isolation.

Q2. Speciation has two requirements. (a) Name the two requirements, and state which of them comes first in allopatric speciation. (b) State what happens to gene flow when a population becomes isolated. (c) Name the three processes that cause two separated gene pools to diverge.

Q3. The Galapagos finches are a study-design example of one pattern of speciation. (a) Name the geographic barrier that isolates the finch populations from one another. (b) Explain why the finches on one island came to have a different beak from those on another island. (c) Name the pattern of speciation this illustrates, and state the feature of the situation that allows you to classify it that way.

Q4. Two species of *Howea* palm grow on Lord Howe Island. (a) Name the pattern of speciation this illustrates, and state why. (b) Name the isolating mechanism that stops the two palms interbreeding, and state what caused the two kinds of palm to come to differ in this way. (c) Explain how that mechanism stops gene flow even though the palms grow side by side.

Q5. Consider these two situations.

- **Situation P** — the sea rises and floods a low neck of land, turning the end of a peninsula into an island and dividing a population of ground-dwelling skinks in two.
- **Situation Q** — in one grassland, males in a cricket population begin to produce two different songs, and females respond only to the song of their own group.

(a) Classify each situation as allopatric or sympatric speciation.
(b) State the single feature that allowed you to tell them apart.
(c) State the two requirements that both situations must still satisfy before two species exist.

Q6. Biologists look for evidence that speciation has occurred. (a) State one kind of evidence that two separated gene pools have undergone genetic divergence. (b) State one result of a breeding trial that would show two populations are reproductively isolated. (c) Explain why two populations isolated 8000 generations ago are more likely to be separate species than two populations isolated 200 generations ago.

Unscaffolded

Q7. Distinguish between allopatric speciation and sympatric speciation. *(2 marks)*

Q8. Explain why **both** isolation and genetic divergence are required for speciation, referring to what would happen if only one of them occurred. *(3 marks)*

Q9. Explain how the Galapagos finches provide evidence of **allopatric** speciation, referring to both requirements for speciation. *(4 marks)*

Q10. Explain how the *Howea* palms of Lord Howe Island provide evidence of **sympatric** speciation, referring to both requirements for speciation. *(4 marks)*

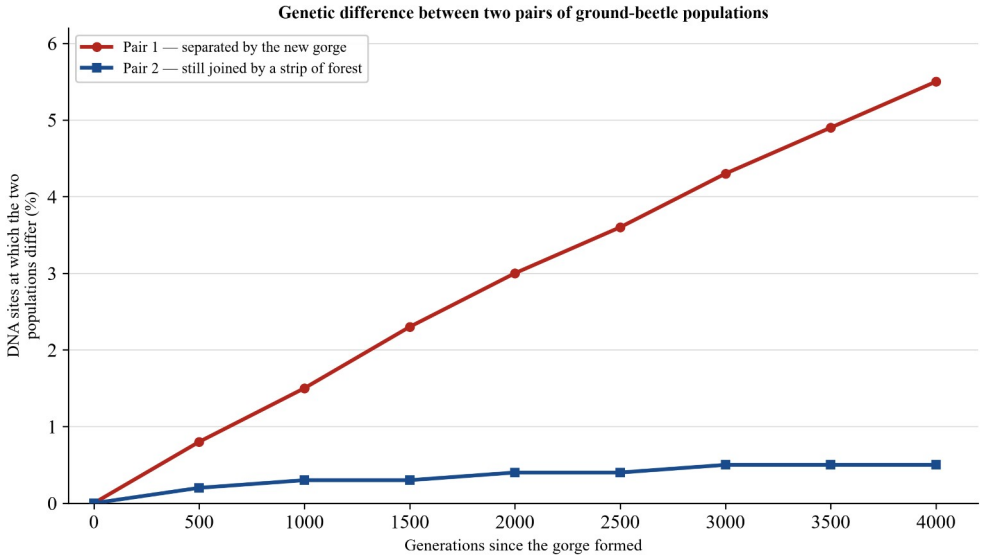
Q11. A student writes: "The Galapagos finches are an example of sympatric speciation, because all of the islands belong to one island group, and the *Howea* palms are allopatric because the two palms grow on different soils."

Identify the error, correct it, and explain the reasoning that gives the correct classifications. *(3 marks)*

Q12. A gorge separated two populations of a native frog for 400 generations. The gorge is then bridged by a landslide, the two populations meet, and they interbreed freely and produce offspring that go on to breed normally.

Deduce whether speciation has occurred, and state what would need to have been different for the outcome to be two species. *(3 marks)*

Q13. *(Data interpretation.)* A gorge formed across a forest, separating one pair of ground-beetle populations (Pair 1). A second pair of populations of the same beetle (Pair 2) was left joined by an unbroken strip of forest. For each pair, researchers estimated the percentage of DNA sites at which the two populations differ, at intervals after the gorge formed.



(a) Describe the trend shown by each pair.

(b) Explain the difference between the two pairs.

(c) State which pair is more likely to have become two species, and why. *(5 marks)*

Q14. (a) Suggest a geographic barrier that could isolate two populations of each of the following, and in each case state why it would stop gene flow: (i) a ground-dwelling snail living in one patch of rainforest; (ii) a fish living in one river system; (iii) a low shrub growing on one mountain top. (b) Explain why a landscape feature that is a barrier to one species may be no barrier at all to another. *(4 marks)*

Q15. *(Data — table.)* Four populations of a small native fish (W, X, Y and Z) were tested by placing individuals from two populations together and recording the results.

Cross	Did they mate?	Offspring produced?	Were the offspring fertile?
W × X	Yes	Yes	Yes
W × Y	Yes	Yes	No
W × Z	No	—	—
X × Y	Yes	Yes	No
Y × Z	No	—	—

Deduce how many species are represented by the four populations, and justify your answer using the results. *(4 marks)*

Q16. Explain how mutation, natural selection and genetic drift together cause two isolated gene pools to diverge from each other. *(3 marks)*

Q17. In one orchard, a native fly feeds on the fruit of a local tree and mates on that same fruit. Some flies begin to feed and mate on the fruit of a recently planted introduced tree growing in the same orchard.

Explain how this could lead to sympatric speciation. *(3 marks)*

Q18. *(Evaluate.)* Two populations of a native freshwater crayfish have lived in two separate river systems for a long time. A commentator examines them and states: “The crayfish of the two rivers now differ clearly in body size, claw shape and colour, so they must be two separate species.”

Evaluate this statement. *(3 marks)*

Q19. *(Method evaluation.)* Two populations of a native grasshopper have been isolated by a mountain range for a long time. To test whether they are now separate species, researchers place 20 males and 20 females from each population together in one enclosure, record every mating, rear all offspring produced, and then record what proportion of those offspring go on to produce young of their own.

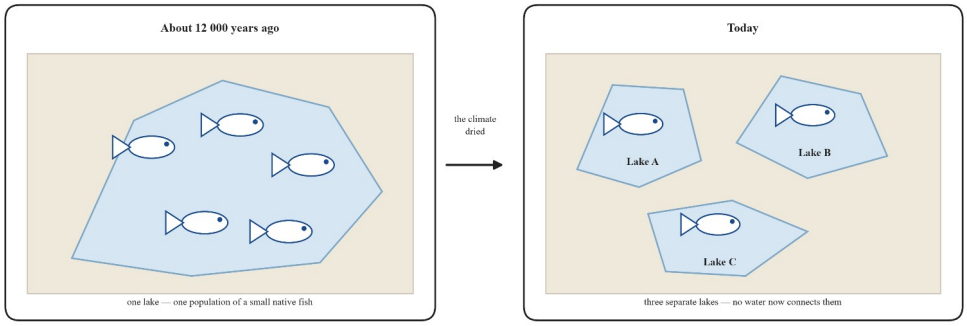
(a) Identify the independent variable.

(b) Identify the dependent variable that shows whether the populations are separate species.

(c) State one variable that should be controlled.

(d) State one limitation of this method. *(4 marks)*

Q20. *(Synthesis.)* About 12 000 years ago a single large lake held one population of a small native fish. The climate then dried, and the lake shrank into the three separate lakes shown below. No water now connects them.



Researchers compared the fish now living in the three lakes. The percentage of DNA sites at which the populations differ is: A and B, 3.1%; A and C, 3.4%; B and C, 0.9%. Fish from lakes A and B were placed together in a tank: they courted and mated repeatedly over a full breeding season, but no eggs were ever fertilised and no offspring were produced. Fish from lakes B and C mated, produced offspring, and those offspring bred normally.

(a) Identify the pattern of speciation occurring in these lakes, and justify your answer.

(b) Explain how the two requirements for speciation have been met since the lake dried.

(c) Using the breeding results, state how many species are present in the three lakes today and justify your answer.

(d) Suggest one reason why the fish of lakes B and C differ from each other less than either differs from the fish of lake A. *(7 marks)*

Answers

Q1. (a) A group of organisms whose members can interbreed under natural conditions to produce fertile offspring. (b) The process by which one species gives rise to two or more new species. (c) The condition in which two groups can no longer interbreed to produce fertile offspring, even if they meet. **Q2.** (a) Isolation (gene flow stops) and genetic divergence; in allopatric speciation isolation comes first. (b) Gene flow stops — no alleles pass between the two groups. (c) Mutation, natural selection and genetic drift. **Q3.** (a) The ocean between the islands. (b) The main food differs between islands, so natural selection favoured a different beak on each island and those alleles became common there. (c) Allopatric speciation — the populations were separated by a geographic barrier. **Q4.** (a) Sympatric — the two palms grow in the same location on one small island, with no geographic barrier between them. (b) A difference in flowering time (about six weeks apart), caused by natural selection acting differently on the two soils the palms grow on — calcareous and about pH 8 on the low ground, volcanic and about pH 6 higher up. (c) The palms are wind-pollinated; one has finished shedding pollen before the other becomes receptive, so hardly any pollen is exchanged and gene flow stops. **Q5.** (a) P = allopatric; Q = sympatric. (b) Whether a geographic barrier separates the populations. (c) Isolation — gene flow between the two groups must stop — and genetic divergence of the two gene pools until reproductive isolation results. **Q6.** (a) The percentage of DNA sites at which the two populations differ increases with time since separation (or accumulating differences in structure, physiology or behaviour). (b) They do not mate, or they mate but produce no offspring, or the offspring produced are sterile. (c) The longer the isolation, the more generations of mutation, selection and drift have acted independently, so the gene pools have diverged further and are more likely to have reached reproductive isolation. **Q7.** Allopatric = a geographic barrier separates the populations; sympatric = no geographic barrier, the populations remain in the same location and gene flow is stopped some other way. **Q8.** Isolation alone leaves one species, because the separated populations could still interbreed successfully; divergence alone cannot reach reproductive isolation while gene flow continues, because the constant mixing of alleles keeps the two gene pools similar; both together produce reproductive isolation and therefore two species. **Q9.** The ocean between the islands is a geographic barrier, so gene flow between island populations stops; different foods on each island select for different beaks, and drift and separate mutations add further independent change; the populations diverge until they no longer interbreed successfully; the barrier was geographic, so this is allopatric. **Q10.** Both palms grow on one small island with no geographic barrier; selection on the two soils (calcareous, pH 8, low ground versus volcanic, pH 6, higher up) favoured different alleles in the two groups and produced a difference in flowering time of about six weeks, so hardly any pollen is exchanged and gene flow stops; the two gene pools then diverge further and independently; the result is reproductive isolation in the same location, so this is sympatric. **Q11.** The two classifications are swapped. The finches are allopatric (isolated by the ocean, a geographic barrier) and the palms are sympatric (same island, no geographic barrier — isolated by flowering time). Classification depends only on whether a geographic barrier was involved, not on the number of islands or on a difference in soil. **Q12.** No — speciation has not occurred; they are still one species, because they interbreed and produce fertile offspring, so there is no reproductive isolation. The gene pools would have had to diverge much further (more generations of isolation, selection, drift and mutation) for two species to result. **Q13.** (a) Pair 1 increases steadily throughout, from 0% to about 5.5% at 4000 generations; Pair 2 rises only slightly and more and more slowly — about 0.3% by 1000 generations and about 0.5% by 3000 — then stays level at about 0.5% to 4000 generations. (b) Pair 1 is separated by the gorge so gene flow has stopped and the gene pools diverge independently; Pair 2 is still joined by forest so gene flow continues and mixing keeps the two gene pools similar. (c) Pair 1 — its gene pools have diverged far more, so reproductive isolation is far more likely. **Q14.** (a) (i) A wide band of dry, open ground opening up through the forest — the snail cannot survive crossing bare, dry ground, so no snails pass between the two patches of rainforest. (ii) A waterfall, or the drying of the channel joining two parts of the system — the fish cannot pass along the water. (iii) A deep ravine or a landslide scar cutting across the summit — it splits the shrubs into two patches too far apart for pollen or seeds to cross. (b) A feature is a barrier only if it stops that organism (or its pollen or gametes) moving across it, and species differ in how far and by what means they can travel. **Q15.** Three species: W and X are one species (fertile offspring); Y is a second species (mates with W and X but the offspring are sterile); Z is a third species (will not mate with W or Y at all). **Q16.** Mutation produces different new alleles in each isolated gene pool; natural selection favours different alleles in each because the conditions differ; genetic drift alters allele frequencies at random and independently in each — so the two gene pools become steadily more different. **Q17.** Both kinds of tree grow in the same orchard, so there is no geographic barrier; because the flies mate on the fruit they feed on, flies using different trees rarely meet to mate, so gene flow stops; the two gene pools then diverge until they are reproductively isolated — sympatric speciation. **Q18.** Partly correct: the visible differences are good evidence that the two gene pools have undergone genetic divergence. But looking different is not the test of a species — the test is reproductive, and the two populations remain one species for as long as they could still interbreed to produce fertile offspring. The statement

should therefore be rejected on the evidence given; a breeding trial is needed. **Q19.** (a) The pairing type — whether the two parents come from the same population or from the two different populations. (b) The proportion of the offspring produced that go on to produce young of their own (offspring fertility). (c) Any one of: temperature, food supply, enclosure size, age of the grasshoppers, number of individuals per cross. (d) Any one of: behaviour in an enclosure may differ from behaviour in the wild; the sample of 20 may not represent the whole population; two breeding seasons may be too short to show fertility. **Q20.** (a) Allopatric — the populations were separated by a geographic barrier (dry land between the three lakes). (b) Isolation: no water connects the lakes, so no fish move between them and gene flow stopped; divergence: each gene pool has since changed independently through selection, drift and its own mutations, shown by DNA differences of up to 3.4%. (c) Two species — the lake A fish are one species (they mate with B fish but no offspring at all are produced) and the fish of lakes B and C together are the other (B and C give fertile offspring). (d) Any reasoned answer, for example: lakes B and C may have remained connected for longer, so their gene pools have had fewer generations of independent change; or their conditions are more similar, so selection has acted on them in the same direction.

Fully-worked solutions

Q1. (a) A **species** is a group of organisms whose members can **interbreed under natural conditions to produce fertile offspring** — offspring that can themselves reproduce. (b) **Speciation** is the process by which **one species gives rise to two or more new species**, through the isolation and genetic divergence of its gene pools. (c) **Reproductive isolation** is the condition in which the members of two groups **can no longer interbreed to produce fertile offspring**, even when they meet; it is the point at which the two groups are separate species.

Q2. (a) **Isolation** (gene flow between the two groups stops) and **genetic divergence** (the two gene pools change independently). In **allopatric** speciation isolation comes first: the barrier appears, and only then can the two gene pools change independently. (In a **sympatric** case it is usually the other way round — selection begins to drive the two groups apart while they are still interbreeding, and it is that divergence which creates the mechanism that stops the gene flow — but both requirements must still be met.) (b) **Gene flow stops**: no alleles pass from one group's gene pool into the other's, so the single gene pool becomes **two separate gene pools**. (c) **Mutation** (which supplies new alleles), **natural selection** (which favours different alleles in each group's conditions) and **genetic drift** (which changes allele frequencies at random in each).

Q3. (a) The **ocean (the stretch of sea between the islands)**. (b) The **main food differs from island to island** — for example large, hard seeds on one island and small, soft seeds on another. On each island, the birds whose beak best handles the food available there **survive and reproduce best**, so the **alleles for that beak shape become common on that island**. Because the ocean prevents birds moving between islands, these changes cannot spread from one island to another, so each island's beak becomes different. (c) **Allopatric speciation**, because the populations were separated by a **geographic barrier** (the ocean between the islands).

Q4. (a) **Sympatric speciation**, because the two palms grow **in the same location** — on one small island, side by side where their soils meet — with **no geographic barrier** between them. (b) A difference in **flowering time**: the two kinds of palm flower about **six weeks apart**. That difference was produced by **natural selection acting differently on the two soils** — one group grows on the calcareous soil of the low ground (about **pH 8**), the other on the more acidic volcanic soil higher up (about **pH 6**) — so **different alleles were favoured in each group**, and a change in flowering time was one of the differences that resulted. (c) The palms are **wind-pollinated**. Because the first species has **finished shedding its pollen before the second species becomes receptive**, pollen from one is almost never available when the other can be fertilised. So although the palms are close enough for the wind to carry pollen between them, they are effectively separated **in time**, and **gene flow stops**.

Q5. (a) **Situation P = allopatric speciation**; **Situation Q = sympatric speciation**. (b) Whether a **geographic barrier** separates the populations. In P the risen sea is a physical landscape feature dividing the skinks; in Q the crickets remain in the same grassland and nothing physical separates them. (c) Both must still satisfy **isolation** (gene flow between the two groups stops) and **genetic divergence** (the two gene pools change independently through selection, drift and their own mutations) until **reproductive isolation** results.

Q6. (a) The **percentage of DNA sites at which the two populations differ increases** with the time since they were separated (accumulating differences in structure, physiology or behaviour are also acceptable evidence). (b) Any one

of: the two groups **will not mate**; they mate but **no offspring are produced**; or offspring are produced but they are **sterile**. (c) The longer the isolation, the **more generations** over which mutation, natural selection and genetic drift have acted **independently** on the two gene pools. After 8000 generations the gene pools have accumulated far more independent change than after 200, so they are much more likely to have diverged past the point of **reproductive isolation**.

Q7. In **allopatric** speciation, a **geographic barrier** (such as an ocean, mountain range or river) physically **separates the populations**, so they no longer occupy the same place, and gene flow stops. In **sympatric** speciation there is **no geographic barrier**: the populations remain **in the same location**, and gene flow is stopped by another isolating mechanism, such as a difference in flowering or breeding time. *(1 mark: allopatric — geographic barrier / separated in space; 1 mark: sympatric — no geographic barrier / same location.)*

Q8. If only isolation occurred, the two populations would be separated but would still be **one species**, because their gene pools would not yet differ enough to prevent them interbreeding successfully if they met again. **If divergence began but gene flow never stopped**, it could not be carried through: while **gene flow continues**, alleles are constantly exchanged between the two groups, and that mixing pulls the two gene pools back towards each other, so the differences never build up as far as reproductive isolation. Isolation is therefore what allows independent change to continue, and divergence is what actually creates the difference. **Only when both have occurred** — gene flow stopped, and the two gene pools changed independently far enough — do the two groups become **reproductively isolated**, which is what makes them two species. *(1 mark: isolation alone leaves one species; 1 mark: while gene flow continues the mixing of alleles keeps the gene pools similar, so divergence cannot reach reproductive isolation; 1 mark: both together give reproductive isolation / two species.)*

Q9. Isolation. The Galapagos finch populations live on separate islands, and the **ocean between the islands is a geographic barrier**: the birds rarely cross it, so almost no alleles pass between the island populations and **gene flow stops**. Each island holds its own gene pool. **Divergence.** The **main food differs between islands** — large hard seeds, small soft seeds, or insects under bark — so **natural selection favours a different beak** on each island (deep and strong, small and fine, or long and narrow). The favoured alleles become common **on that island only**, and **genetic drift** and each island's own **new mutations** add further independent change. **Outcome.** Over many generations the populations diverge so far in beak shape, body size and song that birds from different islands **no longer interbreed successfully** — they are **reproductively isolated** and are therefore **separate species**. Because the isolating agent was a **geographic barrier**, this is **allopatric** speciation. *(1 mark: ocean = geographic barrier, gene flow stops; 1 mark: different foods select for different beaks on each island; 1 mark: drift and separate mutations add independent change; 1 mark: reproductive isolation / separate species, classified allopatric because the barrier was geographic.)*

Q10. No geographic barrier. Both *Howea* species grow on **one small island**, and where the calcareous and volcanic soils meet the two kinds of palm grow **side by side**, so nothing separates them in space. **Divergence begins.** The two groups grow on **different soils** — calcareous and about **pH 8** on the low ground, volcanic and about **pH 6** higher up — so **natural selection favoured different alleles in each group**, and one of the differences it produced was a change in **when each group flowers**. **Isolation.** The palms are **wind-pollinated**, and that shift has left the two kinds **flowering about six weeks apart**; the first has finished shedding pollen before the second becomes receptive, so hardly any pollen passes between them and **gene flow stops** — with no geographic barrier anywhere in the story. **Further divergence and outcome.** With gene flow now stopped, the two gene pools change completely **independently** — each stays under selection suited to its own soil, and each accumulates its own **genetic drift** and its own **new mutations** — until the palms are **reproductively isolated**: two species occupying the **same location**. Because **no geographic barrier** was involved, this is **sympatric** speciation. *(1 mark: one island, no geographic barrier; 1 mark: selection on the different soils/pH favoured different alleles and produced the difference in flowering time; 1 mark: flowering about six weeks apart, so pollen is not exchanged and gene flow stops; 1 mark: the gene pools diverge on to reproductive isolation in the same location, classified sympatric.)*

Q11. The error: the student has **swapped the two terms**. **The correction:** the Galapagos finches are **allopatric** and the *Howea* palms are **sympatric**. **The reasoning:** the two patterns are distinguished by **one thing only — whether a geographic barrier separated the populations**. The finch populations are separated by the **ocean between the islands**, which is a geographic barrier, so their speciation is **allopatric**; the fact that the islands form one group is irrelevant. The two palms grow on the **same small island with no geographic barrier between them** and are isolated only by a difference in **flowering time**, so their speciation is **sympatric**; growing mainly on different soils in the same place is a habitat preference, not a geographic barrier. (The difference in soil is not irrelevant — selection on the two

soils is what produced the difference in flowering time — but a soil boundary does not separate the palms **in space**, which is what "allopatric" requires.) *(1 mark: identifies that the two classifications have been swapped; 1 mark: correct classifications stated; 1 mark: reasoning — classification depends only on the presence of a geographic barrier.)*

Q12. Speciation has not occurred — the two populations are **still one species**. When they met, they **interbred freely and produced offspring that went on to breed normally**, that is, the offspring were **fertile**. This is exactly the test of a single species, so the two groups are **not reproductively isolated**. The gorge supplied **isolation** for 400 generations, but that alone is not speciation. For the outcome to have been two species, the **genetic divergence** of the two gene pools would have had to go much further — many more generations of independent **mutation**, **natural selection** and **genetic drift** — until the two groups could no longer mate, or produced no offspring, or produced only sterile offspring. *(1 mark: no speciation / still one species; 1 mark: because they produce fertile offspring, so there is no reproductive isolation; 1 mark: far greater divergence over many more generations would have been needed.)*

Q13. (a) **Pair 1 (separated by the gorge)** shows a **steady increase** in genetic difference across the whole period, rising from **0% at the time the gorge formed to about 5.5% after 4000 generations**. **Pair 2 (still joined by forest)** shows only a **small increase, and one that slows as it goes** — about **0.3% by 1000 generations** and about **0.5% by 3000 generations** — after which it **stays level at about 0.5%** to 4000 generations. (b) The difference is caused by **gene flow**. In Pair 1 the gorge stopped individuals moving between the populations, so **gene flow stopped** and each gene pool has since accumulated its **own** mutations and been shaped by its **own** selection and drift — the differences therefore build up generation after generation. In Pair 2 the unbroken strip of forest allows beetles to keep moving between the populations, so **gene flow continues** and the constant exchange of alleles **keeps the two gene pools similar**, which is why the difference stops rising. (c) **Pair 1**, because its two gene pools have **diverged much further** (about eleven times the difference of Pair 2), so they are far more likely to have reached **reproductive isolation**. *(a: 2 marks — 1 for each pair's trend, with data quoted; b: 2 marks — 1 for no gene flow allowing independent divergence in Pair 1, 1 for continuing gene flow keeping Pair 2's gene pools similar; c: 1 mark — Pair 1, with reason.)*

Q14. (a) **(i) Rainforest snail:** a **wide band of dry, open ground** — a strip of grassland or bare rock opening up through the forest as the climate dries — cutting the forest patch in two. The snail must stay damp and cannot survive the crossing, so no snails pass between the two patches. **(ii) Fish:** a **waterfall**, or the **drying of the channel** that joined two parts of the river system — the fish cannot travel along the water past the obstacle, so the two groups never meet. **(iii) Mountain-top shrub:** a **deep ravine or landslide scar cutting across the summit** — it divides the shrubs into two patches that are too far apart, and too inhospitable in between, for pollen or seeds to cross, leaving each patch breeding only within itself. (b) A landscape feature counts as a barrier only if it actually **stops that particular organism (or its pollen, seeds or gametes) crossing**. Species differ enormously in how far and by what means they can travel, so a mountain range that divides a population of burrowing frogs completely is no obstacle at all to an eagle that crosses it in minutes. *(a: 3 marks — 1 for each organism, requiring a plausible barrier and why it stops gene flow; b: 1 mark.)*

Q15. There are **three species** among the four populations.

- **W and X are the same species.** They mate, produce offspring, and those offspring are **fertile** — which is the definition of a single species, so alleles can still flow between their gene pools.
- **Y is a separate species from W and X.** It mates with both W and X and offspring are produced, but those offspring are **sterile**. Because the hybrids cannot reproduce, no alleles pass between Y's gene pool and the W/X gene pool, so Y is **reproductively isolated** from them.
- **Z is a third species.** Z **will not mate at all** with either W or Y, so no offspring are possible and it is reproductively isolated from every other population tested.

The three species are therefore **W together with X**, **Y alone** and **Z alone**. *(1 mark: W and X are one species, because their offspring are fertile; 1 mark: Y is separate, because its hybrids with W and X are sterile; 1 mark: Z is separate, because it does not mate at all; 1 mark: correct total of three species.)*

Q16. Once two gene pools are isolated, all three processes act on each of them **separately**. **Mutation** is the only source of **new alleles**, and because mutations occur at random, the new alleles that appear in one gene pool are **not the same** as those appearing in the other. **Natural selection** then acts on each population according to **its own conditions** — food, predators, climate — so a different set of alleles is favoured in each, and different alleles become common. **Genetic drift** changes allele frequencies by **chance** from generation to generation, and because it is random it pushes

the two gene pools in **different, unrelated directions** (its effect is strongest in small populations). Because no gene flow can carry any of these changes from one gene pool to the other, the differences **accumulate**, and the two gene pools steadily diverge. *(1 mark: mutation supplies different new alleles in each; 1 mark: selection favours different alleles because conditions differ; 1 mark: drift changes frequencies at random and independently in each.)*

Q17. Both trees grow in the **same orchard**, so the two groups of flies are in the **same location** and there is **no geographic barrier** between them. Because the flies **mate on the fruit they feed on**, a fly that has switched to the introduced tree meets and mates almost only with other flies on that tree, and flies on the local tree do the same — so the two groups **rarely interbreed** and **gene flow between them stops**. With gene flow stopped, the two gene pools change **independently**: selection favours the flies best suited to the timing, chemistry and texture of their own host fruit, and drift and separate new mutations add further change. If this continues long enough the two groups become **reproductively isolated** and are two species living in one orchard — **sympatric speciation**. *(1 mark: same location, no geographic barrier; 1 mark: mating on the host fruit stops interbreeding, so gene flow stops; 1 mark: gene pools then diverge to reproductive isolation — sympatric.)*

Q18. The statement is **partly supported but not justified**. Its strength is that the differences in size, claw shape and colour are **evidence of genetic divergence**: once the two river systems separated the crayfish and gene flow stopped, each gene pool accumulated its own mutations and was shaped by its own selection and drift, and accumulating differences in **structure** are exactly what that produces. Divergence is one of the two requirements for speciation, so the commentator has real evidence of it. Its weakness is that the commentator has used the **wrong test**. A species is defined **reproductively**, not by appearance: the two populations are still **one species** for as long as their members could **interbreed to produce fertile offspring**. Looking different does not show that **reproductive isolation** has been reached — two populations can differ visibly and still interbreed freely, and part of the difference may even be caused by the different environments rather than by the genes. **Judgement: the statement should be rejected on the evidence given.** To support it the commentator would need a **breeding trial** showing that the two groups will not mate, produce no offspring, or produce only **sterile** offspring. *(1 mark: what is correct — the structural differences are evidence that the gene pools have diverged; 1 mark: what is wrong — the test of a species is reproductive, so visible difference does not establish reproductive isolation; 1 mark: a clear judgement, with the breeding evidence that would be needed.)*

Q19. (a) The **independent variable** is the **pairing type** — that is, whether the two parents come from the **same population** or from the **two different populations**. (The two combinations are the levels of the variable, not the variable itself.) (b) The **dependent variable** is the **proportion of the offspring produced that go on to produce young of their own** — that is, the **fertility of the offspring**. This is the variable that matters, because two populations are separate species if their hybrid offspring are sterile; simply counting matings or offspring would not answer the question. (c) Any one of: **temperature**; **food supply and water**; **enclosure size**; **the age and health of the grasshoppers**; **the number of individuals used in each cross**. (d) Any one of: grasshoppers may **behave differently in an enclosure** from how they behave in the wild, so a mating observed in captivity may not occur naturally; **20 individuals may not represent** the genetic variation of the whole population; **two breeding seasons may be too short** for slow-maturing offspring to show whether they are fertile. *(1 mark each for (a), (b), (c) and (d).)*

Q20. (a) This is **allopatric speciation**. The single lake dried into **three separate lakes with no water connecting them**, so the fish populations are separated by a **geographic barrier** — the dry land between the lakes — which the fish cannot cross. (b) **Isolation:** once the lakes separated, no fish could move between them, so **gene flow stopped** and the one gene pool became three separate gene pools. **Genetic divergence:** each gene pool has since changed **independently** through its own new **mutations**, **natural selection** suited to the conditions of its own lake, and **genetic drift**. The DNA evidence confirms this has happened: the populations now differ at up to **3.4%** of DNA sites, whereas 12 000 years ago they were one interbreeding population. (c) There are **two species** in the three lakes today. Fish from **A and B** courted and mated over a whole breeding season but **no offspring were produced at all** — no eggs were fertilised — so no alleles can pass from one of those gene pools into the other and A and B are **reproductively isolated**: they are different species. Fish from **B and C** produced offspring that **bred normally**, so their offspring are **fertile** and B and C are still **one species**. A and C were not tested directly, but they must also be different species, because C belongs to the same species as B and A does not. The two species are therefore the **lake A population** and the **combined lake B and lake C populations**. (d) The fish of lakes B and C may have remained **connected to each other for longer** than either was connected to lake A as the lake dried, so their gene pools have had **fewer generations of independent change**. (Alternatively: lakes B and C may have **more similar conditions**, so natural selection has acted on both gene pools in the **same direction**.) ∴ isolation by the drying of the lake, followed by

independent divergence of the separated gene pools, has already produced two species from one. *(a: 2 marks — allopatric, and the justification that a geographic barrier separates the lakes; b: 2 marks — 1 isolation/gene flow stops, 1 divergence by mutation, selection and drift with the DNA evidence; c: 2 marks — 1 for two species, 1 for justifying both the failure of A × B to produce any offspring and the fertile B × C offspring; d: 1 mark — any reasoned suggestion.)*