Course Companion

International Baccalaureate · IB Diploma · Biology · HL

Every topic handout in one volume

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Water

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Scope and prerequisites

A drop can stand on a leaf while water inside the same plant moves up a narrow stem. A seal can swim in water that would cool an unprotected human rapidly. These observations connect water's molecular structure with the conditions organisms face.

This handout teaches water as life's medium, polarity and hydrogen bonding, cohesion and adhesion, solvent behaviour, and the consequences of water's physical properties for animals. Sections marked **AHL** are for Higher Level only.

Prerequisites: atoms, molecules, shared electrons, positive/negative charge and energy transfer. All numerical datasets below are original, invented teaching examples unless a physical constant is explicitly given.

Water molecules and the medium for life

The earliest cells arose in an aqueous environment. Water remains the main medium for most cellular reactions: reactants can dissolve, move and meet enzymes in the watery contents of cells. Blood plasma and plant transport fluids also carry dissolved substances. Living on land does not remove this dependence; terrestrial organisms maintain internal aqueous environments and must replace water losses.

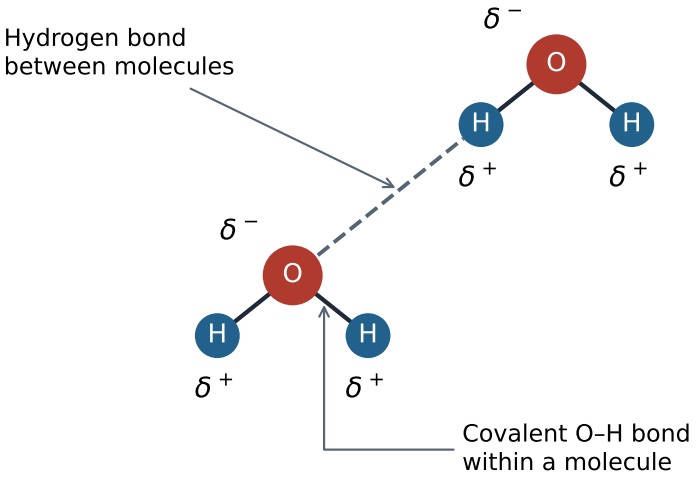
Water is not simply empty space between molecules. Its interactions affect protein folding, membrane organization, transport and temperature. A dry seed may survive with greatly reduced activity, but this does not mean its active metabolism is independent of water.

■ From shared electrons to partial charges

In each O–H **polar covalent bond**, electrons are shared unequally: oxygen attracts the shared electrons more strongly than hydrogen. Oxygen has a partial negative charge, δ^-; the hydrogens have partial positive charges, δ^+. These are partial charges within a neutral molecule, not evidence that ordinary water consists entirely of separate hydrogen and oxygen ions.

Water has a bent shape. Its bond polarities do not cancel, so the molecule has a positive side and a negative side. A **hydrogen bond** is an attraction between a partially positive hydrogen bonded to an electronegative atom and a suitable electronegative atom on another molecule. In water, this is commonly represented as an attraction between H on one molecule and O on another.

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Solid O–H bonds join atoms within a molecule; the dashed attraction joins different molecules.

When drawing water, label O and both H atoms, show the partial charges, and place the dashed hydrogen bond between the appropriate H and O on different molecules. Do not draw it between two hydrogens or use the same symbol for a covalent bond and a hydrogen bond.

Individual hydrogen bonds are weaker than covalent O–H bonds. In liquid water they continually form and break as molecules move. Many interactions together produce strong effects at the scale of a droplet or a column of water. Heating increases molecular motion and changes these interactions. Ordinary boiling separates intact water molecules; it does not normally decompose them into hydrogen and oxygen by breaking their covalent bonds.

Cohesion, surface tension and water transport

Cohesion is attraction between molecules of the same substance. Hydrogen bonding contributes to cohesion between water molecules. Inside a liquid, a molecule has neighbours in many directions; at the surface the environment is different. Creating more surface requires energy, so the surface tends to resist stretching. This is **surface tension**. The surface is still liquid, not a solid skin.

A pond skater can use the water surface as a habitat. Its water-repellent legs spread the load and deform the surface, allowing surface-tension forces to help support it. This does not mean any animal can walk on water: mass, contact shape, wetting and the liquid's surface properties all matter. Detergent can alter surface tension and wetting, so it changes the system rather than merely colouring the water.

In a plant's **xylem**, evaporation from leaf surfaces helps create tension, meaning a

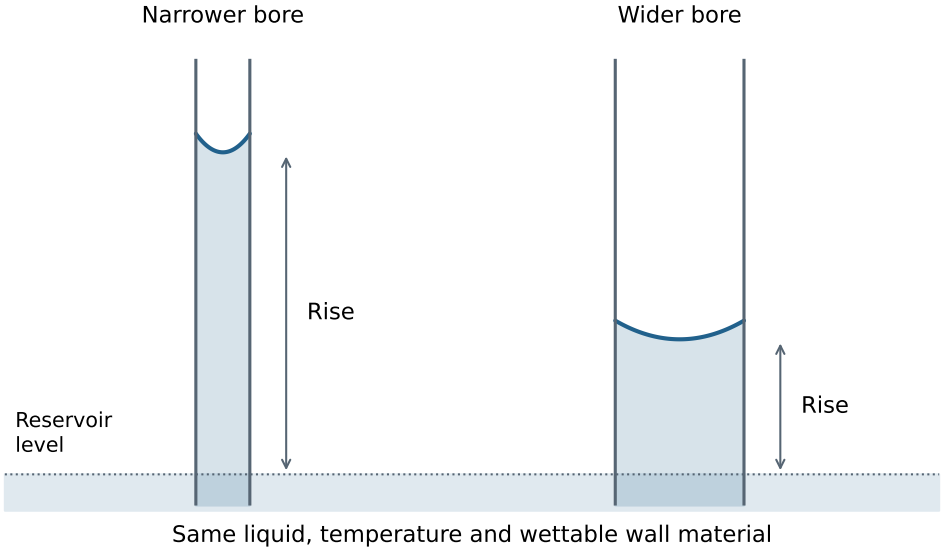
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pulling stress or pressure below the surrounding reference pressure. Cohesion helps the water column transmit this pull through the plant. Water molecules do not actively climb because they "want" to reach the leaves. The explanation links evaporation, tension and an interconnected column of water. A gas-filled break can interrupt that column; cohesion is strong but not unlimited.

Adhesion and capillary action

Adhesion is attraction between different substances. Water can interact with polar or charged groups on plant cell-wall materials and soil particles. It does not adhere equally to every surface; a nonpolar waxy surface may resist wetting.

In a narrow wettable space, adhesion at the wall and cohesion within the liquid help produce **capillary action**: water can rise against gravity, with a curved meniscus. In an ideal wettable tube, a narrower bore can support a higher equilibrium rise. The upward surface-tension force is eventually balanced by the weight of the raised water. Surface chemistry and tube radius matter; "narrow" alone does not guarantee that a liquid rises.



The sketch compares equilibrium rises under the same conditions; it is not a scale drawing of a whole plant.

Capillary spaces in soil and plant cell walls help retain and distribute water. Distinguish this from explaining the entire height of water transport in a tall tree. Adhesion helps maintain contact with walls and cohesion links the column, but transpiration-driven tension is central to long-distance ascent. A glass-tube demonstration does not by itself prove a complete biological transport mechanism.

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Water as a solvent: what dissolves and why?

A solvent is the medium in which a solute dissolves. Water's partial charges interact with ions and with suitable polar groups. Around a dissolved positive ion, the oxygen sides of nearby water molecules tend to face the ion; around a negative ion, the hydrogen sides tend to face it. The ions become surrounded by water rather than turning into water molecules.

Hydrophilic substances interact favourably with water. Many ions, sugars and other small polar molecules dissolve sufficiently to move in aqueous transport fluids. Glucose carried in blood plasma can reach tissues; mineral ions move in xylem sap. Dissolved reactants can diffuse and participate in enzyme-controlled metabolism. Solubility still depends on the substance, temperature and concentration: "polar" does not mean unlimited solubility.

Hydrophobic substances interact poorly with water and often aggregate rather than dissolve. Lipids provide useful examples. The nonpolar regions of membrane lipids avoid water while their polar regions interact with it, helping organize a boundary around the cell's aqueous interior. Hydrophobic regions also contribute to protein folding. Insolubility can therefore be biologically useful; water need not dissolve everything to support life.

Do not confuse dissolving with melting, reacting or forming a suspension. Fine insoluble particles can make a cloudy mixture without becoming molecularly dissolved. A clear solution is also not necessarily pure water: dissolved ions or molecules may be invisible.

Water and air as animal habitats

Compare each physical property with the biological consequence it actually explains. Several properties operate at once, so one adaptation can have more than one benefit or cost.

Property	Water versus air	Main consequence
Buoyancy	Larger at equal displaced volume	Supports body weight
Dynamic viscosity	Higher	Resists motion
Thermal conductivity	Higher	Faster heat exchange
Specific heat capacity	Higher per kilogram	Buffers temperature change

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Support and movement

Buoyancy is an upward force caused by the surrounding fluid's pressure distribution. For a simple fully submerged object, it equals the weight of displaced fluid. Floating at rest involves balance between weight and upthrust. A greater upthrust does not mean an animal has no mass or that every animal automatically floats.

A ringed seal uses flippers for propulsion and has a streamlined body suited to movement through water. When swimming, it must overcome drag; buoyancy helps support its weight but does not remove resistance to motion. A loon, such as the black-throated loon, also moves between an aquatic habitat and air. Webbed feet provide propulsion in water, while wings provide aerodynamic forces in flight. The same animal faces different support and resistance conditions in the two media.

Viscosity is not identical to density. Drag depends on speed, size, shape and the flow conditions as well as the fluid's properties. Do not claim that one numerical viscosity ratio must equal the ratio of swimming and flying energy costs.

Heat movement and temperature stability

Thermal conductivity describes heat transfer down a temperature gradient through a material. Specific heat capacity describes energy needed to change a mass's temperature. These are different quantities. High conductivity can increase heat exchange; high heat capacity can reduce the temperature change caused by a given energy input.

A ringed seal's blubber helps reduce heat loss in cold water. A layer of insulating material slows transfer; it does not make the animal's body completely waterproof to heat. Air trapped by suitable dry feathers can also provide insulation in a bird. Actual heat loss depends on water movement, insulation, body size and the temperature difference, not conductivity alone.

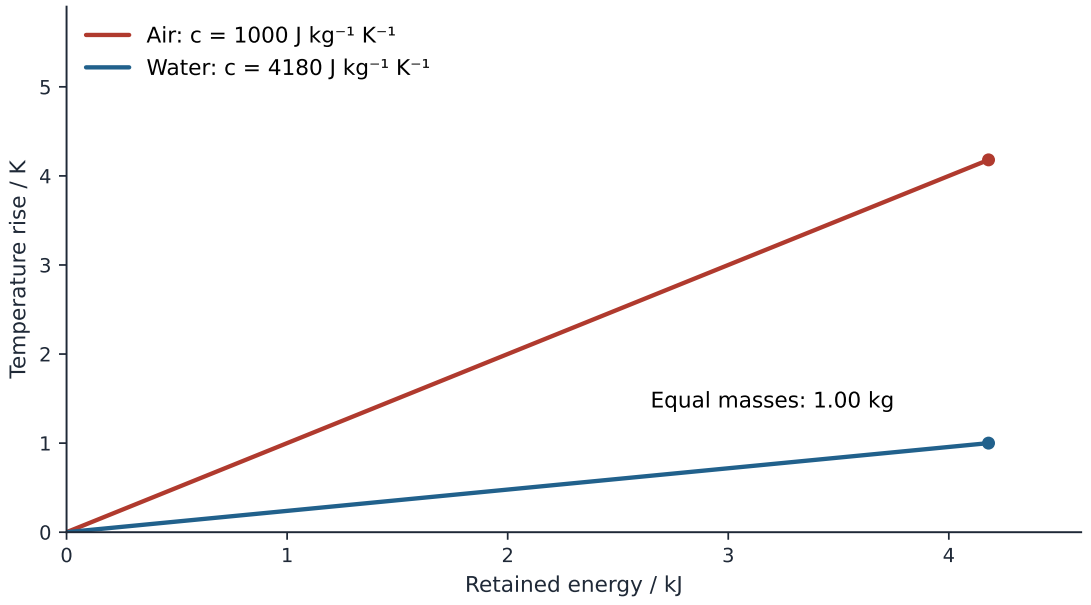
Worked example: an equal energy input

Use the given approximate specific heat capacities: water $c = 4180\ \mathrm{J\,kg^{-1}\,K^{-1}}$ and air $c = 1000\ \mathrm{J\,kg^{-1}\,K^{-1}}$. Suppose 4180 J is retained by 1.00 kg of each, without phase changes or losses.

$$\Delta T = \frac{Q}{mc}.$$

Water warms by $4180/(1.00 \times 4180) = 1.00$ K; air warms by $4180/(1.00 \times 1000) = 4.18$ K. This is an **equal-mass** comparison. Equal volumes contain very different masses, so they would give a different comparison. It is not a calculation of a seal's cooling rate.

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For equal mass and retained energy, the larger specific heat capacity gives the smaller temperature rise.

Hydrogen bonding helps explain the energy required to warm water: energy changes molecular motion and intermolecular arrangements. Water therefore helps buffer organisms and habitats against rapid temperature changes. It does not keep temperature perfectly constant or remove the need for temperature regulation.

Investigate a water property fairly

To compare spreading, place equal-volume drops of water on clean samples of two surfaces at the same temperature. Define a measurement such as contact angle from a side photograph. Keep drop volume, surface preparation, time after placement and camera geometry consistent. Use repeated trials and report the scatter.

Contact angle describes wetting under these conditions; it is not a direct numerical measurement of cohesion alone. If investigating detergent concentration, change that concentration systematically while holding the surface fixed. A result can support an explanation involving surface interactions, but a difference might also arise from contamination or unequal volumes. Match the conclusion to what the method measured and give a specific improvement for each limitation.

AHL: Earth's water and the search for life

One hypothesis proposes that water-bearing asteroidal material delivered water during Earth's early history. Finding water-related minerals in asteroids supports the plausibility of such a source, but does not mean a single asteroid supplied all Earth's oceans. Distinguish the proposed source from the conditions that allowed water to

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remain over geological time.

Earth's gravity helps retain material, including an atmosphere, rather than allowing it all to escape readily. Conditions cool enough for water vapour to condense allow liquid reservoirs to form. Gravity alone is insufficient if surface conditions keep water in vapour form or promote its loss; temperature alone is insufficient to explain retention on a small body. Long-term water availability gave biological evolution a persistent medium.

The **habitable zone** around a star, sometimes called the Goldilocks zone, is a range where surface liquid water could be possible on a suitable planet. It is not a fixed universal distance, because stars differ. Atmosphere, pressure and other planetary properties also affect whether liquid water actually persists. Being in the zone does not prove that water is present or that the planet supports life.

Liquid water is a useful search target because known life depends on it as a reaction medium. It is evidence of possible suitable conditions, not a detection of life. Water, an energy source and suitable chemistry must be considered together. Subsurface liquid water may also occur outside a star's surface-habitable zone, so the zone is a search guide rather than the only possible location worth investigating.

Practice

All numerical observations below are invented. Use the stated models and constants.

1. Explain why a land animal still needs an internal aqueous medium. Give one metabolic role and one transport example.
2. Draw two water molecules. Label their partial charges, distinguish covalent and hydrogen bonds, and explain why the molecule is polar but electrically neutral overall.
3. Explain how hydrogen bonding contributes to cohesion and how this helps transmit a pull through xylem. Identify where the pulling stress originates.
4. A student calls a pond surface "a solid skin that holds up all insects". Correct both parts using surface tension and the conditions for support.
5. Distinguish cohesion from adhesion. Explain capillary rise in a wettable narrow tube and why this alone is not a full explanation of tall-tree water transport.
6. Explain how water can surround sodium and chloride ions. Contrast this with a hydrophobic region of a membrane lipid, and explain why insolubility can be useful.
7. Explain one consequence each of buoyancy and viscosity for a swimming seal. Why does buoyancy not eliminate drag?
8. Compare thermal conductivity and specific heat capacity. Explain how cold water can cool a warm animal rapidly while a large lake can resist rapid temperature change.

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9. Using $c = 4180\ \text{J}\,\text{kg}^{-1}\,\text{K}^{-1}$, calculate the energy retained when 0.200 kg water warms by 5.0 K. State two energy transfers omitted if this is used as a simple heating experiment model.
10. Equal 0.500 kg samples retain 2090 J. Use $c_{water} = 4180$ and $c_{air} = 1000$ in J kg^{-1} K^{-1}. Calculate both temperature rises and explain why the comparison must specify equal mass.
11. A spreading experiment gives contact angles 68°, 71° and 69° on surface A, and 108°, 112° and 110° on B. Find each mean, identify which is more readily wetted, and give two controls and one limitation.
12. **AHL.** Distinguish the asteroidal-source hypothesis from two reasons Earth could retain water. Why would a claim that "gravity produces liquid water" be incomplete?
13. **AHL.** A planet is reported to orbit in its star's habitable zone. Explain what this suggests and what it does not establish. Why might a moon outside that zone still interest a search for life?

■ Worked answers

1. Enzyme-controlled reactions usually occur in an aqueous environment where reactants can move and meet. Blood plasma carries dissolved substances such as glucose to tissues. Life on land still depends on maintaining water within cells and transport fluids.
2. Each O bears δ^- and each H δ^+. Solid O–H bonds are within molecules; a dashed bond links an appropriate H on one to O on another. Oxygen attracts shared electrons more strongly; the bent arrangement gives a molecular polarity, while the molecule's total positive and negative charges still balance.
3. Intermolecular attractions link water molecules. Evaporation from leaf surfaces generates tension; cohesion helps transmit that pulling stress through the connected water column. The molecules are not using metabolic energy to crawl upwards.
4. Surface tension is a property of a liquid surface, not a solid layer. It can provide supporting forces for a suitable small animal with appropriate mass, contact geometry and water-repellent legs; it cannot support every insect or unlimited weight.
5. Cohesion acts between water molecules; adhesion acts between water and another material. At wettable walls, surface interactions can support a rise until the raised water's weight balances the upward force. Narrow spaces in walls and soil matter, but long-distance ascent also requires transpiration-driven tension in a cohesive column.
6. Water's oxygen side tends to face Na^+; its hydrogen sides tend to face Cl^-. Nonpolar lipid regions do not interact similarly and tend to avoid the aqueous phase. This helps membrane lipids organize a boundary rather than disperse completely in water.

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7. Buoyancy provides an upward force that supports some or all of the seal's weight under the relevant conditions; viscosity contributes to resistance during movement. Upward support and resistance to forward motion are different effects, so swimming still requires propulsion.
8. Conductivity concerns heat transfer across a temperature gradient; specific heat capacity relates warming to retained energy per mass. Water can carry heat from a warm body while its large mass and heat capacity limit its own warming. Insulation and currents affect the transfer rate.
9. $Q = mc\Delta T = 0.200(4180)(5.0) = 4180$ J, or 4.18 kJ before final significant-figure rounding (about 4.2 kJ to two significant figures). Energy absorbed by the container and transferred to the wider surroundings are omitted.
10. Water: $2090/(0.500 \times 4180) = 1.00$ K. Air: $2090/(0.500 \times 1000) = 4.18$ K. Their ratio is 4.18. Equal volumes contain unequal masses, so substituting equal volumes would not preserve this result.
11. A: $(68 + 71 + 69)/3 = 69.3°$; B: $(108 + 112 + 110)/3 = 110°$. A is more readily wetted. Control drop volume and temperature; prepare and photograph surfaces consistently. Contact angle measures wetting, not cohesion alone; contamination or camera geometry could bias it.
12. **AHL.** Asteroidal material is a proposed source of water. Gravity helps retain material; sufficiently cool conditions allow condensation. Gravity does not itself supply water or ensure suitable temperature and pressure for a persistent liquid phase.
13. **AHL.** Its orbital region may permit surface liquid water under suitable planetary conditions. The report does not establish an atmosphere, actual liquid water or life. A moon outside the surface-habitable zone could have a subsurface liquid-water habitat maintained by internal heating.

Exam tips

- Link molecular structure → property → named biological consequence. Distinguish cohesion/adhesion, viscosity/density and conductivity/heat capacity.
- Label partial charges and both bond types. Boiling normally leaves covalent O–H bonds intact.
- State comparison conditions and calculation units; match experimental conclusions to measured outcomes.
- **AHL.** Liquid water suggests possible suitable conditions; a habitable-zone position does not detect life.

References: IB A1.1; NOAA insulation; NASA habitable zones.

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Nucleic acids

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Scope and prerequisites

How can a molecule made from only four kinds of base carry so many different instructions? The answer lies in their sequence, the structure that keeps the sequence intact, and complementary pairing that allows information to be copied and used.

This handout teaches nucleotides, DNA and RNA structure, information storage, base pairing and the genetic code. Sections marked **AHL** are for Higher Level only. Prerequisites: covalent bonds, hydrogen bonds and condensation reactions. All sequence examples and numerical datasets below are original teaching examples, not real gene sequences or historical measurements.

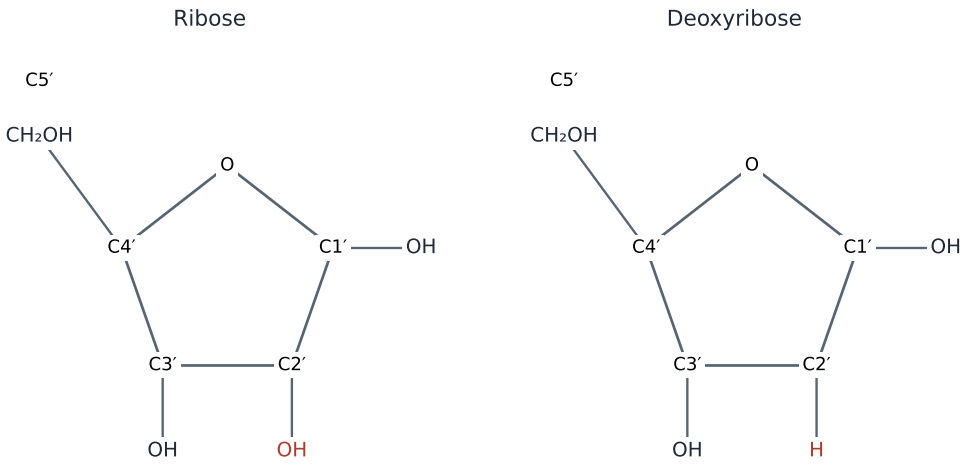
Nucleotides: the units of nucleic acids

DNA is the genetic material of living organisms. RNA also has important cellular roles; some viruses have RNA genomes, but viruses are not classified as living organisms in this course. "DNA is genetic material" does not mean every mature cell retains a nucleus or that RNA never stores genetic information in a virus.

A **nucleotide** contains a phosphate group, a five-carbon pentose sugar and a nitrogen-containing base. Use a circle for phosphate, a pentagon for sugar and a rectangle for the base in a simplified diagram. Connect the base to the sugar and the phosphate to the sugar; the base is not inserted between sugar and phosphate in the backbone.

DNA contains deoxyribose; RNA contains ribose. In a sketch of the sugars, number the carbon positions and compare the 2′ position: ribose has an –OH group there, while deoxyribose has H in its place. "Deoxy" does not mean the sugar contains no oxygen. The prime mark distinguishes sugar-carbon numbering from other numbering in a nucleotide.

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2′: OH in ribose; H in deoxyribose.
Other H and stereochemistry omitted.

The important comparison is the group at 2′; the diagram highlights connectivity and is not a full stereochemical formula.

The DNA bases are adenine (A), thymine (T), cytosine (C) and guanine (G). RNA uses adenine, uracil (U), cytosine and guanine. A base name is not the name of a whole nucleotide: adenine alone has neither a sugar nor a phosphate.

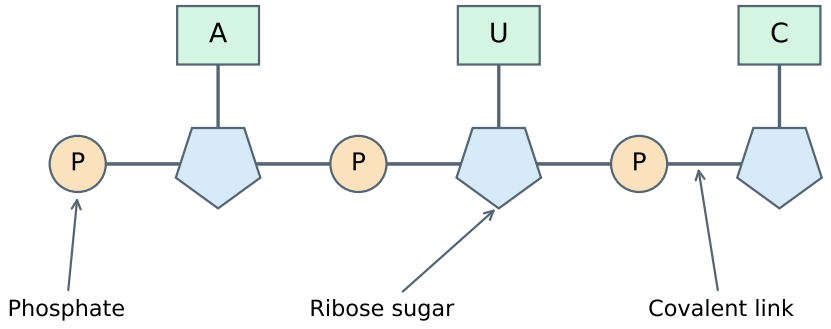
Build a strand: a continuous covalent backbone

Nucleic acids are polymers of nucleotides. In a simplified condensation model, linking nucleotide units forms a sugar–phosphate connection with water released. The link is a covalent **phosphodiester bond**. Repeated sugar–phosphate links form the backbone, with bases projecting from the sugars. RNA can be represented as a single chain of these units.

When drawing an RNA polymer, preserve three relationships: a continuous alternating sugar–phosphate backbone, a base attached to each sugar, and no thymine in the RNA sequence. A chain is not held together mainly by hydrogen bonds between successive bases. Hydrolysis can break covalent links in a backbone; separating paired DNA strands is a different process.

For a simple linear condensation model, n separate mononucleotide units need $n - 1$ new links to make one chain. Thus six units need five links. This counting model is not a complete description of intracellular polymerization: cells use activated nucleotide substrates, and their reaction products must be considered separately. Do not assume one classroom condensation diagram describes every biochemical step.

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Continuous sugar–phosphate backbone.
Each base attaches to a sugar.

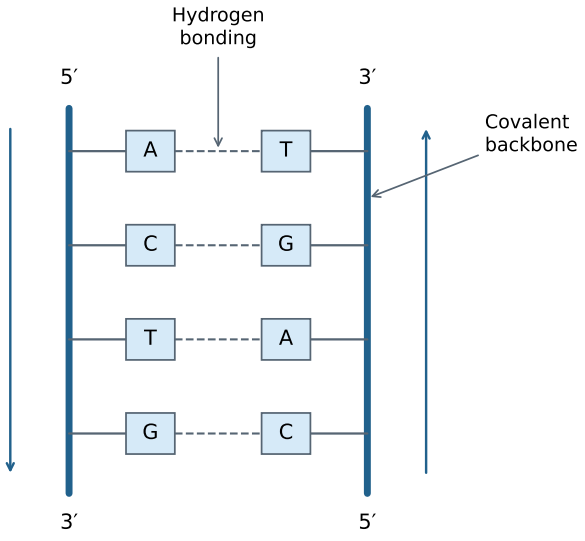
Bases attach to sugars; the strand is connected through covalent sugar–phosphate links.

DNA: complementary strands form a double helix

A DNA molecule normally contains two polynucleotide strands twisted into a double helix. Their sugar–phosphate backbones are on the outside and paired bases lie inside. The strands are **antiparallel**: they run in opposite directions. In a flat drawing, show opposing end labels or arrows rather than drawing both strands pointing the same way. A ladder sketch can represent the pairing without reproducing the helical twist.

Complementary base pairing means A pairs with T and C pairs with G in the standard DNA model. Hydrogen bonds form between the bases on different strands. Covalent bonds hold each backbone together. These two kinds of bond have different positions and roles; do not draw hydrogen bonds as the backbone links.

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Reading across a pair gives its complement; reading along a strand gives a sequence.

For the invented top strand A–T–G–C–G–A, the paired bases in the aligned opposite strand are T–A–C–G–C–T. The matching is determined, but the sequence along a strand can vary. "Complementary" means matching by the pairing rule, not identical.

In ordinary double-stranded DNA, total A equals total T and total C equals total G because every pair contributes one of each matching base. This does **not** require A to equal G or every base to be 25%. It also does not require each individual strand to contain equal A and T. State whether a composition describes one strand or the entire double-stranded molecule.

■ Worked example: count both strands

Suppose an invented DNA molecule has 800 base pairs and 28% of its nucleotides contain adenine. It has $2 \times 800 = 1600$ nucleotides in total. A and T each make up 28%; together they are 56%. The remaining 44% is split equally between C and G, giving 22% each.

$$N_A = N_T = 0.28(1600) = 448,$$

$$N_C = N_G = 0.22(1600) = 352.$$

Check: $448 + 448 + 352 + 352 = 1600$. There are 448 A–T pairs and 352 C–G pairs, totaling 800 pairs. Mixing base pairs and individual nucleotides would give a factor-of-two error.

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Compare DNA and RNA

Feature	DNA	RNA
Sugar	Deoxyribose	Ribose
Bases	A, T, C, G	A, U, C, G
Typical cellular arrangement	Two paired strands	One strand, often folded
Examples	Chromosomal DNA	mRNA, tRNA, rRNA

Messenger RNA carries sequence information used in protein synthesis. Transfer RNA helps match amino acids to codons, and ribosomal RNA is a structural and functional part of ribosomes. A single RNA strand can fold so that complementary regions pair; "single-stranded" does not mean it must remain a straight line. These are typical cellular comparisons, not a claim that every viral genome follows the same strand pattern.

Copy and express information by base pairing

Complementary pairing makes a template useful. During DNA replication, the strands can separate and each can guide the formation of a complementary strand. The sequence of an existing strand constrains the new sequence. Accurate copying also depends on enzymes and checking processes; complementarity does not make mutations impossible.

Transcription produces RNA using a DNA template. RNA nucleotides pair with the template: DNA A directs RNA U, DNA T directs RNA A, and C pairs with G. The coding DNA strand has the same base sequence as the RNA transcript in the corresponding region, with T in DNA in place of U in RNA. Always state which DNA strand you were given.

Translation uses the codon sequence in mRNA to assemble a polypeptide. A **codon** is a group of three mRNA bases. Transfer RNA helps implement complementary matching during translation. A codon table is normally read using mRNA codons, not an unexplained DNA triplet. Stop codons signal termination; they do not specify an additional amino acid.

For the invented coding DNA segment ATG–GAA–TTC–TAA, the mRNA segment is AUG–GAA–UUC–UAA. Using the stated meanings AUG = methionine, GAA = glutamate, UUC = phenylalanine and UAA = stop, the model peptide has three amino acids. The four codons do not give four amino acids.

In a simplified complete coding segment with 90 mRNA bases, including one terminal stop codon, there are $90/3 = 30$ codons and $30 - 1 = 29$ amino acids. This assumes the segment begins with the initiating codon, contains no intron or untranslated region, and is translated as stated. A whole gene's DNA length cannot automatically be divided by three: genes can include other sequences.

The genetic code is **degenerate**: more than one codon can specify the same amino

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acid. For example, GAA and GAG both specify glutamate. A substitution between them can be silent with respect to the amino-acid sequence. Inserting or deleting a number of bases not divisible by three within a translated region can shift the reading frame. A mutation's consequence depends on position and context; not every sequence change alters the protein.

Sequence diversity and shared ancestry

With four possible bases at each of n specified positions, the number of possible sequences on one defined strand is 4^n. Six positions give $4^6 = 4096$ sequences; twenty give $4^{20} = 1,099,511,627,776$. The count grows rapidly even though the alphabet is small. The complementary strand is determined by the first; it is not a second independent choice of four bases at each position.

A particular molecule has a finite length and information capacity. The enormous general capacity of DNA comes from varying sequence and length, not from a single finite molecule holding literally infinite information. Not every mathematically possible sequence forms a functioning gene or an organism with high fitness.

Across living organisms, codons generally have the same meanings. This strong conservation of the genetic code supports common ancestry: related information-processing systems are inherited and modified. Small known code variations do not erase the broad pattern. Distinguish a shared code from identical DNA sequences; different organisms can use the same code to specify different proteins.

AHL: Directionality and molecular organization

The two ends are chemically different

The 5′ and 3′ labels refer to sugar-carbon positions. The backbone connects the 3′ position of one sugar through phosphate to the 5′ position of the next. A conventional sequence is written 5′ to 3′ unless the question states otherwise.

DNA polymerases extend a strand by adding to its 3′ end, so new DNA is made 5′ to 3′ while its template is read in the opposite direction. Transcription similarly makes RNA 5′ to 3′. During translation, the ribosome reads mRNA codons in the 5′ to 3′ direction. These are distinct processes with a common need to track sequence direction.

If the top DNA strand is 5′–ATGCGA–3′, the aligned complement is 3′–TACGCT–5′. Written independently 5′ to 3′, that lower strand is 5′–TCGCAT–3′. Complementing without reversing when asked for an independently written 5′-to-3′ strand is a common error.

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Pair widths and packaging

Adenine and guanine are double-ring **purines**; cytosine and thymine are single-ring **pyrimidines**. RNA's uracil is also a pyrimidine. Each standard DNA pair combines one purine with one pyrimidine, helping maintain a consistent helix width across different sequences. A purine–purine pair would be too wide and a pyrimidine–pyrimidine pair too narrow in the ordinary helix geometry.

In a eukaryotic nucleosome, DNA wraps around a core of eight histone proteins. Linker DNA joins neighbouring nucleosomes; an additional linker histone associates near the DNA entry/exit and linker region. That additional histone is not a ninth member of the eight-protein core. Packaging organizes a long DNA molecule and influences access to its sequence.

Use a molecular viewer, such as the RCSB view of structure 4QLC, with an experimentally determined nucleosome structure to distinguish DNA from protein, rotate the structure and identify the wrapped DNA path. In 4QLC the linker-associated component is a globular H5 domain, not a complete additional core histone. A cross-section may hide parts of the molecule, so inspect more than one view. Record the structure identifier and representation settings. A diagram alone does not replace this three-dimensional inspection.

AHL: Evidence for DNA as genetic material

Hershey and Chase: track different components

Hershey and Chase investigated bacteriophage T2 infection of bacteria. In separate preparations, radioactive phosphorus-32 labelled DNA, while sulfur-35 labelled sulfur-containing phage proteins. DNA contains phosphorus in its backbone but no sulfur; proteins containing cysteine or methionine can incorporate sulfur. Keep the two labels and preparations distinct.

After phages attached and infection began, blending detached much of the external phage coat. Centrifugation separated a bacterial pellet from the supernatant containing detached material. Much of the DNA-associated label remained with the bacteria, whereas much of the protein-associated label was outside them. DNA-associated material also contributed to progeny phage. This supported DNA, rather than the bulk of the external protein coat, as the material directing phage reproduction.

The experiment did not mean a phage contains no protein, that the protein coat has no function in attachment, or that every labelled molecule crossed the bacterial membrane. Radioactive tracing allowed components to be followed through a process; it was evidence interpreted with controls and the experimental separation, not a photograph of a gene controlling a cell. This is a historical explanation, not a school procedure involving radioisotopes.

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Chargaff: test a prediction with composition data

A rigid repeating tetranucleotide model containing equal numbers of A, T, C and G predicts about 25% of each base regardless of the organism. Chargaff's compositional findings showed that base proportions differed across biological sources, while matching relationships between A/T and G/C were evident. These observations contradicted the equal-four-base prediction and helped constrain later structural explanations.

For an invented comparison, sample X has A = T = 30%, C = G = 20%; sample Y has A = T = 21%, C = G = 29%. Both satisfy complementary-pair totals but neither has four equal proportions. Even a single reliable counterexample can falsify a universal equal-proportion claim. A sample with four 25% values would fit that prediction, but would not prove that every DNA molecule consists of a repeating four-base unit. Composition also does not reveal the order of the bases.

Practice

All sequences and numerical data are invented. Use the stated simplified models.

1. Draw a nucleotide using the standard three shapes. Name each component and show where the base attaches.
2. Sketch ribose and deoxyribose to show their key difference. Explain why deoxyribose does not mean "no oxygen".
3. Draw three RNA nucleotide units as one chain. Identify the covalent backbone, attached bases and the number of new links in the simple condensation model.
4. Compare DNA and RNA in sugar, bases and usual cellular strand arrangement. Name two functional types of RNA and explain why folded RNA can still be one strand.
5. For the aligned DNA strand A–T–G–C–G–A, write the six paired bases. Distinguish complementary pairing from an identical sequence.
6. Double-stranded DNA has 800 base pairs and 28% adenine among all nucleotides. Calculate the total number of each base and check the sum.
7. For coding DNA ATG–GAA–TTC–TAA, give the mRNA sequence and peptide using the codon meanings supplied above. Why is the coding strand not the template strand?
8. A complete mRNA coding segment has 90 bases including one terminal stop codon. Find peptide length. Repeat for 63 bases, and state the assumptions that make these calculations valid.
9. GAA and GAG both encode glutamate. Explain degeneracy and a possible silent substitution. Contrast this with inserting one base inside the translated region.
10. Calculate the number of possible six-base sequences on one defined strand. Explain why knowing its complementary strand does not double the number of independent positions.

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11. Explain how conserved codon meanings support common ancestry. Distinguish this from a claim that all organisms have the same base sequence.
12. **AHL.** Write the complement of 5′–ATGCGA–3′ first aligned with it, then independently 5′ to 3′. State synthesis and mRNA-reading directions, and explain how purine–pyrimidine pairing helps helix geometry.
13. **AHL.** Describe a nucleosome's eight-protein core, wrapped DNA and linker region. What would you inspect in a molecular viewer to distinguish a ninth core protein from a linker-associated protein?
14. **AHL.** Explain what the Hershey–Chase label separation supported. Then use invented samples X and Y above to test an equal-four-base hypothesis. State one limitation of each type of evidence.

■ Worked answers

1. A phosphate circle links to a pentose pentagon; a base rectangle attaches to the sugar. One complete three-component unit is a nucleotide, whereas the base alone is only one component.
2. At 2′, ribose has –OH and deoxyribose has H instead. Both still contain oxygen elsewhere, including in the ring. Label the compared carbon rather than replacing every oxygen in the sugar.
3. Draw an alternating sugar–phosphate chain with a base attached to each of three sugars. Its backbone links are covalent phosphodiester bonds. Three separate units need $3 - 1 = 2$ new links in this stated linear condensation model.
4. DNA: deoxyribose, A/T/C/G, typically two paired strands. RNA: ribose, A/U/C/G, typically one strand. mRNA carries coding information; tRNA helps match amino acids to codons, or rRNA forms part of ribosomes. Intramolecular pairing can fold one continuous RNA chain without adding a second chain.
5. T–A–C–G–C–T, aligned opposite the given sequence. Each base follows A–T or C–G pairing; complementarity is a specific matching relationship rather than copying the same letters across the pair.
6. $N = 2(800) = 1600$ nucleotides. $N_A = N_T = 0.28(1600) = 448$. C and G each make up $(100 - 56)/2 = 22\%$, so $N_C = N_G = 352$. The sum is 1600; there are 800 pairs.
7. AUG–GAA–UUC–UAA gives methionine–glutamate–phenylalanine, then termination: three amino acids. The template pairs with the RNA; the coding strand matches the RNA's corresponding sequence except T replaces U.
8. $L = 90/3 - 1 = 29$ amino acids; for 63 bases, $L = 63/3 - 1 = 20$. Assume one complete translated coding region beginning at initiation, one terminal stop and no untranslated region or intron included in the count.

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9. Degeneracy means multiple codons can specify the same amino acid. GAA → GAG preserves glutamate and can leave the amino-acid sequence unchanged. A one-base insertion within the translated sequence can shift subsequent codon grouping, though the exact effect depends on position and sequence.
10. $N = 4^6 = 4096$. There are six independent positions on the defined strand; each paired base is then constrained. A finite molecule has finite capacity even though varying sequence and length allow an enormous range of information.
11. Shared codon meanings are a conserved feature of the information-processing system, consistent with descent from shared ancestors. Sequences can differ while the same codon-to-amino-acid mapping is retained. The inference comes from a broad pattern, not a requirement that every code variant be identical.
12. **AHL.** Aligned: 3′–TACGCT–5′; independently: 5′–TCGCAT–3′. DNA/RNA synthesis extends 5′ to 3′ by adding to the 3′ end; a ribosome reads mRNA 5′ to 3′. One double-ring purine with one single-ring pyrimidine helps maintain standard pair width.
13. **AHL.** DNA wraps around a core of eight histones; linker DNA connects cores, with an additional histone associated near entry/exit and the linker. Rotate a viewer model and distinguish the core's protein chains from the separate linker-associated chain; the latter is not a ninth core member.
14. **AHL.** DNA-associated phosphorus label remained largely with infected bacteria and contributed to progeny; sulfur-labelled coat material was largely outside, supporting DNA's reproductive-information role in this system. Separation was not perfect and protein still had roles. X and Y depart from four equal 25% proportions while preserving A/T and C/G matches, contradicting the rigid equal-proportion prediction. Composition alone gives no base order, and one matching sample could not establish a universal repeating sequence.

Exam tips

- Identify sugar, phosphate and base separately. Distinguish backbone covalent bonds from hydrogen bonds across a pair.
- Count base pairs and total nucleotides carefully; state the strand or whole-molecule conditions behind a percentage calculation.
- Identify coding DNA, template DNA and mRNA before using codons. A stop codon adds no amino acid; a whole gene is not automatically all coding sequence.
- **AHL.** Keep 5′/3′ ends explicit, and connect an experiment's observation to the precise hypothesis it supports or contradicts.

References: IB A1.2; Hershey and Chase, original research; Chargaff, original research.

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Origins of cells

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A membrane bubble is not automatically alive, and making an amino acid is not the same as making a cell. The origin-of-cells problem asks how chemistry could acquire compartments, catalysis and inherited information that could evolve together.

This is the HL-only A2.1 unit; there is no SL counterpart. It teaches early-Earth models, requirements for cellular life, prebiotic chemistry, vesicles, the RNA-world hypothesis, evidence for common ancestry, dating methods and hydrothermal-vent hypotheses. Prerequisites: water, membranes, nucleic acids and enzymes. All numerical datasets and timelines used for calculations below are original, invented teaching examples.

AHL: Early Earth and a testable origin problem

Early Earth's atmosphere contained very little free molecular oxygen compared with today's atmosphere. Consequently, there was little protective ozone, allowing more ultraviolet radiation to reach the surface. Models include higher carbon dioxide and methane concentrations than today's atmosphere, contributing to greenhouse warming. The precise composition varied across environments and time and is reconstructed from evidence; it should not be treated as one directly measured global gas recipe.

Energy from ultraviolet light, electrical discharges, heat or chemical gradients could drive reactions among simpler substances. Some resulting carbon compounds could supply ingredients for later biological chemistry. "Organic" describes a category of carbon chemistry; it does not mean that a compound must have been made by an organism. Conversely, showing that a reaction is chemically possible under one set of conditions does not show that those conditions existed everywhere or produced the first cells.

An origin hypothesis should generate predictions that evidence can test. For example, a proposed gas mixture and energy source might predict the production of particular compounds. Researchers can vary the mixture, include controls and analyse products. Reproducing every detail of the historical setting is impossible, and the earliest small compartments may have left no recognizable fossil. Those limitations make the problem difficult; they do not make every explanation equally supported.

AHL: What must a cellular system do?

A cell is the smallest structural unit capable of sustaining the integrated processes of life under suitable conditions. A cell exchanges matter and energy with its surroundings, maintains an internal organization, carries information and participates in

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reproduction through its lineage. "Self-sustaining" does not mean isolated from an environment or able to make every required molecule from nothing.

A virus can carry genetic information and evolve, but it relies on a host cell's machinery and energy supply for reproduction and other functions. It has no autonomous cellular metabolism. These dependencies help explain why viruses are not classified as living cells in this course. A single feature such as movement is not a reliable living/non-living test: fire spreads and crystals grow without being cells.

Present-day cells arise by division of pre-existing cells. This observation describes current cell production; it does not by itself explain how the earliest cellular systems arose before a lineage of cells existed. **Abiogenesis** concerns that historical transition from non-living chemistry. It is different from claiming that modern, fully formed organisms routinely appear in sterile food or mud.

A plausible route needs interacting capacities:

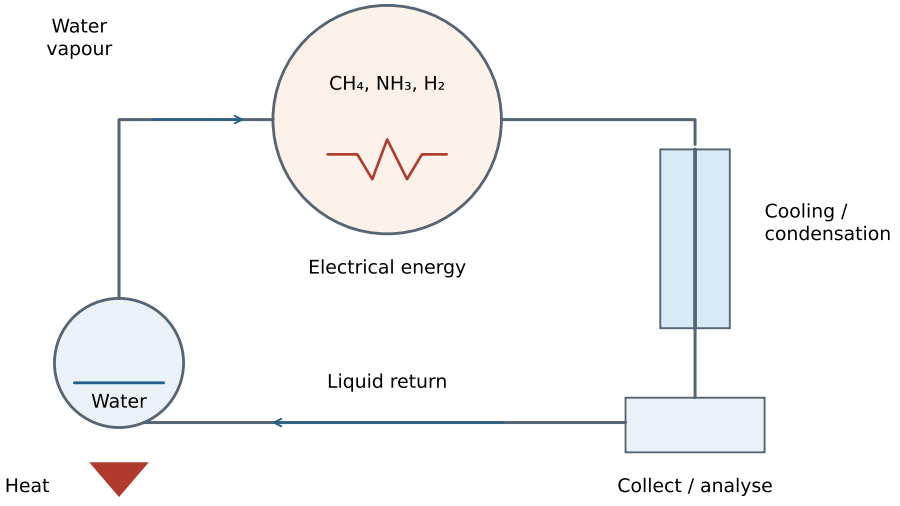
- **Catalysis:** reactions must proceed sufficiently fast under the available conditions; a catalyst speeds a route without being used up overall.
- **Replication and heredity:** information-bearing molecules must be copied with enough fidelity to preserve useful features, while variation remains possible.
- **Self-assembly:** suitable components can organize into larger structures through their interactions, without a pre-existing cell individually placing every component.
- **Compartmentalization:** a boundary can keep interacting components together and make internal chemistry differ from the surroundings.

A compartment that grows but cannot transmit an inherited functional difference is not yet a complete model of Darwinian cellular evolution. Conversely, a replicating molecule in solution does not automatically explain how a stable, regulated cell forms. Investigations can solve parts of the problem without having solved the whole transition.

AHL: Evaluate the Miller–Urey experiment

In the classic spark-discharge investigation, heated water supplied vapour to a circulating system containing methane, ammonia and hydrogen. Electrical sparks supplied energy. Cooling condensed vapour, and collected liquid could be analysed. The apparatus modelled selected possible early conditions rather than reproducing the entire early planet.

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The diagram shows a laboratory model and its material flow, not a photograph of an early-Earth environment.

Amino acids and other carbon compounds were detected among the products. This supports the possibility of abiotic synthesis of some building blocks under the tested conditions. It did not produce living cells, prove a complete origin pathway, or show that finished proteins and genomes arose spontaneously in that apparatus.

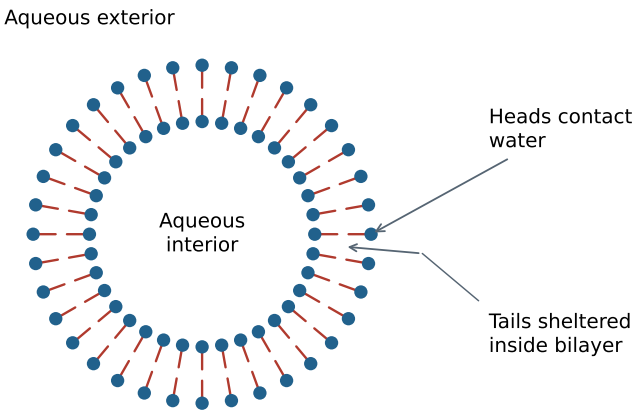
Evaluate the method through a claim–evidence–limit chain. Supplying a defined gas mixture and an energy source makes the conditions testable. Collection and chemical analysis identify products. Controls without the energy input, checks on starting materials and procedures to exclude biological contamination help distinguish synthesis from contamination. Repeatability strengthens a chemical claim.

The historical gas mixture is a limitation when applying the result to all early environments: the true composition and suitable energy inputs remain questions. A successful model reaction demonstrates feasibility within its conditions. A failed trial with a different mixture would limit that trial's pathway; it would not alone disprove every abiogenesis hypothesis. Do not confuse scientific evaluation with saying an experiment was either "perfect proof" or "worthless".

AHL: Boundaries can form by self-assembly

Suitable amphipathic molecules have a water-interacting region and a water-avoiding region. In appropriate aqueous conditions, fatty acids can aggregate into bilayers and closed vesicles. Water-avoiding tails are sheltered within the membrane while water-interacting heads contact the aqueous regions on either side. The resulting compartment can enclose a water-based interior.

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A boundary can self-assemble.
Heredity and metabolism need further explanation.

Self-assembly can create a boundary; it does not by itself create heredity or a working metabolism.

A boundary can keep catalysts and reactants close, alter concentrations and permit a different internal chemical environment. It must also allow adequate exchange of matter and energy. A perfectly impermeable container would prevent nutrient entry and waste removal; a boundary that leaks everything would lose useful components.

Assembly depends on molecular structure and conditions such as pH, concentration and dissolved ions. An observed vesicle is therefore evidence that a certain kind of compartment can form under particular conditions. It is not proof that every early membrane had the composition of a modern phospholipid membrane, or that the vesicle is already alive. Growth, division and the inheritance of contents must be connected to the chemistry inside.

AHL: RNA as an early information-and-catalysis system

Modern cells divide tasks among DNA, RNA and proteins. That division creates a historical question: how could hereditary information be used before the complete modern translation system existed? The **RNA-world hypothesis** proposes an earlier stage in which RNA had central roles in both information storage and catalysis.

RNA can carry information in its sequence and serve as a template for complementary copying. Some RNA molecules, called **ribozymes**, can catalyse reactions. The ribosome's peptide-bond-forming centre is based on ribosomal RNA, showing that catalytic RNA is relevant to modern biology as well as a laboratory possibility.

These properties make a dual-role system plausible. They do not establish that an isolated RNA molecule can already carry out every step of self-replication, metabolism and membrane maintenance without assistance. Researchers still investigate nu-

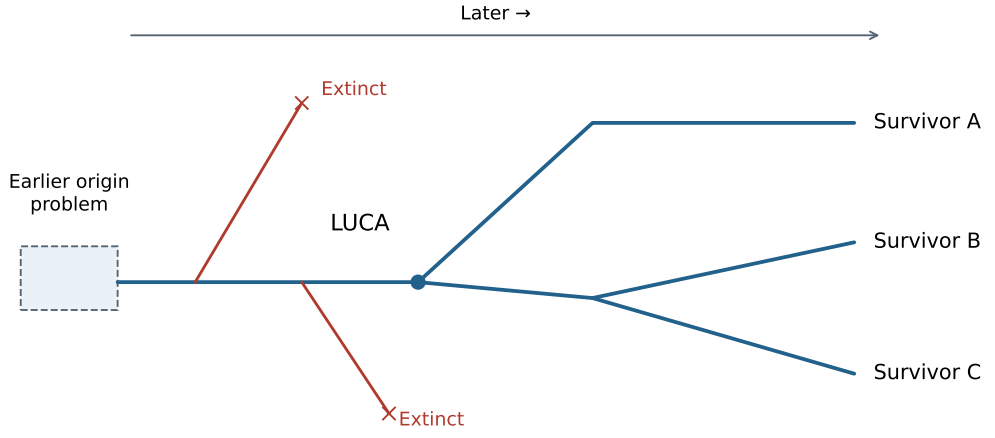
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cleotide supply, polymer formation, copying accuracy, stability and the connection to compartments. “RNA can act as a template” is a more precise claim than “every RNA molecule copies itself”.

A useful model links copying with variation and selection. If different sequences produce different catalytic effects, and compartments retain those differences through reproduction, some lineages could leave more descendants under particular conditions. This describes how selection could improve a heritable system; it does not mean the molecules deliberately seek progress or that evolution has a predetermined goal.

AHL: LUCA is not the first cell

The last universal common ancestor, or **LUCA**, is the shared ancestral lineage from which all present-day cellular life descends. It need not have been the first living system, the only organism alive at its time, or one uniquely identifiable fossil cell. Earlier lineages could have existed and left no surviving descendants. Other forms could also have been outcompeted or lost; common ancestry of survivors does not prove that only one origin event was chemically possible.



Surviving cell lineages A–C; schematic relationships.
No time scale.

The schematic shows ancestry, not a dated family tree; LUCA is the common ancestor of surviving lineages.

Evidence includes the broadly conserved genetic code and shared genes involved in fundamental information-processing functions. Compare sequences from many organisms and several independent genes, with methods that account for change. Similarities that form a consistent, branching pattern support common ancestry more strongly than one superficial resemblance.

An **inference** is an explanation drawn from observations and reasoning. A

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modern sequence does not directly display the whole history of its lineage. Gene transfer between lineages, gene loss, different evolutionary rates and uncertainty in ancient reconstructions complicate the inference. The evidence remains informative when these limits are recognized and alternative explanations are tested.

■ **Worked example: a similarity is not a date**

An invented alignment has 18 matching sites out of 24 comparable sites:

$$\text{identity} = \frac{\text{matching sites}}{\text{compared sites}} \times 100\% = \frac{18}{24} \times 100\% = 75\%.$$

This describes similarity in that alignment. It does not specify the rate of change, which differences are repeated changes at one site, or the relevant calibration. It therefore cannot alone give a date for LUCA or prove that one sampled organism was another's direct ancestor.

AHL: How do scientists estimate ancient dates?

Different evidence answers different dating questions. Fossils and chemical traces can show that life existed **by** a certain time, provided their biological origin and geological context are established. They rarely identify the instant when life first appeared. Older evidence may be absent because rocks were destroyed, altered or never preserved the relevant features.

Rock ages can be constrained using radiometric methods and the relationships between rock layers. A date for the surrounding geological material is not automatically a direct date for the formation of every object inside it. Investigators must rule out younger contamination, later fluid movement and deformation that could change the interpretation.

A chemical signature, such as a carbon-isotope pattern compatible with biological processing, can be suggestive but needs testing against non-biological explanations. Shapes resembling cells or filaments also require contextual and chemical evidence. A convincing interpretation combines lines of evidence rather than treating one familiar-looking shape as decisive.

Molecular clocks use accumulated sequence differences, calibrated with other information, to estimate divergence times. They require assumptions about evolutionary rates, relationships and calibrations. Ancient reconstructions are uncertain and can change when the model or evidence changes. The estimated age of a common ancestor is not simply the age of the oldest fossil found so far.

To appreciate scale, an invented interval from 3.8 to 3.2 billion years ago lasts 0.6 billion years, or 600 million years. A diagram that compresses that interval into a centimetre can hide an enormous number of generations. This numerical example illustrates elapsed time; it is not a proposed date for life's origin or LUCA.

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AHL: Why investigate hydrothermal settings?

Hydrothermal systems circulate water through rock and create environments with mineral surfaces and chemical gradients. Some vent settings offer differences in redox conditions or proton concentrations that could provide energy for reactions. Mineral pores can offer small spaces that concentrate substances. A useful hypothesis must connect the proposed conditions to particular chemistry rather than saying only that vents are warm.

Ancient seafloor hydrothermal precipitates contain structures and chemical associations that researchers have interpreted as evidence of early microorganisms. Biological and non-biological processes can sometimes make similar structures, and the rocks' ages and alteration histories need careful evaluation. Such evidence can support life in an ancient hydrothermal environment without proving the exact birthplace of the first living system.

Genomic reconstructions also investigate whether conserved sequences and inferred metabolisms are consistent with an early ancestor using chemical energy in a vent-related setting. These are model-based inferences from surviving organisms, not a direct reading of LUCA's entire environment. Fossil context and genomic evidence are different lines of reasoning; agreement can strengthen a hypothesis, but shared assumptions must not be counted as independent confirmation.

AHL: Distinguish later endosymbiosis

Endosymbiosis is an association in which one organism lives within another. Evidence that mitochondria and chloroplasts descend from bacterial lineages includes their own DNA, bacterial-type ribosomes and division within cells. Several consistent comparisons are more persuasive than one feature alone.

This model addresses the later history of eukaryotic cell organization. It is not a complete explanation for the origin of the first cells: bacteria and a host lineage already existed in the proposed association. It also does not mean every organelle descended from an engulfed bacterium. Keep the question being answered distinct from the wider origin-of-life problem.

Practice

All calculations use invented teaching data. Answer with an observation, inference and limitation where relevant.

1. Link little free oxygen to ozone and ultraviolet exposure on early Earth. Why should one laboratory gas recipe not be treated as a directly measured universal atmosphere?
2. Explain why a virus is not classified as a living cell here. Why are crystal growth and fire spreading insufficient evidence of cellular life?

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3. Explain how current cells arising from pre-existing cells differs from the historical origin problem. Name four capacities a plausible early cellular system must bring together.
4. Describe the roles of water heating, the gas mixture, electrical energy, cooling and collection in the classic spark-discharge model. State what the product evidence supported and what it did not show.
5. Suggest one control and one contamination check for a model chemical-synthesis investigation. Explain why failing to produce cells is not itself a failure to test building-block synthesis.
6. Explain how fatty acids can form a vesicle in suitable conditions. Why does its boundary need both retention and exchange, and why is the vesicle not automatically alive?
7. Explain how RNA can be relevant to both heredity and catalysis. Give one modern example and two unresolved requirements of an early RNA-based cellular model.
8. Distinguish LUCA from the first living system and from the only organism alive at its time. Give two kinds of evidence for common ancestry.
9. An alignment has 21 matches among 28 compared sites. Calculate identity. Repeat for 16 out of 20, and explain why neither percentage alone dates a common ancestor.
10. Explain how fossils, rock dating and molecular clocks answer related but different questions. Why does the oldest known evidence usually give a minimum age for the existence of life rather than an exact origin date?
11. An invented geological interval runs from 3.8 to 3.2 billion years ago. Find its duration in millions of years. Explain one way a small timeline can mislead a reader about evolutionary timescales.
12. Explain two potentially useful features of a hydrothermal environment. Distinguish evidence for ancient life at a vent from proof that all life originated there.
13. Explain how evidence for mitochondrial endosymbiosis differs from evidence for abiotic synthesis. Why does a single matching gene not prove the complete origin of life?

■ Worked answers

1. Little molecular oxygen means little ozone could form, so more ultraviolet radiation could reach the surface. Early atmospheric models are reconstructed and vary across settings and time; a defined gas recipe tests a possible chemical setting rather than directly measuring the whole ancient atmosphere.
2. A virus relies on host cellular machinery and energy for reproduction and lacks autonomous cellular metabolism. Crystals and fire can grow or spread without the integrated hereditary, compartmentalized metabolic organization of a cell.

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3. Cell division describes how cells arise today, not how the first cellular lineage originated. An origin model must connect catalysis, replication/heredity, self-assembly and compartmentalization with exchange of matter and energy.
4. Heating supplies vapour; the methane/ammonia/hydrogen mixture supplies reactants; sparks provide energy; cooling condenses material; collection allows analysis. Amino-acid production supports abiotic building-block synthesis under those conditions, not production of living cells or a complete historical pathway.
5. A matched no-energy-input control tests whether the energy source affects products. Analyse clean starting materials and blanks, and use procedures that exclude biological contamination. A building-block experiment can answer its chemical question without generating a complete cell.
6. Amphipathic molecules can arrange with heads contacting water and tails sheltered in a bilayer that closes around an aqueous interior. Retention keeps useful components together; exchange supplies reactants and removes products. A boundary alone supplies neither inherited functional information nor an integrated metabolism.
7. RNA sequences can store information and act as copying templates; some folded RNAs catalyse reactions. Ribosomal RNA contributes to peptide-bond formation. Early models still need workable nucleotide supply and accurate copying, for example, as well as suitable stability and connection to compartments.
8. LUCA is ancestral to surviving cellular lineages, not necessarily the first cell or the only contemporary organism. The conserved genetic code and shared fundamental genes support common ancestry; earlier or competing lineages may have become extinct.
9. $I = 21/28 \times 100 = 75\%$; $I = 16/20 \times 100 = 80\%$. Identity alone supplies no calibrated change rate and does not account for multiple changes, gene transfer or the full relationship among samples.
10. Fossils/chemical traces can establish life's presence in context; radiometric and stratigraphic methods constrain geological ages; calibrated molecular clocks estimate divergence times from sequences. An older origin could predate the oldest preserved, discovered evidence, so that evidence usually establishes life existed by a time.
11. $\Delta t = (3.8 - 3.2) \times 1000 = 600$ million years. A short drawn interval can conceal many generations; apparent spacing in an unscaled diagram must not be read as a measured duration.
12. Chemical/proton gradients could supply energy, and pores/mineral surfaces could concentrate reactants or support reactions. Biological-looking structures and suitable chemistry in an ancient deposit can support life there, but neither identifies the unique origin site nor rules out alternative environments or abiotic structures without further tests.
13. Endosymbiosis concerns existing cell lineages combining in later eukaryotic evolution; abiotic synthesis concerns chemical building blocks forming without organ-

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isms. One gene comparison has a limited history and possible alternative explanations. A complete origin model needs several processes and convergent lines of evidence, not one similarity.

Exam tips

- State the question a model actually tests. Building blocks, compartments, common ancestry and eukaryotic organelles are distinct explanatory targets.
- Separate observations from inferences; add a limitation without discarding valid evidence. Feasibility under defined conditions is not proof of a unique historical pathway.
- LUCA is the ancestor of surviving lineages, not automatically the first cell. Similarity percentages are not dates without a model and calibration.
- For early-life claims, connect chemical, geological and sequence evidence while considering contamination, alteration and non-biological alternatives.

References: IB A2.1; Miller, original research; Dodd and colleagues, hydrothermal evidence.

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Cell structure

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A photograph can make a bacterium look as large as a leaf cell. To compare them, we need a scale; to explain how they live, we need their internal organization.

This unit covers A2.2: cell theory, microscopy and measurements, common cell features, prokaryotic and eukaryotic structure, unicellular life, plant/animal/fungal comparisons, atypical cells, and interpreting and drawing micrographs. SL understandings plus endosymbiosis, differentiation and multicellularity (marked AHL). Prerequisites: water, DNA/RNA, membranes and basic unit conversions. Numerical examples are original, invented teaching data. Diagrams are models, not micrographs.

Cells as units of life

Cell theory connects three ideas: organisms consist of one or more cells; cells are the basic structural and functional units of organisms; present-day cells arise from pre-existing cells. An organism's activities depend on the coordinated activities of its cells. A single-celled organism must carry out the essential processes within one cell; a multicellular organism distributes work among cells.

A theory can generate a prediction by deduction. If a newly discovered organism is living and cell theory applies, we predict that examination at sufficient resolution will reveal one or more cells. This prediction guides observations; it is not a substitute for examining the organism. Viruses depend on host cellular machinery and are not cells. Exceptions to a typical cell diagram need explanation rather than being hidden.

Typical cells share DNA carrying heritable information, an aqueous cytoplasm in which reactions occur, ribosomes for protein synthesis, and a lipid-based plasma membrane. The membrane separates internal conditions from the surroundings while allowing regulated exchange. These are functional connections: a boundary without exchange could not supply continuing metabolism. Specialized mature cells can lose structures during development, as the red-blood-cell example below shows.

Microscopy: prepare, focus and measure

For a temporary mount, place a thin piece of suitable tissue in a drop of water on a slide. Lower a coverslip at an angle to reduce trapped air. A thick folded specimen hides structures in overlapping layers. Use approved preparations and stains, protect eyes and skin, and handle glass carefully. A stain can increase contrast between structures; it does not make every coloured object a nucleus. Staining or preparation may kill cells or alter their appearance.

Start with the lowest-power objective and centre the specimen. Use coarse focus to

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find the image at low power, then fine focus for detail. On changing to high power, use fine focus and avoid driving the objective into the slide. Adjust illumination rather than accepting a bright but washed-out image. Total optical magnification is eyepiece magnification multiplied by objective magnification: a 10× eyepiece and 40× objective give 400×.

magnification is image length divided by actual length. **resolution** is the ability to distinguish close points as separate. Enlarging a blurred photograph increases magnification without recovering detail that was never resolved. Conventional light microscopy resolves details down to roughly $0.2\,\mu$m under favourable conditions; preparation, optics and contrast affect the result. A bacterial outline can be visible while its ribosomes remain unresolved.

■ Worked example: use one unit throughout

An image is 30 mm long and represents a cell 50 micrometres long. Since $1\,\text{mm} = 1000\,\mu\text{m}$,

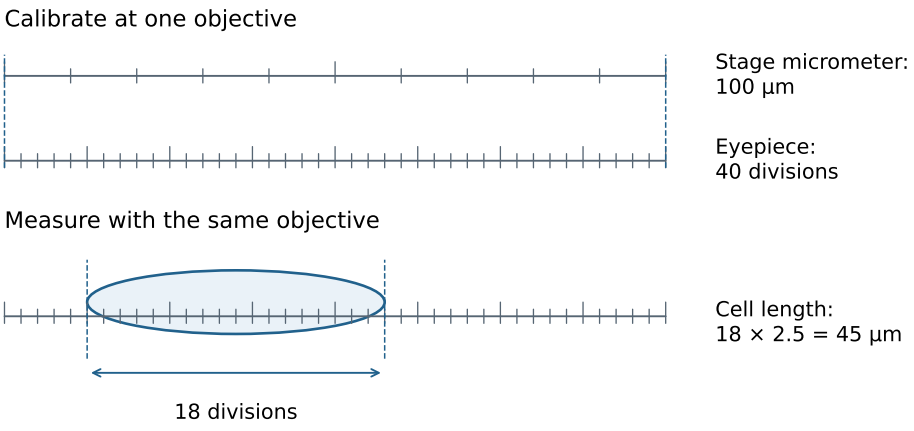
$$M = \frac{I}{A} = \frac{30 \times 1000}{50} = 600.$$

The ratio has no unit. A separate photograph with a 10 mm bar representing 20 micrometres has $M = 500$. A cell measuring 25 mm on that photograph is therefore $25\,000/500 = 50\,\mu$m long. Do not transfer the first photograph's magnification to the second.

■ Calibrate an eyepiece graticule

An eyepiece graticule has arbitrary divisions, not fixed micrometres. Superimpose it on a stage micrometer with a known scale. At one objective, suppose 40 graticule divisions align with 100 micrometres. Then one division represents $100/40 = 2.5\,\mu$m. A cell spanning 18 divisions is $45\,\mu$m long. At a different objective, recalibrate: the same graticule division covers a different actual distance.

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One division = 100 / 40 = 2.5 µm.
Recalibrate after changing the objective.

Calibration belongs to the objective setting. The divisions become distances only after comparison with a known scale.

To add a scale bar, convert a chosen actual distance using the calibrated image scale. At 500×, a 20 μm bar must be 10 mm long on the printed image. If the image and its bar are resized together, their ratio stays valid. A printed "500×" label becomes wrong after resizing; a correctly attached scale bar remains usable. Take photographs with the objective and calibration recorded, keep the original, and resize the bar with the image.

What better microscopes reveal

Electron microscopes use electrons and can resolve much smaller structures than conventional light microscopes. Transmission electron microscopy (TEM) reveals internal details in thin sections; scanning electron microscopy (SEM) mainly reveals surface form. A section is a two-dimensional cut through a three-dimensional object. The same mitochondrion can look long or round depending on the cutting plane.

Conventional electron-microscope preparation involves conditions unsuitable for living cells. The resulting image is evidence about prepared material, not a film of an undisturbed living cell. Colour in an electron micrograph is usually added later; it is not the specimen's natural visible colour.

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Technique	What it adds	Limit when interpreting evidence
Fluorescence light microscopy	Fluorophores emit light after excitation, highlighting selected components	Signals depend on labelling, background and resolution; colour alone does not identify an unlabelled structure
Immunofluorescence	An antibody binds a target antigen; a fluorescent label locates the binding	Specificity needs controls; failure to label does not automatically prove absence
Freeze fracture	Frozen material is fractured, often splitting membranes; replicas reveal membrane organization	Fracture faces are not simply the outside surfaces of intact living cells
Cryogenic electron microscopy	Rapid freezing can preserve hydrated structures without conventional chemical fixation and heavy-metal staining	It still examines frozen material; processing and reconstruction need evaluation

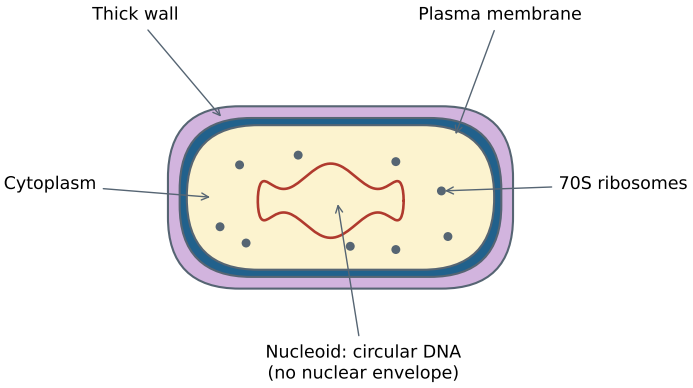
Choose a method for a question. Following a living cell's movement favours light microscopy. Resolving internal membrane folds favours TEM. Localizing one protein favours a validated fluorescent label. More magnification alone cannot solve every problem.

A prokaryotic cell: a Gram-positive model

Use a typical Gram-positive bacterium, such as *Bacillus*, as the model. Its cell wall contains peptidoglycan and lies outside the plasma membrane. The wall supports the cell and helps resist osmotic bursting; the membrane controls exchange. Gram-positive bacteria have a relatively thick peptidoglycan wall, without the additional outer membrane characteristic of Gram-negative bacteria.

The cytoplasm contains 70S ribosomes that synthesize proteins. The main chromosome is represented as a circular DNA molecule in a nucleoid region, with no surrounding nuclear envelope. "Naked DNA" here distinguishes the bacterial model from DNA packaged on eukaryotic histones; it does not mean that no proteins ever associate with bacterial DNA. Some bacteria also contain plasmids. Plasmids, flagella and capsules are not compulsory features of every bacterium.

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Gram-positive model; schematic, not to scale.
Accessory structures are omitted.

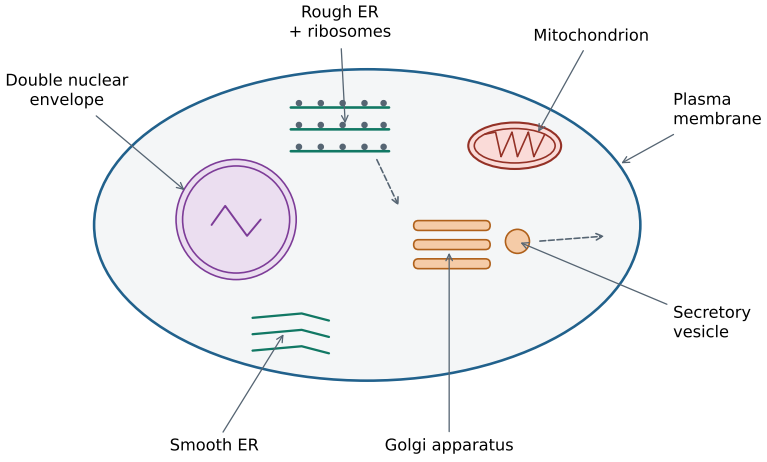
Identify the wall and membrane separately. The nucleoid is a region, not a membrane-bound nucleus.

Prokaryotic cells do not contain the membrane-bound organelles characteristic of eukaryotic cytoplasm. This does not mean that they lack internal organization or cannot respire. Bacterial species vary; the model is not a claim that every prokaryote has the same wall, shape or accessory structures. Never identify a bacterium solely because a blurred image shows no nucleus.

Eukaryotic compartments and their functions

A typical eukaryotic cell has a nucleus bounded by a double nuclear envelope containing pores. Chromosomes contain DNA associated with histone proteins. Pores allow regulated traffic between nucleus and cytoplasm. The cytoplasm outside the nucleus contains 80S ribosomes and membrane-bound compartments. Ribosomes themselves have no enclosing membrane.

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Animal-cell model, not to scale.
Dashed arrows trace part of a secretion route.

Trace a secreted protein from a ribosome on rough ER, through vesicles and the Golgi apparatus, to the plasma membrane.

Structure	**Recognition clue in a suitable image**	**Function to annotate**
Nucleus	Large compartment, double envelope; dense chromatin or chromosomes may be visible	Stores DNA and is a site of transcription
Mitochondrion	Double envelope and inner-membrane folds called cristae	Aerobic respiration and ATP production
Rough endoplasmic reticulum (ER)	Membrane sacs with attached ribosomes	Synthesis and processing of proteins entering the secretory pathway
Smooth ER	Tubular membranes without attached ribosomes	Lipid synthesis; other roles depend on cell type
Golgi apparatus	Stack of flattened sacs with associated vesicles	Modifies, sorts and packages cell products
Secretory vesicle	Small membrane-bound compartment	Carries material towards secretion by exocytosis
Lysosome	Membrane-bound compartment; identification needs context	Intracellular digestion using enzymes

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Structure	Recognition clue in a suitable image	Function to annotate
Chloroplast	Double envelope and internal thylakoid membranes, often stacked	Photosynthesis
Sap vacuole	Large fluid-filled compartment bounded by the tonoplast	Storage and maintenance of turgor

The mitochondrial matrix is the region inside the inner membrane; it contains enzymes for some respiration reactions. In a chloroplast, the stroma surrounds the thylakoid membranes. Condensed chromosomes can appear as dense rods during division; their function is to carry DNA into daughter cells. Ribosomes appear as much smaller dense particles in suitable electron micrographs, beyond ordinary light-microscope resolution.

The cytoskeleton includes microtubules and microfilaments. It helps maintain organization and shape, moves materials and participates in cell division. A microvillus is an extension of the cell surface supported by microfilaments, increasing surface area for exchange. It is not a cilium. Cilia and eukaryotic flagella have microtubule-based internal structures and commonly move the cell or surrounding fluid.

A lysosome cannot always be identified confidently from a single round dark profile. Use several structural clues and the preparation context. A drawing's neat colours and labels may make identification much easier than it is in a real micrograph.

One cell can carry out the processes of life

A freshwater *Paramecium* provides a connected example. Cilia move the cell and help bring food towards an oral region. Food enters food vacuoles, where digestion supplies nutrients. Enzyme-controlled metabolism uses these materials and releases energy. Waste products such as carbon dioxide leave across the cell surface; removal of metabolic waste is excretion, different from loss of undigested food.

Water tends to enter from the dilute surroundings. Contractile vacuoles collect and expel excess water, helping maintain internal conditions: an example of homeostasis. The organism responds to stimuli, grows by making cellular material, and reproduces by cell division. These functions happen in one cell; a contractile vacuole is not a miniature kidney and a food vacuole is not a multicellular stomach.

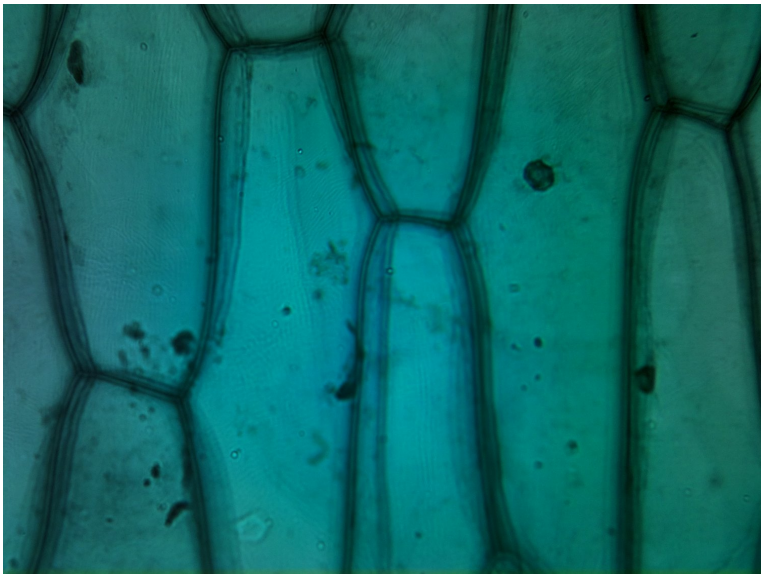
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Compare animals, plants and fungi

Feature	Typical animal cell	Typical plant cell	Typical fungal cell
Plasma membrane	Present	Present inside the wall	Present inside the wall
Cell wall	Absent	Mainly cellulose	Chitin and other polysaccharides
Vacuoles	Usually smaller compartments; no single large sap vacuole	Often a large central sap vacuole	Vacuoles involved in storage and regulation
Chloroplasts/plastids	Absent	Chloroplasts in photosynthetic cells; other plastids also occur	No chloroplasts
Centrioles	Common in many animal cells	Absent from most higher-plant cells	Vary among fungal groups; not universal
Cilia/flagella	Present in some cell types, such as ciliated epithelia or sperm	Absent from most vegetative cells; some plant gametes are motile	Present in some groups or stages, absent from many familiar fungi

A plant root cell can lack chloroplasts and still be a plant cell. Onion bulb epidermis is useful for observing cell boundaries and nuclei, but is a poor choice for demonstrating chloroplasts. A fungal wall does not make a fungus a plant; wall composition and nutrition differ. Presence/absence statements should name the cell type rather than treating one textbook sketch as universal.

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Observe the repeated cell outlines and darker nuclei. This photograph has no calibrated scale bar: do not calculate cell size from the online display width.

Atypical cells refine a model

An aseptate fungal hypha is a long tube with many nuclei sharing cytoplasm, without cross walls dividing it into the usual sequence of separate compartments. A skeletal muscle fibre is also multinucleate: it forms through fusion of precursor cells. Neither is well represented by a tiny "one nucleus, one round cell" sketch.

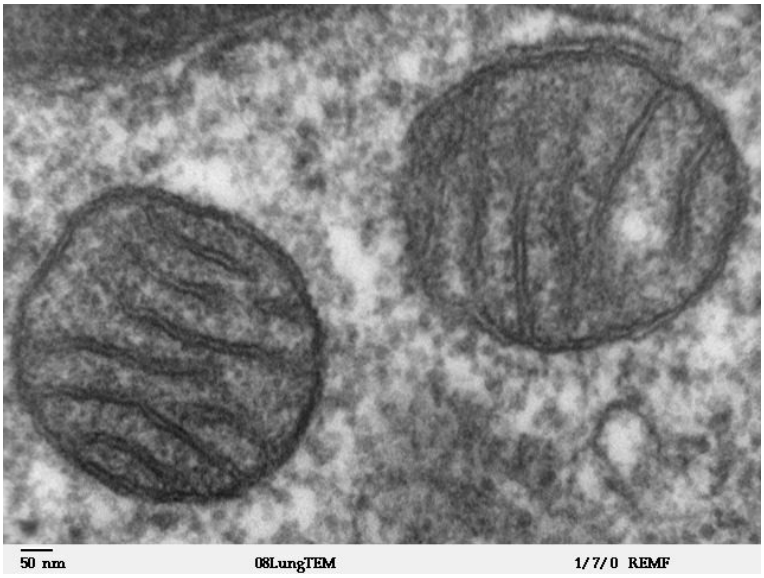
A mature mammalian red blood cell has no nucleus and lacks mitochondria. Losing organelles leaves space for haemoglobin and supports its specialized transport role, but limits repair and prevents normal cell division. This is a mature mammalian example, not a statement that all vertebrate red blood cells lack nuclei.

A mature phloem sieve tube element also lacks a nucleus and loses many organelles. It remains living and depends on associated companion cells for support. It is not an empty dead tube like a mature xylem vessel element. These examples show why "every living cell now contains a nucleus and every organelle" is a poor rule.

Read micrographs and draw what is there

First identify the image method and scale, then examine boundaries, compartments and internal membranes. Plant identification is stronger when a wall, large vacuole and/or plastids agree. A nucleus with membrane-bound organelles supports a eukaryotic identification. A nucleoid and bacterial envelope support a prokaryotic identification. Shape alone is insufficient, and a missing feature may simply lie outside the section or below the instrument's resolution.

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Follow the organelle boundaries and internal cristae. The scale bar, rather than the displayed pixel width, supports length estimates.

Make a large, clear line drawing with continuous outlines and no decorative shading. Preserve relative sizes and draw the structures actually visible, not every organelle recalled from a model. Use straight ruled label lines that end on the intended structure and do not cross. A drawing of a mitochondrion should show the outer boundary, inner membrane/folds and internal region when visible. A chloroplast drawing should distinguish its envelope, internal stacks and surrounding stroma; a nucleus should show envelope, pores or chromatin only where supported by the image.

Annotations add functions to labels: "cristae: increase inner-membrane area for ATP-producing processes" explains more than "cristae" alone. Label a cell wall outside the plasma membrane, a vacuole inside its tonoplast, and microvilli as projections of the surface. Give an accurate scale bar or magnification calculated for your drawing. Record image source and distinguish direct observations from functional interpretations.

AHL: Endosymbiosis explains organelle origins

The endosymbiotic model links mitochondria to bacteria that became permanent residents within an ancestral cell. A later association with a photosynthetic bacterial lineage explains chloroplasts in some eukaryotes. The shared ancestry of eukaryotes includes an ancestor with a nucleus and sexual reproduction; the precise early sequence of host and organelle evolution remains a research question. Do not present an imagined photograph of an engulfment event as direct evidence.

Mitochondria and chloroplasts retain DNA, commonly represented as naked circular molecules in this course model, bacterial-type ribosomes and the ability to replicate within cells. Their double envelopes and molecular relationships also fit the model.

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These observations jointly support descent from bacterial lineages. The 70S bacterial-type ribosome model is used here; actual mitochondrial ribosomes vary among eukaryotic groups.

Organelle replication is not complete independence: many necessary proteins are encoded by nuclear genes and imported. Several consistent observations are stronger than one superficial resemblance. The model explains particular organelles; it does not imply that the nucleus, Golgi apparatus and every membrane compartment arose from engulfed bacteria.

AHL: Differentiation and multicellularity

Differentiation produces cells with specialized structures and functions through different patterns of gene expression. A muscle cell and a nerve cell generally retain the same genome, but use different subsets of genes and make different amounts of particular proteins. Signals and environmental conditions can trigger changes in expression. Specialization does not usually require deleting every gene a cell does not currently use.

Multicellularity evolved repeatedly in different lineages. Animals and plants are multicellular, as are many fungi and eukaryotic algae. Cells remaining together can build a larger body, divide work and create a controlled internal environment. Adhesion, communication and coordinated resource supply become important. Cells deep inside a large body cannot all rely on rapid diffusion directly from the external surroundings, so larger size brings transport and coordination demands as well as benefits.

A multicellular organism is more than a loose crowd of independent cells. Specialized cells depend on one another: transport cells supply tissues, while other cells process nutrients or provide support. Loss of independent reproduction by a mature specialized cell can benefit the organism's coordinated functions.

Practice

All numerical data below are invented. Show conversions and state the evidence needed for identifications.

1. State three central ideas of cell theory. Use the theory to predict a property of a newly discovered organism, and explain why the prediction still needs observation.
2. Describe how to make a temporary mount, find a specimen at low power and change safely to high power. Explain what a stain changes and why an air bubble can mislead.
3. A cell image is 12 mm long and the actual cell is 40 micrometres long. Calculate magnification. Repeat for a 15 mm image of a 20 micrometre cell.
4. In another photograph a 10 mm bar represents 20 micrometres. A cell is 24 mm long on the same print. Calculate actual cell length. The whole photograph is enlarged twofold: state the new image length, bar length and actual cell length.

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5. At one objective, 50 graticule divisions correspond to 125 micrometres. A cell spans 22 divisions. Find its length. At a second objective, 50 divisions correspond to 50 micrometres; the same cell spans 55 divisions. Explain whether the measurements agree.
6. Distinguish magnification from resolution. Select a method for observing a living cell's movement, internal mitochondrial membranes and the location of a particular protein. Give a limitation for the last method.
7. Describe five structures in the Gram-positive bacterial model and link each to its function. Explain why "no nucleus" does not mean "no DNA" or "no protein synthesis".
8. Trace a protein destined for secretion through a eukaryotic cell. Distinguish the roles of ribosomes, rough ER, Golgi apparatus, vesicles and plasma membrane. Name two cytoskeletal components.
9. Link four processes of life to structures or activities in *Paramecium*. Distinguish excretion from removal of undigested food.
10. An image shows a wall, a large vacuole and a nucleus but no chloroplast. Explain why this does not rule out a plant cell. Compare plant and fungal walls, and explain two atypical examples that challenge a one-nucleus-per-cell sketch.
11. Explain how to identify and draw a mitochondrion from a suitable TEM image. Give two functional annotations and explain why drawing every memorized organelle would weaken the observation record.
12. **AHL:** Give three observations supporting endosymbiosis. Explain why a mitochondrion replicating within a cell is not fully independent, and why the evidence does not imply that every organelle was once a bacterium.
13. **AHL:** Explain how two specialized cells can retain the same genome but differ in structure. Give two benefits and two coordination demands associated with multicellularity.

■ Worked answers

1. Organisms consist of one or more cells; cells are their basic structural/functional units; present-day cells come from existing cells. Predict that the organism has at least one cell. Examine it at suitable resolution: deduction generates a testable expectation, not an observation.
2. Put thin tissue in water on a slide and lower the coverslip at an angle. Start with low power, centre and focus, then use fine focus at high power without driving the lens into the slide. Stain increases contrast and can alter or kill the sample. A bubble can imitate a circular boundary but is not a cell.
3. $M = 12\,000/40 = 300$; $M = 15\,000/20 = 750$. Both are dimensionless ratios, with image and actual lengths in micrometres.

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4. $M = 10\,000/20 = 500$; actual length $= 24\,000/500 = 48\,\mu$m. After enlargement, cell image = 48 mm and bar = 20 mm. Actual cell length remains $48\,\mu$m; the magnification becomes 1000.
5. First calibration $= 125/50 = 2.5\,\mu$m per division, so length $= 22(2.5) = 55\,\mu$m. Second calibration $= 50/50 = 1\,\mu$m per division, giving $55\,\mu$m. The readings agree after using the correct calibration.
6. Magnification enlarges an image; resolution separates close details. Use light microscopy for movement, TEM for internal membranes, and validated fluorescence/immunofluorescence for a protein. Antibody non-specificity or background can mislead; include controls. A larger blurry image need not reveal more detail.
7. Wall: support/resistance to osmotic bursting; membrane: regulated exchange; cytoplasm: reaction medium; circular chromosomal DNA in a nucleoid: heritable information; 70S ribosomes: protein synthesis. The nucleoid has no nuclear envelope, but DNA and ribosomes are present.
8. A ribosome on rough ER synthesizes the protein into the secretory pathway; ER processes it; transport vesicles carry it to the Golgi for modification/sorting; secretory vesicles fuse with the plasma membrane to release it. Microtubules and microfilaments contribute to organization and movement. Ribosomes are not membrane-bound organelles.
9. Any four connected examples: cilia produce movement; food uptake/digestion supplies nutrition; enzyme-controlled reactions provide metabolism; contractile vacuoles support water balance/homeostasis; growth adds cellular material; cell division reproduces; altered activity responds to stimuli. Excretion removes metabolic waste, such as carbon dioxide; undigested food was not produced as a metabolic waste.
10. Non-photosynthetic plant cells can lack chloroplasts; the combined features fit a plant cell, though identification needs context. Plant walls contain cellulose; fungal walls contain chitin. Aseptate fungal hyphae and skeletal muscle fibres have many nuclei in shared cytoplasm. Mature mammalian red cells and sieve tube elements instead lack nuclei; sieve elements depend on companion cells.
11. Look for a double envelope and folded inner membrane in a suitable thin section. Draw the observed outlines and folds in proportion, add straight non-crossing labels and a scale. Annotate cristae as increasing inner-membrane area for ATP-producing processes, and the matrix as containing enzymes for some respiration reactions. Invented extra organelles would mix recall with observation.
12. **AHL:** Organelle DNA, bacterial-type ribosomes and replication within cells support bacterial ancestry; molecular comparisons add evidence. Many required proteins are nuclear-encoded and imported, so replication is not full autonomy. The combined evidence concerns mitochondria/chloroplasts, not every membrane compartment.
13. **AHL:** Different expression patterns produce different proteins and structures without generally changing the genome. Larger body size and division of labour are

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benefits. Coordinated transport/resource supply and communication/control are demands; adhesion also keeps cells organized. Signals can help trigger differentiation.

Exam tips

Use matching length units; distinguish magnification from resolution and recalibrate after changing objective. Identify cells from several features, allowing for sectioning and resolution limits. Draw observed structures with a scale, source and clear labels; annotate functions separately. Qualify typical-cell models: not every plant cell has chloroplasts, and specialized mature cells can lose nuclei or organelles.

References: IB Biology guide, A2.2; image credits recorded with figures.

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Viruses

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

Why can a tiny infectious particle use a much larger cell to make thousands of copies, and why can yesterday's effective immune response become less effective against a changed virus?

This is the HL-only A2.3 unit; there is no SL counterpart. It covers viral structure and diversity, host dependence, the lytic and lysogenic cycles of bacteriophage lambda, possible multiple origins, and rapid evolution using influenza and HIV. It also uses infection rates to practise fair comparisons. Prerequisites: DNA/RNA, gene expression, membranes and natural selection. Numerical data below are invented teaching examples, not measurements from patients or named outbreaks.

AHL: A virus is not a small bacterial cell

A virus contains a nucleic-acid genome protected by a protein capsid. A complete particle outside a host cell is a virion. Unlike a cell, it has no cytoplasm containing ribosomes and a complete metabolic system. It does not grow into a larger cell and divide by binary fission. New particles are assembled from components made during infection.

The genome is DNA or RNA, depending on the virus, and can be single- or double-stranded. "All viruses contain DNA" is therefore wrong. A virus can make different nucleic-acid forms during its replication cycle; distinguish the genome packaged in the particle from intermediate molecules inside an infected cell.

Some viruses have a lipid envelope acquired from host membranes during formation. Viral proteins inserted into the envelope help recognize and enter suitable host cells. The envelope is not a bacterial cell wall, and it does not supply a cell's cytoplasm or autonomous metabolism. Other viruses have no envelope. Capsid structure, genome type and entry mechanism vary widely, so one spherical diagram cannot represent every virus.

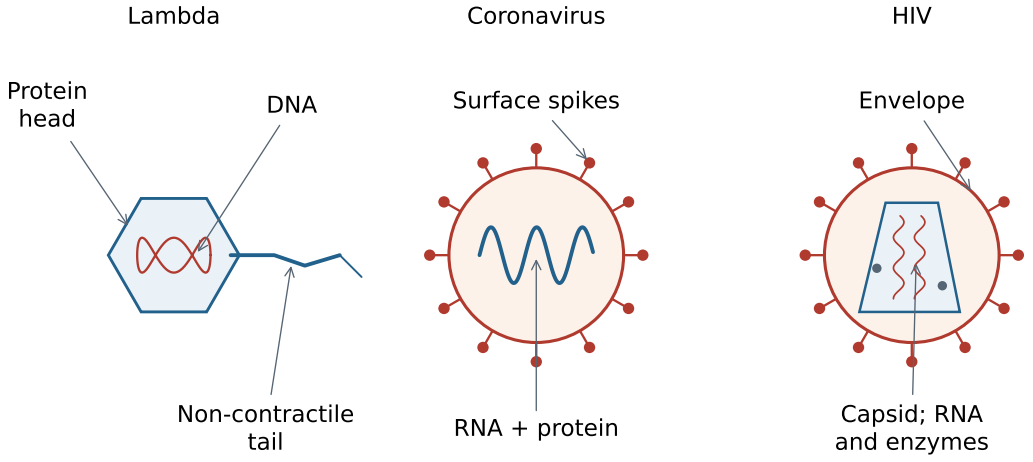
Viruses carry relatively few enzymes compared with cells; some carry enzymes essential for the first steps of infection. HIV, for example, carries reverse transcriptase. Having an enzyme does not make a virus independent: it still needs host resources and ribosomes to synthesize its proteins.

A **pathogen** is an agent that causes disease. Not every virus infects humans, and an infection does not necessarily cause immediate visible illness. Bacteriophages infect bacteria. The word "virus" describes a kind of infectious agent, while "pathogen" describes a disease-causing role.

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AHL: Compare lambda, coronavirus and HIV

Feature	Bacteriophage lambda	Coronavirus	HIV
Usual host example	*Escherichia coli* bacterium	Susceptible animal cells; host range depends on the coronavirus	Susceptible human cells, especially certain immune cells
Packaged genome	Double-stranded DNA	Single-stranded RNA	Two copies of single-stranded RNA
Main structural model	Protein head and long non-contractile tail	Envelope with surface spike proteins and an internal RNA–protein complex	Envelope with surface proteins and a capsid containing RNA and enzymes
Envelope	Absent	Present	Present
Important replication feature	Can follow lytic or lysogenic pathways	Uses RNA copying during replication	Reverse transcription produces DNA that can integrate into host DNA



Schematic organization; no shared scale.
Envelope proteins and genomes are simplified.

Compare organization rather than colour. Lambda's tail is not the contractile syringe drawn for some other phages.

Binding depends on interactions between viral surface components and suitable host receptors. A virus that cannot attach to, enter or use a cell may fail to reproduce there.

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Host range is therefore a biological relationship, not a virus choosing a preferred victim. Receptor binding alone may be insufficient if later steps cannot occur.

■ **Worked example: a particle on a printed image**

An invented micrograph shows a 9.0 mm particle whose actual diameter is 90 nm. Convert both lengths to nanometres:

$$M = \frac{9.0 \times 10^{6}\,\text{nm}}{90\,\text{nm}} = 1.0 \times 10^{5}.$$

The ratio is dimensionless. At this scale an ordinary light microscope would not resolve the particle's internal capsid and genome. A large printed image is not evidence that the original instrument resolved every drawn feature. For an electron micrograph, do not infer natural colours from added colour coding.

AHL: Host dependence and assembly

During infection the viral genome directs production of viral components using an infected cell. The host supplies amino acids and other building materials, energy, and ribosomes for translation. Different viruses use different combinations of viral and host enzymes for copying and expressing their genomes.

A useful chain of reasoning is genome → expression of viral proteins → genome copying → assembly → release. This is a functional outline, not one universal sequence of identical molecular steps. RNA genomes do not all enter the same pathway as DNA genomes. HIV uses reverse transcriptase to make DNA from RNA; this is the reverse of ordinary DNA-to-RNA transcription.

Some particles are released by cell lysis, others by budding or related membrane processes. Budding can supply a lipid envelope containing viral proteins. Do not use the lambda lytic model to claim that every virus always bursts its host cell immediately. An infected cell may continue functioning for a time while producing particles or carrying viral genetic material.

Antibiotics target bacterial structures or processes, such as cell-wall synthesis or bacterial ribosomes. Viruses do not have these targets as independent cellular systems. Antiviral approaches instead target stages of viral replication or entry. This distinction explains a treatment principle; it does not mean that every antibiotic works against every bacterium or every antiviral works against every virus.

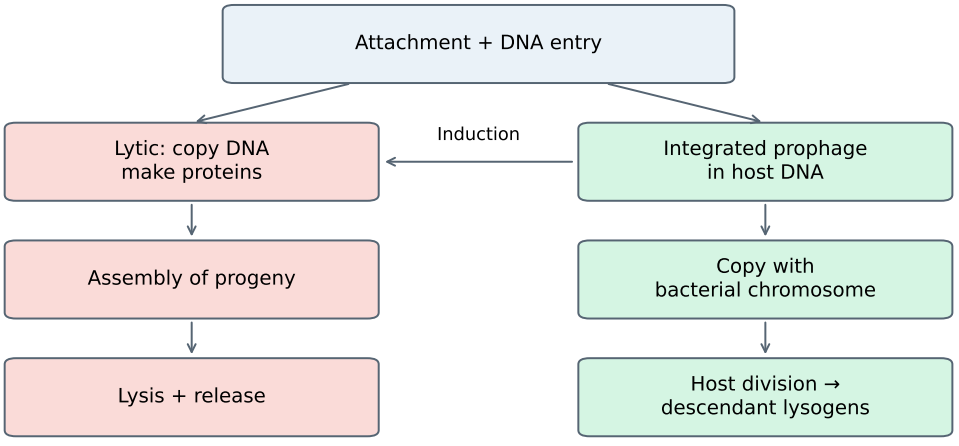
An **antigen** is a structure recognized by a specific immune response; viral surface proteins can be antigens. Vaccination exposes the immune system to antigens so that memory cells can support a later response. This is different from an immediate cure for an established infection. Changes to antigens can affect recognition, connecting immune memory to viral evolution.

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AHL: The lambda lytic cycle

Bacteriophage lambda is a temperate phage: following infection it can enter either a lytic pathway or a lysogenic state. In the lytic pathway, distinguish the particle outside the cell from the viral genome and components inside it.

1. **Attachment:** the tail region binds suitable receptors on the bacterial surface.
2. **Entry:** phage DNA enters the bacterium. The whole head and tail are not injected as a ready-made new cell.
3. **Biosynthesis:** phage genes are expressed; viral DNA is copied and phage proteins are made using host resources.
4. **Assembly:** genomes are packaged into new heads and tails are attached to form progeny particles.
5. **Lysis and release:** phage-directed processes disrupt the bacterial envelope, releasing progeny able to infect other susceptible bacteria.



Lysogeny copies integrated DNA with the host.
It does not itself require a burst of free particles.

Follow the DNA in the two pathways. Integration and host-cell division are different from immediate particle assembly.

The infecting particle is not enlarged until it splits. The host cell manufactures components that assemble into many new particles. "Replication" can refer to genome copying; explain whether a question asks about DNA replication, particle production or increase in infected cells.

■ **Worked example: estimate burst size under stated conditions**

In an invented single-cycle model, 80 infected bacterial cells lyse and release 4,800 newly produced infectious particles. Residual input particles have been removed and no second infection cycle occurs.

$$\text{mean burst size} = \frac{4800}{80} = 60 \text{ infectious particles per infected cell.}$$

This is an average under the model, not a fixed property of all viruses or even every infected cell. A plaque-based count measures infectious units under the assay conditions; it need not equal the number of all physical particles, some of which may be non-infectious. Do not mix a whole-culture titre with a count per infected cell.

AHL: Lysogeny preserves a different state

In lambda lysogeny, viral DNA integrates into the bacterial chromosome as a prophage. The bacterium carrying it is a lysogen. Viral regulation suppresses immediate production of a lytic burst. As the bacterium copies its chromosome and divides, the integrated phage DNA is copied and passed to descendant cells.

The prophage is not a fully assembled particle hiding inside the nucleus: bacteria have no membrane-bound nucleus, and the integrated material is DNA. The term "dormant" should not suggest that the bacterial cell stops growing or that every viral gene must be silent.

Under suitable induction conditions, such as particular DNA-damage responses, lambda can leave the integrated state and enter the lytic pathway. Excision, renewed viral gene expression, genome copying, assembly and release then occur. Induction is a regulated switch, not the phage deciding to become aggressive. Not all viruses integrate their genomes, and HIV integration should not be labelled the lambda lysogenic cycle without explaining the differences.

■ **Worked example: follow a prophage through divisions**

Begin with one lysogen containing one integrated prophage. In a simplified model there is no induction, loss or cell death. Four rounds of binary division give $2^4 = 16$ descendant cells carrying the prophage. This does **not** imply that 16 free virus particles have been released: the counted objects are bacteria containing integrated viral DNA.

AHL: Why viruses may have several origins

Viruses differ greatly in genomes, replication systems, capsid organization and host relationships. These differences are consistent with several origins rather than requiring one simple ancestral virus from which every viral feature descended unchanged. Proposed routes include genetic elements escaping cells and cellular parasites losing

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functions as dependence increased. Ancient exchanges of genes can complicate the history.

The shared genetic code connects viral gene expression to cellular translation systems. It does not mean that all viruses share identical genes or genomes. Similar needs can also favour similar solutions: genome protection and transfer between hosts are useful to unrelated obligate parasites.

Convergent evolution produces similar functional features independently. A protective protein shell is therefore not, by itself, proof that all viruses descend from one recent common viral ancestor. Compare sequences, replication mechanisms and structural evidence across several groups. Conversely, diversity alone does not identify the exact historical route for any one virus. State an explanation as a hypothesis with evidence and limits, not a directly witnessed origin story.

AHL: Rapid evolution needs variation and selection

Evolution is a change in inherited characteristics of populations across generations. In some viruses, rapid copying, large populations and relatively error-prone genome copying generate many variants over short times. Mutations are not produced because a virus knows which change it needs. Many mutations are harmful or neutral; only some improve reproduction in a particular environment.

Selection acts when variants differ in reproductive success. An immune response may reduce the success of one surface-antigen variant more than another. A drug can inhibit susceptible variants more effectively than resistant variants. A resistant variant can then form a larger fraction of the population. Conditions determine the advantage; resistance can carry costs when the selective pressure is absent.

Do not turn this into "all RNA viruses evolve equally fast". Copying accuracy, proofreading, population bottlenecks, genome structure and selection differ. A measured mutation rate is also not automatically the same as a long-term evolutionary substitution rate.

■ Influenza: drift and shift are different mechanisms

Small genetic changes can alter influenza surface antigens, including haemagglutinin and neuraminidase. Accumulation of such changes is antigenic drift. Changes can reduce recognition by antibodies produced after an earlier infection or vaccination, without making the immune system entirely useless against every variant.

Influenza has a segmented RNA genome. If different influenza viruses infect the same cell, progeny can receive a new combination of genome segments: reassortment. In influenza A, a major antigenic change can produce antigenic shift. Shift is not simply one more small mutation, and not every reassortment causes a pandemic. Transmission ability and population immunity also matter. Seasonal influenza change creates a continuing need for surveillance and assessment of vaccine strain matching.

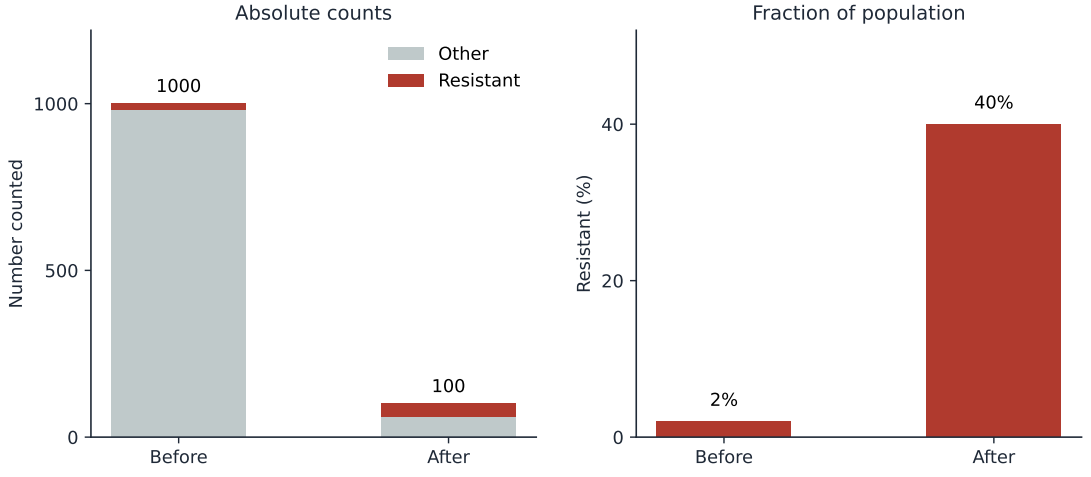
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HIV: copying errors and selection complicate control

HIV copies its RNA genome through a DNA intermediate using reverse transcriptase, which lacks the proofreading accuracy of many cellular DNA-copying systems. Frequent replication and genetic variation provide material for selection. Variants can differ in susceptibility to immune responses or antiretroviral drugs; recombination can also contribute to variation.

Drug exposure does not deliberately instruct HIV to produce a useful resistance mutation. Where susceptible variants are inhibited more strongly, variants already able to reproduce under those conditions can become more common. Combination antiretroviral therapy targets multiple aspects of replication, helping suppress reproduction and limiting opportunities for resistance. Latent integrated viral DNA is another reason why suppressing replication is different from removing every viral genome.

AHL: Interpret counts before making a claim



Invented data: resistant 20/1000 → 40/100.
Total count falls 90%; resistant count doubles.
Resistant fraction rises 20-fold.

Resistance rises from 2% to 40% while total count falls. Frequency, absolute number and total population answer different questions.

In this invented example, the resistant fraction rises twentyfold, but resistant count rises only twofold. The total population falls from 1,000 to 100, a 90% reduction. Calling this simply "the virus increased" would hide three different quantities. The pattern is consistent with differential success, but counts alone do not identify the resistance gene or prove that no other process affected the sample.

For infection comparisons, keep population size and observation period consistent. In an invented group A, 18 cases among 900 people over one month give $18/900 \times 1000 = 20$ cases per 1,000 people. Group B has 10 cases among 250 over the same month: 40

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per 1,000. B has fewer cases but a higher proportion. Equal denominators improve comparison; they do not remove differences in exposure, testing, age or immunity. Distinguish existing cases at one time from new cases during an interval.

Use published datasets or simulations for school analysis, not collection of unknown pathogens. A model of differential reproduction is enough to investigate the logic of selection without handling infectious material.

Practice

All numerical data are invented. Identify the objects counted and distinguish observations from explanations.

1. Describe the minimum structural model of a virus and explain why it is not a bacterial cell. Distinguish a genome from a capsid and a virion from an infected cell.
2. Compare lambda, coronavirus and HIV for packaged genome, envelope and overall organization. Explain why a lambda diagram should not automatically have a contractile tail.
3. Name three resources a virus obtains from a host cell. Explain why HIV carrying reverse transcriptase does not make it an autonomous living cell.
4. Put attachment, assembly, entry, biosynthesis and lysis into the order of a lambda lytic infection. State what enters the bacterium and what is made inside it.
5. Explain integration, a prophage, a lysogen and induction. In a no-loss/no-induction model, how many prophage-carrying cells descend from one lysogen after five rounds of division? How many free particles must this imply?
6. An invented single-cycle investigation finds 6,300 newly produced infectious particles from 90 infected cells. Input particles were removed. Calculate mean burst size and give two reasons why this number is not a universal physical-particle count per cell.
7. A virus image is 12 mm long and actual length is 120 nm. Calculate magnification. Explain why digital enlargement does not establish better resolution.
8. Explain how viral diversity and convergent evolution are relevant to possible multiple origins. Why is a protein capsid alone insufficient to establish one common origin?
9. Distinguish influenza antigenic drift from shift. Explain the role of genome segments and why a major change does not automatically prove pandemic spread.
10. In the selection figure, calculate the resistant percentage before and after, its fold increase, the change in resistant count, and the percentage fall in total count. Explain the selection interpretation without saying the drug teaches the virus to mutate.

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11. Explain two reasons HIV can evolve rapidly and two biological difficulties for controlling it. Distinguish suppression of replication from removal of every integrated genome.
12. Calculate cases per 1,000 for 12 cases among 600 people and 8 among 500 over the same month. Which group has the higher proportion, and why would different testing practices limit the comparison?
13. Explain why an antibiotic aimed at bacterial cell-wall synthesis does not directly stop viral replication. Distinguish vaccination-induced immune memory from an immediate cure for an established infection.

■ Worked answers

1. A nucleic-acid genome is protected by a protein capsid; some viruses also have envelopes and enzymes. The genome carries information; the capsid protects/packages it. A virion is a complete particle, not a cell with cytoplasm, ribosomes and autonomous metabolism. An infected cell is the host system used to produce new viral components.
2. Lambda: double-stranded DNA in a protein head with a long non-contractile tail, no envelope. Coronavirus: single-stranded RNA with associated protein, inside an envelope bearing spikes. HIV: two RNA genome copies plus enzymes inside a capsid and envelope. Contractile tails occur in some other phages; they are not the lambda model.
3. Host ribosomes translate proteins; host building materials supply components; host energy supports synthesis. Reverse transcriptase catalyses RNA-to-DNA copying but cannot supply the entire protein-synthesis and metabolic system.
4. Attachment → entry → biosynthesis → assembly → lysis/release. Lambda DNA enters; the whole particle is not injected as a cell. DNA and proteins are produced, then assembled into progeny particles.
5. Viral DNA integrates into bacterial DNA as a prophage; its bacterial carrier is a lysogen. Induction switches towards the lytic pathway. $2^5 = 32$ descendant cells carry the prophage under the assumptions. This implies no necessary release of free particles; the model can have zero release throughout.
6. $6300/90 = 70$ infectious particles per infected cell on average. Cells can differ in output, and infectious units need not include all physical particles. Assay conditions and viral/host identity also affect the estimate; residual input or a second cycle would invalidate the simple calculation.
7. $12\,\text{mm} = 12 \times 10^6\,\text{nm}$, so $M = 12 \times 10^6/120 = 100\,000$. Enlarging the displayed image cannot reconstruct details not resolved in the original recording.
8. Different genomes, structures and replication systems are consistent with several origins; similarities can also arise because obligate parasites face similar functional demands. One protective shell is not enough: compare independent sequence and mechanistic evidence. Proposed origins remain historical explanations evaluated with evidence.

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9. Drift involves smaller accumulated genetic changes affecting antigens. Shift is a major antigenic change in influenza A, often linked to reassortment of segments when different viruses co-infect a cell. A new combination does not guarantee efficient transmission or the population conditions for pandemic spread.
10. Before $20/1000 = 2\%$; after $40/100 = 40\%$. Resistant fraction rises $40/2 = 20$ times; resistant count doubles from 20 to 40. Total count falls $(1000-100)/1000 = 90\%$. Differential inhibition favours variants able to reproduce under the conditions; useful mutations are not directed by need.
11. Error-prone reverse transcription and frequent replication generate variation; selection can favour resistant or immune-escape variants. Variation and long-lived integrated genomes complicate control. Combination therapy suppresses replication through multiple targets; low replication does not establish that every integrated viral genome has been removed.
12. $12/600 \times 1000 = 20$ and $8/500 \times 1000 = 16$ per 1,000. The first proportion is higher. Different testing can change how many infections are detected, so the difference need not reflect transmission alone.
13. Viruses have no bacterial peptidoglycan cell-wall synthesis to target and use host cellular machinery. Vaccination can establish antigen-specific memory that supports a later response; it is not an instant cure after infection. A bacterial co-infection would be a separate question about treatment targets.

Exam tips

- Follow the genetic material: particle genome, copied genome, integrated prophage and released particle are different objects.
- State lambda's two pathways precisely. Host-cell division during lysogeny is different from immediate assembly and lysis.
- Explain rapid evolution through inherited variation and differential reproductive success, without intention or need-directed mutation.
- Keep counts, fractions, rates and observation periods separate. A higher resistant fraction can coexist with fewer total particles.

References: IB Biology guide, A2.3; CDC: influenza drift and shift; NIH: HIV drug resistance.

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Diversity of organisms

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

Two organisms can look alike yet belong to different species. Two cells can contain very different amounts of DNA without the organism with more DNA being more complex. Diversity needs several kinds of evidence, with a clear definition of what is being compared.

This unit covers A3.1: variation, species concepts and names, chromosome numbers and karyograms, evidence for human chromosome 2 fusion, genome diversity and size, and whole-genome sequencing. SL understandings plus limits of species concepts, chromosome pairing, local identification keys and environmental DNA (marked AHL). Prerequisites: DNA, chromosomes, cell division and percentages. Worked datasets and chromosome/leaf diagrams are invented unless a named database or study is explicitly cited.

Variation and the problem of defining a species

Variation means differences among individuals. Some differences are inherited; others arise through environmental conditions or development. Leaf length in one species may vary with genotype, light, water and age. A measured difference therefore does not automatically identify a genetic difference or a different species. Record the character, measurement method, life stage and environmental conditions before comparing samples.

A species is a way of recognizing and naming groups in this diversity. Under a morphological species concept, organisms are grouped by shared structural traits. This is useful for field identification and fossils, where breeding tests or living DNA may be unavailable. Use several stable diagnostic characters rather than one colour or size. Young and adult forms, male and female forms, and environmental effects can make members of one species look different. Conversely, unrelated organisms can evolve similar features, and some distinct species look very similar.

Under the biological species concept, a species consists of populations whose members can interbreed and produce viable, fertile offspring, with reproductive isolation from other such groups. It concerns populations and their potential gene flow; it does not require every individual to mate with every other individual. Age, sex or temporary geographical separation of two individuals does not by itself define different species.

The concepts answer related but different questions: “Can I distinguish these forms?” and “Do their populations form a reproductive group?” There is no single simple test that works perfectly for every organism and every historical sample. State which concept a question uses and which evidence it makes available.

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■ **Worked example: one visible trait is insufficient**

In an invented population, adults have two different colour forms and readily produce fertile offspring with one another under natural conditions. Colour alone is weak evidence for splitting the forms into separate biological species. If a second population looks similar but does not exchange genes because of consistent reproductive barriers, appearance alone could also hide a real species boundary. The conclusion depends on population-level evidence, not an assumption that similar means identical.

Scientific names identify the group precisely

A binomial name contains a genus name followed by a specific epithet. In *Homo sapiens*, *Homo* is the genus and the complete two-word name identifies the species. Capitalize the genus, use lowercase for the epithet, and italicize both when typing. When handwriting, underline the two parts separately. After first writing the full name, the genus may be abbreviated when unambiguous: *H. sapiens*. "Sapiens" alone is not the full species name.

Pan troglodytes and *Pan paniscus* share a genus but are different species. Sharing a genus does not mean sharing every feature or having the same chromosome sequence. Common names can vary between languages and regions, so a recorded scientific name improves communication and makes a database search more precise.

A **taxon** is a named classification group, such as a species or genus. A **clade** comprises a common ancestor and all its descendants. Naming a group and inferring ancestry are linked tasks, but a name alone is not evidence for one branching pattern. A modern species is not automatically the direct ancestor of a different modern species.

Speciation can make boundaries difficult to draw

Speciation is the splitting of an ancestral species into two or more species. Reduced gene flow allows separated populations to diverge through mutation, selection and other population processes. Differences in mating behaviour, breeding time or compatibility can develop gradually. Populations at an intermediate stage may be partly isolated rather than perfectly connected or perfectly separate.

A judgement about whether to name two populations as species may therefore depend on the evidence and the species concept. Consistent genetic differences do not automatically establish complete reproductive isolation; occasional hybridization does not automatically make all populations one species either. Compare fertility, patterns of gene flow, morphology and molecular relationships in context.

An observed sequence comparison supplies one piece of evidence. Six differences among 40 aligned sites give $6/40 = 0.15 = 15\%$. This is a difference proportion for that alignment, not a universal species threshold or an evolutionary date. Reliable comparison

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needs homologous regions and appropriate alignment. Test whether additional independent characters or genes support the proposed grouping. On an unscaled branching diagram, rotating branches at a node does not change ancestry, and branch lengths must not be read as measured time.

Chromosome number, DNA amount and copying

Chromosome number varies among species. Typical human diploid body cells have 46 chromosomes, arranged as 23 pairs; chimpanzees typically have 48, arranged as 24 pairs. Human gametes normally have 23 chromosomes, while chimpanzee gametes normally have 24. More chromosomes does not automatically mean more genes, more total DNA or greater complexity.

In the usual diploid model, homologous chromosomes occur in pairs, one inherited from each parent. They carry corresponding genes, although their alleles can differ. The paired model gives $2n$ chromosomes; gametes produced by meiosis have n. This describes the normal model, not a claim that chromosome-number abnormalities or unusual sex-chromosome systems cannot exist.

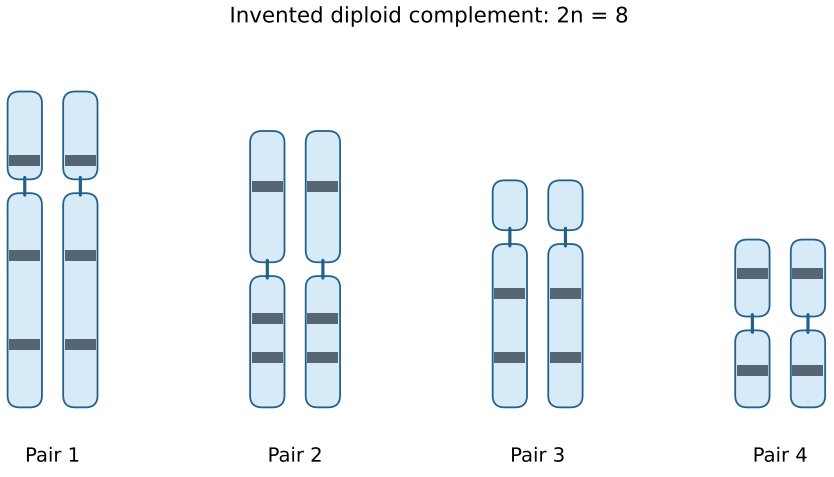
Count chromosomes by centromeres in a standard chromosome-count question. After DNA replication, a human cell still has 46 chromosomes, now with two sister chromatids each, giving 92 chromatids before their separation. Homologous partners are not the same thing as the two sister chromatids of one copied chromosome. The amount of DNA doubles during replication even though the chromosome count at that stage does not.

Build and interpret a karyogram

A karyogram displays chromosomes arranged into matching groups. A karyotype describes the chromosome complement, including number and appearance. In a typical preparation, cells are observed when chromosomes are condensed, stained or otherwise imaged, then chromosomes are identified and arranged. A spread of dark rods needs analysis; it is not already a labelled karyogram.

Match homologues using relative length, centromere position and banding pattern. Length alone is not enough, and orientation can be reversed in an image. In a copied metaphase chromosome, the two joined chromatids belong to one chromosome. Pairing homologues in a karyogram is a comparison made by the analyst, not an assertion that the photographed chromosomes were already physically paired along their lengths.

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Pair by length, centromere position and bands.
Eight chromosomes; four homologous pairs.

Four pairs mean eight chromosomes, not four. Each rod represents one chromosome; chromatids are not shown separately. This is not a real human karyogram.

■ Worked example: classify an invented complement

Suppose a sample contains two long chromosomes with matching bands, two medium chromosomes with a central centromere, and two short chromosomes with matching bands. The complement has three homologous pairs: $2n = 6$, so a normal haploid set has $n = 3$. After replication but before chromatid separation, there are still six chromosomes, with 12 chromatids. If the image instead contains a third copy of one type, identify the extra chromosome explicitly rather than forcing everything into pairs.

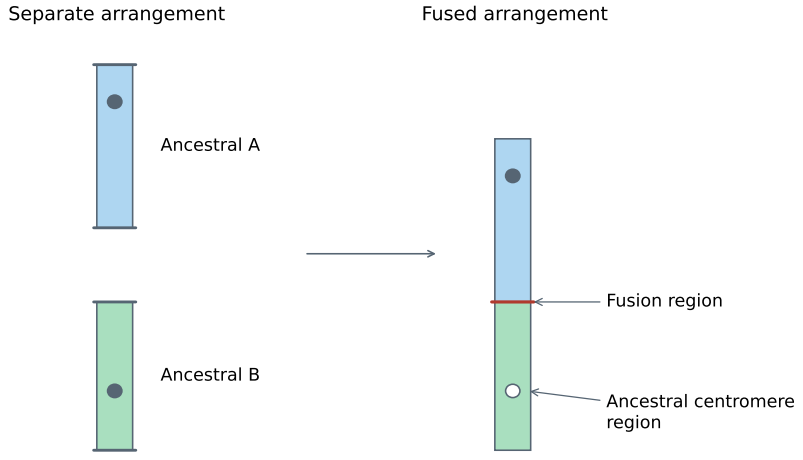
A karyogram can reveal large number changes and some structural changes. It usually cannot reveal a single-base substitution, distinguish every gene variant, or by itself prove that an individual will show a particular trait. Match the claim to the resolving power of the evidence.

Human chromosome 2: test a fusion hypothesis

The chromosome-number difference between humans and chimpanzees suggests a question, not the full answer. If two chromosomes present separately in an ancestral ape lineage fused into one chromosome in the human lineage, corresponding blocks of genes should lie in the appropriate order along human chromosome 2. This is a testable prediction from a historical hypothesis.

Human chromosome 2 corresponds to two chimpanzee chromosomes now called 2A and 2B, labelled 12 and 13 in older nomenclature. Modern chimpanzees are not our direct ancestors: the inference concerns changes in lineages after shared ancestry. Do not

describe a modern chimpanzee suddenly losing two chromosomes to become a human. Evidence includes correspondence of sequence and banding blocks, internal telomere-like repeats near the inferred fusion region, and evidence of an additional ancestral centromere region. Telomeres normally occur at chromosome ends, so oppositely oriented telomere-repeat arrays inside a chromosome are relevant to an end-to-end fusion hypothesis. The 1991 study linked below analysed such internal repeat sequences; later genomic comparisons add context.



Ancestral blocks; schematic map.
Internal telomere-like repeats test fusion.

The drawing models a testable relationship between chromosome blocks. It does not show a newly observed fusion event or an exact sequence map.

A chromosome count alone would be insufficient: other changes can alter counts. Combining independent observations makes a stronger explanation. The simple fusion model predicts a reduction of two in the diploid number when both homologous sets carry the fused arrangement: two pairs become one pair, while much of their sequence remains represented. It does not predict that half the genome disappears.

Genomes share information and contain variation

A genome is the complete genetic information of an organism. In eukaryotes, distinguish the nuclear genome from DNA in mitochondria or chloroplasts when a comparison specifies the nucleus. A genome includes non-coding regions as well as protein-coding genes. Genome size is an amount of DNA, not the number of chromosomes or genes.

Members of one species share much of their genome but can differ in base sequence. A single-nucleotide polymorphism (SNP) is variation at a particular nucleotide position in a population. Individuals may have different bases at that site. In a diploid indi-

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vidual, a site can carry the same allele on both homologues or different alleles. Not every sequence difference changes a protein, and not every protein change produces a visible trait.

Between species, sequences and the total amount of DNA can differ substantially. For a selected comparison, state whether you counted all sites, only coding sites, one sequence region or an entire assembly. A reported similarity percentage without its denominator and method is incomplete. Similar sequence does not mean identical organisms; sequence difference does not by itself date divergence.

Compare genome sizes using a database

Use a named record and version so another student can repeat a comparison. The following values come from NCBI assembly records, rounded for teaching. They describe assembled sequence, not DNA mass measured in a diploid cell.

Organism / group	**NCBI assembly accession**	**Reported total sequence length, rounded**
Escherichia coli K-12 MG1655 / bacterium	GCF_000005845.2	4.64 million bp
Saccharomyces cerevisiae S288C / fungus	GCF_000146045.2	12.1 million bp
Homo sapiens / animal	GCF_000001405.40	3,100 million bp

To reproduce the comparison, open the linked NCBI record, record accession/version, organism/strain, assembly name and the statistic used. Distinguish "total sequence length" from "ungapped length", and check which chromosome sequences and organellar sequences are included. A human reference can include both X and Y sequence types; its count of chromosome sequence types is not the 46 chromosomes in an ordinary diploid body cell. An incomplete assembly can underestimate sequence not yet assembled.

Compare across groups without ranking organisms by DNA quantity. Many differences in genome size involve repeated and other non-coding sequences, duplication and loss. A larger genome is not automatically a larger gene set or a more complex organism. As a plant counterexample, a published study measured about 160.75 billion base pairs per unreplicated nuclear complement in the fork fern *Tmesipteris oblanceolata*. That DNA-content estimate uses a different measurement approach from the assembly totals above; avoid treating every displayed number as exactly the same statistic.

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■ **Worked example: compare stated quantities**

An invented plant sample has a haploid nuclear DNA amount of 150 million bp, while an invented animal sample has 3,000 million bp measured on the same basis. The ratio is 3000/150 = 20. This says the animal sample has 20 times the DNA amount in the stated comparison. It does not show 20 times the complexity, 20 times the genes, or the chromosome number. If one sample were diploid and the other haploid, first correct the comparison rather than dividing unlike quantities.

Whole-genome sequencing: uses and limits

Whole-genome sequencing determines the base sequence across a genome rather than testing only one short marker. Many short or long reads must be checked, assembled or aligned, and interpreted. Improvements have generally increased throughput and reduced cost compared with early genome projects, but data generation and accurate interpretation remain different tasks.

Current research uses include comparing evolutionary relationships, finding variants, investigating inherited conditions, studying pathogen transmission and improving crop research. Genome-scale comparisons can test whether a relationship inferred from one gene is supported by many others. Different parts of a genome can have different histories, so careful analysis still matters.

Personalized medicine aims to use relevant genomic variation with clinical and environmental information to guide prevention or treatment. Sequencing does not guarantee that every detected variant has a known effect or that a person's future can be predicted precisely. Some results are uncertain, and privacy, consent and access matter. A sequence record is evidence to interpret, not an automatic diagnosis or a complete account of an organism's phenotype.

AHL: Limits of the biological species concept

Asexual organisms do not normally form reproductive groups by mating, so the biological species concept cannot be applied by a simple fertile-offspring test. Fossils also cannot be bred. Other evidence and species concepts are needed. Bacteria add a further difficulty: horizontal gene transfer can move genes between lineages outside ordinary parent-to-offspring inheritance. One transferred gene may therefore suggest a relationship different from the broader genome history.

Chromosome number is often shared within a species and can affect the success of crosses. During meiosis, homologous chromosomes must align and separate appropriately. Different numbers or structural arrangements can interfere with pairing, giving unbalanced gametes and reduced fertility in a hybrid. Equal chromosome number does not guarantee compatibility, and a count difference is not an infallible universal species test. Combine the mechanism with evidence from the particular populations.

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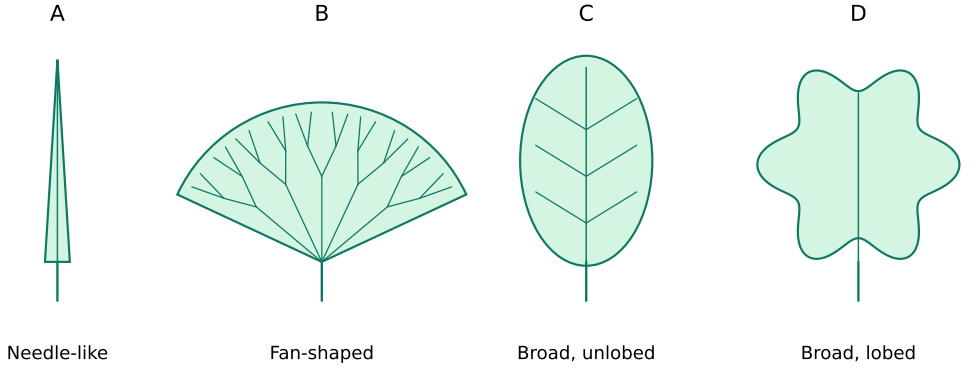
AHL: Build a key from local observations

A dichotomous key identifies a specimen by a sequence of paired alternatives. Each pair should describe observable, contrasting states of the same character. "Large" versus "green" is a poor pair: a leaf can be both. Avoid using habitat or an invisible feature when the key is intended for a detached leaf. State the set of organisms and life stages for which the key was built.

For a local practical, select four permitted plant species or non-destructively observed animal species from the school grounds or a nearby accessible habitat. Confirm their names with a suitable field guide or authoritative collection. Photograph the actual specimens, record several individuals where possible, and make a character table. Do not claim an unverified species name merely because a phone application proposes it. Test the draft key with another student's specimens and revise ambiguous couplets.

The following is an invented leaf dataset, not a claim about species observed at this school:

Sample	Observable state
A	Needle-like leaf
B	Broad fan-shaped blade with veins repeatedly dividing into two
C	Broad blade, not fan-shaped, with no lobes
D	Broad blade, not fan-shaped, with lobes



Invented character set A–D.
Test a local-species key against observed, verified specimens.

The key identifies A–D within this declared dataset. Replace labels with verified local species only after observing and testing the relevant characters.

One valid key is: 1a needle-like → A; 1b broad → 2. Then 2a fan-shaped with repeatedly dividing veins → B; 2b not fan-shaped → 3. Finally 3a lobed → D; 3b unlobed → C. Every sample should reach one endpoint. An identification key is a decision tool, not automatically a phylogenetic tree: an efficient first split need not represent the earliest ancestral divergence.

AHL: DNA barcodes and environmental DNA

A DNA barcode is a standardized, relatively short sequence used for identification by comparison with reference sequences. A reference must be linked to a reliably identified organism. Different taxonomic groups may require different suitable markers; no single barcode automatically separates every species. A close database match is stronger when the reference coverage, sequence quality and discriminatory power of the marker are known.

Environmental DNA (eDNA) is DNA recovered from environmental samples such as water or soil rather than necessarily from a captured whole organism. Organisms shed cells and other material; sequencing suitable barcode regions can reveal evidence of taxa in a habitat. This can investigate biodiversity with less disturbance and can detect organisms difficult to observe directly.

Use collection blanks, extraction controls and comparison samples to assess contamination and method performance. DNA can move with water and persist after an organism has left. A detection does not automatically locate a living individual at that exact point and time. Failure to detect DNA does not prove absence if sampling was limited, the DNA degraded or the chosen marker amplified poorly.

Sequence-read abundance is not automatically the number or biomass of organisms: shedding, transport, extraction and amplification differ. Use eDNA results alongside repeated sampling and other ecological observations. Barcoding supports identification; whole-genome sequencing examines much more information and answers additional questions.

Practice

Use stated species concepts and units. All unreferenced numerical datasets are invented.

1. Explain how inherited and environmental differences can both contribute to variation. Give one strength and two limitations of grouping species by morphology alone.
2. Write the scientific name *Pan troglodytes* with the correct capitalization and explain both parts. Compare it with *Pan paniscus*. Why is an epithet alone an incomplete name?
3. Explain the biological species concept and why two colour forms producing fertile offspring are not automatically separate species. Describe one difficulty when two populations are only partly reproductively isolated.
4. State typical human and chimpanzee diploid and gamete chromosome numbers. For a human cell after DNA replication but before chromatid separation, state chromosome and chromatid numbers and explain the difference.

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5. The invented karyogram has four homologous pairs. Find $2n$, n, and the number of chromatids after replication but before separation. Give three features used to pair the chromosomes.
6. Explain two predictions or observations relevant to the human chromosome 2 fusion hypothesis. Why are chromosome count alone and the claim "humans came from modern chimpanzees" inadequate?
7. Define genome and SNP. Explain why genome size, gene count and chromosome count are different measurements, and why not every SNP produces a visible trait.
8. An alignment has 8 differences among 50 sites; another has 7 among 25. Calculate both percentages. Explain why these are neither universal species thresholds nor calibrated dates. What does rotating branches at an unscaled node change?
9. Use the three NCBI assembly rows to identify the largest and smallest reported assembled sequence totals. Explain why the reference's chromosome sequence-type count must not be substituted for human diploid chromosome number.
10. An invented plant and animal have respectively 150 and 3,000 million bp per haploid nuclear complement. Calculate the ratio. If the plant measurement instead represents a diploid cell containing 300 million bp, show the correction needed for a fair comparison.
11. Give two research uses of whole-genome sequencing and one potential use in personalized medicine. Explain two reasons a sequence does not provide a complete prediction of phenotype.
12. **AHL:** Explain why asexual reproduction and horizontal gene transfer challenge a fertile-offspring species test. Describe how chromosome-number differences can reduce hybrid fertility, without treating count as a perfect species boundary.
13. **AHL:** Write a key for A–D in the leaf table, trace sample D through it, and explain how to replace the invented labels with a tested local-species key. Why is this key not automatically an evolutionary tree?
14. **AHL:** Explain how barcodes and eDNA can identify taxa. Give two controls or validation steps, two reasons a positive detection need not locate a living organism exactly, and one reason read number is not organism number.

■ Worked answers

1. Different inherited alleles and different conditions such as light or water can affect a trait. Morphology enables practical identification, including fossils. Limits include variation with age/sex/environment and similar-looking distinct species or convergent structures. Use multiple diagnostic characters and suitable comparison samples.
2. *Pan* has an initial capital; *troglodytes* is lowercase; italicize both. *Pan* is the genus and the whole binomial names the species. *P. paniscus* shares the genus but is a different species. The epithet alone omits the genus needed for the complete species name.

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3. Populations belong to a potentially interbreeding group producing viable, fertile offspring and reproductively isolated from other groups. Fertile interbreeding colour forms can be variation within one species. Partial isolation can develop gradually; different concepts or evidence may give different boundary decisions.
4. Humans: 46 diploid, 23 in a normal gamete. Chimpanzees: 48 and 24. A replicated human cell has 46 chromosomes and 92 chromatids before separation. DNA copying makes sister chromatids without yet separating their centromeres into different chromosomes.
5. $2n = 8$, $n = 4$ and 16 chromatids after replication before separation. Match relative length, centromere position and banding pattern. The four displayed pairs are eight chromosomes, not four.
6. Corresponding sequence/banding blocks should align with two separate ancestral chromosome types; internal oppositely oriented telomere-like arrays and an additional ancestral centromere region fit fusion. Count alone cannot specify the structural event. Modern chimpanzees and humans share ancestors; one modern species is not automatically the other's direct ancestor.
7. A genome contains complete genetic information, including coding and non-coding regions; specify nuclear versus organellar DNA when needed. A SNP is nucleotide-position variation in a population. DNA amount differs from how DNA is divided among chromosomes or how many genes it contains. A variant can be non-coding, have a silent coding effect or depend on biological context.
8. $8/50 \times 100 = 16\%$ and $7/25 \times 100 = 28\%$. These describe the selected alignments, without a universal species cutoff or a calibrated change-rate model. Rotating branches changes page order, not branching relationships; arbitrary branch lengths do not measure elapsed time.
9. The human reference row has the largest total, about 3,100 million bp; the bacterial row the smallest, about 4.64 million bp. An assembly is a sequence representation with a declared component set, not an ordinary diploid cell. A reference can represent both X and Y types without containing a real 46-chromosome body-cell complement.
10. $3000/150 = 20$. For the alternative plant measurement, a comparable haploid amount is $300/2 = 150$ million bp, again giving 20. Dividing $3000/300 = 10$ would mix haploid and diploid quantities. No ratio establishes a complexity or gene-count ratio.
11. Examples include evolutionary comparisons, variant discovery, inherited-condition research, pathogen studies and crop research. A potential medical use is considering relevant variants with other evidence when choosing prevention or treatment. Unknown variant effects and environmental/developmental influences limit prediction; sequence quality and interpretation also matter.
12. **AHL:** Asexual lineages cannot be grouped through normal interbreeding tests, while horizontal transfer can move genes outside parent–offspring descent. Different chromosome numbers/arrangements can disrupt meiotic alignment and pro-

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duce unbalanced gametes, reducing hybrid fertility. Equal counts do not ensure compatibility, and count differences require case-specific interpretation.

13. **AHL:** 1a needle-like → A; 1b broad → 2. 2a fan-shaped/dividing veins → B; 2b not fan-shaped → 3. 3a lobed → D; 3b unlobed → C. D follows 1b, 2b, 3a. Observe and verify local species, record characters across samples, then test and revise the key. Its splits are identification decisions, not necessarily ancestry claims.
14. **AHL:** Compare a suitable marker from a sample with reliably identified reference sequences. Use collection/extraction blanks and validated references or positive controls, for example. DNA transport and persistence can separate detection from a current living individual. Unequal shedding and amplification can make read counts differ from organism counts; non-detection also needs cautious interpretation.

Exam tips

Name the species concept and the evidence it needs. Keep chromosome, chromatid, gene and genome-size counts separate. Match karyogram chromosomes by several features, and evaluate fusion using several predictions. Record database versions and compare the same DNA quantity. For a key or eDNA result, state the organisms and conditions for which identification is supported.

References: IB Biology guide, A3.1; chromosome 2 fusion study; chimpanzee genome comparison; NCBI bacterial assembly, yeast assembly, human assembly; fern DNA-content study.

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A.3.2 · Classification and cladistics

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A dolphin and a shark have similar swimming shapes. A mushroom and a green plant both stay in one place. Which similarities help us identify an organism, and which reveal its ancestry? A useful classification must do more than collect things that look alike.

This is the HL-only A3.2 unit; there is no SL counterpart. It covers the need for classification, the limits of fixed ranks, clades, molecular evidence and clocks, constructing and reading cladograms, revising plant classification and the three domains. Prerequisites: species and genomes from A3.1 and base sequences from A1.2. All numerical datasets and letter-labelled organisms below are invented teaching examples, not records from real investigations.

AHL: Why classify such diverse organisms?

Classification arranges organisms into groups so we can identify, compare and study them. A named group is a taxon. A common system lets researchers connect observations made in different places, find information about a newly identified organism and ask whether a feature is shared across a group. Scientific names reduce the ambiguity of common names.

The traditional nested ranks are kingdom, phylum, class, order, family, genus and species. Domain is placed above kingdom in the three-domain classification. A species belongs within a genus, that genus within a family, and so on. A genus contains one or more species; species in one family need not belong to the same genus. The **binomial system** gives genus followed by specific epithet, for example *Homo sapiens*. The epithet alone does not name the species.

The ranks help organize information, but a rank is not a fixed amount of evolutionary time or genetic difference. Two families in different lineages need not be equally old or equally diverse. Evolution produces branching at many levels, without a natural rule that exactly seven named ranks must fit every branch. Assigning the same rank therefore does not establish that two groups diverged at the same time.

Traditional classifications sometimes emphasize a useful visible trait while excluding some descendants of the same ancestor. For example, a traditional grouping of reptiles that excludes birds does not include all descendants of the reptile group's common ancestor. Birds belong within the reptile lineage in a classification based on ancestry. This does not mean birds descended from today's crocodiles, or that birds lack their own distinctive features.

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AHL: Clades connect classification to ancestry

Cladistics uses evidence of common ancestry to group organisms. A clade includes a common ancestor and **all** its descendants. Clades can be nested and can be named without assigning each one a traditional rank. This avoids forcing every evolutionary split into an arbitrary rank system.

A classification that follows evolutionary relationships supports predictions. If several members share a feature inherited from their common ancestor, we can investigate whether other members have it too. For example, a group sharing an enzyme pathway suggests a useful starting point for studying a newly identified member. Such predictions are testable: later loss, modification or incomplete knowledge can produce exceptions. A clade does not require every descendant to look identical.

A **shared derived character** is a character state inherited from the common ancestor of a group and different from the ancestral state used for comparison. It supports a particular branching hypothesis. An ancestral state shared much more widely may tell us little about the closest relationships inside the group. A suitable outgroup, outside the group being studied, helps infer which state is ancestral; the choice must itself have evidence.

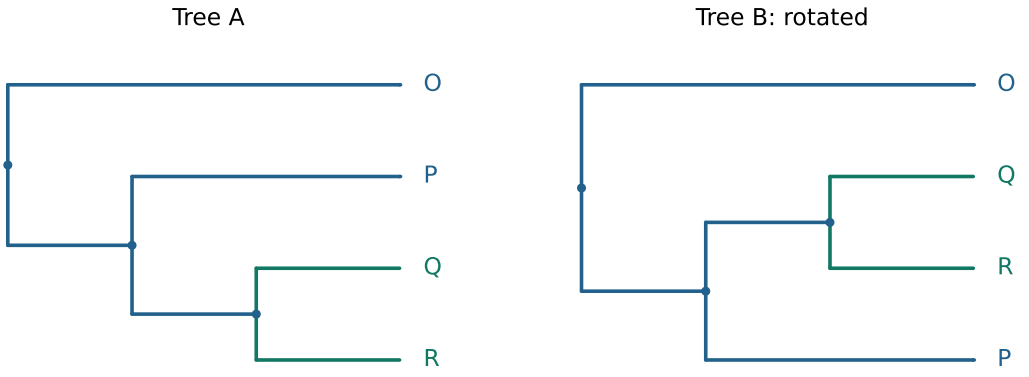
Similarity can also arise independently through convergent evolution. Similar swimming outlines in dolphins and sharks are useful in water, but do not place them together as the closest pair among mammals and fish. Study multiple characters and distinguish a shared inherited structure from a similar function. Reversals and losses can also obscure history. One striking trait should not override all other evidence automatically.

AHL: Read the branching, not the positions on the page

A **cladogram** is a hypothesis about branching relationships. Trace connections rather than counting the gaps between tips.

Part	What it represents	What it does not establish by itself
Root	The ancestral end of a rooted tree	The identity of a directly observed first organism
Internal node	A hypothetical common ancestor at a split	A named modern species at a different tip
Terminal branch	A lineage leading to a displayed terminal taxon	A ladder of improvement from lower to higher life
Branch length	Time or amount of change only if an appropriate scale is given	An age measured from the drawing's unscaled length

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Invented taxa. Same nodes, same clades.
No branch-length scale is given.

Both trees have the same relationships. The coloured Q–R branches identify one complete clade; their position on the page changes.

In the illustrated tree, Q and R share a more recent common ancestor with each other than either shares with P. The clade beginning at their shared node contains Q and R. The next deeper clade contains P, Q and R. Grouping P and Q while excluding R omits a descendant of their common ancestor, so that set is not a complete clade on this tree.

Rotating branches around a node preserves the connections. It may move two tips next to each other without making them sister taxa. P is not an ancestor of Q merely because P's branch leaves a deeper node; P represents its own descendant lineage. Modern terminal taxa have all continued evolving since their shared ancestors.

An unscaled cladogram does not say whether a long-looking branch took longer or accumulated more mutations. A dated tree or a phylogram can supply such information, but the key must say what the scale measures. Identify the root before using "more recent"; an unrooted diagram alone does not specify the direction of ancestry.

AHL: Build a tree from sequence evidence

Base sequences of homologous genes, or amino acid sequences of homologous proteins, allow many comparable characters to be studied. Molecular evidence can reveal relationships that visible form hides, especially among microorganisms. The comparison still needs care: use corresponding regions, align equivalent positions, check sequence quality and avoid mixing unrelated genes.

The following six aligned DNA sites are invented. O is the outgroup, and for this simple exercise its A states are taken as ancestral. A column is a character; its bases are states. A gap in a real alignment must be handled by a stated method, not silently counted as whichever base is convenient.

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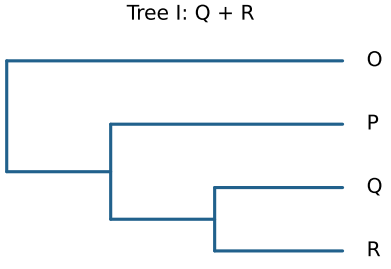
Taxon	Site 1	Site 2	Site 3	Site 4	Site 5	Site 6
O	A	A	A	A	A	A
P	G	G	A	A	A	A
Q	G	G	C	C	A	A
R	G	G	C	C	T	T

Sites 1 and 2 support grouping P, Q and R relative to O. Sites 3 and 4 support Q with R. Sites 5 and 6 distinguish R, but a state unique to one terminal taxon does not by itself choose its sister group. The same reasoning works with amino acid states, although different DNA sequences can encode the same amino acid sequence.

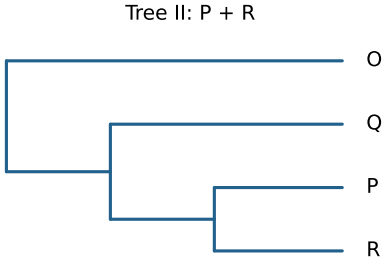
■ Worked example: choose by parsimony

Parsimony compares the minimum number of changes needed on candidate trees. Here assume equal cost per base substitution and count each column independently.

- Tree I has the rooted branching O, then P, then the Q–R pair. Each of sites 1–2 needs one A-to-G change on the P–Q–R stem. Each of sites 3–4 needs one A-to-C change on the Q–R stem. Each of sites 5–6 needs one A-to-T change on R. Total: $2 + 2 + 2 = 6$ changes.
- Tree II has O, then Q, then the P–R pair. Sites 1–2 still need two changes in total. For each of sites 3–4, Q and R share C but are separated by P's A state: the minimum is two changes per site, by independent gains or a gain plus reversal. Sites 5–6 still need one change each. Total: $2 + 4 + 2 = 8$ changes.



Sites 1–2	2
Sites 3–4	2
Sites 5–6	2
Total changes	**6**



Sites 1–2	2
Sites 3–4	4
Sites 5–6	2
Total changes	**8**

Invented alignment; O states taken as ancestral.
Minimum substitutions; equal cost per site.

The shorter explanation is selected under the stated equal-cost model. Six changes do not mean six observed mutation events in a real history.

Tree I is more parsimonious for these data. This is a reasoned choice among hypotheses,

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not proof that evolution always takes the fewest possible steps. Multiple substitutions at one site, convergence and reversals can conceal changes. Different genes may have different histories, and a short alignment may supply weak or conflicting support. Add independent genes or characters and ask whether the grouping persists; do not repeat the same six sites and call that independent confirmation.

A percentage difference is useful but is not itself a complete tree-building method. P–Q and Q–R each differ at two of six sites here, yet only Q–R share the derived C states at sites 3–4. A tied pairwise distance cannot select between these relationships without further reasoning. Real analyses may use models beyond equal-cost parsimony.

AHL: A molecular clock estimates a date

Over generations, sequence changes can accumulate in diverging lineages. A molecular clock uses an estimated rate to relate divergence to elapsed time. The observed fraction of different sites is

$$d = \frac{\text{number of differing aligned sites}}{\text{number of sites compared}}.$$

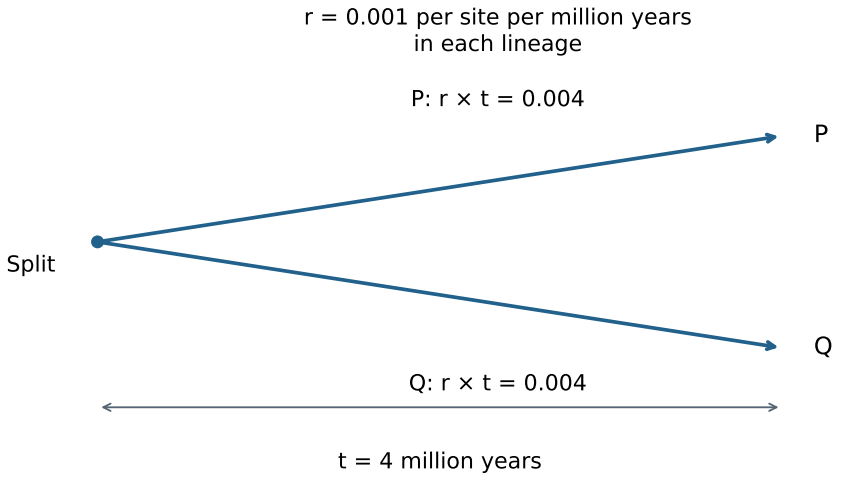
Observed difference is not automatically the total number of historical substitutions. A site can change more than once or change back. For distant lineages, simply counting present differences may underestimate the changes that occurred. Select a suitable region and model, and calibrate rates using independent evidence where possible.

■ Worked example: keep the rate definition explicit

In an invented comparison, two lineages differ at 8 of 1,000 aligned sites. Assume a constant substitution rate $r = 0.001$ substitutions per site per million years **in each lineage**, equal in both lineages, with negligible repeated changes. Both lineages accumulate differences after their split, so

$$d = \frac{8}{1000} = 0.008, \qquad d \approx 2rt, \qquad t = \frac{0.008}{2(0.001)} = 4 \text{ million years}.$$

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Invented model: equal constant rates; negligible repeated changes.
Pairwise difference ≈ 0.004 + 0.004 = 0.008.

Add the two lineage contributions. A rate defined for pairwise divergence already includes both; do not double it again.

If the stated rate were instead a **pairwise** divergence rate of 0.002 per site per million years, use $t = d/0.002 = 4$ million years. Check the definition before substituting. A percentage and a fraction must also be consistent: 0.8% is 0.008, not 0.8.

A clock gives an estimate, not an exact historical timestamp. Rates can vary with generation time, population processes, selective pressures and sequence function. Errors in alignment, sampling and calibration add uncertainty. If the per-lineage rate in this example could be 0.0008–0.0012, the same model gives about 5.0–3.3 million years. A faster rate implies less time to produce the same difference. Without a rate or calibration, a difference percentage alone supplies no numerical date.

AHL: Classification changes when evidence changes

A cladistic analysis can test whether an existing named group contains one ancestor and all its descendants. A traditional family may prove to contain several separate lineages, or a group excluded because it looks different may belong inside the same clade. Revision changes the classification to better fit the evidence; it does not make the organisms themselves suddenly evolve into different organisms.

The figwort family, Scrophulariaceae in its older broad sense, provides a real example. A published 2001 study compared sequences from three plastid genes and found that the broad grouping included at least five distinct monophyletic groups. Several traditional family boundaries therefore needed revision. You do not need to memorize all the transferred genera to explain the scientific reasoning. Similar-looking flowers alone had not reliably identified one complete ancestry group. The study's abstract and bibliographic record identify the actual analysis; the invented trees in this handout

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are not its data.

To evaluate a proposed revision, ask which organisms and genes were sampled, how the tree was inferred, and whether additional evidence agrees. One unusual sequence might reflect an error or a particular gene's history. Agreement across independent evidence is stronger. A well-supported new tree can falsify a specific claim that an old family is one complete clade. It does not falsify the broader conclusion that organisms evolve. Classification is a scientific framework open to improvement, rather than a list that must remain unchanged.

AHL: Three domains from ribosomal RNA evidence

Bacteria and archaea are both prokaryotic in cell organization: neither has a membrane-bound nucleus. This shared absence does not establish that they form one uniform evolutionary group. Molecular comparisons of ribosomal RNA revealed major differences hidden by that simple structural description.

Ribosomal RNA is useful because cellular organisms share ribosomes, and corresponding rRNA regions can be compared across very diverse groups. Conserved regions help alignment; variable positions supply information about divergence. Researchers compare many positions, rather than deciding from the presence of one base. rRNA is a structural and functional RNA component of ribosomes, not the messenger RNA encoding one protein.

The three-domain classification distinguishes Bacteria, Archaea and Eukarya (also written Eucarya). Domain lies above kingdom; animals, plants and fungi belong within Eukarya, not in three separate domains. Archaea are not simply bacteria living in extreme habitats: their molecular distinctions matter, and not all archaea are extremophiles. Viruses have no ribosomes of their own and are not assigned to these three domains of cellular life.

Historically, Woese and Fox's 1977 rRNA work identified three major lineages; Woese, Kandler and Wheelis formally proposed the domain rank and names in 1990. Distinguish the underlying discovery from the later naming proposal. The three-domain scheme demonstrates how molecular evidence can transform a familiar classification. Deep relationships continue to be investigated; three named domains should not be read as a claim that every detail of life's rooted history is settled.

Practice

Use the invented six-site alignment and trees above where specified. State assumptions in calculations.

1. Give three reasons for classification. Write the traditional ranks from kingdom to species and explain why two families need not be equally old.

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2. Define a clade. On the tree O, then P, then Q–R, decide whether each set is a complete clade: Q and R; P and Q; P, Q and R. Explain using their common ancestors.
3. Explain root, internal node and terminal branch. Does rotating Q–R around their shared node alter their relationship? Does the longest unscaled branch identify the oldest taxon?
4. Distinguish an ancestral character, a shared derived character and a convergent similarity. Explain how an appropriate outgroup helps a character matrix.
5. For the six-site alignment, identify the sites supporting P–Q–R, those supporting Q–R, and those unique to R. Why do the last sites not identify R's sister taxon alone?
6. Recalculate the minimum equal-cost scores for trees I and II. Explain why two changes per site are required for sites 3–4 on tree II. Which tree is preferred?
7. Calculate P–Q, Q–R and P–R percentage differences across the six sites. Why is choosing one tied shortest distance insufficient to settle the tree?
8. Two invented alignments have 8 differences in 50 sites and 12 in 60 sites. Calculate both percentages. Can these alone establish which pair split earlier? Explain.
9. Two lineages differ at 12 of 600 sites. Each has a constant rate of 0.0025 substitutions per site per million years. Estimate their divergence time, stating the simple model. What changes if 0.005 is instead given as the pairwise rate?
10. For $d = 0.008$, calculate the time range when each lineage's rate is between 0.0008 and 0.0012 per site per million years. Explain the direction of the relationship and give two limitations of clock estimates.
11. An old plant family appears in three separated parts of a well-supported new tree. Explain how to test the old grouping and revise the classification. Distinguish revising a family from rejecting evolution.
12. Name the three domains, locate animals and fungi, and explain why "both lack a nucleus" is insufficient to combine archaea and bacteria. Why is rRNA suitable evidence, and why are viruses excluded from this scheme?
13. Evaluate: "Molecular data always give the true tree; a shorter tree proves the exact history." Propose two genuinely independent checks, and distinguish the 1977 rRNA finding from the 1990 domain proposal.

■ Worked answers

1. Classification supports identification and communication, organizes comparisons, and helps predict or investigate shared features. Kingdom → phylum → class → order → family → genus → species. Ranks are conventions, not units of time or divergence; evolutionary splitting does not occur at just seven fixed levels.
2. A clade includes an ancestor and all its descendants. Q–R is complete; P–Q is incomplete because their common ancestor also has descendant R; P–Q–R is complete. Include the ancestor concept even when only terminal labels are listed.

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3. Root: ancestral end of a rooted tree; internal node: hypothetical common ancestor at a split; terminal branch: lineage to a terminal taxon. Rotation leaves the connections unchanged. Unscaled branch length gives no date, and one modern tip is not automatically another's ancestor.
4. An ancestral state predates the group of interest; a shared derived state originated in its common ancestor relative to the comparison. Convergence produces similar states independently. An evidence-based outgroup helps infer ancestral states, but is not assumed to be a surviving direct ancestor.
5. Sites 1–2 support P–Q–R; sites 3–4 support Q–R; sites 5–6 uniquely distinguish R. A state present only in R can be placed on its terminal branch on either candidate tree, so it does not select R's sister group.
6. Tree I: $2 + 2 + 2 = 6$; tree II: $2 + 4 + 2 = 8$. On II the C-bearing Q and R do not form a clade excluding A-bearing P. At least independent C gains, or one gain and one reversal, are needed per affected site. Prefer I under equal-cost parsimony, without treating its score as an observed event count.
7. P–Q: $2/6 \times 100 \approx 33.3\%$; Q–R: also 33.3%; P–R: $4/6 \times 100 \approx 66.7\%$. The equal shortest distances do not uniquely choose a topology. The distribution of derived states, including the outgroup comparison, supports Q–R in this dataset.
8. $8/50 \times 100 = 16\%$ and $12/60 \times 100 = 20\%$. The larger percentage does not alone prove an earlier split: regions, rates, selection and repeated substitutions can differ. A date needs calibration or a rate model.
9. $d = 12/600 = 0.020$; assuming equal constant per-lineage rates and negligible repeated changes, $t = d/(2r) = 0.020/0.005 = 4$ million years. A pairwise rate of 0.005 already includes both contributions: $t = 0.020/0.005 = 4$ million years, with no extra factor of two.
10. At 0.0008, $t = 0.008/0.0016 = 5.0$ million years; at 0.0012, $t = 0.008/0.0024 \approx 3.3$ million years. The range is about 3.3–5.0 million years. A faster rate requires less time. Variable rates and repeated substitutions are two limitations; alignment, sampling and calibration uncertainty also matter.
11. Locate the group's common ancestor and test whether the named family includes all and only that ancestor's descendant lineages. Check sampling, sequence quality and independent genes or characters. Revise family boundaries to follow supported clades, which may require moving species. This rejects a particular grouping, not common descent itself.
12. Bacteria, Archaea and Eukarya/Eucarya; animals and fungi are in Eukarya. A shared absence of a nucleus hides major molecular differences. rRNA has homologous conserved and variable regions and occurs in all cellular life. Viruses lack their own ribosomes, so this cellular classification does not place them in the three domains.
13. Sequence quality, alignment, short samples, convergence, reversals and gene-specific histories limit inference. Parsimony chooses fewer required changes under a model, not a proven history. Test different independent genes and a separately

observed character dataset, rather than reuse the original sites. The 1977 work identified major lineages using rRNA; the formal domain proposal and names followed in 1990.

Exam tips

Name the common ancestor and all its descendants when identifying a clade. Trace nodes rather than tip spacing. Count changes site by site under the stated model. Write a difference as a fraction before using a rate, and check whether that rate is per lineage or pairwise. Explain classification revisions through evidence and a specific rejected grouping.

References: IB Biology guide, A3.2; 1977 rRNA study; 1990 domain proposal. The figwort study is linked in its teaching section.

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Evolution and speciation

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A plant closes its stomata during a dry afternoon. Over many generations, a plant population becomes more likely to inherit a thick leaf cuticle. Both changes may reduce water loss, but only the second necessarily describes evolutionary change. We need to identify what changed, which organisms were compared, and whether the difference is inherited.

This unit covers A4.1: evolution, molecular evidence, artificial selection, homologous and analogous structures, speciation, reproductive isolation and differential selection. SL understandings plus allopatric and sympatric speciation, adaptive radiation, hybrid barriers and plant polyploidy (marked AHL). Prerequisites: inheritance, meiosis, variation and species concepts. All numerical datasets, chromosome models and letter-labelled populations below are invented teaching examples. Named studies are identified separately.

Evolution changes populations across generations

Evolution is change in the heritable characteristics of a population across generations. An individual's growth, learning or temporary physiological adjustment does not by itself show that its population has evolved. An inherited allele can become more or less common; a population can change even while each individual keeps the same alleles throughout its life.

An **adaptation** is a heritable feature associated with improved survival or reproduction in particular conditions. A thick cuticle can reduce water loss; reduced stomatal opening can conserve water but also limit carbon dioxide entry for photosynthesis. There is no universally best leaf: a benefit under dry conditions may carry a cost elsewhere.

Acclimatization is an individual's adjustment to changed conditions during its lifetime. A useful response is not automatically a newly evolved inherited feature. To investigate inheritance, compare offspring under controlled common conditions, use appropriate genetic evidence and consider environmental effects. A functional story alone is a hypothesis to test.

Natural selection can explain adaptive population change when individuals vary, relevant variation is heritable, and variants differ in reproductive success in an environment. Variation is not created because an organism knows what it needs. Mutation supplies new variants; inheritance passes variants on; selection changes their relative representation. Not every evolutionary change is adaptive: chance and other population processes also matter.

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■ **Worked example: count the next generation**

In an invented model, a starting population has 100 individuals of inherited form A and 100 of inherited form B. In one environment, the next generation contains 300 A and 100 B offspring. Assume forms are faithfully inherited, generations do not overlap, and there is no migration or mutation in this simple example.

- Initially, the frequency of A is $100/(100 + 100) = 0.50$.
- Among the offspring, it is $300/(300 + 100) = 0.75$.
- The increase is 25 percentage points, or a 50% relative increase in frequency: $(0.75 - 0.50)/0.50 \times 100 = 50\%$.

The model illustrates differential reproductive success changing an inherited population characteristic. It does not mean B deliberately turned into A. In a real study, test the inheritance and alternative causes before attributing every frequency change to selection.

Molecular evidence connects organisms through ancestry

All cellular organisms use DNA as inherited information and RNA in gene expression. Similarities in homologous DNA, RNA and protein sequences provide powerful evidence of common ancestry. Shared complex sequence patterns are more informative than noticing that both samples contain the same four DNA bases.

Use corresponding genes or regions and align equivalent positions. A shared sequence difference can support a particular relationship when compared with other organisms. Different genes, many sites and other evidence can test whether the same branching pattern is supported. The shared genetic code also connects living systems, while allowing known variations rather than requiring every detail to be identical.

Suppose two invented aligned sequences have 6 differences among 40 sites. Their observed percentage difference is $6/40 \times 100 = 15\%$, so 85% of these aligned positions match. This describes the chosen alignment, not a universal percentage of the whole genomes. Neither "15% different" nor one matching gene automatically provides a divergence date. Rates, repeated changes, sampling and gene histories affect interpretation.

A molecular tree is a hypothesis inferred from evidence. Modern species at its tips share ancestors; one modern species need not be the direct ancestor of another. Agreement among independent sequence datasets and anatomical evidence is stronger than repeatedly analysing the same short region.

Artificial selection demonstrates heritable change

Artificial selection occurs when people choose which organisms reproduce based on desired inherited traits. If breeders repeatedly select parents with a heritable characteristic, its frequency or average expression can change in subsequent generations. Dog breeds differ greatly in body form but share ancestry; crop varieties can also differ

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markedly from their wild relatives and from one another.

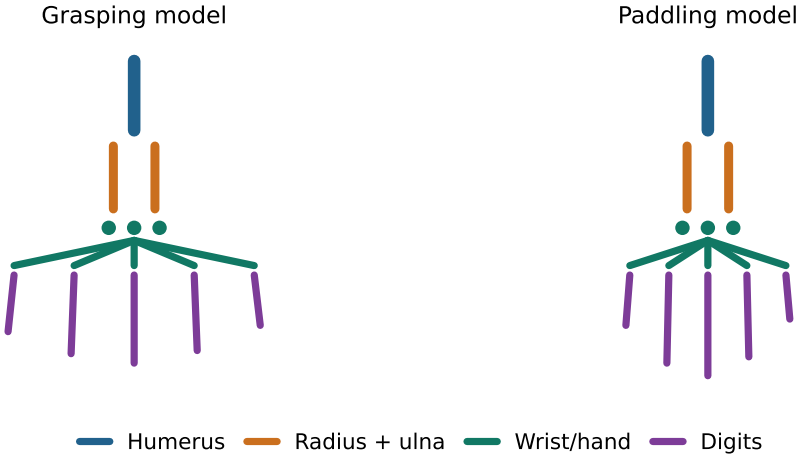
The important explanation is selection of breeding individuals, inheritance and cumulative population change. Breeders do not simply train a parent's body into a new shape and transmit that acquired shape to every offspring. If a trait is strongly affected by environment, compare offspring under consistent conditions and repeat the selection across generations.

Artificial selection shows that populations can change relatively quickly when selection is strong and variation is available. It supports the possibility of evolutionary change, but it is not a complete recreation of every past event. Natural selection has no breeder choosing a goal: environmental conditions influence which inherited variants leave more offspring.

Homologous structures share an inherited plan

Homologous structures derive from the corresponding structure in a common ancestor, even when their functions differ. The pentadactyl limb illustrates this: an ancestral five-digit forelimb plan has been modified for functions such as grasping, running, swimming and flying.

In a typical tetrapod forelimb comparison, identify one upper-arm bone (humerus), two forearm bones (radius and ulna), wrist bones and a hand region with digits. Their relative positions and developmental relationships matter more than identical lengths. Some descendants reduce, lose, elongate or fuse parts; not every modern limb must display five equally obvious fingers.



Five digit rays in each generalized model.
Different proportions; not measured skeletons.

These teaching models show a shared structural plan, not measured skeletons of named species. Changes in proportions can support different functions.

A flipper and a human arm need not perform the same task to be homologous. Their

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corresponding bones are evidence of common ancestry with modification. The inference is stronger when it agrees with developmental and molecular evidence. Do not conclude that one living animal's arm gradually turned into another living species' flipper; both lineages descend from ancestral populations.

Analogous features arise through convergence

Analogous features have similar functions but different evolutionary origins as those functional features. Bird wings and insect wings both support flight, but an insect wing is not a modified bony vertebrate forelimb. Similar selection pressures can favour independently evolved ways of solving a similar problem. This is convergent evolution.

Distinguish the comparison being made. Bird and bat forelimb bones are homologous as tetrapod forelimbs, while powered flight and the wing surfaces evolved separately in their lineages. A bird's flight surface uses feathers; a bat's uses a skin membrane supported largely by elongated digits. A label such as "homologous" must refer to a specified structure and level of comparison.

Convergence explains why organisms that live similarly can look alike without being each other's closest relatives. A streamlined outline is useful in water, but dolphin–shark similarity alone does not override the other evidence connecting dolphins with mammals. Use several independent features and molecular comparisons to test relationships.

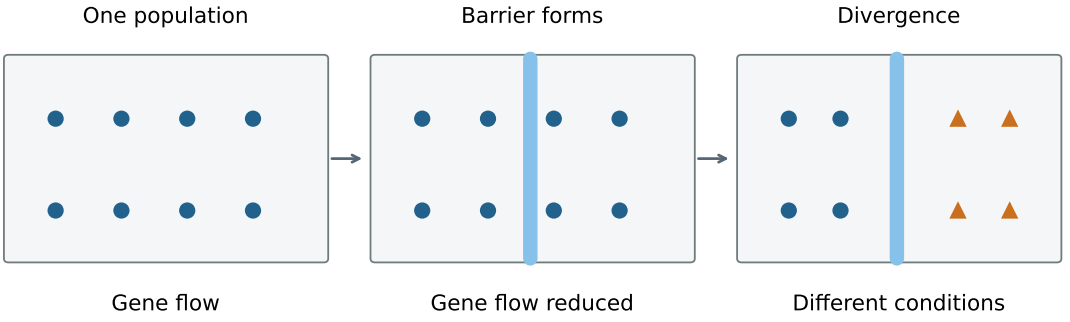
Speciation splits an existing lineage

Speciation produces two or more species from pre-existing lineages. A population can change substantially over time without splitting; that is evolution, but not necessarily speciation. A new species does not normally appear as an unrelated organism from nowhere. Speciation increases species number, while extinction decreases it; both processes affect biodiversity.

In the biological species framework, reproductive isolation limits gene flow between populations. A geographical barrier may reduce contact. Differences in mating signals, breeding time or compatibility can also reduce successful reproduction. A river separating populations is a possible cause of isolation, not by itself proof that two species already exist.

When gene flow is reduced, populations can diverge through mutation, selection and other population processes. Different environments can impose differential selection: inherited variants favoured in one population may be less successful in the other. Over generations, differences affecting mate recognition or hybrid fertility may strengthen reproductive isolation.

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Invented history: separation need not create species immediately.
Test reproductive isolation using suitable evidence.

Geographical separation reduces opportunities for gene flow. Different appearance alone does not prove complete reproductive isolation.

A real example: chimpanzees and bonobos

Common chimpanzees (*Pan troglodytes*) and bonobos (*Pan paniscus*) share ancestral populations. The Congo River separates much of their present ranges and is an important geographical barrier to gene flow. Separation allowed populations experiencing different ecological and social conditions to diverge. This illustrates geographical isolation and the opportunity for differential selection, rather than one modern species deliberately changing into the other.

Keep the historical claim proportionate to the evidence. Genomic research also supports episodes of ancient gene flow after their main divergence. A river need not have been perfectly impermeable throughout history, and their evolutionary history is not explained by one observation of today's ranges. Molecular data, geography and trait comparisons contribute different kinds of evidence. The research institute's account of the genome study explains this added complexity.

Test an adaptive explanation with fair comparisons

To investigate water-loss differences, normalize measurements by leaf area and observation time, then control other influences. In invented observations, sample A loses 12 g from $0.20\,\mathrm{m}^2$ over 2 h; sample B loses 8 g from the same area over the same time. Define the area-time-normalized rate R:

$$R = \frac{\text{mass lost}}{\text{leaf area} \times \text{time}}.$$

Thus $R_A = 12/(0.20 \times 2) = 30\,\mathrm{g/(m^2\,h)}$ and $R_B = 8/(0.20 \times 2) = 20\,\mathrm{g/(m^2\,h)}$. B's

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measured rate is one-third lower under these conditions, a reduction of about 33.3% relative to A.

This does not by itself prove that B's leaves are an inherited drought adaptation. Control humidity, temperature, airflow, light, leaf age and water supply; use repeated measurements and independent plants. Compare relatedness and growth conditions, and test inheritance if the claim is evolutionary. Lower water loss may also carry a photosynthetic cost. Observe permitted local plants without removing protected species. An observational environmental gradient can suggest hypotheses, but correlated conditions limit causal conclusions.

AHL: Allopatric and sympatric routes

Allopatric speciation begins with geographical separation that reduces gene flow. Both populations then evolve; different selection pressures and chance processes can contribute to divergence. A barrier need not create immediate reproductive incompatibility. If populations meet again, successful mating and the viability and fertility of offspring help test whether reproductive isolation has developed.

Sympatric speciation occurs without geographical separation of the diverging populations. Gene flow must still be reduced by another mechanism, such as mating preferences, different breeding times or, in plants, a change in chromosome-set number. Merely living in one area does not mean all individuals exchange genes freely.

The two routes share divergence and reproductive isolation but differ in the role of geography. Behavioural isolation involves differences such as courtship signals; temporal isolation involves different reproductive periods. A strong explanation identifies the mechanism, how it limits gene flow, and how divergence can persist. "They look different" is insufficient for either route.

AHL: Adaptive radiation fills different ecological roles

Adaptive radiation is diversification from a common ancestral lineage into multiple species associated with different ecological roles. New habitats or underused resources can create ecological opportunities. Divergent selection can favour different ways of feeding, moving or using habitat, while reproductive isolation allows differences to persist.

For example, an invented colonizing bird lineage encounters large hard seeds, small seeds and insects in different parts of an island group. Inherited variation in feeding structures and behaviour can influence access to these resources. Over many generations, divergence and isolation can produce several related species with different niches.

Do not claim that individuals choose to evolve new beaks, that every colonization causes radiation, or that competition disappears completely. Resource partitioning can reduce overlap and permit coexistence, but species can still compete for other limiting resources. Demonstrate common ancestry, divergence, ecological differences

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and species boundaries rather than treating a list of differently shaped animals as sufficient evidence.

AHL: Barriers before and after fertilization

Some mechanisms reduce hybrid formation before fertilization. Different courtship signals may prevent mating; different flowering seasons may prevent pollen transfer between plant populations. These are barriers to the mixing of alleles even when organisms occupy overlapping areas.

Other barriers act after fertilization. A hybrid may have reduced viability, or it may survive but have low fertility. A mule, the usual hybrid of a female horse and a male donkey, is generally sterile. Its existence therefore does not establish continuing free gene flow between the parental species. Chromosome differences can disrupt pairing and segregation in meiosis, so producing an adult hybrid is different from producing fertile gametes.

Such barriers need not be absolute in every organism. Some related species hybridize occasionally, and gene flow may be limited rather than zero. Evaluate the frequency, direction and reproductive success of crosses instead of treating one hybrid as an automatic disproof of every species boundary.

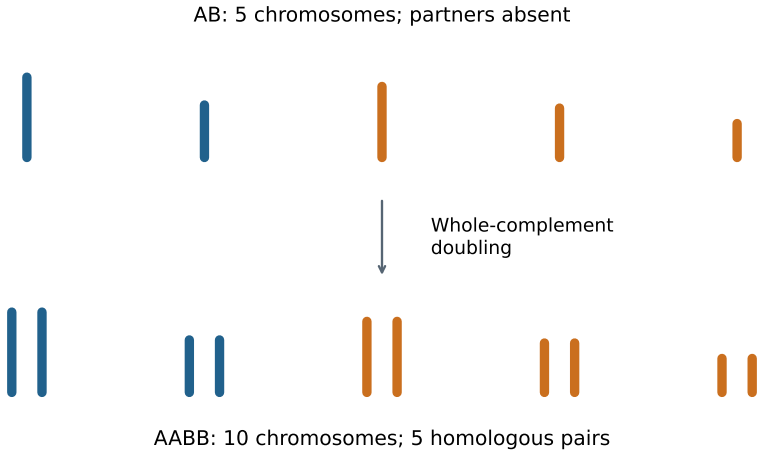
AHL: Hybridization and polyploidy in plants

Polyploidy means having more than two complete chromosome sets. Hybridization combines genetic contributions from different species. Together they can produce a new reproductively isolated plant lineage much more abruptly than gradual divergence of separated populations.

In an invented model, parent species A has two copies of an A set containing two chromosomes: $2n_A = 4$. Parent B has two copies of a B set containing three chromosomes: $2n_B = 6$. Their normal gametes contain two and three chromosomes respectively. Fusion gives an AB hybrid with five chromosomes. If A and B chromosomes cannot pair effectively as homologues, meiosis is irregular and fertility is low.

If all chromosomes in this hybrid are doubled, the new plant has AABB: ten chromosomes, with a homologous partner for each of the five chromosome types. Regular pairing can allow viable AB gametes with five chromosomes. Compatible doubled plants, or a self-compatible plant, can reproduce while crosses with the original diploid parents may produce unbalanced complements. This can establish reproductive isolation. Doubling creates chromosome partners; it is not merely ordinary DNA replication followed by an ordinary mitosis.

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Invented model: blue = A types; orange = B types.
Regular pairing can restore fertility; establishment still needs reproduction.

Chromosome numbers are invented. Pairing and establishment are model conditions, not a guarantee that every hybrid becomes a new species.

The knotweed/smartweed genus *Persicaria* supplies real evidence for this process. A 2008 molecular study supported multiple allopolyploid origins, using different nuclear gene copies to identify contributions from different parental lineages. For *P. punctata*, the evidence supported *P. hydropiper* and a lineage related to *P. hirsuta* or *P. setacea* as contributors. The uncertainty between potential contributors is part of the inference. Our five-to-ten chromosome model is not that study's measured chromosome dataset. See the primary study.

Practice

All numerical datasets are invented. State what the evidence supports and what remains untested.

1. Define evolution and adaptation. Distinguish a plant's afternoon stomatal response from a population-level inherited change, and explain one cost of reduced stomatal opening.
2. A model starts with 80 inherited form A and 120 form B individuals. The next generation contains 240 A and 160 B. Find A's initial and later frequencies, the change in percentage points and its relative percentage increase. State two model assumptions needed for a selection interpretation.
3. Two homologous sequences differ at 9 of 60 aligned sites. Calculate percentage difference and percentage matching positions. Explain why the results are neither a whole-genome identity claim nor a divergence date.

4. Explain how selective breeding can produce heritable population change, and why it is evidence relevant to evolution. Distinguish selection from a breeder transmitting an acquired body change directly.
5. Describe the shared pentadactyl forelimb plan. Explain how different functions can accompany homology and why a modern flipper need not be the ancestor of a modern arm.
6. Compare bird and insect wings as analogous flight structures. Explain why bird and bat forelimb bones can nevertheless be homologous while their flight surfaces differ.
7. A population's average inherited body size changes for many generations but it remains one interbreeding group. Is this evolution, speciation, both or neither? Explain. Distinguish the effects of speciation and extinction on species number.
8. Explain how geographical isolation and differential selection can lead to speciation. Why are a river and different appearance insufficient proof of complete reproductive isolation?
9. Use the chimpanzee–bonobo example to connect geography and divergence. Why should the explanation allow for evidence of ancient episodes of gene flow?
10. Two plant samples each have leaf area $0.25\,m^2$ and are measured for 4 h. A loses 15 g; B loses 10 g. Calculate both normalized rates and B's percentage reduction relative to A. Give three controls, one replication requirement and a reason this alone does not prove inherited adaptation.
11. Design a stronger test of the claim that a leaf trait is an inherited adaptation to dry conditions. Include evidence of inheritance, reproductive benefit and a possible trade-off; explain why a correlation along a habitat gradient is insufficient alone.
12. **AHL** Compare allopatric and sympatric speciation, giving a mechanism limiting gene flow in each. Explain how adaptive radiation involves more than different-looking descendants and why niche differences need not eliminate all competition.
13. **AHL** Distinguish a courtship barrier, a seasonal breeding barrier and hybrid sterility. Use a mule to explain why producing a surviving hybrid is different from maintaining fertile gene flow.
14. **AHL** In the invented A/B plant model, state parental diploid counts, gamete counts, AB hybrid count, AABB count and the number of paired chromosome types after doubling. Explain restored pairing, possible isolation from parents, and why the *Persicaria* study supports rather than directly observes every inferred historical event.

■ Worked answers

1. Evolution is population change in heritable characteristics across generations. An adaptation is an inherited feature improving survival or reproduction in particular conditions. Afternoon closure is an individual response, not by itself inherited population change. Reduced carbon dioxide uptake can limit photosynthesis even while closure saves water.

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2. Initially $f_A = 80/200 = 0.40$; later $f_A = 240/400 = 0.60$. Increase: 20 percentage points; relative increase $(0.60 - 0.40)/0.40 \times 100 = 50\%$. Assume relevant forms are inherited and the counts represent successive generations without confounding migration or mutation; reproductive success differs under the model. In real data, test these assumptions and alternative causes.
3. Difference $9/60 \times 100 = 15\%$; matching positions $51/60 \times 100 = 85\%$. These apply to this homologous alignment. Other genome regions can differ, and a date needs a rate/calibration model with appropriate assumptions.
4. Breeders choose parents with a desired heritable trait; offspring inherit relevant variants, and repeated selection changes their representation. Differences among breeds and varieties show population change is possible on observable timescales. Training or changing a parent's body does not itself establish inheritance of that acquired change.
5. One humerus, radius and ulna, wrist bones and a hand region with a five-digit ancestral plan. Corresponding positions can be retained while proportions and functions change, with later reductions or losses possible. Modern arms and flippers descend from ancestral structures in different lineages; neither living endpoint must be the other's ancestor.
6. Bird and insect flight structures have different origins: a bony feather-bearing forelimb versus an insect wing without that vertebrate bone plan. Similar flight function evolved independently. Bird and bat forelimbs retain homologous bones; feathers and a bat's digit-supported skin membrane provide different flight surfaces.
7. Evolution, because inherited population characteristics change; not necessarily speciation, because no lineage split is established. Speciation adds species through splitting; extinction removes species. A change in trait average alone supplies no new-species count.
8. A barrier reduces gene flow; different selection pressures, mutation and other processes permit divergence; reproductive barriers may accumulate. A river may separate still-compatible populations. Different appearance can reflect environment or within-species variation. Test mating patterns and offspring viability/fertility in suitable evidence, rather than forcing risky or unnecessary crosses.
9. The Congo River is a major barrier separating much of the present ranges of *P. troglodytes* and *P. paniscus*. Reduced gene flow allowed divergence under different conditions. Genomic evidence of later ancient mixing means the barrier was not perfectly impermeable throughout history. Both modern species share ancestors; one is not simply the other's ancestor.
10. $R_A = 15/(0.25 \times 4) = 15$ and $R_B = 10/(0.25 \times 4) = 10\,\mathrm{g/(m^2\,h)}$. Reduction $(15 - 10)/15 \times 100 \approx 33.3\%$. Control, for example, temperature, humidity and airflow; also consider light, age and water supply. Use several independent plants and repeat measurements. Conditions or plasticity can explain differences; inheritance and reproductive benefit remain untested.
11. Compare related plants or offspring under common controlled growth conditions

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to investigate inheritance, supported where appropriate by genetic evidence. Test replicated dry and wetter conditions, measuring survival or reproductive output as well as the leaf trait and water loss. Look for a photosynthetic or growth cost. Habitat gradients confound temperature, soil, ancestry and other factors, so correlation alone cannot establish the mechanism or inheritance.

12. **AHL** Allopatric: geography reduces gene flow; sympatric: separation arises without a geographic barrier, for example through breeding-time differences or plant chromosome-set changes. Both need persistent divergence and reproductive isolation. Radiation combines common ancestry, multiple species and ecological diversification. Niche partitioning can reduce resource overlap without eliminating competition for every resource.
13. **AHL** Courtship differences can prevent mating; different breeding seasons can prevent reproductive contact. Both reduce fertilization between groups. Sterility acts after a hybrid is formed: a mule can survive but is generally unable to produce offspring. Reduced meiotic pairing/segregation success limits onward allele mixing; occasional hybrid formation does not demonstrate unrestricted fertile gene flow.
14. **AHL** A: four chromosomes, gametes two; B: six, gametes three. AB: five; AABB: ten, with five paired types. Doubling supplies homologous partners and can permit five-chromosome AB gametes. Reproduction among compatible doubled plants can maintain a lineage isolated from parental complements. Establishment and fertility are conditions, not automatic. Different *Persicaria* gene copies support contributions from different ancestral lineages, with uncertainty about some contributors; the historical crosses were inferred, not directly watched.

Exam tips

Identify the population, generations and evidence of inheritance. Explain a benefit and a cost in a stated environment. Distinguish homology of a structure from similarity of function. For speciation, explain reduced gene flow and reproductive isolation; geographical separation or a changed average alone is insufficient. State the denominator in frequency and rate calculations.

References: IB Biology guide, A4.1. Named genomic and plant studies are linked in their teaching sections.

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Conservation of biodiversity

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A field contains thousands of plants, yet almost all belong to one crop variety. A nearby wetland has fewer large plants but many species and habitats. Which place has more biodiversity, and which evidence would justify spending a limited conservation budget there?

This unit covers A4.2 for both SL and HL, with no additional higher-level content: levels of biodiversity, comparisons through time, human-caused extinctions and ecosystem loss, evidence of a biodiversity crisis, its causes, complementary conservation methods and EDGE priorities. Prerequisites: species, variation, evolution and ecological sampling. All numerical survey datasets and maps below are invented teaching examples unless a named published source is identified. They are not observations from the school grounds.

Biodiversity exists at several levels

Biodiversity is the variety of life, including genetic variation within species, differences among species, and differences among ecosystems. A count of visible animals does not measure all three levels.

Level	What varies	A conservation question
Genetic	Alleles and inherited variation within a species	Does a breeding population retain enough variation for future change?
Species	Species present and their relative abundances	Are rare species disappearing even while common species increase?
Ecosystem	Communities, habitats and their physical environments	Are wetlands, forests and grasslands being replaced by one land use?

Losing several genetically distinct populations can reduce biodiversity even before their species becomes globally extinct. Conversely, adding many individuals of one already common species does not necessarily restore lost species or ecosystems. A useful conservation goal identifies the level being protected and how success will be measured.

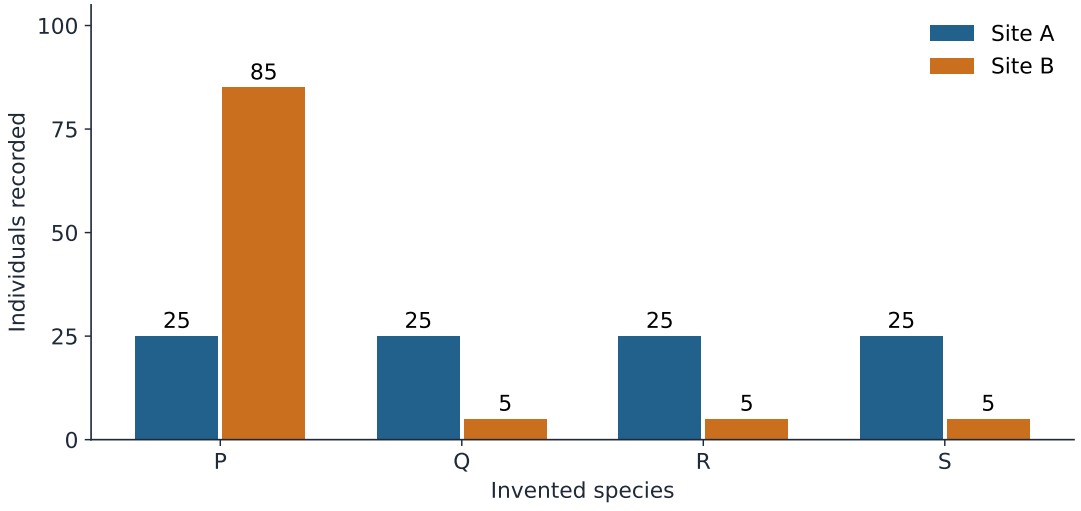
Species richness is the number of species recorded. **Evenness** concerns how evenly individuals are distributed among those species. Total abundance counts individuals. These quantities answer different questions and must not be substituted for one another.

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Worked example: equal richness is not equal composition

In invented standardized surveys, sites A and B each contain 100 individuals in four species. A has counts 25, 25, 25 and 25. B has 85, 5, 5 and 5.

- Both have total abundance 100 and recorded richness 4.
- The dominant species represents $25/100 = 25\%$ at A but $85/100 = 85\%$ at B.
- A is more even. B's high total abundance does not remove the dominance of one species.



Both invented samples: total = 100; richness = 4.
Dominant species: A 25%; B 85%.

Use the same sampling effort and identification rules. The bars describe these samples, not a complete census of every organism at each site.

A diversity index can combine aspects of a community into one value, but its formula and convention must be stated. An index does not identify the cause of a change. Counts, uncertainty and species composition remain useful even when a summary index is calculated.

Compare the present with the past carefully

Many species have been described, while many others remain undiscovered or undescribed. Fossils record past organisms and show diversification interrupted by major losses. Evidence supports long-term increases in diversity in many groups and very high present richness, but the fossil record is not an equally complete census of every period and habitat.

Hard parts preserve more readily than many soft tissues; accessible rocks and extensively studied regions supply more records. Changes in sampling and classification also affect apparent counts. "More recorded species" can reflect a better survey or a

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revised species boundary rather than a sudden ecological increase. Define whether a comparison concerns described species, estimated total richness, a particular group or a local sample.

High present diversity and a current biodiversity crisis are compatible. A large total can be declining rapidly. A population can fall sharply before global extinction occurs, and habitats can lose ecological functions before every species disappears. We therefore need repeated measurements, not just one impressive count of living species.

Three human-caused extinction cases

Extinction is the loss of a species everywhere. Local disappearance is extirpation: a species may still survive elsewhere. "Extinct in the wild" differs from global extinction because individuals remain in captivity or cultivation. A date of official assessment is not necessarily the date when the last individual died.

■ North Island giant moa: loss of terrestrial megafauna

The North Island giant moa, *Dinornis novaezealandiae*, was a large flightless bird of New Zealand. Its remains occur widely in archaeological sites. Human hunting for food was the main cause of its extinction. Sustained removal can prevent a population from replacing its losses. This is an example of over-exploitation of terrestrial megafauna, not evidence that a large body automatically prevents extinction. The Te Papa/Birds New Zealand species account identifies overhunting as the main cause.

■ Caribbean monk seal: loss of a marine species

The Caribbean monk seal, *Neomonachus tropicalis* (called *Monachus tropicalis* in older records), was hunted heavily, including for oil and meat, and suffered human disturbance. A marine habitat did not protect it from sustained exploitation. The last confirmed sighting was in 1952; later searches and the absence of convincing records supported an extinction conclusion. NOAA's 2008 status review concluded that the species was extinct. Do not report 2008 as a directly observed death date.

■ Chinese paddlefish: a Yangtze example

The Chinese paddlefish, *Psephurus gladius*, inhabited the Yangtze system. Its decline involved overfishing and fragmentation of its river habitat; dams could obstruct movement between feeding and spawning areas. A 2020 study combined historical records with basin surveys and inferred extinction by the early twenty-first century. The study explains the evidence; the species was treated as extinct in the 2022 sturgeon-group assessment announcement.

This case is relevant to the river basin near the Yangtze delta region. A river population needs connected habitats and successful reproduction, not simply water remaining in its channel. Do not confuse the extinct paddlefish with other Yangtze fishes that have different conservation statuses.

Across the three cases, human pressures reduced survival or reproduction faster than

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populations could replace losses. Several pressures can interact. Explaining the causal chain is more useful than memorizing a list of species names without their habitats, threats or evidence.

Ecosystem loss removes relationships as well as area

An ecosystem includes organisms interacting with one another and with the physical environment. Replacing a diverse forest with a single-species plantation can leave many trees while removing much of the original community and its processes. Loss of area, degradation of quality and fragmentation are related but different changes.

■ Mixed dipterocarp forest in Southeast Asia

Lowland forests in parts of Southeast Asia contain diverse trees, including many dipterocarps. Timber extraction changes forest structure; roads can increase access and divide habitats. Subsequent clearance for oil-palm or other plantations replaces the forest ecosystem rather than merely removing selected trunks.

Distinguish logged forest from cleared land. Logged forests can retain substantial biodiversity and deserve protection; calling them "degraded" does not mean they are empty or worthless. Research in Sabah, Borneo compared unlogged, once-logged and repeatedly logged forests, showing the conservation value of forests that had already been logged. The primary study supports this distinction. Conservation must address both further clearance and the quality and connectivity of remaining habitat.

■ Yangtze estuary tidal flats

Intertidal flats are exposed and covered by the tides. They contain feeding habitats for waterbirds and communities adapted to changing water and sediment conditions. Coastal land reclamation can replace these habitats with enclosed land. Reduced river sediment supply can also change the balance between deposition and erosion.

Studies of the Yangtze estuary and delta identify reclamation and altered sediment supply as important causes of tidal-flat loss. See the estuary study and the delta analysis. A protected label alone cannot maintain the ecosystem if sediment processes and connected feeding areas are lost. Local gains in one place also need not offset losses elsewhere for the same organisms.

■ Worked example: area is only one indicator

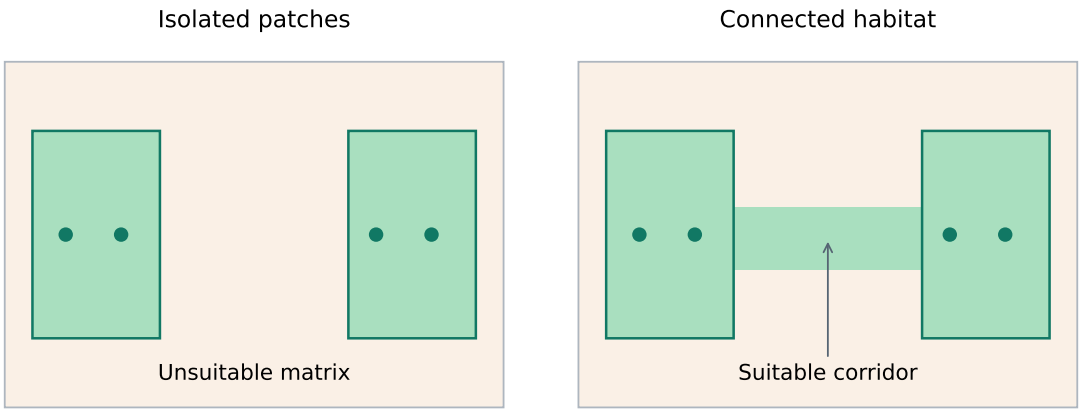
An invented wetland map records 1,000 ha of suitable habitat initially and 760 ha later, using the same definition and mapping method. The loss is $1000 - 760 = 240$ ha, or $240/1000 \times 100 = 24\%$. The remaining fraction is 76%.

This does not establish that 24% of species became extinct. Some can persist temporarily, others may disappear disproportionately, and the remaining habitat may be fragmented or degraded. Species surveys and measures of habitat quality are needed alongside area.

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Fragmentation can reduce movement and gene flow

When roads, farms or buildings divide habitat, individuals may have fewer opportunities to find mates, migrate or recolonize empty patches. Smaller isolated populations may be more vulnerable to chance losses and loss of genetic variation. Edge conditions can differ from the interior in light, temperature, disturbance or predation.



Invented plan: green = habitat; tan = surrounding land use.
Test movement and breeding; a drawn connection is not proof.

The corridor represents a management hypothesis to test. It does not guarantee movement or successful reproduction for every species.

A corridor can improve connection when it contains suitable habitat and crosses barriers effectively. Width, vegetation, roads and the species' movement requirements matter. A strip useful to one organism may fail for another or spread unwanted organisms. Compare actual movement, survival and breeding before and after management rather than assuming a line on a map solves isolation.

Evidence for a biodiversity crisis needs repeated surveys

The IPBES 2019 global assessment estimated that around one million species were threatened with extinction. This is an estimate of risk, not a count of one million already extinct species. The assessment's summary combines evidence across many groups and habitats; a single local survey cannot establish a global rate of extinction.

A strong local comparison uses repeated surveys with similar area, duration, season, weather constraints, sampling method and identification rules. Record uncertain identifications and keep a consistent taxonomic basis. Use independent sites or sampling units and report variation, not only one total.

Invented comparable surveys of species P, Q, R and S give counts 40, 30, 20, 10 initially and 65, 25, 10, 0 later. Both totals are 100. Recorded richness falls from 4

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to 3, while dominance of P rises from 40% to 65%. Equal total abundance therefore hides a change in composition and reduced evenness. Failure to record S is evidence of possible local loss, not proof that S is globally extinct.

Citizen scientists can expand coverage and supply long time series. Training, photographs, expert checks and consistent protocols strengthen those records. Uneven observer effort, changes in skill, difficult species and preferential reporting can bias trends. Published methods and accessible evidence allow other researchers to check the inference. Citizen data are valuable when their limits are handled, rather than rejected or accepted automatically.

Human pressures act through specific mechanisms

Human population growth can increase demands for food, water, energy and land. Its effects depend on consumption, technology and management, so population size alone does not fully predict a site's damage. Link each pressure to a mechanism and evidence.

Pressure	How biodiversity can decline
Over-exploitation	Hunting, harvesting or fishing removes organisms faster than replacement
Land-use change	Urbanization, agriculture and clearance replace or divide habitats
Pollution	Toxic materials or excess nutrients change survival and ecosystem conditions
Transport of organisms	Introduced pests, diseases or invasive species can disrupt communities
Climate change	Altered temperature, rainfall and sea conditions can shift suitable habitat

Not every introduced species becomes invasive, and not every species responds identically to warming or pollution. Interactions matter: a small fragmented population may be less able to move when climate changes. Identify the scale and affected species before making a management claim.

Combine conservation methods around the actual threat

In situ conservation protects species within their natural habitats. Nature reserves can protect communities and ecological processes, but they need suitable boundaries, management, enforcement and connections. A reserve that excludes important breeding or seasonal feeding areas may protect only part of a species' needs.

Restoration repairs degraded ecosystems by addressing causes and rebuilding suitable conditions. Rewilding aims to restore ecological processes and, where appropriate, native organisms. Removing a pressure, restoring water flow or recovering native

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vegetation may be more important than simply planting a large number of trees. Set a baseline, choose suitable reference conditions and monitor outcomes. Reintroduction needs appropriate habitat and ecological assessment; release alone is not evidence of success.

Ex situ conservation maintains organisms outside their natural habitats, for example in zoos, botanic gardens or managed breeding collections. It can protect a population during an immediate threat and support later recovery. Small populations still need management of breeding, genetic diversity, health and suitable behaviour. Captive survival does not replace a functioning habitat or guarantee a self-sustaining wild population.

Germplasm is inherited biological material that can be conserved for future reproduction. Seed banks and tissue collections can preserve genetic resources. Seeds of many species can be dried and stored, but some cannot tolerate this treatment and require other approaches. A collection must maintain viability, identify its material correctly and retain suitable genetic representation. A freezer is not a substitute for every ecosystem interaction.

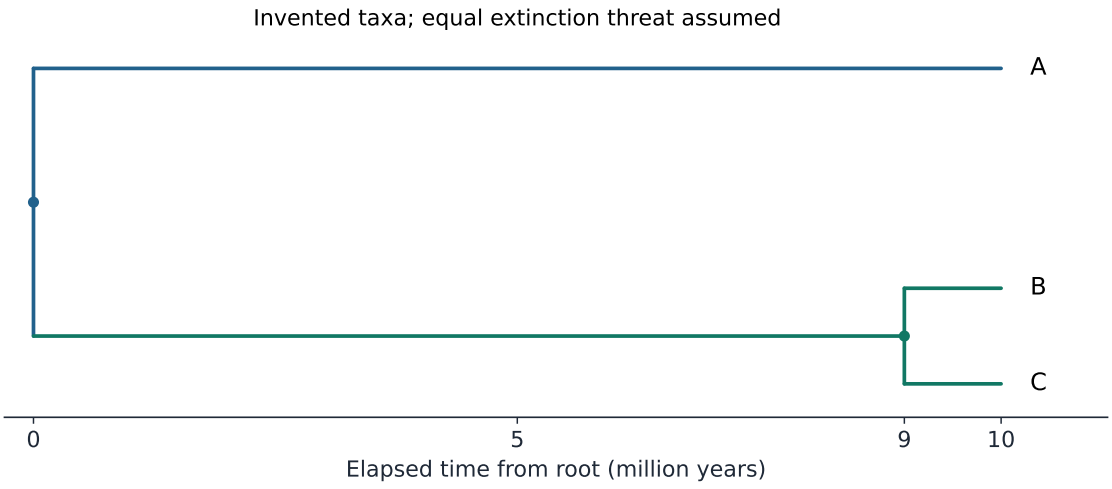
No single method is sufficient for every species. For an invented plant threatened by drainage and habitat clearance, a sensible package might protect and restore the wetland in situ, retain seed or tissue material ex situ as appropriate, and monitor establishment and reproduction. Banking seeds while allowing the last suitable habitat to disappear leaves the main cause unresolved.

Prioritize threatened evolutionary history with EDGE

The EDGE of Existence programme combines Evolutionary Distinctiveness with Global Endangerment. Evolutionary distinctiveness concerns how much relatively unique evolutionary history a species represents, using relationships on a dated phylogeny. Global endangerment concerns its extinction risk. A species with few close relatives can represent a large branch of history that would be lost if it disappeared.

Consider three invented equally threatened species in the figure. A has no close relative among these taxa, while B and C share a recent common ancestor. If threat and other conditions are comparable, conserving A may protect more unique evolutionary history. This explains a rationale, not a complete real EDGE score: do not invent a score from appearance or simply count individuals. The programme's explanation describes how phylogeny and threat are combined.

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A has a long independent lineage; B and C are close relatives.
This illustrates priority, not a real EDGE score.

With equal threats in this simplified example, losing A removes more uniquely represented history. This is not a real EDGE ranking or a complete calculation.

Priorities also involve feasibility, ecological roles, cost, local knowledge, cultural values and effects on people. An evolutionary argument contributes evidence but does not remove ethical choices. State the goal, compare options transparently, involve affected communities and explain the trade-offs. Success requires a reduction in the actual threat, not merely a high position on a list.

Practice

All numerical datasets and management scenarios below are invented. Explain limits as well as calculations.

1. Distinguish genetic, species and ecosystem biodiversity. Give one loss that could occur at each level, and explain why a field with many crop plants need not be highly diverse at all three levels.
2. Sites A and B have counts 25, 25, 25, 25 and 85, 5, 5, 5. Calculate total abundance, richness and dominant-species percentage for both. Which is more even, and why does abundance not settle the comparison?
3. Explain two limits of comparing present and past recorded species counts. Why can high present richness coexist with a biodiversity crisis?
4. For each of the North Island giant moa, Caribbean monk seal and Chinese paddlefish, identify the habitat, an important human pressure and a causal route to extinction. Distinguish a last sighting from an assessment date.

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5. Compare the loss of mixed dipterocarp forest with the loss of Yangtze tidal flats. Explain why many plantation trees do not necessarily replace a forest ecosystem, and why logged forest may still deserve protection.
6. Suitable habitat falls from 1,000 to 760 ha. Calculate area lost, percentage lost and percentage remaining. Why is it invalid to conclude that the same percentage of species became globally extinct?
7. For a fragmented habitat, propose one corridor benefit and two conditions needed for success. Give a possible limitation and one measurement that would test the proposal.
8. Comparable surveys change from counts 40, 30, 20, 10 to 65, 25, 10, 0. Compare total abundance, recorded richness and dominance. Does the zero prove global extinction? Explain.
9. Design a repeated biodiversity survey using citizen scientists. Give four features to standardize and two ways to check identification or observer bias. Explain what a published method adds.
10. Explain how human population growth can increase biodiversity pressures while not being a complete explanation by itself. Link over-exploitation, habitat change, pollution and invasive organisms to distinct mechanisms.
11. A threatened wetland plant loses habitat through drainage. Propose an in situ measure, a restoration measure and an appropriate ex situ backup. Give one limit of each and a measure of recovery beyond counting planted individuals.
12. Explain germplasm conservation. Why cannot all plant species be conserved in the same dry-seed storage conditions, and why does stored genetic material not preserve an entire ecosystem?
13. Two equally threatened species differ in evolutionary distinctiveness. Explain the EDGE rationale for prioritizing the more distinct species, why appearance alone is insufficient, and two other considerations that could affect the final decision.

■ Worked answers

1. Genetic biodiversity: variation within a species, reduced when distinct populations or alleles are lost. Species biodiversity: variety and relative abundance of species, reduced by species losses or increased dominance. Ecosystem biodiversity: variety of ecological systems, reduced when different habitats are converted to one land use. Many individuals can belong to one species or cultivar and occupy one simplified ecosystem.
2. A: total 100, richness 4, dominance $25/100 \times 100 = 25\%$. B: total 100, richness 4, dominance $85/100 \times 100 = 85\%$. A is more even because its individuals are equally distributed. Both totals are identical, but their composition differs greatly.
3. Preservation and discovery differ among periods, habitats and organisms; survey effort and taxonomic boundaries also change. Recorded counts are not complete equal-quality censuses. A large present total can still be declining, with population and habitat losses occurring before global extinctions are confirmed.

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4. Moa: terrestrial New Zealand, overhunting removed individuals faster than replacement. Monk seal: Caribbean marine/coastal habitats, heavy hunting and disturbance reduced survival and reproduction. Paddlefish: Yangtze river system, overfishing and barriers to movement/spawning fragmented the population. A last confirmed sighting records detection; an assessment later evaluates all evidence. Neither is automatically a directly observed date of the final death.
5. Forest clearance replaces diverse communities and structure with another land use; plantation tree abundance does not reproduce the original species and processes. Logged forest can retain important biodiversity. Tidal-flat loss involves replacement by reclaimed land and changes in sediment supply and erosion. Both require attention to habitat quality and ecological processes, not area alone.
6. Loss $= 1000 - 760 = 240$ ha. Percentage lost $= 240/1000 \times 100 = 24\%$; remaining $= 760/1000 \times 100 = 76\%$. Species respond differently, some persist temporarily, and local habitat loss is not a census of global extinctions. Survey composition, population trends and remaining habitat quality too.
7. A suitable corridor may improve movement, mating or recolonization. It needs appropriate habitat and effective continuity across barriers; width and species-specific behaviour matter. It may fail for a species or spread undesirable organisms. Compare movement, survival or breeding using consistent before-and-after or managed-and-comparison surveys.
8. Both totals are 100. Recorded richness falls $4 \rightarrow 3$; dominant P rises $40/100 = 40\%$ $\rightarrow 65/100 = 65\%$, a 25-percentage-point rise. The later sample is less even and S is not detected. Detection failure or local loss is possible; there is no evidence here that S disappeared everywhere.
9. Standardize area, effort/duration, season and method, with comparable identification rules and suitable conditions. Train observers, use photographs or vouchers where appropriate without harmful collection, request expert verification and record uncertain identifications. Track observer effort and repeat independent units. Published methods allow other people to examine and repeat the comparison rather than relying on an unexplained total.
10. More people can increase demand for land, food, water and energy, but consumption, technology and management alter impacts. Exploitation exceeds replacement; habitat conversion removes or fragments suitable places; pollution changes biological conditions; an invasive organism may outcompete, consume or infect native organisms. Introduction does not automatically imply invasiveness.
11. Protect remaining habitat: reserve management can reduce clearance but may miss water flows beyond its boundary. Restore hydrology and native conditions: recovery takes time and may be incomplete. Preserve appropriately stored seeds or tissue and manage a backup population: viability and genetic representation need maintenance, and this does not replace habitat. Measure natural recruitment, survival, reproduction and continuing threat reduction.
12. Germplasm stores inherited material for future reproduction. Many seeds tolerate

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drying and storage, while others require different methods such as suitable living or tissue collections. Storage needs viability tests and representative material. Genes in a collection do not recreate food webs, habitats, water flows or species interactions.

13. A more distinct lineage may carry more unique evolutionary history that would vanish with extinction. EDGE combines phylogenetic information with extinction risk; unusual appearance or low abundance alone is not a score. Feasibility and cost, ecological roles, local priorities and effects on people can also alter the decision. State the goal and trade-offs and address the actual threat.

Exam tips

Name the level of biodiversity and distinguish individuals, species, populations and ecosystems. Compare consistent repeated surveys before attributing a trend. Connect threats to mechanisms and measures to those threats. Distinguish assessment dates, detection dates and extinction itself. For a conservation decision, combine evidence, limits and an explicit goal.

References: IB Biology guide, A4.2. Named assessments, case studies and the EDGE method are linked in the relevant sections.

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Carbohydrates and lipids

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A potato stores starch, a seed stores oil, and a plant cell wall contains cellulose. All three materials contain carbon, hydrogen and oxygen. Why can one be digested as a quick source of glucose, another store much energy in a small mass, and another resist stretching?

This unit covers all B1.1 understandings for SL and HL: carbon compounds, condensation and hydrolysis, monosaccharides, storage and structural polysaccharides, cell recognition, triglycerides, phospholipids, fatty-acid saturation, insulation, bilayers and steroids. There is no additional higher-level content in this unit. Prerequisites are covalent bonds, water polarity, cell structure and basic ratios. Molecular cartoons simplify three-dimensional molecules; numerical practice data are invented unless stated otherwise.

Carbon supports many different molecular structures

A covalent bond involves a shared pair of electrons. Carbon usually forms four covalent bonds in biological compounds. Four single bonds, or two single bonds and one double bond, both give a total bond order of four. A carbon–carbon double bond counts as two, not one, in this accounting. Carbon can bond to other carbon atoms and to elements such as hydrogen, oxygen, nitrogen, phosphorus and sulfur.

Carbon skeletons can be unbranched chains, branched chains or rings. Several rings can be joined, as in steroids. Different arrangements of the same types of atoms can produce different properties. An element list is therefore insufficient to identify a carbohydrate, lipid or protein. Check the actual bonds and functional groups. Proteins usually contain nitrogen as well as carbon, hydrogen and oxygen; some also contain sulfur.

Biological diagrams use conventions. In a skeletal formula, an unlabelled corner or line end normally represents carbon; hydrogens attached to carbon may be omitted. An explicitly labelled O is oxygen, not an extra carbon. A ring containing five atoms can contain only four carbons if the fifth atom is oxygen. Count carbon atoms outside the ring too.

Metric prefixes also follow agreed conventions: kilo means a thousand, centi one hundredth, milli one thousandth, micro one millionth and nano one billionth of the base unit. Thus 1 mm = 1,000 μm and 1 μm = 1,000 nm. A membrane thickness of 7 nm is 0.007 μm, not 0.007 mm. Convert to the same unit before comparing a molecule with a cell.

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Condensation builds links; hydrolysis breaks them

A monomer is a small building unit; a polymer contains many linked units. Polysaccharides are polymers of monosaccharides, polypeptides contain amino-acid residues, and nucleic acids contain nucleotide residues. A residue is the part incorporated into a larger molecule; it is not necessarily chemically identical to the original free monomer.

In a simplified condensation model, two units become joined by a covalent linkage while a water molecule is released. Hydrolysis breaks a linkage by using water: H and OH from water become incorporated into the products. For a glycosidic linkage, distinguish the covalent bond between sugar residues from hydrogen bonds between neighbouring molecules or chains.

The linkage names matter: a glycosidic bond joins sugar residues; a **peptide bond** joins amino-acid residues; a phosphodiester linkage joins nucleotides in a nucleic-acid backbone. Lipids such as triglycerides contain ester linkages but are not repeating-monomer polymers. "Large biological molecule" and "polymer" are not interchangeable.

■ Worked example: count bonds before counting water

One connected, unbranched chain made from 20 monomers has 19 links between successive units. In the simple condensation accounting model, joining those free units releases 19 water molecules; completely hydrolysing those links uses 19 water molecules. For n units in a linear chain, there are $n - 1$ links.

Two separate chains of ten residues each initially have $2 \times 9 = 18$ links. Joining the chains end to end adds one new link, giving 19 links in the 20-residue chain. A calculation must state whether it concerns forming all links or making just the final connection. This is molecular accounting, not a complete description of enzyme-mediated biosynthesis in cells.

Digestion hydrolyses large food polymers into smaller soluble products that can be absorbed. Cutting a polymer into pieces does not automatically mean every piece is already a monomer. For complete hydrolysis, every relevant linkage must be broken.

Monosaccharide form explains useful properties

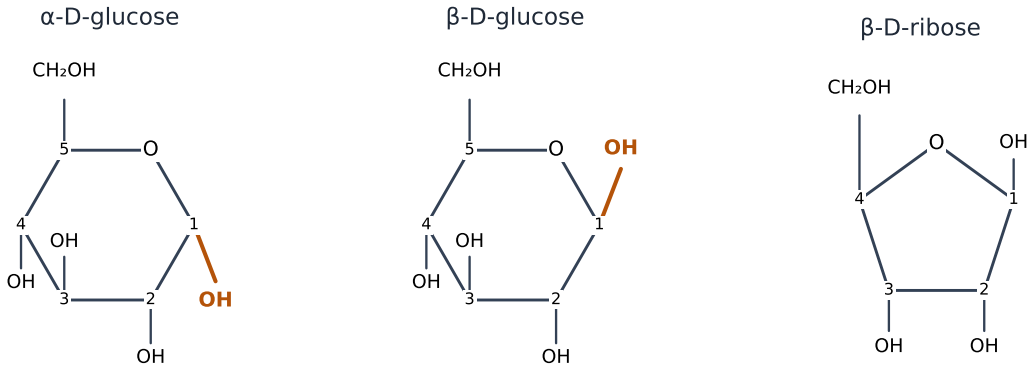
Monosaccharides are single sugar units. Pentoses contain five carbon atoms, as in ribose; hexoses contain six, as in glucose. In ring forms, glucose commonly has five carbons and one oxygen in its ring, with the sixth carbon outside it. Ribose commonly has four carbons and one oxygen in its ring, with one carbon outside. A pentagon is not automatically a pentose, and a hexagon is not automatically a hexose: inspect the labels and side groups.

Glucose has several hydroxyl groups that interact with water, making it soluble. It can be transported in aqueous body fluids and supplied to cells. It is sufficiently chemically stable for controlled transport and storage in suitable forms, yet enzymes can catalyse its reactions. Oxidation in respiration can transfer energy to processes

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such as ATP production. Glucose does not release a useful burst of energy merely because it dissolves.

Alpha- and beta-glucose differ in the orientation of the hydroxyl group at carbon 1 in the usual ring representation. For D-glucose drawn as below, the alpha C1 hydroxyl is on the opposite side to the C5 hydroxymethyl group; the beta C1 hydroxyl is on the same side. Rotating the whole drawing does not turn alpha into beta. Compare the relative positions, not just "up" on the page.



Orange: glucose C1 hydroxyl. Carbon-bound hydrogens omitted.
Glucose has six carbons; ribose has five. Count the external carbon too.

These are simplified Haworth representations. Carbon-bound hydrogens are omitted. Glucose C6 and ribose C5 lie outside their rings. Orange highlights the glucose C1 hydroxyl.

A glucose molecule and a fructose molecule share the formula $C_6H_{12}O_6$ but differ in structure. Their formula alone does not identify which sugar is present. The different arrangement of glucose units in polysaccharides likewise changes function even when the building units have the same overall formula.

Starch and glycogen store glucose compactly

Plants store glucose as starch. Amylose consists mainly of unbranched alpha-glucose chains with alpha-1,4 links that coil. Amylopectin also contains alpha-1,6 branch points. Animals and fungi store glycogen, a more highly branched alpha-glucose polymer. A starch sample is not necessarily one perfectly unbranched chain.

Coiling and branching help pack many glucose residues into a relatively small volume. Glycogen's many branch ends provide sites at which residues can be added or removed during regulated metabolism. Do not confuse a branch point with a free chain end. The arrangement allows mobilization of a store when energy demand increases.

Large storage polysaccharides are relatively insoluble compared with glucose. They can be retained as granules instead of diffusing away as many small dissolved molecules. Converting many free glucose molecules into a smaller number of large storage particles

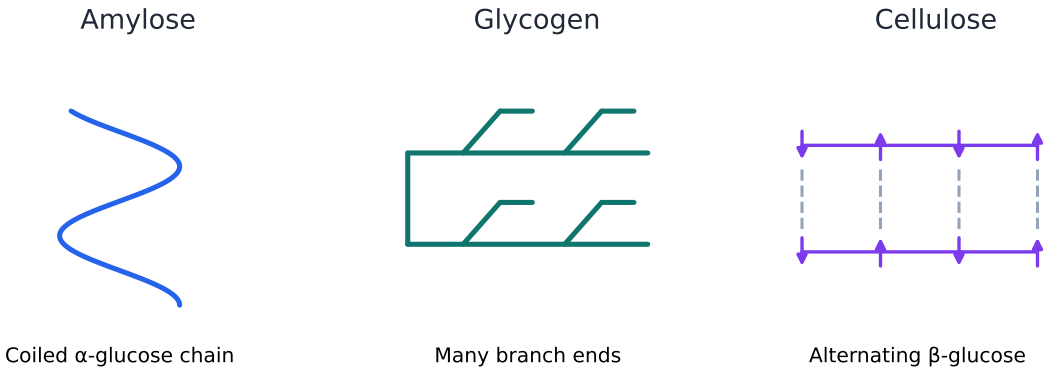
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reduces their contribution to the concentration of dissolved particles and therefore helps limit osmotic effects. This does not mean a living cell has no water movement or no osmotic regulation.

Hydrolysis of glycosidic bonds can ultimately yield glucose units for respiration. Condensation accounting describes building the store; hydrolysis describes breaking its links. In cells, enzymes and activated intermediates control these processes. A storage molecule does not choose when to break down.

Cellulose resists tension through a different arrangement

Cellulose is a structural polysaccharide in plant cell walls. It contains beta-glucose residues joined by beta-1,4 glycosidic bonds. Alternate residues have opposite orientations, producing straight chains rather than the coiled arrangement of amylose. Many chains lie alongside one another. Hydrogen bonds between chains help hold them in bundles called microfibrils.



Schematic organization, not atomic formulas.
Solid: covalent chain links. Dashed: hydrogen bonds between cellulose chains.

Lines along a chain represent covalent glycosidic links. Dashed cross-links between cellulose chains represent hydrogen bonds. The cartoon shows organization, not a full atomic formula.

The covalent backbone and collective interactions between chains give cellulose high tensile strength. A plant cell wall can resist forces as water enters the cell. The wall is not the selectively permeable cell membrane, and its strength does not mean every small substance is excluded.

Humans digest starch using suitable enzymes but do not produce cellulase to hydrolyse cellulose's beta-1,4 links. "Both are glucose polymers" does not imply "both are digested by the same enzyme". Cellulose contributes dietary fibre; some organisms use microbial partners to help digest it. Heating a material or disrupting some hydrogen bonds is not equivalent to completely hydrolysing every glycosidic bond.

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Surface carbohydrates contribute to cell recognition

A glycoprotein has carbohydrate groups attached to a protein. Carbohydrate chains on the outer cell surface can act as recognition features. Different terminal sugar arrangements can be recognized by specific binding molecules, allowing cells or immune components to distinguish surfaces.

ABO blood-group antigens provide an example. A and B antigens differ in their terminal carbohydrate structures. They occur on membrane glycoproteins and glycolipids, so it is inaccurate to call every ABO antigen simply a free protein. Group O lacks the A and B terminal modifications but still has other cell-surface molecules and antigens. "O means no antigens anywhere" is false.

Specific recognition depends on molecular structure and binding, not on a cell reading a written label. A change in a terminal sugar can change recognition without changing every molecule in the membrane. The ABO example connects carbohydrate structure with recognition; it is not a complete set of rules for clinical transfusion decisions.

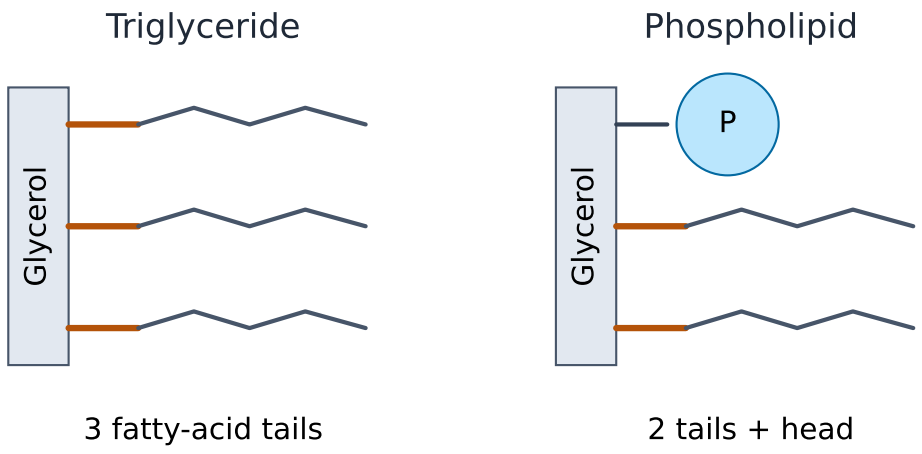
Lipid structures explain hydrophobic behaviour

Lipids include fats, oils, waxes and steroids. They are generally poorly soluble in water and more soluble in non-polar solvents. Much of a typical lipid consists of non-polar hydrocarbon regions that do not interact favourably with water in the same way as polar or charged groups.

A triglyceride consists of glycerol linked to three fatty acids. Glycerol has three hydroxyl groups; a fatty acid has a carboxyl group and a hydrocarbon chain. In a simple esterification model, each fatty acid's carboxyl group reacts with a glycerol hydroxyl, forming an ester linkage and releasing water. Three linkages give three water molecules per triglyceride. Complete hydrolysis of those three ester links uses three waters and yields glycerol and three fatty acids.

A typical glycerophospholipid contains glycerol, two fatty-acid tails and a phosphate-containing head group. It is not a triglyceride with an extra phosphate simply added on top of three tails. The head is polar or charged, while the tails are largely non-polar. The detailed head-group chemistry varies among phospholipids.

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The molecular cartoons label components rather than every atom. A typical glycerophospholipid has two hydrophobic tails and a hydrophilic phosphate-containing head.

> ■ **Worked example: conservation of mass during condensation**
>
> For an invented molar-mass calculation, use glycerol 92, each of three identical fatty acids 256, and water 18, all in $\mathrm{g\,mol^{-1}}$. The triglyceride's molar mass is $92+3(256)-3(18)=806\,\mathrm{g\,mol^{-1}}$. The reactants total 860; the products are triglyceride 806 plus water 54. Matter is conserved. The released water has not vanished, and its mass must not be subtracted twice.

Saturation changes packing and melting behaviour

A saturated fatty acid has no carbon–carbon double bonds in its hydrocarbon chain. A monounsaturated fatty acid has one; a polyunsaturated fatty acid has two or more. The carbon–oxygen double bond of the carboxyl group does not make a fatty acid "unsaturated" in this classification.

A cis carbon–carbon double bond introduces a bend that tends to disrupt close packing. For otherwise comparable chains, more cis unsaturation generally lowers the melting point. Saturated chains pack more closely and often have higher melting points. Chain length and the mixture of fatty acids matter too. A trans double bond has a different shape, so "every double bond produces the same kink" is false.

Many plant storage oils contain substantial unsaturated fatty acids and remain liquid at ordinary room temperatures. Many animal storage fats contain larger saturated fractions and are more solid under those conditions. These are tendencies, not universal plant-versus-animal rules: some plant fats are solid, and animal fats also contain

unsaturated components. Endotherms maintain warm bodies, so a material's state must be compared at a stated temperature, not inferred from its origin alone.

Triglycerides provide energy storage and insulation

Triglycerides contain many reduced carbon–hydrogen bonds and have a high energy yield per unit mass when oxidized. They are stored with little associated water compared with hydrated carbohydrate stores. These features suit long-term energy storage, especially where carrying mass matters. They are hydrophobic and are not readily transported as free dissolved molecules in water; organisms use appropriate packaging and transport mechanisms.

Adipose tissue contains cells that store triglycerides. A layer of this tissue reduces heat transfer between a warm body and colder surroundings, providing thermal insulation. It slows heat loss; it does not create heat by itself. Stored lipid can also be metabolized to supply energy. Insulation needs depend on body temperature, habitat and other adaptations such as fur, feathers or behaviour.

■ Worked example: compare equal energy, then hydration

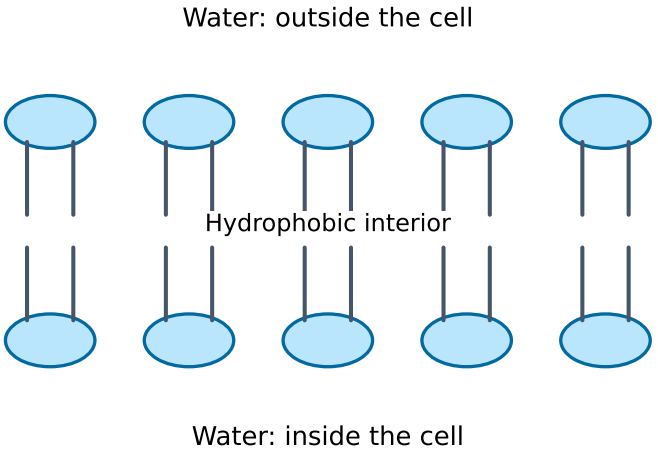
Use invented comparison values of 38 kJ per gram of lipid and 17 kJ per gram of carbohydrate, with all stated energy available. To store 380 kJ requires $380/38 = 10.0$ g lipid or $380/17 = 22.4$ g dry carbohydrate, to three significant figures. If the model assigns 3 g water per gram of carbohydrate store, its hydrated mass is $4(380/17) = 89.4$ g. The 3:1 hydration assumption is supplied for this calculation; it is not a universal measured constant.

This comparison does not establish that carbohydrate stores are useless. Glucose availability, speed of mobilization, oxygen supply and the organism's physiology also influence which fuel is used. Energy density and immediate accessibility answer different questions.

Amphipathic phospholipids form bilayers in water

An amphipathic molecule has both a hydrophilic region and a hydrophobic region. In water, phospholipid heads interact with the aqueous environment while tails associate away from it. A bilayer places heads toward water on both outer surfaces and tails toward each other in the interior. Water can be present inside a cell and outside it; both surfaces need hydrophilic heads.

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Blue heads are hydrophilic; tails point inward.
Schematic cross-section; real membranes contain other components.

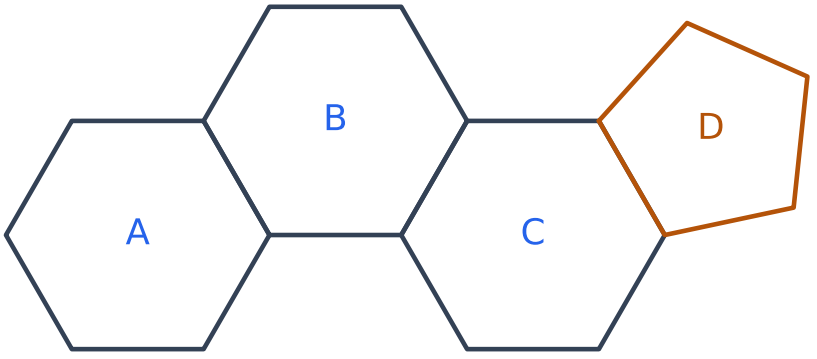
Cross-section through a bilayer: both surfaces meet water. The tails form the hydrophobic interior; this is not a layer with water on only one side.

Bilayer formation follows the molecules' interactions in their environment; it is not purposeful behaviour. The interior creates a barrier to many ions and polar substances. It is not absolutely impermeable to every molecule. Proteins, cholesterol and other components modify real membranes, which are dynamic structures rather than rigid sheets.

Steroid ring structure relates to membrane passage

Steroids have a characteristic skeleton of four fused carbon rings: three six-membered rings and one five-membered ring. Fused rings share atoms and a bond; four separate circles are not an adequate molecular structure. Side groups and bond arrangements distinguish individual steroids.

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A–C: six atoms each. D: five.

This is a simplified steroid nucleus, not a complete formula of a hormone. Oestradiol and testosterone have this ring framework but different substituents and bonding.

Oestradiol and testosterone are steroid hormones whose overall lipid solubility allows them to pass through the phospholipid bilayer. Their structures still contain oxygen-containing groups; "lipid-soluble" does not mean "made only of carbon and hydrogen". A charged water-soluble molecule faces a different barrier. Recognition by an appropriate receptor is needed for a specific cellular response: membrane passage alone does not tell every cell to respond in the same way.

Practice

All numerical values below are invented teaching data. State the molecular model or assumption used.

1. Explain how carbon can form diverse biological molecules. A carbon is drawn with one double bond and three single bonds. Identify the problem. Convert 2.5 μm to nm and 7 nm to μm.
2. Distinguish a monomer, polymer and residue. Identify the linkage joining sugar residues and the linkage joining amino-acid residues. Why is a triglyceride not a repeating-monomer polymer?
3. A linear carbohydrate chain contains 35 monosaccharide residues. How many glycosidic links join them, and how many water molecules are used in complete hydrolysis? Two separate chains each contain ten residues; how many new links are needed to join them end to end?

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4. A 30-residue linear peptide has its first six residues removed as a signal sequence. How many residues and peptide bonds remain in the mature chain? Distinguish this from the number of peptide bonds in the original chain.
5. A ring diagram contains one oxygen and five carbons within its ring, plus one carbon outside. Is it a pentose or hexose? Explain why counting ring corners alone fails. Distinguish alpha- and beta-D-glucose using the C1 hydroxyl and C5 hydroxymethyl group.
6. Explain three properties that make glucose useful to cells, then explain why storing the same number of glucose units as a polysaccharide can reduce osmotic problems.
7. Compare amylose, amylopectin and glycogen. Explain how coiling, branching and relative insolubility support storage. Distinguish a branch point from a chain end.
8. Explain why cellulose is suited to resisting tension. Identify the covalent links within chains and the interactions between chains. Why can a human digest starch but not cellulose using their own digestive enzymes?
9. Explain how carbohydrate groups on glycoproteins contribute to cell recognition, using ABO as an example. Correct the statement "group O red cells have no surface antigens".
10. State the components and ester-link count of a triglyceride and compare them with a typical glycerophospholipid. Using glycerol 92, three fatty acids each 256, and water 18 in the stated molar-mass units, calculate the triglyceride molar mass and account for the water.
11. Classify fatty-acid chains with zero, one and three C=C bonds. For otherwise comparable cis fatty acids, predict the broad effect of increasing unsaturation on melting point. Give two reasons that source alone cannot identify whether a mixture is solid at a specified temperature.
12. Using 38 kJ per gram of lipid and 17 kJ per gram of dry carbohydrate, calculate the masses needed for 190 kJ. If the carbohydrate store carries 3 g water per gram of dry store, calculate its hydrated mass. Explain how adipose tissue insulates a warm animal.
13. Explain phospholipid bilayer orientation in water on both sides. Identify the steroid ring pattern and give two steroid-hormone examples. Explain why their passage through a bilayer does not imply that ions cross equally easily or that every cell responds identically.

■ Worked answers

1. Carbon forms four covalent bonds and can link into branched or unbranched chains and rings with different side groups. One double plus three single bonds gives bond order five, inconsistent with the usual carbon valence shown here. $2.5\,\mu\text{m} = 2500\,\text{nm}$; $7\,\text{nm} = 0.007\,\mu\text{m}$.

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2. A monomer is a building unit; a polymer contains many linked units; a residue is the incorporated part of a unit. Sugar residues have glycosidic links; amino-acid residues have peptide bonds. A triglyceride combines one glycerol with three fatty acids rather than a long chain of repeating monomers.
3. $35 - 1 = 34$ links; complete hydrolysis consumes 34 waters. The two ten-residue chains initially have $9+9 = 18$ links and need one new link to produce a 20-residue chain with 19 links. Only one new water is released in the final joining model.
4. $30-6 = 24$ residues remain, so the linear mature chain contains $24-1 = 23$ peptide bonds. The original chain contained 29. Removing a connected six-residue segment is not the same as hydrolysing every bond in that segment.
5. Six carbons in total make it a hexose. One ring corner is oxygen, while an external carbon must also be counted. In the usual D-glucose ring representation, alpha has the C1 hydroxyl opposite the C5 hydroxymethyl group; beta has them on the same side. Whole-molecule rotation does not change this relationship.
6. Hydroxyl groups support solubility; dissolved glucose can be transported; it is sufficiently stable for controlled handling and can be oxidized through enzyme-catalysed respiration. Any three explained properties are suitable. A relatively insoluble large polymer produces fewer dissolved particles than many separate glucose molecules and can be retained as granules. This reduces the store's osmotic contribution without abolishing all water movement.
7. Amylose is mainly unbranched and coiled; amylopectin is branched; glycogen is more highly branched. All contain alpha-glucose residues. Coiling and branching compact the store, and branch ends provide sites for mobilization or addition. A branch point is where a side chain begins; a free chain end is the terminus of a chain. Relative insolubility helps retain a store with a smaller osmotic effect than free glucose.
8. Beta-1,4 glycosidic links connect alternating beta-glucose residues into straight chains. Parallel chains form hydrogen-bonded bundles; these collective interactions and covalent backbones provide tensile strength. Human digestive enzymes hydrolyse suitable starch linkages but humans do not produce cellulase for cellulose's beta-1,4 links. Same building unit does not imply same enzyme specificity.
9. Exposed carbohydrate groups have shapes recognized by specific binding molecules. A and B antigens have different terminal carbohydrate structures on surface glycoproteins and glycolipids. Group O lacks A and B terminal modifications, not every surface antigen or every membrane carbohydrate.
10. One glycerol plus three fatty acids gives three ester links; complete ester hydrolysis uses three waters. A typical glycerophospholipid has two fatty-acid tails and a phosphate-containing head. $92 + 3(256) - 3(18) = 806\,\mathrm{g\,mol^{-1}}$. Reactants total 860; triglyceride 806 plus three waters totalling 54 gives 860. These are simplified condensation-accounting quantities.
11. Zero C=C: saturated; one: monounsaturated; three: polyunsaturated. More cis bends generally disrupt packing and lower melting point for comparable chains.

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Chain lengths, cis/trans geometry and mixture composition affect the result. Plant and animal origins are broad tendencies, not fixed molecular compositions; compare the actual temperature with the material's melting range.

12. Lipid mass = 190/38 = 5.00 g; dry carbohydrate = 190/17 = 11.2 g; hydrated mass = 4(190/17) = 44.7 g, to three significant figures. Keep unrounded intermediate values. Adipose tissue reduces heat transfer from a warmer body to colder surroundings. It slows loss rather than generating heat by insulation alone.
13. Hydrophilic heads face water at both surfaces; hydrophobic tails face inward toward one another. A steroid has three fused six-membered rings and one fused five-membered ring. Oestradiol and testosterone are examples. Their overall lipid solubility permits bilayer passage, whereas a charged ion interacts unfavourably with the hydrophobic interior. A response also requires the appropriate receptor and cellular machinery.

Exam tips

Read bond types, carbon counts and side groups before naming a molecule. Distinguish a monomer from its residue, a glycosidic bond from a hydrogen bond, and a storage lipid from an amphipathic membrane lipid. Count links before calculating water or molar mass. Explain a function through structure and a mechanism; avoid claiming that every plant lipid is liquid, every animal lipid solid, or every large molecule a polymer.

Scope reference: IB Biology guide, B1.1. Numerical comparisons and molecular cartoons are original teaching examples.

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B.1.2 · Proteins

International Baccalaureate · IB Diploma · Biology · HL

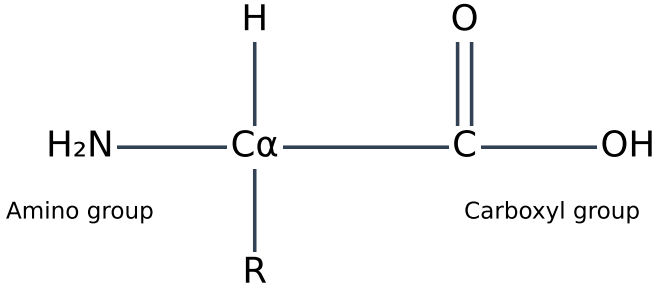
Scope and prerequisites

An egg white becomes opaque and firm when heated. Cooling it does not restore the original liquid. Yet digestion can still break its proteins into smaller molecules. Why are changing a protein's shape and cutting its chain different processes?

This unit covers B1.2 SL understandings plus the additional higher-level content (marked AHL): amino-acid structure, peptide formation, dietary amino acids, sequence variety, denaturation, side-chain chemistry, protein structure and structure–function examples. Prerequisites are covalent bonds, condensation, hydrolysis and interactions with water. Numerical datasets and short letter sequences are invented teaching examples. Sections marked **AHL** are for Higher Level only.

Amino acids have a common framework and a variable side chain

A **protein** contains one or more folded polypeptide chains. The amino acids used to build these chains share a central alpha carbon attached to four groups: an amino group, a carboxyl group, a hydrogen atom and a variable R-group or side chain. In the neutral drawing convention these groups can be shown as $-NH_2$ and $-COOH$. In aqueous solution their charge states depend on pH; a common form has $-NH_3^+$ and $-COO^-$.



Neutral representation; charge states depend on pH.

The R-group varies among amino acids. This neutral structural convention makes the four attachments clear; the amino and carboxyl groups can gain or lose hydrogen ions in solution.

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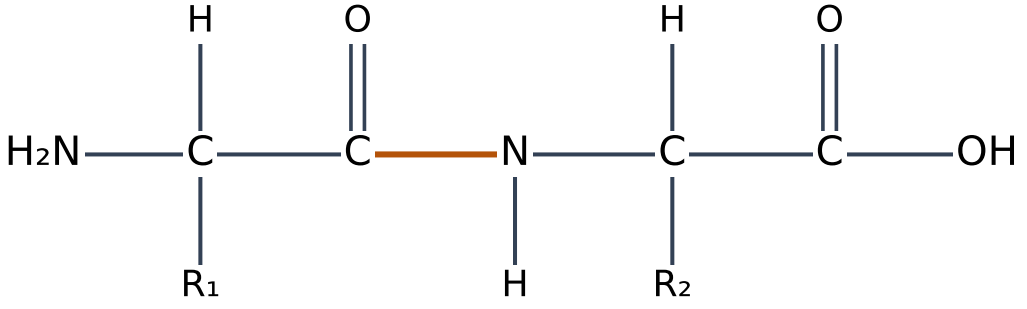
The R-group distinguishes one amino acid from another. The R is a placeholder for a chemical group, not an extra element called "R". Glycine has H as its side chain, so its alpha carbon is attached to two hydrogens. Most other standard amino acids have a more complex side chain. Counting the amino group and carboxyl group as part of R would erase the useful distinction between the shared framework and the variable part.

A good generalized drawing shows all four attachments. For the carboxyl group, show the carbonyl C=O and the hydroxyl –OH attached to the same carbon. An oxygen atom cannot silently replace that carbon. Follow the bonds rather than copying a decorative shape.

Peptide bonds join residues into a chain

In the simple condensation model, the carboxyl group of one amino acid reacts with the amino group of another. A **peptide bond** forms between the carboxyl carbon and amino nitrogen. OH from the carboxyl group and H from the amino group account for one released water molecule. The products are a dipeptide and water:

$$\text{amino acid} + \text{amino acid} \longrightarrow \text{dipeptide} + \text{water}.$$



Orange C–N link: peptide bond.
Two residues; one peptide bond; one water released in the model.

The highlighted carbon–nitrogen link is the peptide bond. A carbonyl C=O remains beside it. The R-groups remain attached to their own alpha carbons.

A residue is the part of an amino acid incorporated into the chain. Repeated linkage produces a polypeptide with a repeating backbone and side chains projecting from it. The amino end and carboxyl end are different, so sequence order has a direction. Hydrolysis uses water to break peptide bonds, producing shorter peptides or free amino acids depending on how many links are cut.

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The condensation equation is an accounting model. Protein synthesis in cells uses ribosomes, tRNAs, activated amino acids and energy-transfer reactions; it is not simply free amino acids spontaneously releasing water in a beaker. The model is useful for identifying the linkage, counting residues and conserving atoms.

■ Worked example: residue count is not bond count

One linear chain of 35 residues has $35 - 1 = 34$ peptide bonds. In the simple model, making the chain from free amino acids releases 34 water molecules; complete peptide-bond hydrolysis uses 34. A 120-residue chain has 119 peptide bonds, not 120.

Two separate chains of 12 and 18 residues contain $11 + 17 = 28$ peptide bonds. Joining their ends by one additional peptide bond gives one 30-residue chain with 29. Merely holding two chains together by other interactions does not create that extra backbone bond. Cross-links between side chains must not be counted as peptide bonds.

Dietary amino acids supply building material

Digestion breaks dietary proteins into smaller products, including amino acids that can be absorbed and used to synthesize the body's own proteins. Eating a particular protein does not mean that intact protein is placed directly into a matching body tissue. Cells use amino-acid supplies according to their own gene expression and metabolism.

An essential amino acid cannot be synthesized by the body in sufficient amounts and therefore must be obtained from food. A non-essential amino acid can be synthesized, for example through reactions involving other amino acids. "Non-essential" does not mean unnecessary: both types are needed for appropriate protein synthesis.

Different foods provide different proportions of amino acids. A high total protein mass alone does not prove that every essential amino acid is supplied adequately. A vegan diet needs attention to the availability and balance of essential amino acids as well as total protein. Diverse plant sources can contribute different amino-acid profiles; "plant proteins contain no essential amino acids" is false.

■ Worked example: identify the limiting supply

In an invented synthesis model, each copy of a short peptide requires two residues of amino acid X and three of Y. A cell has 50 available X residues and 60 Y residues, with every other required resource abundant. X permits $50/2 = 25$ copies; Y permits $60/3 = 20$ copies. Y limits production to 20 complete copies, leaving $50 - 2(20) = 10$ X residues. This is a simplified resource calculation, not a recommended diet or a claim about a real cell.

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Length and sequence create enormous variety

The standard genetic code specifies 20 common amino acids used in polypeptide synthesis. Chains can be short or contain hundreds to thousands of residues. Both the number of residues and their order can vary. The **primary structure** is the amino-acid sequence, read in a specified direction. Two chains can have the same length and amino-acid composition but different sequences.

For example, the invented three-residue sequences A–B–C and A–C–B use the same three types but place them differently. They are not the same primary structure. With three available types, repetition allowed and three positions, there are $3^3 = 27$ possible sequences. With 20 types and four positions, there are $20^4 = 160000$ possibilities. "20 amino acids" does not mean "only 20 proteins". For any fixed length the number is finite; allowing different lengths produces an enormous range of possible chains.

Different sequences can support different structures and functions. Insulin is a protein hormone, collagen is a structural protein, and many enzymes are proteins that catalyse reactions. Not every protein is an enzyme; some catalysts are RNA molecules rather than proteins. A functional protein also depends on appropriate folding and conditions, not just on having a long chain.

Temperature and pH can alter protein structure

Proteins interact with surrounding water and with groups within their own chains. A specific folded arrangement helps create a binding surface, active site or structural role. **Denaturation** is disruption of the normal functional conformation, often with loss of activity. Heating can disturb interactions stabilizing a fold and increase unfolding or aggregation. A pH change can alter charges and interactions. The effect depends on the protein, conditions and duration of exposure.

Ordinary denaturation does not require hydrolysis of the peptide backbone. A protein can lose its working shape while retaining its amino-acid sequence. Digestion, by contrast, cuts peptide bonds. This distinction does not mean every bond survives every extreme chemical treatment: severe conditions can cause additional damage. Explain the process actually being tested.

Some proteins can regain a functional conformation after suitable conditions are restored. Others aggregate or undergo changes that prevent recovery. Heating egg-white proteins causes unfolding and association into an opaque network; ordinary cooling does not reverse the cooked structure. "Every denaturation is reversible" and "no protein can ever refold" are both too strong.

■ Worked example: test recovery under the same assay conditions

An invented experiment exposes equal enzyme samples to different temperatures for five minutes. Every sample is then assayed at 25°C, at the same pH and substrate concentration. Relative activities are:

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Pre-treatment	Activity after return to 25°C
Kept at 25°C	100 units
Heated to 45°C	87 units
Heated to 65°C	20 units
Kept cold at 5°C	98 units

The 65°C sample retains $20/100 \times 100 = 20\%$ of control activity, an 80% loss. Because all assays take place at the same temperature, the result is not explained simply by particles moving faster or slower during the assay. It supports lasting damage to function after the hot treatment. Activity alone does not reveal the complete molecular structure or prove which interaction changed. Repeats, an unheated control and checks for evaporation or changed concentration strengthen the comparison.

A low reaction rate while an enzyme is cold would not by itself establish denaturation. The recovery test asks whether function returns after the original conditions are restored. Keep treatment conditions separate from measurement conditions.

AHL: R-groups influence folding and interactions

Side chains differ in polarity, charge, size and bonding possibilities. Hydrophobic side chains are largely non-polar. Hydrophilic side chains can be polar without a net charge or carry a positive or negative charge. Acidic side-chain groups can lose a hydrogen ion and become negatively charged; basic groups can bind a hydrogen ion and become positively charged. Their charge states depend on pH, so a change in pH can alter interactions without changing the amino-acid sequence.

The sequence fixes which side chains occur at which positions. Interactions among them, the backbone and the environment constrain the conformation. Many functional proteins adopt reproducible structures under appropriate conditions, but they are not rigid statues: movement and conformational changes can be part of function. Folding may require cellular assistance, and an isolated chain is not guaranteed to fold correctly in every environment.

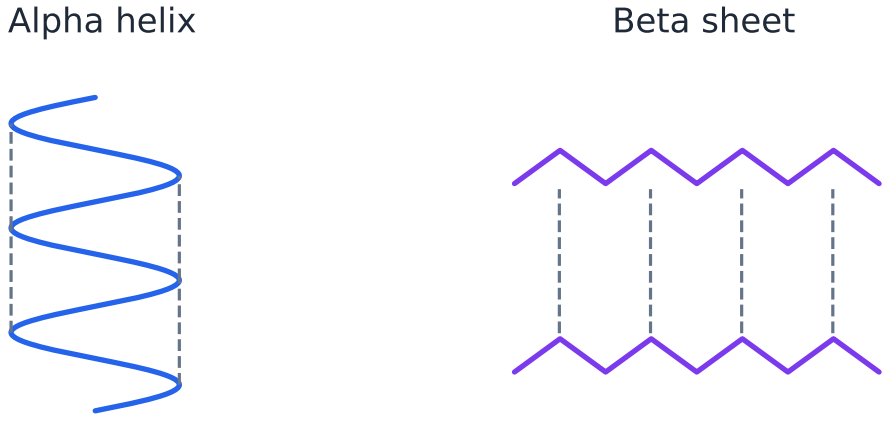
A substitution replacing one side chain with another can change packing, charge, binding or stability. Its effect depends on the position and chemical difference. One substitution can have a large effect, a small effect or little measurable effect in a chosen assay; it is not valid to predict complete loss of function from "one amino acid changed" alone.

AHL: Secondary structure uses regular backbone hydrogen bonds

Alpha helices and beta-pleated sheets are recurring local arrangements of a polypeptide backbone. Hydrogen bonds between backbone carbonyl oxygens and amide hydrogens stabilize these structures. An alpha helix coils one segment of chain, with regular hydrogen bonding within that helix. A beta sheet contains extended strands held

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together by hydrogen bonds; the strands can come from different regions of one chain or from different chains.



Solid: backbone paths. Dashed: hydrogen bonds.
Schematic organization; backbone atoms omitted.

Solid curves represent backbone paths; dashed lines represent stabilizing hydrogen bonds. This is an organizational cartoon, not an atom-by-atom bond diagram.

These hydrogen bonds are not the covalent peptide bonds that connect successive residues. Nor are they simply a line of R-group-to-R-group bonds. The sequence and side chains influence which secondary arrangements are favoured, but the regular stabilizing pattern shown here concerns backbone groups.

AHL: Tertiary structure depends on several interactions

Tertiary structure is the overall three-dimensional arrangement of one polypeptide chain, including how its helices, sheets and other regions pack together. Several interactions contribute:

Interaction	Structural contribution
Hydrogen bonds	Attractions involving suitable polar groups
Ionic interactions	Attractions between oppositely charged groups
Hydrophobic interactions	Non-polar groups tend to cluster away from water
Disulfide bonds	Covalent S–S cross-links formed between suitable cysteine side chains

A disulfide bond is covalent, not a weak hydrogen bond. It can connect different regions of one chain or connect different chains. Side-chain cross-linking does not turn the ordinary polypeptide backbone into a branching tree of peptide bonds. Keep the

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bond type and the structural level distinct.

In a water-soluble globular protein, many hydrophobic side chains cluster in the interior while hydrophilic groups interact with water at the surface. This is a broad pattern, not a claim that every surface residue is charged or every internal atom non-polar. A membrane-spanning region of an integral protein presents hydrophobic side chains toward the lipid tails. A water-filled pore can instead have hydrophilic groups facing its lumen. The same "hydrophobic groups always face inward" rule cannot describe both environments.

AHL: Quaternary structure and protein examples

Quaternary structure is the arrangement and association of two or more polypeptide chains. Each chain has its own sequence and conformation. A protein consisting of one folded chain can have tertiary structure without quaternary structure. A non-protein group does not count as an extra polypeptide chain.

A conjugated protein includes a non-protein component needed for its normal function, such as a prosthetic group. A non-conjugated protein lacks such an associated prosthetic component in this classification. "Non-conjugated" does not mean "one chain", "unfolded" or "contains no covalent cross-links".

■ Insulin: a compact signal with two chains

Mature human insulin contains an A chain of 21 residues and a B chain of 30 residues. Disulfide bonds stabilize the structure, including links between the chains. Its compact globular form suits transport in aqueous blood and specific binding to receptors. It is a non-conjugated protein in this syllabus comparison: the mature hormone does not require a haem-like prosthetic group to make its two-chain structure. Zinc-associated storage assemblies are a separate level of organization, not a reason to call each A–B pair a six-chain hormone.

The two chains contain $(21 - 1) + (30 - 1) = 49$ peptide bonds. They do not contain 50 merely because $21 + 30 = 51$ residues are present. The peptide-bond rule must be applied to each separate chain. The RCSB insulin structure resource shows the two-chain organization and distinguishes it from the precursor.

■ Collagen: a fibrous structure that resists tension

A collagen molecule contains three polypeptide chains wound into a triple helix. Repeating sequence features, including glycine at every third position in the triple-helical region, allow close packing. Associations and cross-links in collagen fibrils help create strong structures that resist pulling forces. This suits collagen's role in tendons, skin and other supporting tissues. A collagen triple helix is not simply an alpha helix renamed because it contains three chains.

Collagen is the fibrous, non-conjugated example in the syllabus comparison. Its elongated structure serves a mechanical role; it does not need to be a compact soluble messenger like insulin. Chemical modifications of residues, such as hydroxylation,

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must not automatically be confused with adding a separate prosthetic group. The RCSB collagen account explains the three-chain structure.

■ Haemoglobin: globular chains plus haem

The main adult human haemoglobin has two alpha and two beta globin chains. Each chain carries a non-protein haem group containing iron that can bind one oxygen molecule reversibly. Four haem groups therefore give a maximum of four bound oxygen molecules per haemoglobin molecule. The protein is conjugated, and its four-chain association is quaternary structure. The four haem groups do not make it an eight-polypeptide protein.

Its soluble globular form supports transport in the red-cell cytoplasm, while interactions between subunits help relate oxygen binding at one site to behaviour at others. Haem provides a binding component that the amino-acid chain alone does not supply. The RCSB haemoglobin resource connects structural observations with oxygen binding.

Protein structures can be studied with methods including X-ray crystallography and cryogenic electron microscopy. These techniques use measurements and reconstruction to reveal details invisible to the unaided eye. A ribbon cartoon summarizes a structural model; it is not an ordinary light-microscope photograph, and a two-dimensional drawing does not mean the molecule is flat.

Practice

Numerical supplies, activity data and short letter sequences below are invented. Use the simple condensation-accounting model where specified.

1. Draw a generalized amino acid. Label the alpha carbon, hydrogen, amino group, carboxyl group and R-group. Explain what R represents and why pH can change the charge of the amino and carboxyl groups.
2. Write the word equation for forming a dipeptide. Identify the peptide bond, the groups supplying the atoms of water, and the groups that remain attached to each alpha carbon.
3. A linear polypeptide contains 120 residues. Calculate its peptide bonds and the water molecules released in the simple formation model. How many waters are needed for complete peptide-bond hydrolysis?
4. Two linear chains contain 12 and 18 residues. Count their peptide bonds before and after joining their ends by one peptide bond. Distinguish this from holding the two chains together without a new backbone link.
5. Define essential and non-essential amino acids. Explain why total protein mass alone does not establish the adequacy of every essential amino acid, including in a vegan diet.

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6. A synthesis model requires two X and three Y residues per peptide. Available supplies are 50 X and 60 Y. Calculate the maximum complete peptides and unused X. State one limitation of this model.
7. Using three permitted amino-acid types, repetition allowed, calculate the number of four-position sequences. Explain why same length and composition do not necessarily mean same primary structure.
8. Distinguish denaturation from peptide-bond hydrolysis. Explain why a cooked egg does not regain its initial liquid structure merely on cooling, without claiming that all proteins can never refold.
9. Samples assayed at 25°C after treatment give activities 100, 87, 20 and 98 units after 25°C, 45°C, 65°C and 5°C treatments respectively. Calculate retained activity and percentage loss for the 65°C sample. Explain why assay temperature matters.
10. Design a comparison to test whether low activity after heating persists on cooling. Give four controlled variables and one source of uncertainty or alternative explanation.
11. Correct these claims: every protein is an enzyme; all catalysts are proteins; a low rate while an enzyme is cold proves denaturation; non-essential amino acids are unnecessary.
12. **AHL** Compare alpha helices, beta sheets and tertiary structure. Locate the regular hydrogen bonds in secondary structure. Explain how pH, ionic interactions and cysteine disulfide bonds can affect a fold.
13. **AHL** Compare the likely locations of hydrophobic and hydrophilic side chains in a water-soluble globular protein and a membrane-spanning protein with a water-filled pore. Explain why a substitution's effect depends on position and chemistry.
14. **AHL** Compare insulin, collagen and adult human haemoglobin in shape, chain organization, conjugation and function. Calculate the peptide bonds in insulin's 21- and 30-residue chains and the maximum oxygen molecules bound by one four-haem haemoglobin. Explain why neither haem groups nor a ribbon drawing should be counted as extra chains.

■ Worked answers

1. The alpha carbon has four attachments: H, R, an amino group and a carboxyl group. The carboxyl carbon is bonded to O by a double bond and to OH by a single bond in the neutral representation. R is the variable side chain, not an element. Binding or loss of hydrogen ions changes charge; a common aqueous form has an ammonium and a carboxylate group.
2. Amino acid + amino acid → dipeptide + water. The new C–N link connects a carboxyl carbon with an amino nitrogen. OH from the first carboxyl and H from the second amino group account for water. Each residue retains its own side chain on its alpha carbon. The product still contains a carbonyl beside the peptide C–N link.

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3. $120 - 1 = 119$ peptide bonds; formation from free amino acids releases 119 waters in the simple model. Complete hydrolysis uses 119 waters. Cutting only some bonds produces shorter peptides, not necessarily free monomers.
4. Initially $(12 - 1) + (18 - 1) = 11 + 17 = 28$ bonds. One new end-to-end peptide bond gives 29 in the 30-residue chain and releases one additional water in the model. Association without that new backbone bond leaves two chains and 28 peptide bonds.
5. Essential amino acids must come from food because synthesis is insufficient; non-essential ones can be synthesized but are still needed. Amino-acid proportions differ among foods. Total protein mass does not identify the supply of each required essential type. Vegan diets therefore need attention to amino-acid balance and supply; plant sources do contain essential amino acids.
6. X permits 25 copies; Y permits 20, so the maximum is 20. X used $= 2(20) = 40$; X left $= 50 - 40 = 10$. Real synthesis also depends on other amino acids, energy, enzymes and regulation, which the model treats as non-limiting.
7. $3^4 = 81$ possible sequences. With the same composition, A–B–C–A and A–A–B–C have different orders and thus different primary structures when read in the same direction. Neither length nor composition alone specifies all positions.
8. Denaturation changes functional conformation and can leave the peptide backbone intact. Hydrolysis cuts peptide bonds using water. Heated egg-white proteins unfold and associate into an altered network that ordinary cooling does not undo. Some other proteins can refold under suitable conditions, so irreversibility must be tied to the example and conditions.
9. Retained activity $= 20/100 \times 100 = 20\%$; loss $= (100 - 20)/100 \times 100 = 80\%$. All assays at 25°C allow comparison after treatment instead of confusing pretreatment with an immediate temperature effect during measurement. The result supports persistent functional damage but does not identify a complete molecular mechanism alone.
10. Heat equal samples for a fixed time, return them and an unheated control to the same assay conditions, then compare activity with repeats. Control enzyme concentration/volume, treatment duration, assay temperature, pH, substrate concentration and measurement interval; any four appropriate controls suffice. Evaporation, concentration errors or unequal timing could alter results and should be checked.
11. Proteins include structural proteins and hormones as well as enzymes. Some catalytic RNAs are not proteins. Cold conditions can reversibly slow a reaction; test recovery after restoring conditions. Non-essential means synthesizable, not dispensable.
12. **AHL** An alpha helix is a coiled local backbone arrangement; a beta sheet contains extended strands. Regular backbone C=O $\cdots$ H–N hydrogen bonding stabilizes both. Tertiary structure describes the overall fold of one chain. A pH change can alter side-chain protonation and ionic attractions; hydrogen bonding and hy-

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drophobic interactions also contribute. A disulfide bond is a covalent S–S link between cysteine side chains, not a peptide bond or a weak hydrogen bond.

13. **AHL** A water-soluble globular protein often has hydrophobic groups toward its core and hydrophilic groups exposed to water. A membrane-spanning surface facing lipid tails is hydrophobic, while a water-filled pore can present hydrophilic groups inward. Thus environment determines which surface contacts water. A substitution at a buried packing site, binding site or lipid-facing region can have different effects; not every substitution destroys function.
14. **AHL** Insulin: compact globular hormone, two chains, non-conjugated in the stated comparison, specific receptor binding. Collagen: three-chain triple helix, fibrous, non-conjugated, suited to resisting tension in supporting tissues. Adult haemoglobin: four globin chains and four haem groups, globular and conjugated, reversible oxygen binding. Insulin has $(21 - 1) + (30 - 1) = 49$ peptide bonds, not 50. Four haem groups bind at most four oxygen molecules, not eight oxygen molecules because oxygen is diatomic. Haem is a non-protein component; a ribbon is a way of displaying a structural model, not an extra molecule or a flat physical chain.

Exam tips

Identify which carbon and nitrogen make a peptide link, and retain the carbonyl oxygen in a dipeptide drawing. State the number of separate chains before using residues minus chains to count peptide bonds. Separate treatment conditions from assay conditions, and distinguish loss of a functional fold from cutting a backbone. In a structure–function explanation, connect a specific feature to the interaction or mechanical property it permits.

- **AHL:** Distinguish backbone hydrogen bonds, side-chain interactions and covalent disulfide bonds. A hydrophobic surface can face membrane lipids rather than a protein's core. Count polypeptides separately from prosthetic groups and use the appropriate example for each level of structure.

Scope reference: IB Biology guide, B1.2. Structural examples are linked in their teaching sections; invented data are not measurements from those sources.

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Membranes and membrane transport

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A potato cylinder gains mass in one solution and loses mass in another. A nerve cell admits an ion through one membrane protein but uses energy to export ions through another. The same thin boundary must act as a barrier, permit controlled exchange and respond to signals. How can one structure do all three?

This unit covers B2.1 SL understandings plus additional higher-level content (marked AHL): membrane structure, simple and facilitated diffusion, osmosis, active transport, surface carbohydrates, fluidity, vesicles, gated channels, sodium–potassium pumps, sodium-dependent glucose cotransport and cell adhesion. Prerequisites are water polarity, phospholipid structure and protein interactions. All numerical datasets are invented teaching examples. Sections marked **AHL** are for Higher Level only.

A phospholipid bilayer separates two aqueous environments

Phospholipids are amphipathic: a hydrophilic head interacts with water, while hydrophobic hydrocarbon tails tend to associate away from it. In a bilayer, heads face water on both surfaces and tails form the interior. A cell has water in its cytoplasm as well as outside it. A diagram with heads exposed to water on only one side misses this arrangement.

The hydrophobic core presents a barrier to many hydrophilic particles, especially ions and large polar molecules. Small non-polar molecules generally cross more readily. This difference gives the membrane a boundary function without making it completely impermeable. A cell wall is a separate structure and does not replace this selectively permeable boundary.

Membranes form continuous sheets that can close around compartments. They also contain proteins, carbohydrates and, in animal cells, cholesterol, with different functions.

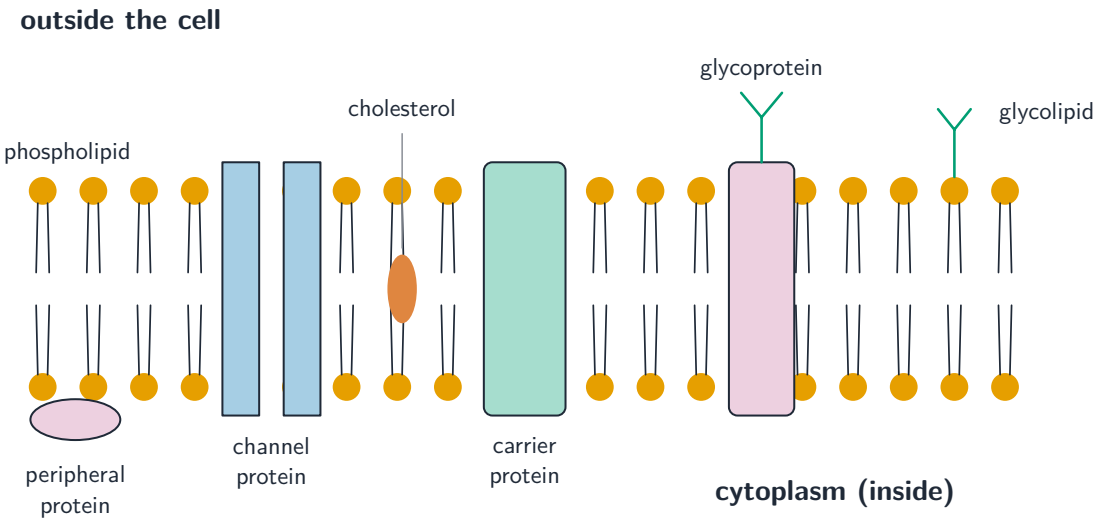
Read and draw the fluid mosaic model

“Fluid” describes the movement of many lipids and proteins within the membrane plane. “Mosaic” describes the variety and distribution of components. The membrane is a dynamic structure, not a rigid row of identical tiles. Movement can be restricted by attachments, so the model does not imply every protein drifts freely everywhere.

An integral protein is embedded in one or both lipid layers; a transmembrane protein spans the bilayer. A peripheral protein attaches at a membrane surface rather than extending through the hydrophobic core. Proteins can form channels, carriers, pumps, receptors, enzymes or attachment sites. Location and molecular shape help explain

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their role; a rectangle in a drawing does not establish its function on its own.



Carbohydrate branches face the extracellular side. Heads contact water; tails lie inside. Protein shapes are schematic. Cholesterol sits among the lipid tails, while a peripheral protein attaches at a surface.

To draw the model, label both aqueous sides. Add two rows of phospholipids, tails inward, integral and peripheral proteins, external carbohydrate and cholesterol. Label hydrophilic and hydrophobic regions; leave the channel pore clear.

Simple diffusion follows random movement and a gradient

Diffusion is the net movement of particles from higher to lower concentration due to their random motion. Individual particles can move in either direction; the net transfer reflects unequal numbers moving across a boundary in each direction. At equilibrium particles continue moving, although there is no net concentration-driven transfer.

Oxygen and carbon dioxide can move between phospholipids by simple diffusion. No transport protein or direct ATP use is needed for each crossing. Net direction depends on the concentration gradient. If oxygen concentration is higher outside than inside, net entry is expected under otherwise comparable conditions; reverse the gradient and net movement reverses. The membrane does not permanently label oxygen as "incoming".

Diffusion rate depends on permeability, concentration gradient, area, distance and temperature. A gradient cannot drive passage through an impermeable barrier. Simple diffusion depends on particle size and polarity rather than specific carrier binding.

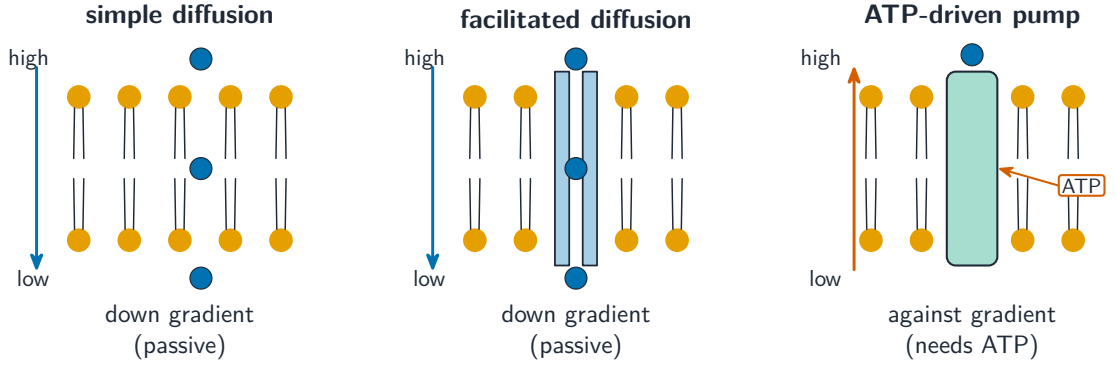
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Channel proteins permit facilitated diffusion

A channel protein provides a hydrophilic pathway through the hydrophobic core. Suitable ions can diffuse through an open channel, while its shape and chemical environment help determine which particles can pass. A potassium channel is not simply a wide hole that admits every particle smaller than a potassium ion.

Facilitated diffusion uses a membrane protein but does not move the transported substance uphill by spending ATP. For an uncharged solute, net movement follows its concentration gradient. For an ion, both concentration difference and electrical attraction or repulsion matter; together these form an electrochemical gradient. An open channel does not automatically mean net entry, and a closed channel does not imply the concentration gradient has disappeared.

Some facilitated transport uses carriers that bind a solute and change conformation instead of forming an open pore. A carrier can also form part of an active transport system, so "carrier" and "active" are not synonyms. Identify energy coupling and net direction, not only the presence of a protein.



The pump panel is an ATP-driven example. Protein-mediated transport is not automatically active, and the same blue symbol is a generic particle rather than the same chemical in every panel.

Osmosis is net water movement through a selectively permeable membrane

Osmosis is net water movement through a selectively permeable membrane in response to a difference in water potential. For solutions at equal pressure, with a solute that cannot cross, net water movement is generally from the more dilute solution toward the more concentrated one. Pressure differences can oppose this tendency, so "toward more solute" is not a complete rule for every situation.

Water molecules move randomly in both directions. A membrane can be permeable to water while restricting a dissolved solute. Aquaporins are channel proteins that allow rapid water movement; they do not pump water using ATP. Some water can cross the

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lipid bilayer directly, but aquaporins greatly increase permeability in many cells.

At no net water movement, crossings can balance rather than stop. A plant cell wall can resist expansion and help generate pressure as water enters. An animal cell lacks that wall, so its response to water entry differs. A whole-tissue mass measurement is evidence about the sample under its experimental conditions; it is not a direct movie of every membrane crossing.

Pumps use energy to maintain unequal distributions

Active transport couples movement of a substance to an energy source and can move it against its gradient. An ATP-driven pump binds particular particles and changes conformation through an energy-coupled cycle. It is not an open channel through which particles simply fall downhill.

Maintaining a concentration difference requires continuing work when diffusion or other transport tends to dissipate it. A cell can therefore consume ATP while an average concentration appears constant: input and loss can balance. Constant concentration is not proof that transport has stopped.

Specific channels and carriers help make a membrane selectively permeable. A membrane may pass one ion rapidly through an open channel, restrict another, and actively transport a third. Changes in channel opening, protein number or energy supply can change the overall permeability and net transfer. “Small molecules always enter” and “large molecules can never cross a membrane” are inadequate generalizations.

Surface carbohydrates support recognition and adhesion

A glycoprotein has carbohydrate groups linked to a protein; a glycolipid has carbohydrate linked to a lipid. Their carbohydrate regions project from the extracellular surface of the plasma membrane. These arrangements help create specific recognition features and can contribute to cell adhesion.

Recognition can involve a receptor binding a particular surface structure. Adhesion involves attachment between cells or between a cell and surrounding material. These are related roles but not identical. A membrane protein used for attachment need not form a transport channel. The external carbohydrate arrangement is part of membrane asymmetry: the two faces have different components and environments.

Test osmosis with a fair potato experiment

Use similar potato tissue and cylinders of equal dimensions, place them in measured concentrations of a suitable solution for a fixed time, then measure initial and final masses. A **control variable** is a factor kept constant so that the comparison can be interpreted. Control temperature, immersion time, solution volume, cylinder dimensions, tissue source and blotting method. Independent repeats help estimate variation.

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Blot each sample consistently before weighing. Liquid left on the surface adds mass without showing that water entered cells. A miscalibrated balance or wrongly prepared solution can bias every repeat; repetition alone does not correct a systematic error. State the assumptions that membranes remain sufficiently intact and that short-term mass changes mainly reflect water transfer rather than substantial solute uptake or tissue loss.

$$\text{percentage mass change} = \frac{\text{final mass} - \text{initial mass}}{\text{initial mass}} \times 100.$$

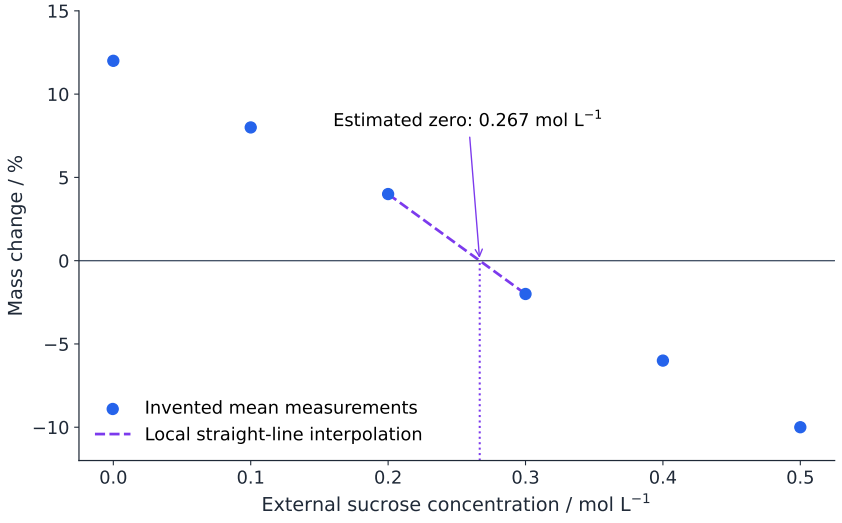
■ Worked example: compare changes relative to the starting mass

A cylinder changes from 2.50 g to 2.75 g. Its change is +0.25 g, so percentage change is $0.25/2.50 \times 100 = +10\%$. A 4.00 g cylinder ending at 3.60 g changes by $-0.40/4.00 \times 100 = -10\%$. A 6.40 g cylinder ending at 8.00 g gives $1.60/6.40 \times 100 = +25\%$.

Raw mass change can mislead when starting masses differ. Sample A gains 0.25 g from 2.50 g: +10%. Sample B gains 0.40 g from 8.00 g: +5%. B gains more grams, but A has the larger proportional gain. Percentage change allows this comparison; it does not correct every difference in tissue properties or surface area.

■ Worked example: estimate a zero crossing rather than invent an exact value

Invented mean percentage changes at external concentrations of 0.00, 0.10, 0.20, 0.30, 0.40 and 0.50 mol per litre are +12, +8, +4, −2, −6 and −10 respectively. Use comparable repeats at each concentration.



The dashed interpolation joins the neighbouring means around zero. It estimates a no-net-change concentration; the plotted data are invented, not measurements from the school laboratory.

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The zero lies between +4% at 0.20 and −2% at 0.30. With a local linear approximation, the fraction of the interval is $4/(4+2) = 2/3$. The estimate is $0.20+(2/3)(0.10) = 0.267$ mol per litre, to three significant figures. Do not select 0.30 simply because it is one of the tested concentrations, or divide by the final mass instead of the initial mass.

A zero-change concentration estimates the external condition at which the tissue has no net mass change over the observation period. It does not prove every cell has exactly that solute concentration, remove pressure effects, or show that water molecules stop moving. More measurements near the crossing and error estimates improve the inference.

AHL: Lipid composition and cholesterol modify fluidity

Cis-unsaturated fatty-acid tails contain bends that tend to reduce close packing. Compared with otherwise similar saturated tails, they often help maintain fluidity at lower temperature. Longer and more saturated chains tend to pack more closely and have higher melting ranges. Chain composition is therefore relevant to a cell's thermal environment.

For a real example, *Escherichia coli* alters membrane lipid composition with growth temperature. Classic experiments found increased proportions of saturated and longer-chain fatty acids at higher temperatures. This primary study of membrane adaptation supports a mechanism for maintaining membrane properties as conditions change. It does not show that every species makes exactly the same adjustment or that a cell can instantly survive any temperature shift.

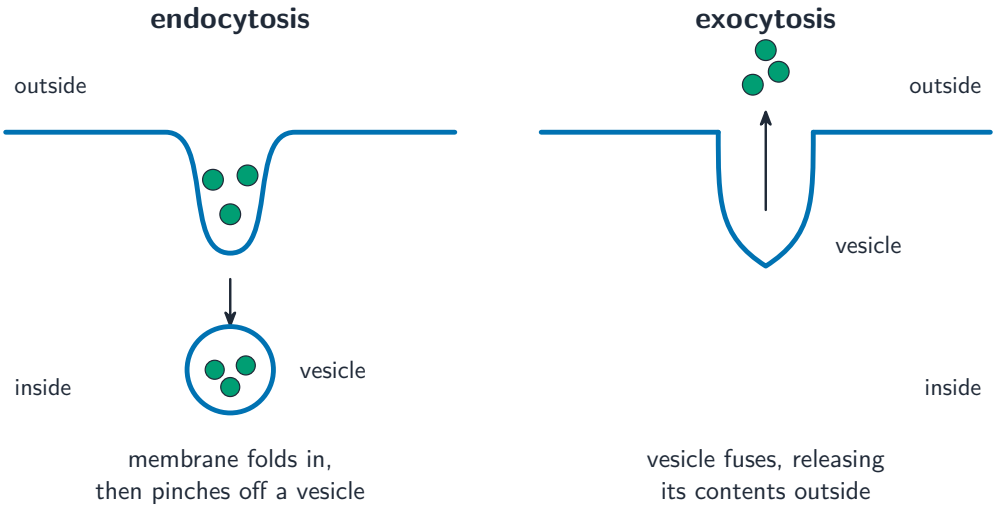
Cholesterol lies among phospholipid tails in animal membranes, with its small polar hydroxyl group near the head region. Its rigid ring region restricts some lipid movements at higher temperatures, while disrupting tight packing helps prevent stiffening at lower temperatures. It acts as a fluidity buffer rather than always increasing or always decreasing fluidity. A change in composition must be interpreted at the stated temperature and in the stated membrane.

AHL: Fluid membranes form and fuse vesicles

Membrane components can rearrange during budding and fusion. In endocytosis, the plasma membrane encloses material and pinches off a membrane-bounded vesicle into the cell. Phagocytosis of a particle by a suitable immune cell is an example. The enclosed material enters a compartment; it has not necessarily dissolved directly into the cytosol.

In exocytosis, a vesicle fuses with the plasma membrane and releases its contents outside. Secretion of a protein hormone such as insulin is an example. Material can leave without passing through the hydrophobic core as individual molecules. Vesicle transport is not the same process as simple diffusion or passage through an aquaporin.

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The boundary line represents a membrane bilayer. Follow the labelled inside and outside, the enclosed cargo and the direction of each process.

AHL: Gated channels respond to different signals

A gated channel changes between conducting and non-conducting states in response to a stimulus. A neurotransmitter-gated channel responds to chemical binding; a voltage-gated channel responds to a change in membrane potential. Opening is different from pumping: ions still move according to their electrochemical gradients.

The nicotinic acetylcholine receptor is a neurotransmitter-gated cation channel. Acetylcholine binding can open it; at a typical negative resting potential, the combined cation movements produce a net inward positive current and depolarization. It is not simply a sodium-only ATP pump. The IUPHAR/BPS channel classification distinguishes these ligand-gated cation channels from other receptor types.

Voltage-gated sodium and potassium channels in neurons respond to voltage rather than acetylcholine binding. Their opening and closing contribute to changes in membrane potential. The opening of one channel does not consume one ATP for every ion that crosses. Energy is needed elsewhere to maintain the gradients that channel currents use.

AHL: Sodium–potassium pumps maintain ion gradients

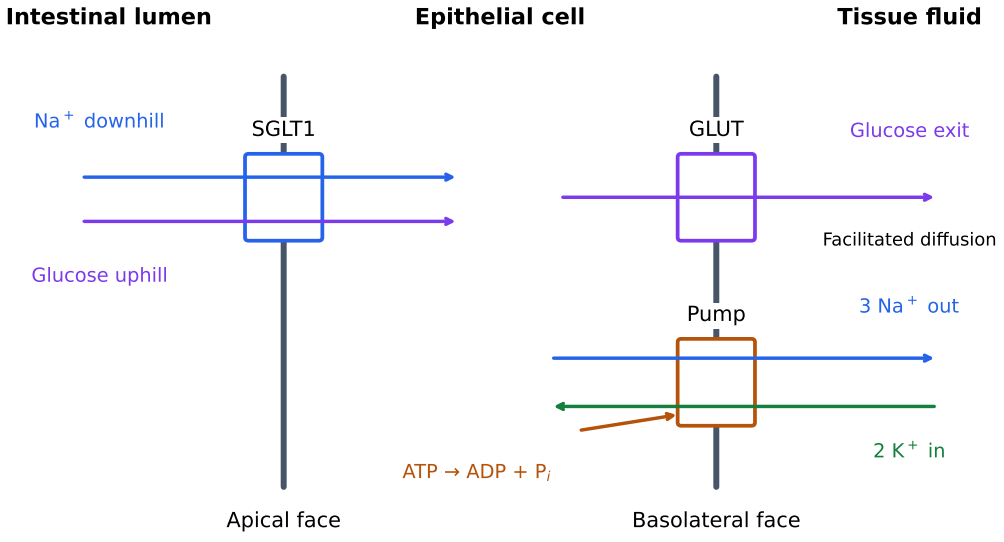
The sodium–potassium pump is an exchange transporter that uses ATP directly. In its usual cycle, three sodium ions move out of the cell and two potassium ions move in for each ATP hydrolysed. Binding, phosphorylation, changes in protein conformation and phosphate release link these movements. The pump does not simply open a pore connecting both sides.

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This maintains relatively low intracellular sodium and high intracellular potassium. Because three positive charges leave while two enter, each complete cycle transfers one net positive charge outward. The gradients and the membrane's selective permeability contribute to membrane potential. The pump helps maintain the conditions for electrical signalling; it is not the rapid voltage-gated sodium current that produces an action-potential upstroke.

In an invented count of eight complete pump cycles, sodium export is $8 \times 3 = 24$ ions, potassium import $8 \times 2 = 16$ ions, ATP use eight molecules, and net outward positive-charge transfer eight elementary charges. These counts describe the stated complete cycles, not all ion movements in a real neuron.

AHL: Sodium-dependent glucose cotransport uses a stored gradient



The pump maintains the Na^+ gradient; SGLT1 does not itself hydrolyse ATP.

Arrows identify the transported substances and membrane face. The cotransporter uses the sodium gradient; the sodium–potassium pump uses ATP directly. This is a pathway schematic, not a full cell drawing.

A sodium-dependent glucose cotransporter binds sodium and glucose and moves them in the same overall direction across a membrane. Downhill sodium movement can drive glucose uptake against its concentration gradient. The cotransporter does not hydrolyse ATP directly, but the sodium gradient depends on ATP-driven pumping. This is indirect or secondary active transport, not energy-free uphill diffusion.

In intestinal epithelial cells, an apical cotransporter takes sodium and glucose from the gut lumen into the cell. A basolateral sodium–potassium pump exports sodium toward the tissue fluid, maintaining the sodium gradient. A separate basolateral glucose

carrier permits facilitated diffusion toward the tissue fluid when the glucose gradient favours that direction, allowing onward transfer to the blood. The membrane faces have different proteins and roles.

A related principle supports glucose reabsorption from the filtrate by epithelial cells of the kidney's proximal tubule. Do not say glucose is pumped into urine during reabsorption: the overall process recovers it from filtrate toward blood. Different sodium–glucose transporter types have different coupling ratios. If a question supplies two sodium ions per glucose for an intestinal SGLT1 model, use that ratio; do not impose it on every nephron transporter.

The cell-biology transport account explains why polarized epithelial transport depends on different carriers and pumps on different surfaces. Removing ATP supply can eventually impair cotransport by dissipating its driving gradient, even though the cotransporter itself is not an ATPase.

AHL: Cell-adhesion molecules help organize tissues

Cell-adhesion molecules, or CAMs, are membrane-associated molecules that contribute to specific attachments. Different forms participate in different cell contacts, helping cells remain organized into tissues. Some contacts provide mechanical linkage; specialized junctions can also create barriers or permit communication. These functions require appropriate molecular arrangements, not simply two bilayers touching.

Adhesion is not synonymous with recognition or transport. A CAM can help attach cells without forming an ion channel. An epithelial tissue with damaged contacts may fail as a barrier even if individual cells still have phospholipid membranes. At this level, connect the type of contact and its tissue role without memorizing every junction protein.

Practice

All numerical data are invented. State assumptions about pressure, membrane permeability and coupling ratios where needed.

1. Explain bilayer orientation in water on both sides and why its interior restricts many ions but permits oxygen more readily. Distinguish the membrane from a cell wall.
2. Draw a labelled fluid mosaic model with phospholipids, integral and peripheral proteins, glycoprotein and cholesterol. Identify hydrophilic and hydrophobic regions and the external side of the carbohydrate chain.
3. Explain net oxygen entry when its concentration is higher outside. What changes if the gradient reverses, and what continues at equilibrium? Why does simple diffusion not need a specific carrier?

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4. Explain how a selective open channel enables facilitated diffusion. Distinguish concentration and electrochemical gradients, and explain why channel opening does not guarantee net entry of an ion.
5. Define osmosis and explain aquaporin function. For equal-pressure solutions separated by a membrane that passes water but not the stated solute, predict the net water direction and explain zero net movement.
6. Compare an ATP-driven pump, an open channel and a facilitated-diffusion carrier. Explain why constant concentration does not prove that no energy-consuming transport occurs.
7. Compare glycoproteins and glycolipids. State which side of the plasma membrane their carbohydrate groups face and distinguish recognition from adhesion.
8. Calculate percentage mass changes for $2.50 \rightarrow 2.75$ g, $4.00 \rightarrow 3.60$ g and $6.40 \rightarrow 8.00$ g. Compare a 0.25 g gain from 2.50 g with a 0.40 g gain from 8.00 g.
9. Invented mean changes are $+4\%$ at 0.20 mol per litre and -2% at 0.30 mol per litre. Estimate the zero crossing by linear interpolation. Explain two limits of interpreting it as an exact intracellular concentration.
10. Plan a fair potato-cylinder investigation. Give five control variables, explain blotting, and distinguish a benefit of independent repeats from a systematic error that repeats cannot remove.
11. A solute enters through a protein while its concentration is lower inside than outside. Does that observation alone establish active transport? Propose evidence needed to distinguish passive movement from energy-coupled transport and give one experimental caution.
12. **AHL** Explain how cis unsaturation and cholesterol affect fluidity at different temperatures. Use the *E. coli* example to explain a temperature-related change without generalizing it to all organisms.
13. **AHL** Compare endocytosis and exocytosis with examples. Distinguish neurotransmitter-gated and voltage-gated channels, using the nicotinic acetylcholine receptor and sodium/potassium channels as examples. Explain why these channels are not ATP pumps.
14. **AHL** For eight complete sodium–potassium pump cycles, calculate sodium export, potassium import, ATP use and net outward charge transfer. Explain intestinal and kidney glucose cotransport and its indirect energy source. If a supplied model uses two sodium ions per glucose, how many sodium ions accompany 25 glucose molecules? Explain one distinct role for CAMs in the epithelial tissue.

■ Worked answers

1. Hydrophilic heads face the aqueous exterior and cytoplasm; hydrophobic tails point inward. Charged ions interact unfavourably with the non-polar core, whereas small non-polar oxygen can pass between lipids. A cell wall is a separate supporting structure, not a substitute for the selectively permeable membrane.

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2. Draw two lipid rows, tails inward, embedded integral proteins, a surface-attached peripheral protein, and a glycoprotein with carbohydrate facing the exterior. Place cholesterol among the tails with its polar region near the heads. Label outside/cytoplasm and hydrophilic/hydrophobic regions. Fluidity means lateral rearrangement of many components; it does not mean every protein moves without restriction.
3. Random motion produces unequal crossing rates when concentrations differ, giving net entry down the gradient. Reversal gives net exit. At equilibrium particles still cross both ways with no net transfer. Simple diffusion depends on physical permeability, including size and polarity, rather than recognition by a specific transport carrier.
4. An open channel provides a hydrophilic, selective path through the core without direct ATP use for each crossing. An ion responds to both concentration difference and membrane voltage. Its electrochemical gradient may favour entry, exit or no net movement. Closing a gate blocks that pathway even if a gradient remains.
5. Osmosis is net water movement through a selectively permeable membrane in response to a water-potential difference. Under the supplied equal-pressure/non-penetrating-solute assumptions, net water moves from the more dilute toward the more concentrated solution. Aquaporins increase water permeability without pumping. No net movement can mean equal opposing crossing rates, not stationary water.
6. A pump couples transport to ATP and can move particles uphill. An open channel permits passive movement down an electrochemical gradient. A facilitated carrier binds and changes conformation for passive transfer, without direct uphill energy coupling. Constant concentration can reflect balanced active input and passive loss, so ATP consumption can continue.
7. A glycoprotein has carbohydrate attached to protein; a glycolipid has carbohydrate attached to lipid. Plasma-membrane carbohydrate groups project extracellularly. Recognition involves specific binding/identification of surface structures; adhesion is attachment. Neither function implies that the component is an open transport pore.
8. $(2.75 - 2.50)/2.50 \times 100 = +10\%$; $(3.60 - 4.00)/4.00 \times 100 = -10\%$; $(8.00 - 6.40)/6.40 \times 100 = +25\%$. The two gains correspond to $0.25/2.50 = 10\%$ and $0.40/8.00 = 5\%$: the smaller gain in grams is the larger proportional gain.
9. The interval fraction is $4/(4+2) = 2/3$, giving $0.20 + (2/3)(0.10) = 0.267$ mol per litre to three significant figures. It is an interpolation from finite, variable tissue measurements, not an exact value. Pressure, tissue compartments, membrane integrity and possible solute exchange complicate a direct inference about every cell's solute concentration. Water still crosses at no net mass change.
10. Use comparable tissue and cylinder dimensions; control solution volume, temperature, immersion time and blotting/weighing procedure. Any five appropriate controls suffice. Vary the prepared external concentration deliberately between

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treatments. Blotting removes external liquid. Independent repeats estimate random variation; they do not repair a wrongly calibrated balance or a consistently misprepared solution.

11. Protein-mediated downhill entry could be facilitated diffusion. Establish the relevant gradient, including voltage for ions, and investigate energy coupling and transport behaviour under controlled conditions. A change after ATP depletion can also reflect indirect effects on gradients, membrane integrity or protein function; it is not sufficient by itself to call the observed protein an ATP-driven pump. Keep the assay conditions and cell viability comparable.
12. **AHL** Cis bends disrupt packing and tend to maintain fluidity at lower temperature compared with otherwise similar saturated chains. Cholesterol restricts some movements at higher temperatures and disrupts close packing at lower temperatures, buffering fluidity. In the cited *E. coli* experiments, warmer growth increased saturated and longer-chain fatty-acid proportions, consistent with compensation for temperature's effect. Different species and membranes can use different adjustments.
13. **AHL** Endocytosis encloses material in an internal vesicle, as in phagocytosis; exocytosis fuses a vesicle with the plasma membrane to release contents, as in insulin secretion. The nicotinic acetylcholine receptor is a ligand-gated cation channel; voltage-gated sodium and potassium channels respond to membrane potential. Open channels allow downhill electrochemical movement rather than coupling each crossing directly to ATP hydrolysis.
14. **AHL** Sodium export $= 8(3) = 24$ ions; potassium import $= 8(2) = 16$ ions; ATP use eight molecules; net outward positive-charge transfer $= 24 - 16 = 8$ elementary charges. ATP-driven sodium export maintains the sodium gradient that can drive uphill glucose entry by a separate cotransporter. Intestinal transport takes glucose from gut lumen toward blood; nephron reabsorption recovers it from filtrate toward blood. The supplied ratio gives $25(2) = 50$ accompanying sodium ions and must not be assumed for every transporter type. CAM-mediated contacts help hold epithelial cells in a tissue and support its barrier organization; that attachment role differs from ion transport.

Exam tips

Identify the particle, pathway, net direction and energy source. Show water on both sides of a bilayer, external carbohydrate branches and the correct protein locations. Use initial mass in percentage change, distinguish net movement from individual crossings, and state the limits of an interpolated zero. A protein in a pathway does not by itself establish active transport.

- **AHL:** Separate channel gating, pumping, cotransport and vesicle movement; trace ATP use.

Scope reference: IB Biology guide, B2.1. Numerical practice data are invented.

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B.2.2 · Organelles and compartmentalization

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A pancreatic cell makes enzymes for release, while a contracting muscle cell needs a continuing supply of ATP. Both are cells, but a micrograph shows different amounts of internal membrane and different organelle arrangements. How can dividing the inside of a cell into regions help it perform a particular job?

This unit covers B2.2 SL understandings plus additional higher-level content (marked AHL): organelle identity, cell fractionation, nuclear separation, local reaction conditions, mitochondrial and chloroplast adaptations, nuclear envelopes, ribosomes, rough ER, Golgi and vesicles. Prerequisites are cell structure, protein function and membrane transport. All numerical datasets are invented teaching examples. Sections marked **AHL** are for Higher Level only.

Identify organelles by structure and function

An **organelle** is a discrete cell subunit adapted to a particular function. The term does not mean only a membrane-bounded sac. In the convention used for this course, nuclei, vesicles, ribosomes and the plasma membrane count as organelles; the cell wall, cytoskeleton and cytoplasm do not. Ribosomes are not surrounded by a membrane. State the convention rather than classifying every useful cell component as an organelle.

Component	Structural evidence	Functional connection
Nucleus	Envelope enclosing chromatin; pores	Separates nuclear activities from the surrounding cytoplasm
Ribosome	Small particle with two subunits; no enclosing membrane	Joins amino-acid residues during translation
Mitochondrion	Two membranes; folded inner membrane	Supports aerobic respiration and ATP production
Chloroplast	Envelope and internal thylakoid membranes	Supports photosynthesis in suitable plant/algal cells
Lysosome	Membrane-bounded digestive compartment	Provides conditions for breakdown and recycling
Vesicle	Small membrane-enclosed compartment	Carries or stores selected contents

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A cell's organelle content depends on its type and state. Some differentiated cells lose particular structures: mature mammalian red blood cells lack nuclei and mitochondria. A secretory cell can have extensive rough ER and Golgi; an active muscle cell can have many mitochondria. These are structure–function relationships, not a rule that one named organelle is the only important structure in that cell.

A schematic uses colour and simplified shapes to explain organization. An electron micrograph gives evidence from a specimen and preparation, usually without the diagram's identifying colours. Look for membranes, folds, stacked sacs and location. One circular profile could be a section through a tube; one image does not reveal a complete three-dimensional shape or prove an organelle's activity.

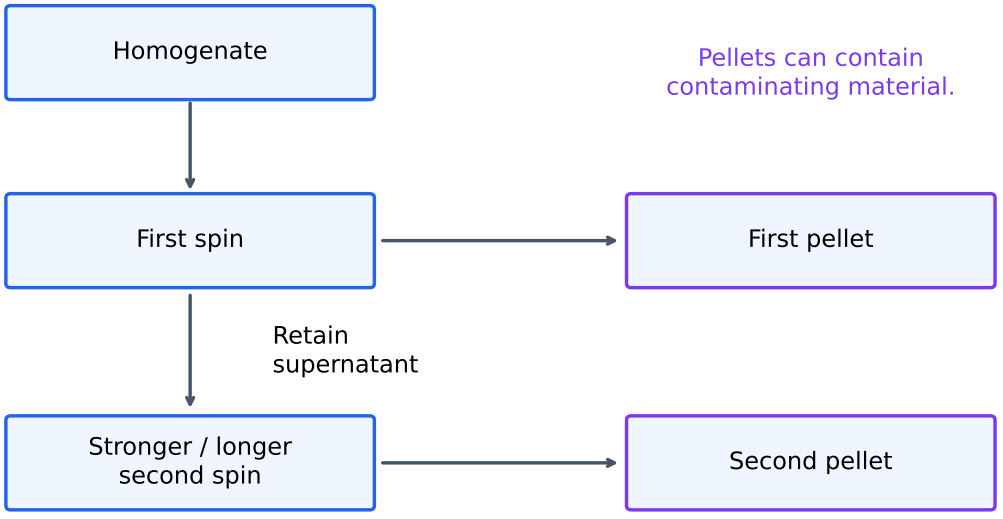
Cell fractionation makes organelle functions testable

A microscope can locate structures, but studying a separated fraction allows different questions. Does the fraction containing a particular organelle also contain a particular enzyme activity? The development of high-speed centrifuges helped researchers test such relationships. A new technique can make a previously inaccessible process measurable.

First homogenize tissue to break cell boundaries and release contents. Use a cold, suitable buffered medium: cooling slows unwanted reactions, buffering limits pH changes, and an appropriate osmotic composition helps preserve organelles. These measures reduce damage; they do not guarantee that every structure survives. Avoid detergent when intact lipid membranes are needed, because detergent can disrupt them.

Centrifuge the homogenate. Material that sediments forms a pellet; the liquid above it is the supernatant. Remove the supernatant and spin it under stronger or longer conditions to collect another fraction. Larger or more readily sedimenting structures can be collected before smaller ones. Sedimentation also depends on density, shape, medium and time, so size alone does not produce an exact universal order.

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Further separation can use the second supernatant.

Carry the supernatant forward; do not spin the discarded pellet and call it the same sequence. Labels describe an experimental separation, not perfect purification.

Test fractions with microscopy and suitable marker assays. A nuclear marker should be enriched with nuclei; an enzyme associated with mitochondria should be enriched in a mitochondrial fraction. Broken organelles and trapped material can contaminate other fractions. Co-occurrence supports an association, but does not establish that a fraction is pure or that a single enzyme explains every function.

■ Worked example: distinguish recovery from enrichment

Invented recoveries, expressed as percentages of each marker's starting total, are:

Fraction	Nuclear DNA marker / %	Mitochondrial marker / %	Cytosolic enzyme / %
First pellet	80	2	5
Second pellet	10	82	5
Small-membrane pellet	5	10	10
Final supernatant	5	6	80

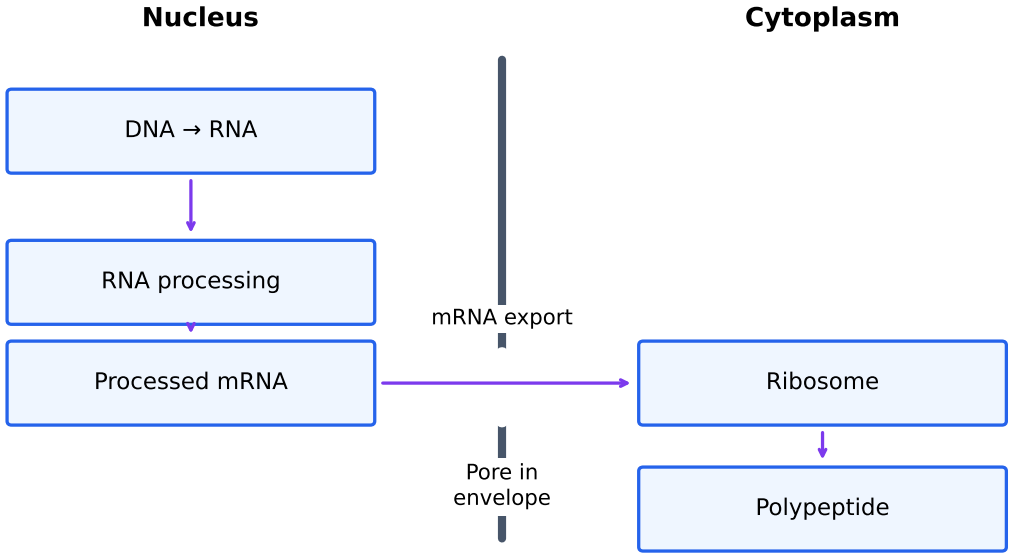
Each marker column totals 100%. The second pellet recovers most of the mitochondrial marker, but also contains some nuclear marker and cytosolic enzyme. It is enriched in mitochondrial material; it is not proved to contain only mitochondria. The final supernatant can also contain small particles not sedimented by the stated procedure.

In a separate invented assay, a starting sample has 30 enzyme units in 15 mg protein: specific activity = 30/15 = 2 units per mg. A collected fraction has 20 units in 2 mg: 20/2 = 10 units per mg. Enrichment is 10/2 = 5-fold, although recovery of enzyme activity is only 20/30 × 100 = 66.7%. A concentrated, enriched fraction need not contain most of the starting material. Assay conditions must be comparable before activities can be divided meaningfully.

Nuclear separation allows RNA processing before translation

Compartmentalization creates regions with different contents or conditions. In a eukaryotic cell, the nuclear envelope separates the main DNA-containing compartment from the cytoplasm. Transcription produces RNA from a DNA template in the nucleus. Translation makes a polypeptide from an mRNA sequence at ribosomes outside the nucleus.

For a typical eukaryotic protein-coding gene, the first RNA transcript can be processed before export. Processing includes removing introns and joining the retained exons by splicing; a cap and poly(A) tail are commonly added. The processed mRNA passes through nuclear pores and can then meet cytoplasmic ribosomes. This separation provides an opportunity for processing and checking before translation. DNA instructions remain in the nucleus; the cell exports an RNA copy, not the gene itself.



The arrow across the envelope carries processed mRNA. The polypeptide is made outside the nucleus. The drawing is a pathway schematic, not a micrograph.

Prokaryotes have no nucleus enclosing their chromosome. Ribosomes may begin translating an mRNA while that RNA is still being transcribed. Do not say prokaryotes have no DNA or no ribosomes, or that all RNA processing is absent from them. The key comparison here is the absence of a nuclear boundary separating transcription

from translation.

A simple timing model illustrates the distinction. Suppose a eukaryotic transcript is made from minute 0 to 3, processing finishes at minute 5, and export finishes at minute 6. In this model cytoplasmic translation cannot start at minute 2: the processed RNA has not arrived. The numbers are invented and do not specify universal cellular rates. They describe the order of events for the stated transcript, not every RNA in every organism.

Compartments concentrate reactants and separate incompatible conditions

An enzyme and its substrate meet more readily when both are concentrated in a suitable region. Keeping them in a smaller compartment can increase their local concentrations without increasing their total amounts. The result still depends on transport, enzyme properties and other requirements; concentration alone does not guarantee the fastest possible reaction.

For an invented example, 12 pmol of a metabolite in 4 pL gives $12/4 = 3$ pmol per pL. The same amount in 12 pL gives 1 pmol per pL: a threefold concentration difference. This is a ratio model, not a realistic concentration claim for a named metabolite. It does not show that the larger compartment has fewer total molecules.

Membranes help preserve different conditions while allowing controlled exchange. Lysosomes maintain an acidic interior suitable for their digestive enzymes. The cytosol has different conditions, so the two environments can support different reactions in the same cell. A lysosomal membrane helps contain digestive activity; it is a selective boundary, not an absolute shield against every form of damage.

A phagocytic cell encloses an engulfed particle in a phagocytic vacuole, also called a phagosome. Fusion with lysosomal compartments supplies digestive enzymes and supports breakdown in the enclosed region. The material does not first have to disperse freely through the cytosol. Useful breakdown products can subsequently be transported out of that compartment. Explain both containment and controlled transfer, rather than describing an isolated bag with no exchange.

This organization has costs. Membranes must be made, maintained and supplied with transport proteins; sorting and transport can require energy. A useful explanation connects a boundary to the conditions it maintains and the process it enables. Simply naming a compartment does not establish its advantage.

Measure structures and report the limits of an image

For a prepared light-microscope slide, locate the specimen at low power first. Centre the region, increase the objective power as appropriate and use fine focus at high power. Draw clear outlines with straight label lines and record the scale. Do not add shading or structures that cannot be seen. Increased magnification does not automatically

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improve resolution.

Magnification is image length divided by actual length, with both lengths in the same unit. A scale bar supplies a known real distance within that image; a captioned magnification becomes wrong if an image is resized without updating it, while a scale bar resized with the image remains usable.

■ Worked example: use a plausible organelle scale

An invented mitochondrion image is 30 mm long; its actual length is 5.0 μm. Convert 30 mm = 30000 μm, then $M = 30000/5.0 = 6000$. Magnification is a dimensionless ratio, written $\times$6000. These numbers do not assert that all mitochondria have that size.

On the same image, a vesicle measures 4.0 mm. Its real diameter is $4000/6000 = 0.667$ μm, about 0.67 μm. An image of a 2.0 μm structure at $\times$15000 is $2.0 \times 15000 = 30000$ μm, or 30 mm. A separate structure shown as 24 mm for an actual 120 μm has magnification $24000/120 = 200$; the arithmetic does not establish that this larger structure is a typical organelle.

If a scale bar labelled 2.0 μm measures 8.0 mm on a print, a structure measuring 18 mm on that same print has actual length $(18/8.0)(2.0) = 4.5$ μm. Doubling both printed lengths leaves the ratio unchanged. Record the measured structure and method; a static micrograph does not directly show a vesicle moving or a mitochondrion making ATP.

AHL: Mitochondrial compartments support aerobic ATP production

The outer and inner membranes enclose an intermembrane space and a matrix. Cristae are folds of the inner membrane. Their large area accommodates respiratory electron-transfer components and ATP synthase. The matrix contains enzymes and substrates used in the Krebs cycle, bringing related reactions into a suitable local environment.

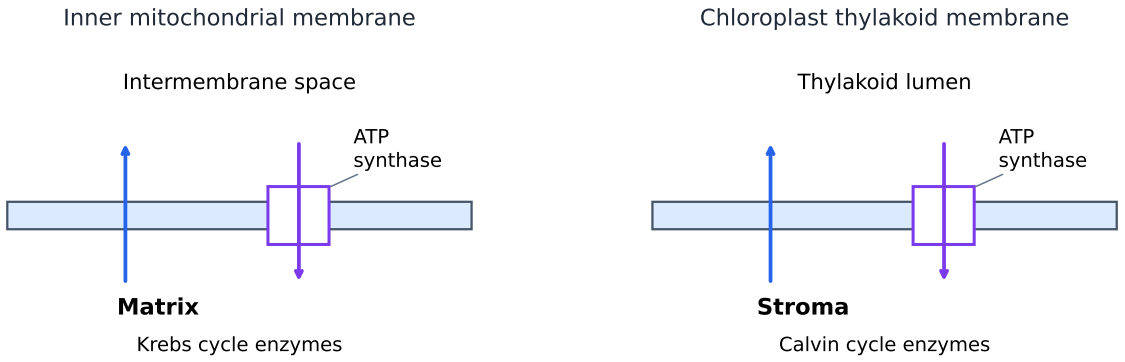
Energy from electron transfer is used to move hydrogen ions from the matrix to the intermembrane space. The relatively small volume of that space allows a given transfer to produce a larger concentration change than it would in a much larger compartment. The inner membrane restricts uncontrolled ion movement, so a gradient can be maintained. Return of hydrogen ions through ATP synthase couples the stored electrochemical difference to ATP production.

Folds increase membrane area; they do not create energy or replace the need for substrates, oxygen in aerobic electron transfer and working proteins. Damaging the inner membrane can dissipate the gradient even if the cell still contains many mitochondria. ATP yield cannot be predicted from a count of cristae alone.

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AHL: Chloroplast compartments support photosynthesis

A chloroplast has an envelope and internal thylakoids, often arranged in stacks called grana. Thylakoid membranes provide a large area for photosystems and other components of the light-dependent reactions. Light energy supports electron transfer and the accumulation of hydrogen ions in the thylakoid lumen. Its small volume helps a given ion transfer change local concentration.



Blue: energy-driven H^+ transfer.
Purple: H^+ return through ATP synthase.

These are local membrane sections, not whole organelles. Identify the two aqueous compartments before following the arrows.

Hydrogen-ion return through ATP synthase into the stroma supports ATP production. The stroma contains enzymes and substrates of the Calvin cycle, which incorporates carbon from carbon dioxide into organic compounds. It is not the thylakoid lumen, and photosystems are not free-floating in it. Carbon fixation and the light-dependent reactions are connected processes in different locations.

Compare functions precisely: mitochondrial ATP synthesis involves the inner membrane, whereas chloroplast light-dependent ATP synthesis involves the thylakoid membrane. A chloroplast's envelope is not a substitute for its thylakoid system. These arrangements illustrate how membranes provide both a surface for proteins and a boundary between regions.

AHL: Nuclear envelopes combine separation with controlled traffic

The nuclear envelope has two membranes with a space between them. Its outer membrane is continuous with the endoplasmic reticulum. Nuclear pore complexes provide controlled routes across the envelope, allowing appropriate RNAs and proteins to move between nucleus and cytoplasm. The boundary separates processes without sealing the DNA compartment away from every required molecule.

An mRNA can leave through a pore; a protein needed for DNA replication can enter

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by a regulated import process. It is not sufficient to say that all small molecules enter and every large molecule is excluded. Pore complexes, recognition and transport machinery contribute selectivity.

During open mitosis or meiosis, as in typical animal-cell examples, the envelope disassembles, permitting spindle access to chromosomes. Nuclear membrane material forms fragments/vesicles and reorganizes with the ER; envelopes later re-form around daughter chromosome sets. This is membrane rearrangement, not destruction of the genetic instructions. Some organisms retain a nuclear boundary during division, so envelope breakdown is not universal. Cell-biology descriptions of nuclear traffic explain why pores and envelope dynamics solve different access problems.

AHL: Ribosomes and rough ER direct proteins toward different destinations

A ribosome contains ribosomal RNA and proteins arranged in two subunits. Free cytosolic and rough-ER-bound ribosomes use the same basic translation mechanism; they are not permanently different species of ribosome. Targeting information in a growing protein can lead a translating ribosome to the ER membrane.

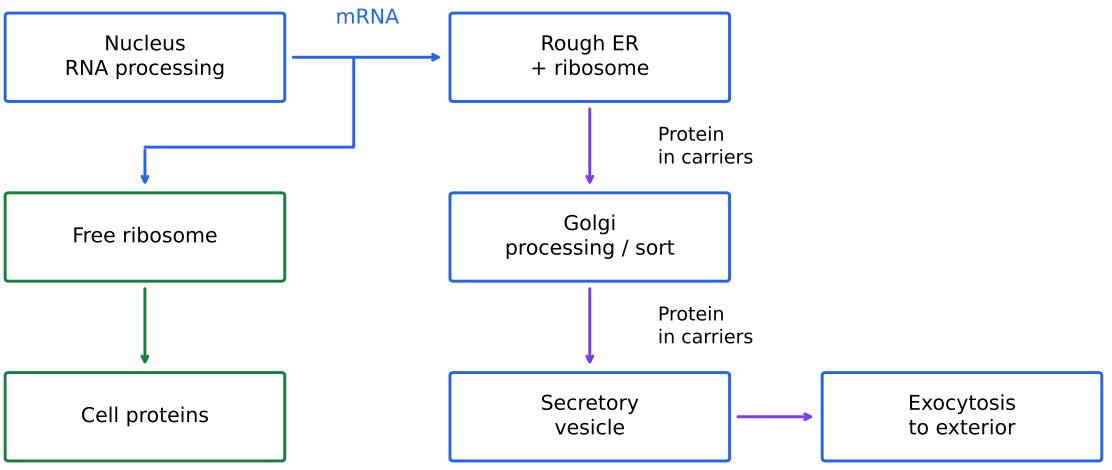
Free ribosomes make many proteins used in the cytosol and proteins subsequently imported into the nucleus, mitochondria or chloroplasts. They do not make only proteins that remain forever in the cytosol. Many proteins entering the secretory pathway are made at ribosomes attached to the cytosolic face of rough ER. The ribosome is outside the ER lumen; the growing protein can be transferred into the lumen or inserted into the membrane.

The rough ER is a membrane system, not a pile of ribosomes. It supports entry, folding and early processing of appropriate proteins. Membrane-bound ribosomes make proteins for secretion and for destinations within the cell, such as parts of the endomembrane system. Not every protein made on rough ER leaves the cell, and not every protein used inside the cell is made on rough ER.

AHL: Golgi and vesicles process, sort and deliver proteins

A protein entering the secretory route passes from ER toward the Golgi in transport carriers. The Golgi contains flattened membrane sacs with receiving and shipping sides. It modifies and sorts appropriate proteins, then packages cargo into carriers for particular destinations. It does not translate an mRNA or synthesize a protein's amino-acid sequence from nothing.

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Blue: RNA route. Purple: secretory protein route. Green: other protein products.

RNA export and protein transport are different arrows. The diagram does not send an already completed secreted protein out of the nucleus.

Vesicle membranes keep cargo separate from the cytosol during transport. Budding, movement, recognition and fusion deliver cargo to a suitable destination; random fusion with every membrane would undermine compartmentalization. Fusion during exocytosis joins the vesicle membrane to the plasma membrane and releases soluble contents outside.

Clathrin is a coat protein that helps form some vesicles, including appropriate endocytic and Golgi-associated carriers. It assembles on the cytosolic side, helping shape a bud and, through associated proteins, organize selected cargo. The coat is removed before later fusion steps. Clathrin is neither the lipid membrane nor a permanent coat on every vesicle; some transport carriers use other coat systems. The intracellular compartment account connects protein sorting with the maintenance of distinct destinations.

Practice

All numerical data are invented. Use a named structure, mechanism and limitation in each explanation.

1. Classify nucleus, ribosome, plasma membrane, cell wall, cytoskeleton and cytoplasm using this course's organelle convention. Explain why a membrane is not required around every organelle.
2. Predict two structural differences between an enzyme-secreting pancreatic cell and a mature mammalian red blood cell. Explain why one organelle count cannot establish a cell's complete activity.

3. Outline cell fractionation from homogenization to successive pellets. Explain the cold buffered medium and why supernatant, rather than the first pellet, proceeds to the next separation.
4. From the marker table, identify the fraction most associated with mitochondria and calculate how much mitochondrial marker remains elsewhere. Explain two reasons it is not a pure mitochondrial sample.
5. A starting assay has 30 enzyme units in 15 mg protein; a fraction has 20 units in 2 mg. Calculate both specific activities, fold enrichment and percentage recovery. Distinguish enrichment from total recovery.
6. Explain how nuclear separation permits RNA processing before translation. Compare with a prokaryote and identify the molecule exported for translation.
7. In the timing model, transcription ends at minute 3, processing at 5 and export at 6. Could cytoplasmic translation of this transcript start at minute 2? Explain without claiming universal timings.
8. Calculate concentrations for 12 pmol in 4 pL and in 12 pL. Explain an advantage and a cost of maintaining different reaction compartments.
9. Explain lysosome and phagocytic-vacuole functions. Distinguish containment from a complete absence of transport across their membranes.
10. Calculate magnification for a 30 mm image of a 5.0 μm mitochondrion, actual vesicle diameter from a 4.0 mm image at that magnification, and image length for a 2.0 μm structure at $\times$15000. Explain low-power focusing and clear scale-labelled drawing.
11. A 2.0 μm scale bar prints as 8.0 mm and a structure as 18 mm. Find actual size. Predict the result after both printed lengths double and explain two limits of a static micrograph.
12. **AHL** Compare mitochondrial and chloroplast ATP-producing membrane systems. Include membrane area, the small receiving compartment for hydrogen ions, enzyme locations, and one consequence of membrane damage.
13. **AHL** Explain nuclear-envelope pores and division-related disassembly. Contrast free and rough-ER-bound ribosomes, including two intracellular destinations for free-ribosome products and one rough-ER product retained inside the cell.
14. **AHL** Trace a secreted protein from its mRNA through translation, ER, Golgi, vesicles and exocytosis. Explain clathrin's role and why a static image of many vesicles does not alone prove a higher secretion rate.

■ Worked answers

1. Nucleus, ribosome and plasma membrane count as organelles in this course convention; cell wall, cytoskeleton and cytoplasm do not. Ribosomes are discrete functional subunits without an enclosing membrane. The classification does not imply excluded structures are unimportant.
2. The pancreatic cell can contain a nucleus, rough ER and Golgi for protein pro-

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duction and processing; the mature mammalian red blood cell lacks these. Its organelle content reflects specialization. Counts alone do not measure protein activity, flux or ATP production; compare cell size, state and suitable functional measurements.

3. Homogenize in cold, appropriate buffered/osmotic medium, then centrifuge and retain the supernatant for stronger/longer spins. Cooling slows unwanted reactions; buffering and suitable osmotic conditions help preserve structures. The first pellet contains already sedimented material. Fractions differ in contents but can be contaminated or damaged.
4. The second pellet contains 82% of the mitochondrial marker. Elsewhere there is $2 + 10 + 6 = 18\%$, also $100 - 82 = 18\%$. Other markers occur in the second pellet, and broken/trapped material can contaminate it. A marker assay and microscopy support enrichment, not perfect purity.
5. Starting specific activity = $30/15 = 2$ units per mg; fraction = $20/2 = 10$ units per mg. Enrichment = $10/2 = 5$-fold. Recovery = $20/30 \times 100 = 66.7\%$. Activity per protein mass increased while about one-third of starting enzyme activity was not recovered in this fraction.
6. Transcription and RNA processing occur in the nuclear compartment before processed mRNA passes through pores to cytoplasmic ribosomes. Introns can be removed before translation. In prokaryotes, lack of a nuclear envelope permits translation to begin on RNA still being transcribed. mRNA, rather than the DNA gene or a completed secreted protein, is exported for translation.
7. No: at minute 2 this transcript has not finished processing or export. Under the model, cytoplasmic translation can start only after mRNA arrival at minute 6. Different real genes and conditions have different timings; the example establishes sequence, not a universal six-minute delay.
8. $12/4 = 3$ pmol per pL and $12/12 = 1$ pmol per pL, a threefold concentration ratio with the same total amount. Concentrating enzymes and substrates can support encounters, and separating incompatible conditions can support different reactions. Making and maintaining membranes and controlled transport require resources.
9. A lysosome supplies an acidic digestive environment. An engulfed particle lies in a phagocytic vacuole; fusion with lysosomal compartments supplies digestive machinery. Contents remain separated from cytosol during digestion, but useful products and other substances can cross through suitable transport pathways. The membranes are selective boundaries, not permanently sealed walls.
10. $30000/5.0 = 6000$, so $\times 6000$. Vesicle diameter = $4000/6000 = 0.667$ μm, approximately 0.67 μm. The 2.0 μm structure gives $2.0(15000) = 30000$ μm = 30 mm. Locate and centre at low power; use fine focus at high power. Draw observed outlines, straight labels and a scale; magnification alone does not improve resolution.

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11. $(18/8.0)(2.0) = 4.5\ \mu$m. After resizing, $(36/16)(2.0) = 4.5\ \mu$m. Section orientation and preparation affect apparent structure; a static image does not directly measure movement or activity. A scale bar must belong to that image and be resized with it.
12. **AHL** Cristae increase mitochondrial inner-membrane area; hydrogen ions accumulate in the small intermembrane space and return toward matrix through ATP synthase. Matrix enzymes support the Krebs cycle. Thylakoids provide photosystem area; hydrogen ions accumulate in the small lumen and return toward stroma through ATP synthase. Stroma enzymes support the Calvin cycle. Damage allowing uncontrolled ion leakage can dissipate gradients; membrane area alone cannot replace working components and an energy source.
13. **AHL** Nuclear pores permit controlled molecular exchange while the double membrane separates processes. During open division the envelope disassembles into rearranging membrane material/fragments/vesicles and later reforms; the DNA is not destroyed. Free ribosomes can supply cytosolic proteins and proteins imported into nucleus or mitochondria/chloroplasts. Rough-ER-bound ribosomes transfer appropriate growing chains into ER or membrane; a lysosomal enzyme is an intracellular example. Ribosomes attach at the cytosolic surface, and the two locations are not permanently different ribosome types.
14. **AHL** Export processed mRNA through a pore; a ribosome translates it and an appropriate targeting signal directs synthesis at rough ER. The protein enters ER, then transport carriers deliver it toward Golgi for modification/sorting. Secretory vesicles deliver selected cargo to plasma membrane; fusion releases contents by exocytosis. Clathrin helps shape some buds and organize cargo through associated proteins, then is removed; it is not every vesicle's permanent membrane. Many visible vesicles could reflect rapid production or blocked onward movement. Use timed labelling or secretion measurements to test flux.

Exam tips

Connect a named structure to a mechanism and function. Distinguish recovery from enrichment, local concentration from total amount, and an RNA route from a protein route. Convert both lengths to one unit and use a scale bar from the same image. Identify what a static specimen can and cannot establish.

- **AHL:** Name both sides of each ATP-producing membrane and the destination of each protein pathway.

Scope reference: IB Biology guide, B2.2. Numerical examples are invented.

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Cell specialization

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A fertilized egg gives rise to cells that carry oxygen, shorten to produce force or pass electrical signals over long distances. Most of these cells retain essentially the same genome, yet their shapes and activities differ. How does one set of instructions support so many structures, and why does cell size matter?

This unit covers B2.3 SL understandings plus additional higher-level content (marked AHL): differentiation, stem-cell properties and niches, developmental potency, cell-size diversity, surface area and volume, exchange adaptations, pneumocytes, muscle cells and human gametes. Prerequisites are gene expression, membrane transport and organelle functions. Numerical models and datasets are invented. Sections marked **AHL** are for Higher Level only.

Differentiation changes gene expression and cell structure

Following fertilization, mitotic divisions produce cells that can contribute to different tissues. **Differentiation** is the development of specialized structures and functions. It involves different patterns of **gene expression** : genes are used to produce functional products, including proteins that affect a cell's activity and shape. An oxygen-carrying cell and a contracting cell need different combinations and amounts of proteins.

Differentiation does not usually require deleting most genes. Most nucleated body cells retain broadly the same DNA sequence while using different parts of it. Mature mammalian red blood cells are an important exception to the usual nucleated-cell pattern: they lose their nucleus during development. Do not extend that exception to every mature animal cell.

Cells also receive different signals. In an early embryo, a signalling substance can form a concentration gradient. Cells at different positions encounter different concentrations and can activate different gene-expression programmes. A threshold model helps explain how position becomes a difference in cell fate. It is not a claim that all embryos use one chemical or one universal concentration threshold.

In an invented model, cells exposed to signal below 2 units express programme A; from 2 up to but not including 5 units they express B; at 5 or above they express C. Exposures of 1, 3 and 6 units predict A, B and C respectively. A cell at exactly 2 belongs to B and one at exactly 5 to C. The genes did not need to disappear for the responses to differ. Testing this explanation needs gene-expression measurements and controlled signal exposure, not just different cell shapes.

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Stem cells combine self-renewal with developmental potential

A stem cell can produce more stem cells by self-renewal and can give rise to specialized descendants. These are different properties. A cell that divides many times but produces only one already specialized type has not thereby demonstrated broad developmental potency. A cell that differentiates once without maintaining a stem-cell population has not demonstrated long-term self-renewal.

Stem cells can sustain repeated division under suitable conditions, potentially maintaining a population for a long period. This does not mean every stem cell divides continuously, survives forever or has unlimited capacity under any culture conditions. Some remain relatively inactive until suitable signals arrive. The NIH stem-cell introduction distinguishes renewal from differentiation; neither property makes a stem cell identical to a cancer cell.

■ Worked example: different division patterns give different populations

Begin with one stem cell. In a simplified asymmetric model, each round produces one continuing stem cell and one specialized descendant. After four rounds along that continuing stem-cell line, there is one stem cell and four specialized descendants, assuming descendants survive and do not divide. There are five cells in total, not sixteen stem cells.

In a different model, every stem cell divides into two stem cells in every round, with no death or differentiation. Four rounds give $2^4 = 16$ stem cells and no specialized descendants. These models hold different assumptions. State which population can divide and what each division produces before applying a power of two.

Adult niches regulate renewal and tissue maintenance

A stem-cell niche is the local environment that influences stem-cell behaviour. Neighbouring cells, extracellular material and local signals can help preserve a stem-cell population or promote proliferation and differentiation. The niche is not simply an empty space in which any cell becomes pluripotent.

Two human examples are bone marrow and hair follicles. Bone marrow contains blood-forming stem cells that supply blood-cell lineages. Hair-follicle niches include epithelial stem cells involved in renewing follicular structures during hair cycles. These stem cells do not normally make every cell type in the body. Different niches provide different signals and support different tissue needs.

A useful experiment separates changes in cell number from changes in differentiation. In an invented culture, three control dishes each begin with 100 comparable cells and yield 8, 10 and 12 cells expressing a differentiation marker. Signal-treated dishes yield 38, 40 and 42. Each dish contains 100 viable cells at measurement. Mean marker-positive proportions are 10% and 40%, a difference of 30 percentage points and a fourfold ratio.

This supports an effect on the measured marker under the tested conditions. It does

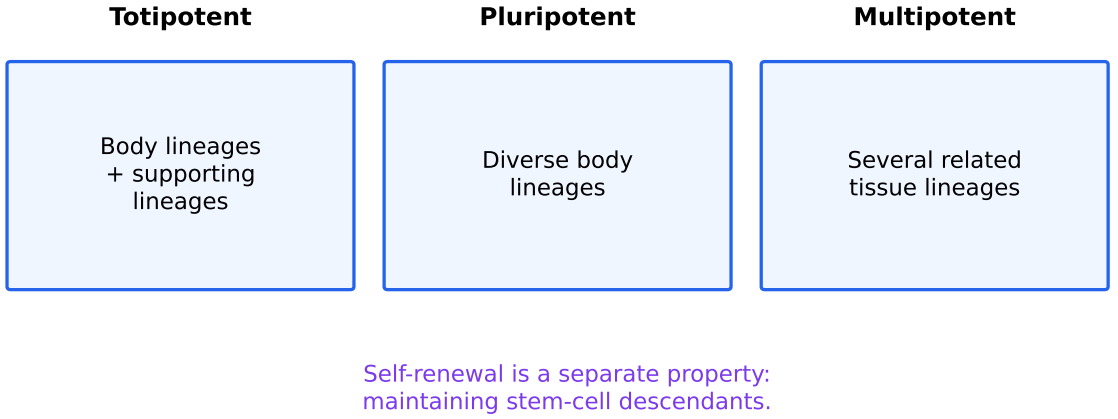
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not by itself prove complete functional differentiation, identify every signalling step or show that the same signal has the same effect in a person. Control starting cells, culture conditions, observation time and marker threshold; measure survival and total cell number as well. A loss of marker-negative cells could change a proportion without producing more specialized cells.

Distinguish totipotent, pluripotent and multipotent cells

Potency describes the range of developmental outcomes a cell can produce under suitable conditions. It is not the rate at which the cell divides.

Potency	Range of outcomes	Suitable context
Totipotent	Embryonic and supporting extraembryonic lineages needed for a complete conceptus	Zygote and certain very early embryonic cells
Pluripotent	The body's diverse cell lineages, without independently supplying every supporting lineage for a complete conceptus	Cells associated with the early embryo; established pluripotent cell lines
Multipotent	Several related cell types within a more restricted developmental range	Blood-forming stem cells in adult bone marrow



The boxes compare developmental ranges, not a universal timetable or stages that every individual cell must pass through.

Early developmental cells soon become more restricted in their usual potential. A blood stem cell can produce several blood-cell types while remaining multipotent; that range does not make it pluripotent. Conversely, producing representatives of three body lineages in one experiment is evidence of breadth, but a few marker readings

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alone do not demonstrate every possible body lineage or a complete organism.

Keep the terms precise when reading a question. If cell X is specified to supply both body and supporting lineages of a complete conceptus, X is totipotent. If Y supplies the body's diverse lineages but not every necessary supporting lineage independently, Y is pluripotent. If Z supplies several related blood-cell lineages, Z is multipotent. The classifications follow the stated capacity, not the age of a donor alone.

Cell size reflects different functional demands

A diameter, cell-body width and total length are different measurements. Representative human red blood cells are about 7–8 μm across, many white blood cells are roughly 10–20 μm across, and a human egg cell is approximately 100–120 μm across. These are approximate biological ranges, not invented practice measurements or fixed sizes of every specimen.

A sperm head is only a few micrometres long, while the whole sperm, including its flagellum, is tens of micrometres long. A neuron may have a small cell body and an axon extending a great distance; a long axon does not imply a spherical cell body of the same diameter. A skeletal muscle fibre can extend centimetres while remaining much narrower than its length. Compare the same dimension and unit before ranking sizes.

Large cytoplasmic volume can supply material for early development in an egg. A long neuronal process supports communication over distance, and an elongated muscle fibre supports force along its length. Small size and thin regions can support exchange. Size therefore has benefits and constraints; there is no single best dimension for every cell function.

Geometry links exchange area to volume-dependent demand

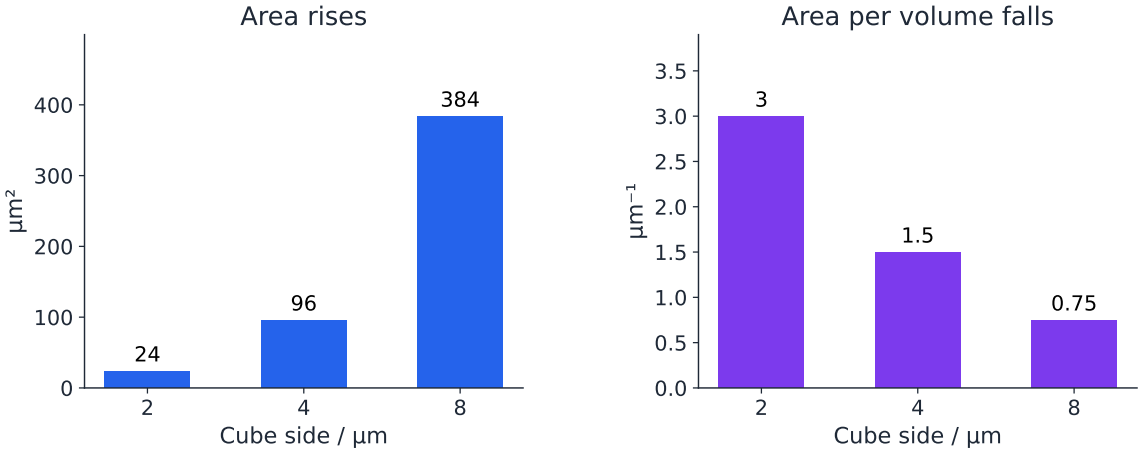
For similarly shaped cells, exchange capacity across the surface depends partly on available area, whereas material requirements and waste production are often related to volume. If linear size increases, volume grows faster than area. The ratio falls even though total surface area increases. Permeability, transport proteins, gradients, temperature and metabolic activity also affect real exchange.

For a cube of side a, surface area is $6a^2$, volume is a^3 and surface area-to-volume ratio is $6/a$. With a measured in μm, the ratio has unit μm^{-1}.

Invented cube side / μm	**Area / μm^2**	**Volume / μm^3**	**Area/volume / μm^{-1}**
2	24	8	3.0
4	96	64	1.5
8	384	512	0.75

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Doubling side length multiplies area by four and volume by eight, halving the ratio. A larger cube has more area in total but less area per unit volume. For a sphere, the corresponding ratio is $3/r$: radii 2, 4, 6 and 10 length units give 1.5, 0.75, 0.50 and 0.30 per length unit. Do not use diameter as r.



All faces are assumed exposed. Cells touching each other in a tissue do not necessarily expose every face to the outside medium.

A 4 μm cube has volume 64 μm^3 and area 96 μm^2. Eight separate 2 μm cubes have the same combined volume, $8(8) = 64$ μm^3, but total area $8(24) = 192$ μm^2. Their exposed area is doubled under this model. This does not mean that cutting real cells into pieces creates eight viable cells: membranes, organelles and other requirements must be maintained.

Diffusion distance adds another constraint. Increasing the distance from surface to centre can slow transfer to the centre. Shape changes, internal transport and metabolic differences can help cells function despite a large length or volume. A cube or sphere isolates geometric scaling; it does not reproduce every cell's shape or predict an exact maximum cell size.

AHL: Flattening and membrane folds improve exchange geometry

At fixed volume, changing shape can increase surface area and reduce a diffusion path. An invented $2 \times 2 \times 2$ μm cube has volume 8 μm^3 and area 24 μm^2. A $0.5 \times 4 \times 4$ μm cuboid has the same volume but area $2(0.5 \times 4 + 0.5 \times 4 + 4 \times 4) = 40$ μm^2. Its area/volume ratio is 5 μm^{-1} instead of 3 μm^{-1}, and the shortest distance from its centre to a face is 0.25 μm instead of 1 μm. This compares model shapes, not a claim that cells are rectangular blocks.

A mammalian erythrocyte has a biconcave, flexible form with thin regions, supporting exchange and passage through narrow capillaries. Loss of nucleus and other organelles leaves space for haemoglobin. These features work together; absence of a nucleus alone

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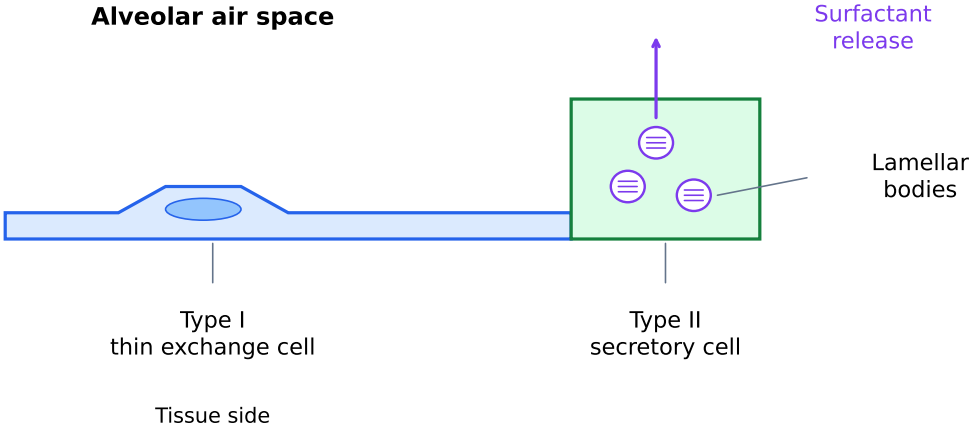
does not explain every aspect of oxygen transport.

Proximal convoluted tubule cells have apical microvilli forming a brush border, increasing area facing the filtrate. Infolding of the basolateral membrane provides additional area for transport proteins; many mitochondria support energy-requiring transport. Microvilli do not beat like cilia. Extra area cannot replace the necessary carriers, pumps and gradients. A root-hair extension offers another area-increasing example, but it serves a different tissue and environment.

AHL: Alveolar tissues combine two pneumocyte adaptations

Type I pneumocytes are extremely thin, spreading over much of the alveolar surface. Their thin regions shorten the diffusion path between alveolar air and blood across the complete exchange barrier. They are not large bags storing oxygen.

Type II pneumocytes are more cuboidal and contain many lamellar bodies, secretory structures that release surfactant to the alveolar lumen. Surfactant lowers surface tension at the moist lining and helps stabilize alveoli during breathing. A type II cell's secretory apparatus serves a different role from the very thin type I exchange surface. Lamellar bodies are not mitochondria or openings through which oxygen is actively pumped.



The two cell types occupy the same epithelial tissue. The diagram is schematic and omits other components of the air–blood barrier.

The tissue therefore contains more than one specialized cell type because its overall function requires both efficient exchange and a stable lining. Do not replace this explanation with "all alveolar cells are thin" or "surfactant provides the oxygen concentration gradient". Ventilation, blood flow, barrier thickness and available area also matter.

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AHL: Cardiac cells and skeletal muscle fibres share contractile machinery

Both cardiac muscle cells and skeletal striated muscle fibres contain myofibrils organized into repeating contractile units, producing striations. The presence of striations does not identify skeletal muscle uniquely. Compare branching, length, cell boundaries and nuclear arrangement as well.

Cardiac muscle cells are relatively short and branched, commonly with one central nucleus and sometimes two. Junctions at intercalated discs help connect cells mechanically and electrically, supporting coordinated contraction of a network. Skeletal muscle fibres are long, generally unbranched and multinucleated, with nuclei near the periphery. Their parallel organization supports force along the fibre's length.

A skeletal muscle fibre forms by fusion of precursor cells into a continuous cytoplasm within one plasma membrane. It is a multinucleate cell, or syncytium, rather than a bundle of many separate mature cells merely placed end to end. It challenges the simplistic rule that every cell has one nucleus, without overturning the usefulness of cell boundaries in identifying cells.

Treat function explanations as hypotheses supported by evidence: branching and connections can help coordinated network activity; long parallel fibres can support directed force; multiple nuclei can support the demands of a large cytoplasmic region. A still image of a branch or nucleus does not by itself measure contraction strength or prove the complete developmental mechanism.

AHL: Human sperm and egg cells have complementary adaptations

A sperm carries a haploid nuclear chromosome set in its head. The acrosome contributes molecules used in interactions with the egg's surrounding layers. A flagellum supports movement, and mitochondria in the midpiece contribute ATP; sperm also use other metabolic pathways, including glycolysis. Avoid treating the head as a tiny complete embryo or the acrosome as a mechanical drill.

The egg cell has a much larger cytoplasm containing organelles, molecules and stored resources supporting the earliest stages of development. Its surrounding layers contribute interactions with sperm. Cortical granule release after fertilization contributes changes that reduce entry of additional sperm. The egg does not swim with a flagellum, and a human egg does not contain enough yolk to feed the whole pregnancy.

The human gametes each contribute a haploid set of 23 nuclear chromosomes; fusion of the parental chromosome sets restores 46 in the usual diploid zygote. This chromosome accounting does not imply identical contributions of cytoplasm or organelles. Fertilization combines genetic material, while subsequent differences in gene expression help produce the embryo's different cell types.

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Practice

All numerical examples below are invented or use the stated geometric model.

1. Explain differentiation through gene expression. State why different protein patterns do not usually require loss of most genes, and name the mature mammalian-cell exception discussed here.
2. Apply the signal thresholds below 2, from 2 to below 5, and 5 or above to exposures 1, 2, 3, 5 and 6. Explain how a gradient can produce positional differences without changing DNA sequence.
3. Distinguish self-renewal from differentiation. Predict populations after four rounds when one continuing stem cell produces one specialized descendant each round, and when all stem cells instead produce two stem cells each round.
4. Describe bone-marrow and hair-follicle niches and explain how a niche can support renewal or differentiation. Why does location alone not prove pluripotency?
5. Calculate mean marker-positive proportions for control counts 8, 10, 12 and treated counts 38, 40, 42, each from 100 viable cells per dish at measurement. Give the difference in percentage points, fold ratio, two controls and a limitation.
6. Classify specified capacities: X supplies body and supporting lineages of a complete conceptus; Y supplies diverse body lineages without independently supplying all necessary supporting lineages; Z supplies several related blood-cell types. Explain why potency is not division rate.
7. Explain why a neuron with a long axon is not equivalent to a huge spherical cell. Compare dimensions needed to describe an egg, sperm, red blood cell and skeletal muscle fibre fairly.
8. Calculate area, volume and area/volume for cubes of sides 2, 4 and 8 μm. Explain why area increases while the ratio falls.
9. Compare one 4 μm cube with eight separated 2 μm cubes: combined volume and total exposed area. State an assumption and a biological limitation.
10. For spheres of radii 2, 4, 6 and 10 units, calculate $3/r$. What happens if a stated diameter of 4 units is mistakenly used as the radius?
11. Explain three reasons that surface area-to-volume ratio alone does not determine an exact maximum cell size. Describe one controlled way of comparing scaled drawings of cells.
12. **AHL** Compare the two equal-volume cuboid models, including areas, ratios and shortest centre-to-face distances. Explain erythrocyte flattening, proximal-tubule microvilli and basolateral infolding without assuming area alone guarantees transport.
13. **AHL** Compare type I and type II pneumocytes, then cardiac muscle cells and skeletal muscle fibres. Link each named structure to function and explain why a skeletal fibre can count as one cell despite many nuclei.

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14. **AHL** Compare human sperm and egg adaptations, including movement, cytoplasm, acrosome, lamellar versus cortical granules, and nuclear chromosome sets. Explain why different gamete shapes do not mean different haploid chromosome counts.

■ Worked answers

1. Different gene-expression patterns produce different functional products and structures. Most nucleated body cells retain broadly the same genome rather than deleting unused genes. Mature mammalian erythrocytes lose the nucleus; this is not the rule for all mature animal cells.
2. Exposures 1, 2, 3, 5 and 6 predict A, B, B, C and C. Cells in different positions can receive different concentrations and activate different programmes. The stipulated thresholds are a model, not universal embryonic values.
3. Self-renewal maintains stem-cell descendants; differentiation produces specialized descendants. Under the first assumptions: one continuing stem cell plus four non-dividing specialized descendants, five cells total. Under the second: $2^4 = 16$ stem cells, no specialized descendants. State survival and division assumptions.
4. Bone marrow supports blood-forming stem cells; hair-follicle niches support relevant epithelial stem cells and tissue renewal. Local signals and contacts can maintain a stem-cell population or promote proliferation/differentiation. A niche's location does not show capacity to produce all body lineages; test developmental potential separately.
5. Controls average $(8+10+12)/3 = 10$ marker-positive cells per 100, or 10%; treated average 40 per 100, or 40%. Difference $= 40 - 10 = 30$ percentage points; ratio $= 40/10 = 4$. Control starting cells, time, medium and marker threshold. A marker need not demonstrate full function; differential survival can also change proportions, so measure total cells and viability.
6. X is totipotent, Y pluripotent and Z multipotent under the stated capacities. Potency describes the range of outcomes; self-renewal and proliferation describe population behaviour. Producing several related types does not imply ability to make a complete conceptus.
7. An axon's length differs from cell-body width and diffusion distance across its narrow diameter; transport within a long cell also matters. Compare diameter with diameter or total length with total length, in common units. Egg diameter, sperm head/whole length, erythrocyte diameter and muscle-fibre length/width must be identified explicitly.
8. Areas 24, 96 and 384 μm^2; volumes 8, 64 and 512 μm^3; ratios 3.0, 1.5 and 0.75 μm^{-1}. Area scales as length squared while volume scales as length cubed. Doubling side multiplies area by four and volume by eight, so area per volume halves.

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9. Single cube: volume 64 μm^3 and area 96 μm^2. Eight small cubes: combined volume $8(8) = 64$ μm^3 and exposed area $8(24) = 192$ μm^2, twice as much. Assume all faces are exposed; touching faces may not be exchange surfaces. Cutting a real cell does not automatically create eight viable cells.
10. Ratios are 1.5, 0.75, 0.50 and 0.30 per length unit. A diameter of 4 means radius 2, giving 1.5. Using 4 as radius gives 0.75, half the correct ratio. State inverse-length units when the length unit is specified.
11. Membrane permeability/proteins, maintained gradients, metabolic demand, diffusion distance, shape and internal transport affect function. Any three linked factors suffice. Use scale bars or calibrated dimensions, compare the same dimension, label observed structures and explain a feature–mechanism–function connection. Geometry alone is not a measured maximum size.
12. **AHL** Both volumes are 8 μm^3. The cube has area 24 μm^2, ratio 3 μm^{-1} and shortest centre-to-face distance 1 μm; the flattened cuboid has area 40 μm^2, ratio 5 μm^{-1} and shortest distance 0.25 μm. Erythrocyte thin regions support exchange; microvilli increase apical area; infoldings increase basolateral area for transport machinery. Protein function, energy and gradients remain necessary.
13. **AHL** Thin type I pneumocytes shorten the exchange path. Type II cells release surface-tension-reducing surfactant from lamellar bodies, supporting a stable lining. Both muscle types have contractile myofibrils and striations. Cardiac cells are short/branched, usually with one or two central nuclei and junctions supporting a coordinated network; skeletal fibres are long, unbranched and multinucleated. A skeletal fibre has continuous cytoplasm within one plasma membrane following precursor fusion, so nuclear number alone does not define the cell boundary.
14. **AHL** Sperm: small head with haploid nucleus, acrosome for interactions with surrounding egg layers, flagellum for movement and metabolic ATP supply including midpiece mitochondria. Egg: large cytoplasm and early-development resources, surrounding layers for interactions and cortical granules contributing a block to additional sperm entry. Lamellar bodies belong to surfactant secretion by type II pneumocytes, not the egg's cortical reaction. Each gamete contributes 23 nuclear chromosomes in the usual human case; their union restores 46 despite unequal cell size and cytoplasm.

Exam tips

Name the cell, structure, mechanism and function. Keep developmental potency separate from division rate, percentage points separate from fold change, and length separate from diameter. In geometry questions report both total area and area per volume, including assumptions about exposed surfaces.

- **AHL:** Compare cell types within a tissue rather than assigning one adaptation to every cell in that tissue.

Scope reference: IB Biology guide, B2.3. Numerical models are invented.

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Gas exchange

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This unit covers B3.1 SL understandings plus additional higher-level content (marked AHL): exchange surfaces and gradients, mammalian lungs and ventilation, lung volumes, leaf tissues, transpiration and stomatal density, and haemoglobin affinity. Sections marked **AHL** are for Higher Level only.

Prerequisites: diffusion, membranes, area/volume and proteins. Numerical data and curves are invented teaching models, not clinical references. Scope: IB Biology guide, first assessment 2025.

Exchange supplies gases across a permeable surface

A person can move air rapidly yet transfer little oxygen if blood flow or the exchange barrier is impaired. Conversely, blood cannot collect a continuing oxygen supply from an isolated, unventilated volume indefinitely. Follow both sides of the surface rather than explaining exchange with "the lungs breathe".

Gas exchange transfers gases between an organism and its surroundings. Aerobic respiration consumes oxygen and releases carbon dioxide; photosynthesis uses carbon dioxide and releases oxygen. Plants respire as well as photosynthesize. A photosynthesizing leaf can have net carbon dioxide uptake while individual cells are still producing carbon dioxide in respiration. Net exchange depends on the balance of processes and conditions, not simply whether the organism is an animal or a plant.

Small organisms can exchange directly over much of their body surface. In a geometrically similar larger organism, volume increases faster than surface area and internal cells lie farther from the exterior. A thin, folded internal exchange surface plus transport can meet these constraints. No universal size cutoff follows from geometry alone: shape, demand and transport also matter.

Effective surfaces combine a large area, a short diffusion path, permeability and moisture. Gases dissolve in the thin aqueous layer before crossing cell membranes. A thin layer helps; a thick layer of fluid adds a diffusion barrier. Area permits many molecules to cross simultaneously. Thin epithelial and capillary layers reduce distance. These features affect transfer without turning diffusion into an ATP-driven pump.

For comparisons with other factors held constant, a useful qualitative model is:

$$\text{transfer rate} \propto \frac{\text{area} \times \text{concentration difference}}{\text{diffusion distance}}.$$

Doubling area doubles the predicted rate; doubling distance halves it. Doubling both leaves the predicted rate unchanged. This simple relationship compares the same gas

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and barrier under controlled conditions; it is not a complete numerical model of a living lung.

Ventilation and blood flow maintain separate gradients

Ventilation moves air over lung surfaces or water over gill surfaces. **Perfusion** is blood flow through tissue, including an exchange surface. These are bulk movements; oxygen and carbon dioxide cross the exchange barrier by diffusion down their own gradients. They need not move in the same direction.

In a mammalian alveolus, fresh air helps maintain a higher oxygen partial pressure than in incoming blood. Oxygen dissolves and diffuses into blood. Incoming blood generally has a higher carbon dioxide partial pressure than alveolar air, so carbon dioxide diffuses the other way. Partial pressure describes the contribution of a gas to the total pressure and indicates its diffusion tendency; compare the same gas across the barrier. Do not compare the oxygen percentage on one side with the carbon dioxide percentage on the other.

Continuous blood flow brings blood requiring oxygen and removes oxygenated blood. It also supplies carbon dioxide for removal. A dense capillary network provides extensive contact with alveoli. Ventilation renews alveolar air, while blood flow prevents the blood immediately next to the surface simply reaching equilibrium with a sealed air space and staying there. Haemoglobin binding helps blood carry oxygen without requiring all of it to remain dissolved.

An original sealed-chamber model starts with 100 oxygen units outside and 40 inside a permeable sac. Oxygen initially moves net inward. If the outside falls to 70 and the inside rises to 70, net transfer ceases although molecules still move both ways. Replacing the outside solution and flowing fresh low-oxygen fluid through the sac can restore a gradient. This model isolates renewal; it omits living tissue, metabolism and haemoglobin.

Mammalian lungs combine branching with thin alveoli

Air passes through the trachea, bronchi and progressively smaller bronchioles to alveolar regions. Branching distributes air through the lung; many alveoli provide a large total surface area in a compact volume. Bronchioles conduct and regulate airflow, rather than serving as the main thin gas-exchange barrier themselves.

An alveolar wall includes very thin type I pneumocytes. Adjacent capillaries also have thin walls; short distances between air and blood support rapid diffusion. A moist lining enables gases to dissolve. Surfactant, secreted by type II pneumocytes, lowers surface tension at the air–liquid interface and helps alveoli remain stable during breathing. It does not provide the oxygen gradient or pump oxygen through the wall. Extensive capillary beds combine a large contact area with ongoing blood renewal.

Link each feature to a mechanism: many alveoli increase area; thin walls shorten distance. Loss of area, thickening, reduced ventilation and reduced perfusion can each

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decrease exchange; breathing rate alone cannot distinguish them.

Ventilation follows muscle, volume and pressure changes

Muscles change the volume of the thorax; air moves because a pressure difference is created between alveoli and the atmosphere. Lungs follow chest movements through the pleural arrangement and elastic tissues. They do not contain muscles that actively expand each alveolus like a small pump.

Stage	Movement and consequence
Inspiration	Diaphragm contracts and flattens; external intercostal activity raises ribs up and out. Thoracic and lung volumes increase.
Air enters	Alveolar pressure becomes slightly lower than atmospheric pressure during inflow. Air moves inward until pressures equalize.
Quiet expiration	Inspiratory muscles relax; diaphragm returns toward its dome and elastic recoil reduces lung volume. Alveolar pressure rises slightly above atmospheric during outflow.
Forced expiration	Abdominal contraction raises abdominal pressure and pushes the diaphragm upward; expiratory intercostal activity can help draw ribs down. Volume falls more forcefully.

Quiet expiration is normally largely passive, whereas inspiration uses muscle contraction. Avoid saying that the diaphragm contracts upward during expiration. Its upward return during quiet expiration follows relaxation and recoil. See the NHLBI explanation of breathing muscles and recoil.

At the end of a breath, airflow can be zero even though lungs still contain air. A larger pressure difference tends to drive greater flow for the same airway resistance; a blocked or narrowed airway changes that resistance. Pressure difference, volume and airflow are related quantities, not interchangeable names for ventilation.

Measure lung volumes and distinguish ventilation from uptake

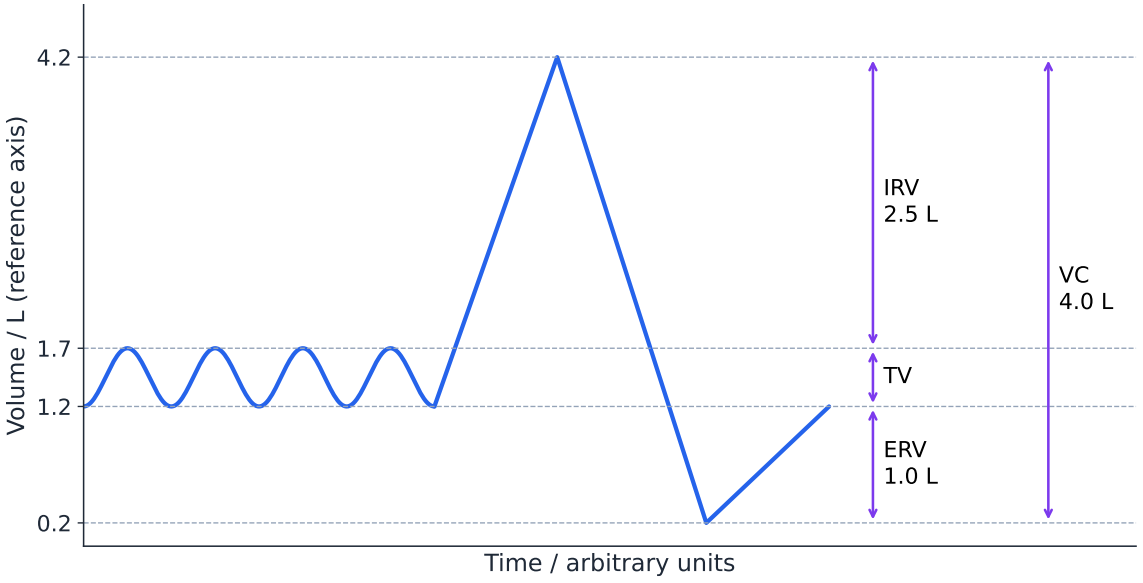
Tidal volume is the air moved in one ordinary breath. Inspiratory reserve volume is the additional volume that can be inhaled after a normal inspiration; expiratory reserve is the additional volume that can be exhaled after a normal expiration. Vital capacity spans the greatest inhalation to the greatest exhalation:

$$\text{vital capacity} = \text{inspiratory reserve} + \text{tidal volume} + \text{expiratory reserve}.$$

Residual volume remains after maximal expiration. A simple spirometer that measures air moving through the mouth does not measure this trapped remaining volume

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directly, so it does not directly provide total lung capacity. Do not put zero lung volume at the lowest point of such a trace unless an independent absolute-volume measurement supports it.



The vertical zero is an arbitrary reference. The trace shows changes in volume, not all the air remaining in the lungs.

In the invented trace, normal expiration is at 1.2 L and normal inspiration at 1.7 L on its reference axis. Tidal volume is $1.7 - 1.2 = 0.5$ L. Maximum inspiration is 4.2 L and maximum expiration 0.2 L. Inspiratory reserve is 2.5 L; expiratory reserve is 1.0 L; vital capacity is 4.0 L. These differences do not reveal residual volume.

Minute ventilation is tidal volume multiplied by breaths per minute. A model with 0.50 L per breath at 12 breaths per minute moves 6.0 L per minute. This is air moved, not 6.0 L of oxygen absorbed. Some air occupies conducting airways, called anatomical dead space, and does not reach alveolar exchange surfaces in that breath. With an assumed fixed dead space of 0.15 L per breath, estimated alveolar ventilation is $(0.50 - 0.15)(12) = 4.2$ L per minute. A different pattern, 0.30 L at 20 breaths per minute, has the same minute ventilation but only 3.0 L per minute alveolar ventilation in this model.

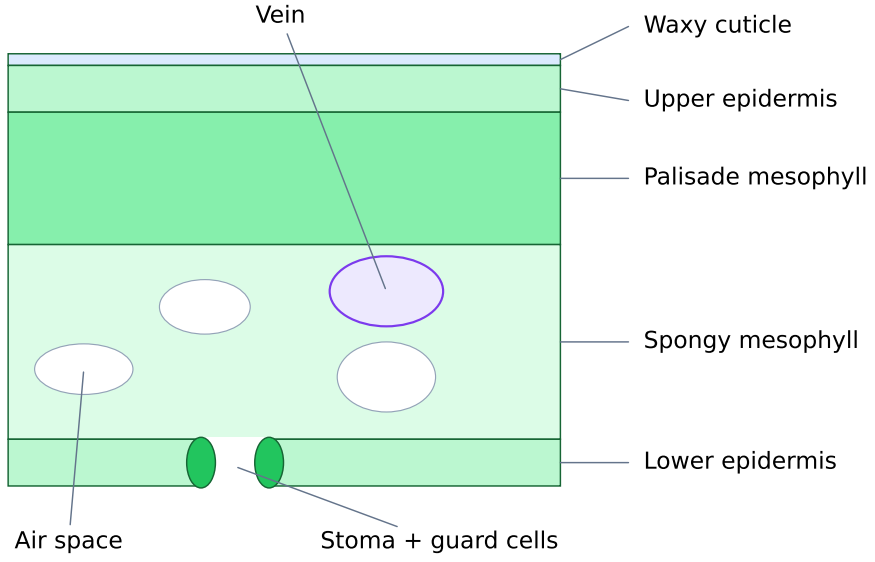
For measurement, calibrate the volume scale, read several regular resting breaths and measure intervals on the time axis. Use clean equipment and the institution's approved supervised protocol. An approved spirometry activity or supplied trace can support identification of reserves and vital capacity. For human demonstrations use voluntary resting measurements; do not induce breathlessness, hyperventilation or unapproved forceful manoeuvres. Explain the distinction using model data when appropriate. Repeated breaths improve an estimate of that person's pattern; they do not represent independent people or establish a population reference range.

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A dicot leaf has connected air spaces and distinct tissues

Leaves exchange gases through stomatal pores, intercellular air spaces and moist mesophyll surfaces. In many terrestrial dicot leaves, the broad thin blade provides area and short distances, while air spaces in spongy mesophyll connect internal surfaces to stomata. Carbon dioxide can diffuse through a pore and air spaces, dissolve on a moist cell wall and enter a photosynthesizing cell. Oxygen can take the reverse path when photosynthesis exceeds respiration.

A typical transverse section has an upper waxy cuticle, upper epidermis, palisade mesophyll, spongy mesophyll with air spaces, and lower epidermis with stomata and guard cells. Veins contain vascular tissues: xylem supplies water and phloem transports assimilates. The cuticle limits uncontrolled water loss but also restricts direct gas exchange over the epidermis. Guard cells regulate pore width, affecting both carbon dioxide entry and water-vapour loss. Stomata are not permanently open holes; the distribution and anatomy vary among species and environments.



This representative arrangement is not universal. A plan diagram shows tissue regions, rather than every individual cell.

When drawing a plan diagram, use clear continuous outlines for tissue boundaries, show relative thickness and position, and label tissues with ruled lines. Indicate a vascular bundle, upper and lower surfaces, mesophyll regions and stomatal position. Do not fill a low-power tissue plan with detailed organelles or invent separate cells where the task asks for tissue distribution. At higher magnification, a cellular diagram can instead show individual guard cells, mesophyll cells and their walls.

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Gas exchange brings a water-loss trade-off

Water evaporates from moist mesophyll walls into leaf air spaces, then water vapour diffuses outward through open stomata when the internal-to-external water-vapour gradient favours loss. This transpiration accompanies access for carbon dioxide. It is not the same process as photosynthesis consuming water, nor is most lost water being converted into oxygen at the pore.

Higher external humidity usually reduces the vapour gradient at a given leaf temperature. Wind can remove the humid boundary layer, increasing loss while other factors remain suitable. Increased temperature can increase evaporative demand; light often promotes stomatal opening as photosynthesis becomes possible. However, water stress can close stomata, so a warmer or brighter treatment does not guarantee a measured increase. Explain both the physical driver and the possible plant response. Species, water supply, leaf temperature and air movement are important controls.

In an invented potometer comparison, corrected water uptake is 0.20 mL in 10 minutes in still air and 0.35 mL in 10 minutes under moving air. Uptake rates are 0.020 and 0.035 mL per minute, an increase of 75%. A potometer measures water uptake, which approximates transpiration under suitable conditions; it does not directly measure every water molecule leaving a leaf. Check leaks, allow equilibration, control leaf area and temperature, and include repeats. A change in uptake alone does not identify whether pore width changed.

Estimate stomatal density using area and replication

Stomatal density is the number of stomata per unit epidermal area. Count pores, not each of the two guard cells as a separate stoma. Use a calibrated micrograph or an approved leaf impression. Apply one boundary rule consistently, such as counting pores touching the top and left edges but not the bottom and right edges. Otherwise overlapping fields can bias counts.

An invented set of four equal fields, each 0.20 square millimetres, contains 32, 36, 28 and 40 stomata. The total is 136 over 0.80 square millimetres; density is 170 per square millimetre. Equivalently, average count 34 divided by 0.20 gives 170. If field areas differ, divide total count by total sampled area rather than averaging unweighted counts. A circular field with radius 0.25 mm has area $\pi(0.25)^2 = 0.196$ square millimetres; 35 stomata correspond to about 178 per square millimetre. Diameter must first be halved.

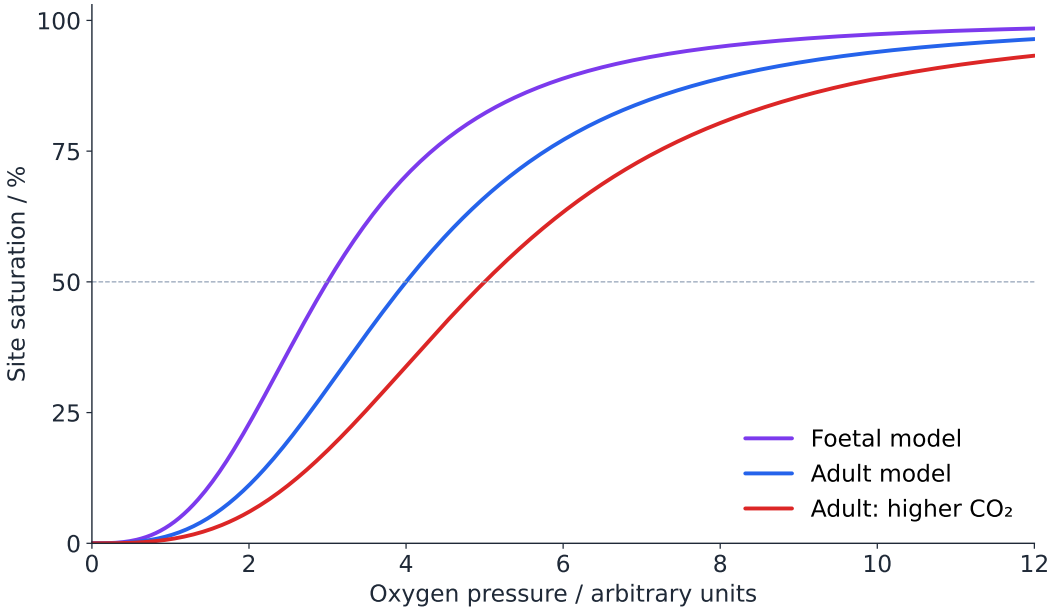
Sample fields systematically or randomly rather than selecting crowded patches. Repeat across leaves and plants, record the leaf side, position and developmental stage, and distinguish within-leaf fields from independent plant replicates. Compare upper and lower surfaces under the same counting and calibration method. Higher density can permit a greater maximum conductance, but actual exchange also depends on pore size, opening and conditions; density alone does not prove a higher current photosynthetic rate.

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AHL: Haemoglobin combines cooperative binding with oxygen release

A haemoglobin tetramer has four haem groups, each able to bind one oxygen molecule reversibly. A fully occupied molecule carries four oxygen molecules, not eight. Binding changes protein conformation so further oxygen binding becomes easier: this positive cooperativity gives a sigmoidal oxygen dissociation curve. At low oxygen availability, relatively little is bound; over the steep region, a small change can cause substantial loading or unloading; at high availability the curve approaches saturation.

The horizontal axis commonly shows oxygen partial pressure; the vertical axis shows percentage saturation of binding sites. Saturation is not the percentage of oxygen in air, the number of red cells or the rate of oxygen uptake. A value of 75% means three quarters of the available sites are occupied across the sample, not that every molecule must have exactly three oxygen molecules attached at that instant.



These illustrative curves use an arbitrary oxygen-pressure scale. They are not clinical measurements or reference ranges.

At a given oxygen partial pressure in the useful comparison region, a left-shifted curve has greater saturation and higher affinity. At half saturation, a lower required oxygen partial pressure likewise indicates higher affinity. “Higher affinity” describes stronger binding under the compared conditions, not automatically better delivery everywhere: blood must also unload oxygen to tissues.

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AHL: Foetal haemoglobin supports transfer across the placenta

Foetal and maternal blood exchange gases across a placental barrier without normally mixing directly as one blood pool. Oxygen must diffuse from maternal blood through the exchange tissues into foetal blood. Under physiological red-cell conditions, foetal haemoglobin has higher oxygen affinity than adult haemoglobin, helping foetal blood load oxygen at the lower partial pressures encountered at this interface.

Adult haemoglobin A has two alpha and two beta chains; foetal haemoglobin has two alpha and two gamma chains. Different protein subunits change interactions with allosteric effectors, including weaker binding of 2,3-bisphosphoglycerate by foetal haemoglobin. This helps account for its higher affinity in red cells. Do not claim purified foetal protein must always have greater affinity under every artificial solution condition. The primary study of human foetal and adult haemoglobin interfaces examines how subunit interactions differ.

In an invented comparison at the same oxygen pressure, adult sites are 50% saturated and foetal sites 70% saturated. With equal capacities of 200 oxygen units, the bound amounts would be 100 and 140 units. This difference is not a universal 40-unit transfer at a real placenta; it isolates affinity while holding capacity fixed. Actual transfer also depends on partial-pressure gradients, perfusion and diffusion conditions.

AHL: Carbon dioxide promotes unloading through the Bohr shift

Actively respiring tissues produce carbon dioxide. Increased carbon dioxide and the accompanying increase in hydrogen-ion concentration lower haemoglobin's oxygen affinity, shifting the oxygen dissociation curve to the right under otherwise comparable conditions. At the same oxygen partial pressure, saturation is lower and more oxygen can be released to tissues. In the lungs, removal of carbon dioxide favours the reverse conditions and oxygen loading.

Carbon dioxide can bind at sites on globin distinct from the oxygen-binding haem sites, contributing to allosteric regulation. It also changes hydrogen-ion conditions through the carbon dioxide–bicarbonate system. Do not describe it as carbon monoxide competing with oxygen for the same haem site, or say that haemoglobin pumps oxygen out using ATP. The Bohr shift changes binding equilibrium; oxygen then diffuses down its gradient.

An invented blood sample enters a tissue at 96% saturation. At the tissue oxygen pressure, a lower-carbon-dioxide condition gives 72% saturation while a higher-carbon-dioxide condition gives 54%. Unloading is 24 versus 42 percentage points of total capacity. The latter is 18 percentage points greater, or $42/24 = 1.75$ times as much under these assumptions. Keep the same starting saturation and capacity in this comparison; differing blood flow or haemoglobin concentration would require additional information.

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Practice

All numerical values below are invented models or datasets. Explain mechanisms as well as giving numbers.

1. Explain why a larger multicellular organism faces exchange challenges. Link four properties of a gas-exchange surface to their functions.
2. Distinguish ventilation, perfusion, diffusion and cellular respiration. Explain how a lung can lose an oxygen gradient even while its wall remains thin.
3. In a controlled transfer model, surface area is multiplied by 3 and diffusion distance by 1.5 while the gradient remains constant. Predict the rate relative to the original and state a limitation.
4. Trace air from trachea to alveoli. Explain bronchiole branching, alveolar area, capillaries and surfactant. Compare inspiration, quiet expiration and forced expiration.
5. A reference trace has normal expiration at 0.9 L, normal inspiration at 1.5 L, maximal inspiration at 3.9 L and maximal expiration at 0.3 L. Calculate tidal volume, both reserves and vital capacity. Explain why residual volume is unknown.
6. Compare 0.60 L breaths at 10 per minute with 0.40 L breaths at 15 per minute. Assume dead space 0.15 L per breath. Calculate minute and estimated alveolar ventilation; explain why neither is oxygen uptake.
7. Label a typical terrestrial dicot leaf tissue plan. Trace carbon dioxide into mesophyll; explain stomata, air spaces, cuticle and veins.
8. Explain humidity, wind, temperature and light effects on transpiration, including possible stomatal closure. Uptake changes from 0.24 to 0.36 mL in equal 12-minute periods. Calculate rates and percentage increase; state a measurement limitation.
9. Four equal 0.15-square-millimetre fields have 18, 24, 21 and 27 stomata. Calculate density. Repeat for 30 stomata in a 0.50-mm-diameter circular field. Improve sampling reliability.
10. One field has 20 stomata over 0.10 square millimetres and another 80 over 0.40 square millimetres. Calculate pooled density and explain why doubling the field diameter changes area fourfold rather than twofold.
11. Critique three claims: plants only absorb carbon dioxide; stomatal density proves photosynthesis rate; breathing rate proves oxygen uptake.
12. **AHL:** Explain haemoglobin cooperativity and the axes of an oxygen dissociation curve. A left-shifted sample has 80% saturation and a right-shifted sample 60% at the same oxygen pressure. With equal 150-unit binding capacities, calculate bound amounts and explain affinity.
13. **AHL:** Compare foetal and adult haemoglobin, their subunits and placental oxygen transfer. Explain why a 70% saturated sample need not have every molecule carrying the same number of oxygen molecules.

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14. **AHL:** Blood enters at 98% saturation. At a fixed tissue oxygen pressure, low-carbon-dioxide blood leaves at 74% and high-carbon-dioxide blood at 50%. Calculate unloading in percentage points, the difference and ratio. Explain the Bohr shift without confusing carbon dioxide with carbon monoxide.

Worked answers

1. Larger similar shapes have lower area/volume and longer internal distances. Exchange requires large area for parallel transfer, thin tissue for short distances, moisture for dissolution, and permeability for crossing. Demand and transport also matter.
2. Ventilation moves air/water; perfusion moves blood; diffusion transfers molecules down gradients; cellular respiration supplies usable energy. Without renewal on either side, oxygen conditions approach equilibrium and net transfer falls even through a thin wall.
3. Relative rate $= 3/1.5 = 2$: twice the original. This assumes the same gas, barrier permeability and maintained gradient. A living system may have changing blood flow, pressure gradients or functioning area.
4. Trachea $\rightarrow$ bronchi $\rightarrow$ bronchioles $\rightarrow$ alveoli. Branching distributes air; alveoli provide area; thin walls shorten distance; capillary flow renews blood; surfactant stabilizes surfaces. Inspiration: diaphragm flattens, ribs rise, volume increases, pressure falls. Quiet expiration: relaxation/recoil. Forced expiration: abdominal and expiratory intercostal contraction.
5. Tidal $1.5 - 0.9 = 0.6$ L; inspiratory reserve $3.9 - 1.5 = 2.4$ L; expiratory reserve $0.9 - 0.3 = 0.6$ L; vital capacity $3.9 - 0.3 = 3.6$ L, also $2.4 + 0.6 + 0.6$. Relative expired-air measurements cannot establish absolute air remaining after maximal expiration.
6. Both minute values are 6.0 L per minute. Estimated alveolar ventilation is $(0.60 - 0.15)(10) = 4.50$ and $(0.40 - 0.15)(15) = 3.75$ L per minute. Airflow includes gases other than oxygen, and uptake across the barrier depends on gradients, diffusion, perfusion and blood binding.
7. Label cuticle, epidermises, palisade and spongy mesophyll, air spaces, vein, guard cells and stoma. Path: pore $\rightarrow$ air spaces $\rightarrow$ moist wall $\rightarrow$ cell membrane. Cuticle restricts water loss; stomata regulate access; air spaces distribute gases; xylem supplies water and phloem transports assimilates. Plans show tissues, not organelles.
8. Humidity reduces the vapour gradient; wind removes humid air; heat increases evaporative demand; light can promote opening. Water stress can close pores. Rates $0.24/12 = 0.020$ and $0.36/12 = 0.030$ mL per minute: $0.010/0.020 \times 100 = 50\%$ increase. Uptake approximates loss; leaks/storage can affect it. Control area/conditions and repeat.

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9. Total $90/0.60 = 150$ stomata per square millimetre. Radius 0.25 mm gives area 0.19635 square millimetres and density $30/0.19635 \approx 153$. Calibrate, use a boundary rule, sample unbiased fields, and repeat across leaves and independent plants.
10. Pooled density $100/0.50 = 200$ per square millimetre, matching both fields individually. Circular area scales with diameter squared: doubling diameter quadruples area.
11. Plants also respire. Net exchange depends on the balance. Photosynthesis also depends on pore size/opening and metabolic conditions. Breathing rate omits tidal volume, dead space, gas composition and transfer/perfusion evidence.
12. **AHL:** Oxygen binding changes conformation and increases subsequent binding affinity, producing a sigmoid rather than a straight line. Axes are oxygen partial pressure and percentage site saturation. Amounts $0.80(150) = 120$ and $0.60(150) = 90$ units. At the same pressure, the left-shifted sample has greater affinity under the specified conditions; this is not automatically greater tissue delivery.
13. **AHL:** Adult A is alpha-two/beta-two; foetal is alpha-two/gamma-two. Foetal haemoglobin's higher affinity under physiological red-cell conditions helps oxygen load across the placental barrier; blood normally remains in separate circulations. Different allosteric interactions contribute. Saturation is a population average over binding sites: 70% of sites occupied does not require identical occupancy of all four-site molecules.
14. **AHL:** Unloading is $98 - 74 = 24$ versus $98 - 50 = 48$ percentage points: 24 points more and twice as much. Higher carbon dioxide/hydrogen-ion conditions lower affinity, shifting the curve right. Carbon dioxide acts through distinct globin sites and acid–base conditions; it is not carbon monoxide competing at haem. Starting saturation and capacity are held constant.

Exam tips

Link structures to mechanisms; separate ventilation, diffusion and uptake. Subtract trace levels; calculate field area and count pores. Use independent plants.

- **AHL:** Compare at equal oxygen pressure. Separate saturation from content and percentage points from relative change. Explain both loading and unloading.

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Transport

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This unit covers B3.2 SL understandings plus additional higher-level content (marked AHL): blood vessels, pulse and coronary evidence, water transport and dicot tissue plans, tissue fluid and lymph, circulation and the heart, root pressure and phloem. Sections marked **AHL** are for Higher Level only.

Prerequisites: membranes, diffusion, osmosis, pressure and gas exchange. All numerical examples below are invented teaching data. Scope follows the IB Biology guide, first assessment 2025. An artery's direction, a vessel's wall and a fluid's pressure answer different questions; keep them separate.

Capillaries provide extensive thin exchange surfaces

A heart can move several litres of blood per minute, yet most body cells are not directly bathed in blood. Materials must cross vessel walls and the surrounding tissue fluid before entering cells. Capillaries connect the delivery system to these local exchanges.

Branching creates a large collective exchange area. Each capillary has a narrow lumen, bringing red cells close to its thin endothelial wall and keeping diffusion distances short. Many parallel branches give a large combined cross-sectional area, so average blood velocity is much lower than in the main arteries for the same total flow. Do not confuse one capillary's small area with the combined area of the whole capillary bed.

The endothelium is one cell layer thick, supported by a basement membrane. Permeability varies with capillary type. Fenestrations in some capillaries provide additional routes for rapid fluid and small-solute exchange, as in specialized kidney or intestinal regions. These are not unrestricted openings that normally let all blood cells pass. Dissolved substances can cross through suitable pathways; oxygen can diffuse through membranes. Size, charge, gradients and the barrier all influence exchange.

For a controlled comparison, flow volume per time equals average velocity multiplied by total cross-sectional area. If flow stays constant while total area rises fivefold, average velocity becomes one fifth. The slower passage can allow exchange time, but adequate gradients, perfusion and permeability are still required. A narrow vessel is not automatically a low-resistance pathway when considered individually.

Arteries and veins have different wall-to-lumen relationships

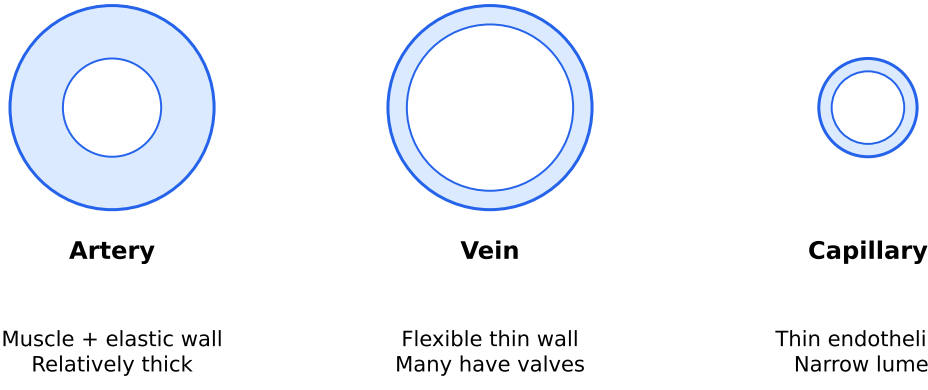
An artery carries blood away from the heart; a vein returns blood toward it. These definitions do not specify oxygen content: pulmonary arteries carry relatively oxygen-poor blood to lungs, while pulmonary veins return oxygenated blood. Vessel names follow direction.

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Feature	Artery compared with a similar-sized vein
Wall	Usually thicker, with more smooth muscle and elastic tissue relative to the lumen
Pressure	Usually higher and more pulsatile in the systemic arterial circuit
Lumen	Often smaller relative to the wall; compare matched vessel sizes
Vein return	Thinner, more compliant wall permits compression by surrounding skeletal muscles
Valves	Many veins, especially in limbs, have valves limiting backward flow; not every vein has them

Arterial walls withstand pressure and stretch as blood enters during ventricular ejection. Elastic recoil helps maintain pressure and flow between beats. Smooth muscle can alter vessel diameter and therefore resistance and distribution. This is not a second independent heartbeat in every artery.

Veins return blood at lower pressure. Skeletal muscle contraction can compress a vein; valves favour movement toward the heart and limit reverse flow when pressure changes. The vein wall's flexibility permits this compression. Valves do not themselves squeeze blood, and the heart, respiratory movements and pressure differences also contribute to return.



Compare equivalent vessel sizes and use several structural clues; colours do not identify oxygen content.

On a micrograph, compare wall thickness relative to lumen diameter, muscle/elastic tissue and whether the lumen appears collapsed. Section angle and processing can distort shape. A rounded lumen alone is insufficient evidence. In an invented matched comparison, A has wall thickness 0.30 mm and lumen diameter 1.20 mm; B has 0.10 mm and 2.00 mm. Wall/lumen ratios are 0.25 and 0.05, supporting A as the artery

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when other tissue features agree.

Pulse measurements estimate heart rate with stated sampling limits

An arterial pulse is a travelling pressure wave associated with ventricular ejection; it is not a discrete packet of blood taking one second to reach each location. In an approved classroom activity, lightly feel the radial pulse at the wrist with fingertips, count over a timed interval and convert to beats per minute. Use voluntary resting measurements and avoid pressing hard. Follow the institution's protocol; a sensor can provide a comparison method.

Counts of 18, 19 and 18 beats in three separate 15-second intervals give 72, 76 and 72 beats per minute. Their mean is about 73.3 beats per minute. One missed beat changes a 15-second estimate by 4 beats per minute, whereas one missed beat over 60 seconds changes it by 1. Longer sampling reduces this particular conversion error, although rate can genuinely vary over the interval.

Keep posture, rest period and timing method consistent. Repeat and report variation. Repeated measurements from one person estimate that person's variability and measurement repeatability, not differences among independent people. A pulse count does not directly measure stroke volume, blood pressure or cardiac output. Use supplied data if a physical measurement is unsuitable.

Coronary occlusion affects the heart muscle's own supply

Coronary arteries supply the myocardium with oxygen and nutrients. Blood inside a heart chamber cannot adequately supply the thick muscle wall by diffusion alone. Atherosclerotic plaque can narrow coronary arteries and limit flow. Plaque disruption can trigger a thrombus that acutely obstructs supply. Reduced oxygen delivery limits aerobic ATP production, impairing contraction; prolonged severe ischaemia can kill heart muscle. The NHLBI explanation of coronary disease distinguishes plaque-related disease from other coronary mechanisms.

Evaluate epidemiological claims with denominators, time periods and comparison groups. In an invented ten-year cohort, 30 of 600 participants in exposure group A develop an outcome, compared with 12 of 600 in B. Risks are 5% and 2%; the difference is 3 percentage points and the risk ratio is 2.5. A ratio is not a 250-percentage-point difference. Check age, smoking, activity, socioeconomic factors, measurement methods and loss to follow-up before interpreting the association.

A correlation coefficient describes the direction and strength of a specified relationship, not proof of causation. A strong positive correlation between a dietary exposure and coronary outcomes could be influenced by confounding. A weak linear correlation does not rule out every nonlinear relationship, poorly measured effect or subgroup pattern. Mechanistic evidence and well-designed controlled or longitudinal studies can strengthen causal evaluation; one food test or sample percentage cannot do that

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alone.

Absorbed nutrients enter transport after digestion

Digestion breaks large food molecules into smaller absorbable products. **Absorption** moves products across an exchange epithelium into the body. Enzymes act on substrates; transport proteins and gradients govern crossing. Large area and thin intestinal exchange surfaces support absorption. Sugars and amino acids can enter blood; much absorbed dietary lipid is packaged and initially enters intestinal lymph. These routes are not identical for every nutrient.

Carbohydrases yield smaller sugars; proteases yield peptides and amino acids; lipases yield fatty acids and monoacylglycerols during intestinal fat digestion. Bile aids emulsification and alkaline intestinal conditions but is not an enzyme. A positive Benedict test for reducing sugars, iodine test for starch, Biuret test for protein or ethanol emulsion test for lipid indicates a component under controlled conditions, rather than proving that a food causes health or illness. Use positive/negative controls, approved heating and keep ethanol away from flames. Nutrient identification, absorption and epidemiological causation are different levels of evidence.

Transpiration creates tension that draws water through xylem

At a moist mesophyll cell wall, evaporation replaces liquid water with vapour that can diffuse out through stomata. As water leaves wall pores, the curved air–water interfaces produce tension in the remaining liquid. Water is drawn from the xylem toward these walls, transmitting a pulling force through a cohesive water column. Cohesion between water molecules helps the column remain continuous; adhesion to hydrophilic walls also supports the system.

The xylem sap can be under negative pressure relative to atmospheric pressure. This is a pull transmitted through liquid, not a vacuum above every leaf and not a leaf muscle pushing water upward. Movement follows water-potential differences through the soil–plant–atmosphere pathway. The pressure component is one contribution; solute conditions also matter. Evaporation is powered by environmental energy, while regulation by living tissues influences the flow.

Xylem vessel elements are dead at maturity and lack cytoplasm and a plasma membrane. Their hollow lumens and absent or perforated end walls reduce resistance along the vessel. Lignified walls withstand tension and resist collapse. Pits are wall regions permitting lateral exchange between conducting elements and adjacent tissues; they are not simply large holes in every side wall without any intervening structure. Cavitation and embolism can interrupt conducting pathways, so continuity is a functional requirement rather than an absolute guarantee.

In an invented pressure comparison at the same height and solute conditions, a leaf xylem pressure of −0.8 MPa is lower than a root xylem pressure of −0.2 MPa. The pressure difference favours movement toward the leaf; the magnitude is 0.6 MPa.

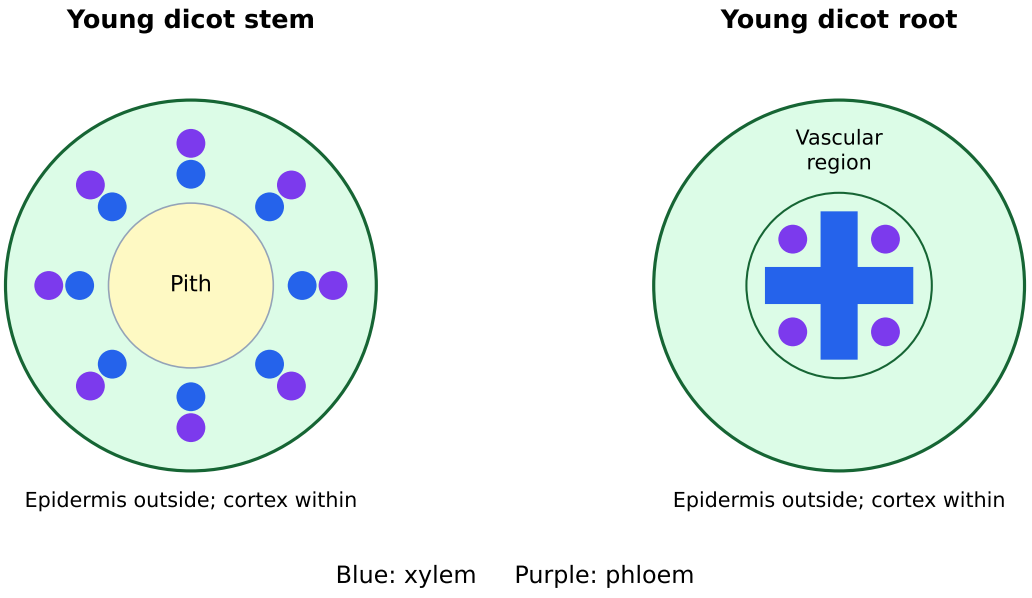
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This simplified comparison omits gravitational and other water-potential contributions. Water does not move upward because "negative numbers rise".

Dicot stem and root plans place transport tissues differently

A young typical dicot stem has an outer epidermis, cortex and vascular bundles arranged around a central pith. Within each bundle, xylem lies toward the centre and phloem toward the outside; a cambial region can lie between. Xylem carries water/minerals and supports the stem; phloem translocates organic solutes. Cortex and pith include storage and supporting tissues. Mature woody stems can look different after secondary growth.

In a typical young dicot root, the epidermis surrounds a broad cortex and the central vascular cylinder. Xylem often forms a central star or cross, with phloem between its arms. The endodermis surrounds the vascular cylinder and controls access through the apoplastic route. Do not copy the stem's ring of separate bundles into a root. Actual patterns vary with species and age; use the supplied micrograph rather than treating the schematic as universal.



Blue marks xylem and purple marks phloem. These plans show tissue distribution, not individual conducting cells.

Draw a low-power plan with continuous tissue boundaries, correct relative positions and clear labels. Annotate a tissue's function separately from its appearance. Do not draw every cell, sieve pore or nucleus in a tissue plan. Higher-power cellular drawings answer a different question. Calibrate any measurements: a 12-mm image of a 0.60-mm stem has magnification 20; a 3.0-mm cortex region in that image corresponds to 0.15 mm in the specimen.

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AHL: Tissue fluid connects capillaries with cells and lymph

Blood plasma contains water, ions, nutrients, dissolved gases, wastes and plasma proteins. Pressure can filter fluid and small solutes across capillary barriers to form tissue fluid. Red cells normally remain within vessels, and most large plasma proteins are retained more strongly than small dissolved substances. Tissue fluid therefore resembles plasma in many small solutes but usually has substantially less protein; it is not simply pure water or plasma with every component unchanged.

Oxygen and nutrients move between tissue fluid and cells down appropriate gradients or through membrane transport. Cells release carbon dioxide and metabolic wastes into this surrounding fluid. Hydrostatic pressure favours outward filtration; plasma proteins contribute an osmotic effect opposing loss. The guide's introductory model contrasts greater filtration near the arteriolar end with conditions more favourable for return as blood pressure falls toward venules.

Model limit: do not turn this into a universal claim that most filtered fluid continuously returns at every venous end. Modern evidence identifies the endothelial glycocalyx and shows sustained slight filtration in many tissues at steady state, with lymphatic return essential; reabsorption can be transient or occur in specialized settings. See Levick and Michel on revised microvascular exchange. The useful syllabus comparison is about pressure forces and possible exchange, rather than a guaranteed percentage recovered locally.

Excess tissue fluid enters thin-walled lymphatic capillaries through permissive endothelial openings. One-way entry arrangements and valves in collecting lymphatic vessels limit reverse flow; surrounding muscle and pressure changes aid movement. Lymph eventually returns to venous blood. If an invented tissue bed filters 4.0 mL per hour and directly reabsorbs 0.8 mL per hour, lymphatic removal must average 3.2 mL per hour for stable volume. If removal is only 2.5, accumulation is 0.7 mL per hour. These assumed flows illustrate conservation, not universal human rates.

AHL: Single and double circulation have different circuit sequences

In a bony fish, blood follows heart → gill capillaries → body capillaries → heart. It passes through the heart once per complete circuit. Pressure falls as blood crosses the gill bed before reaching systemic tissues. The main heart receives relatively oxygen-poor blood; oxygenation occurs downstream at gills.

In a mammal, blood follows right heart → lung capillaries → left heart → body capillaries → right heart. Two linked circuits mean blood passes through the heart twice per complete circuit, with repressurization after the pulmonary capillary bed. This supports a higher systemic pressure while keeping pulmonary pressure lower. Both sides normally pump equal average volumes over time; a thicker left ventricular wall does not mean it continuously pumps more blood than the right.

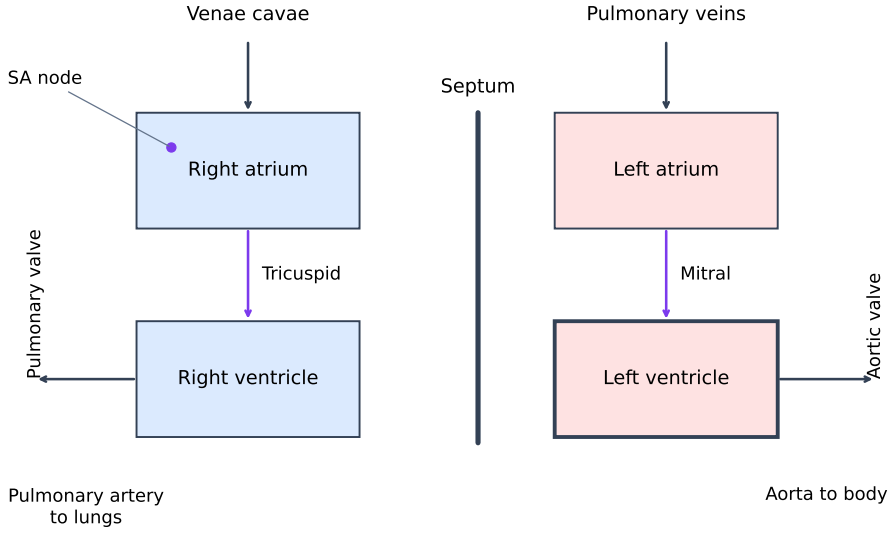
An artery is defined by movement away from the heart in either circuit. Do not label

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every fish vessel after gills as an artery based on oxygen alone, or interpret “double” as two unrelated blood supplies. A complete circuit must return to its starting point.

AHL: Heart structures maintain directed pressurized flow

In a frontal view, the person’s right usually appears on the viewer’s left. Venae cavae deliver systemic venous blood to the right atrium; the tricuspid atrioventricular valve leads to the right ventricle; the pulmonary semilunar valve leads to pulmonary arteries and lungs. Pulmonary veins return to the left atrium; the mitral atrioventricular valve leads to the left ventricle; the aortic semilunar valve leads to the aorta and systemic arteries.



Arrows show flow, not the shape or position of every anatomical vessel. The septum separates the right and left pumps.

Atria receive blood and help complete ventricular filling at relatively low pressure. Ventricles generate the pressures driving blood into arteries. Cardiac muscle contracts rhythmically; the left ventricular wall is thicker because the systemic circuit requires higher pressure, not because left-side blood is “heavier”. The septum separates the two sides and helps prevent mixing. Coronary vessels supply the myocardium itself.

The sinoatrial node in the right atrial wall initiates the normal heartbeat. Excitation spreads through atrial muscle, followed by coordinated conduction to the ventricles; an atrioventricular delay helps atrial contraction precede ventricular contraction. Valve leaflets respond to pressure differences. Tendinous cords and papillary muscles restrain atrioventricular valve inversion during ventricular contraction; they do not pull the valves open. Semilunar valves have a different structure and prevent return from arteries when ventricular pressure falls.

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AHL: Pressure differences explain the cardiac cycle

The left atrium contracts late in ventricular diastole, adding to ventricular filling through the open mitral valve. As the ventricle contracts, its pressure exceeds atrial pressure and closes that valve. Initially both valves are closed, so pressure rises without ejection. When ventricular pressure exceeds aortic pressure, the aortic valve opens and blood is ejected. As the ventricle relaxes, its pressure falls below aortic pressure and the aortic valve closes. Both valves are briefly closed again. When ventricular pressure falls below atrial pressure, the mitral valve opens and filling resumes.

Systole is contraction; diastole is relaxation. The systolic and diastolic values in an arterial blood-pressure reading are the higher and lower pressures during the cycle in that artery, not pressures measured in the left atrium and right ventricle. An invented aortic trace ranging from 78 to 122 mmHg corresponds to 122/78 mmHg. These teaching values are not a diagnosis or treatment threshold.

At one model instant, left atrium is at 10, ventricle at 6 and aorta at 80 pressure units: the mitral valve is favoured open and the aortic valve closed. At another, atrium is 8, ventricle 100 and aorta 90: mitral closed, aortic open. Infer state from the pressure differences, not from whether "all muscles contract together". With 70 beats per minute and 75 mL ejected per beat, cardiac output is 5,250 mL per minute, or 5.25 L per minute. This is each side's average output in a stable double circulation, not a sum of both sides giving twice the systemic flow.

AHL: Root pressure can supplement transpiration-driven flow

Living root cells can use energy-dependent transport to accumulate mineral ions in the vascular region. Lower solute potential favours osmotic entry of water; this can generate positive pressure in xylem. Root pressure can assist movement when transpiration is limited, including humid conditions or some spring situations before a full canopy develops. It is not a universal replacement for cohesion–tension in tall, actively transpiring plants.

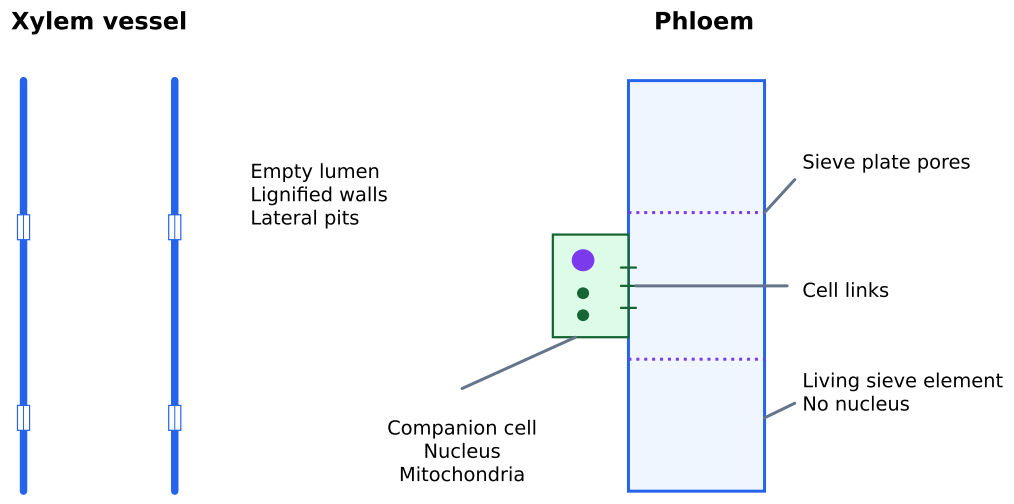
Separate the energy-dependent ion movement from water movement. Ions may be transported against electrochemical gradients using cellular energy directly or indirectly; water enters down a water-potential gradient rather than being pumped by an ATP-driven water motor. Positive root pressure can contribute to exudation or guttation under suitable conditions. Guttation droplets at leaf margins are liquid xylem-derived water, unlike condensation of atmospheric water as dew. Species and conditions determine whether a visible effect occurs.

AHL: Phloem links source loading with sink unloading

Mature sieve tube elements are living conducting cells with reduced cytoplasm, few organelles and no nucleus. End walls form perforated sieve plates, permitting sap passage between elements. Adjacent companion cells retain a nucleus and many mi-

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tochondria; plasmodesmata connect them with sieve elements. This division of labour supports metabolism and transfer while leaving a relatively unobstructed conducting pathway.



Xylem vessels are dead at maturity; sieve tube elements remain living and rely on companion-cell support.

A source supplies organic solutes, such as a photosynthesizing leaf or a mobilizing storage organ. A sink uses or stores them, such as a growing root, fruit or developing bud. Loading solutes at a source can lower sieve-tube water potential, draw in water and raise hydrostatic pressure. Unloading at a sink helps maintain a lower pressure there, allowing bulk sap flow down the pressure gradient. Energy-dependent loading occurs in many systems, but not every species uses exactly the same loading pathway.

The ATP-dependent steps support solute handling; they do not mean each sucrose molecule is individually carried by a pump down the entire tube. Different sieve tubes can carry sap in different directions according to their source–sink connections. An organ's role can change: a storage root can be a sink during accumulation and a source during later mobilization. "Phloem always moves downward" is therefore unreliable.

Practice

All numerical examples are invented. Explain structures and mechanisms alongside calculations.

1. Explain four capillary exchange adaptations: branching, lumen, endothelium and fenestrations. At constant flow, predict velocity when combined cross-sectional area increases eightfold.

2. Compare arterial/venous direction, walls, lumens, pressures and return mechanisms. Wall/lumen measurements are A: 0.24/0.96 mm; B: 0.08/1.60 mm. Calculate ratios, identify the likely artery and state a limitation.
3. Three 20-second counts are 23, 24 and 25 beats. Calculate rates and mean. Quantify one counting error; explain the replication limit.
4. Ten-year outcomes: 24/400 exposed versus 8/400 comparison participants. Calculate risks, difference and ratio. Explain coronary occlusion and two limits on causal inference.
5. Distinguish digestion, absorption and transport, with blood/lymph examples. Why does a food test not establish a health outcome?
6. Trace mesophyll evaporation to upward xylem flow. Explain four xylem vessel adaptations.
7. Compare young dicot stem/root tissue plans. An 18-mm image represents a 0.90-mm specimen; an image cortex region measures 4 mm. Calculate magnification and actual region width.
8. Compare −0.9 and −0.3 MPa xylem pressures under otherwise equal conditions. State flow tendency, pressure difference and one model limit.
9. Correct: every vein has valves; arteries always carry oxygenated blood; one narrow capillary means fast flow through the whole bed.
10. Plan reliable micrograph vessel identification and resting pulse measurement: controls, replication and limits.
11. Distinguish bulk flow from membrane crossing, using nutrients, tissue supply or plant water movement.
12. **AHL:** Compare plasma, tissue fluid and lymph. Explain pressure filtration, the limit of the simple venous-return model and lymphatic drainage. For filtration 5.0, direct reabsorption 0.6 and lymph removal 3.7 mL per hour, calculate accumulation.
13. **AHL:** Trace complete fish and mammalian circuits, then name the four mammalian valves and the direction across each. Compare valve states at left atrium/ventricle/aorta pressures 9/5/82 and 7/110/95. Calculate cardiac output at 72 beats per minute and 80 mL per beat; interpret a 124/76 mmHg arterial reading.
14. **AHL:** Explain root pressure and source-to-sink phloem transport. Compare xylem vessels, sieve elements and companion cells; explain why water is not directly pumped using ATP and why a storage organ can change its transport role.

Worked answers

1. Branching increases collective exchange area; narrow lumens bring red cells close to walls; one-cell-layer endothelium shortens distance; fenestrations support selected rapid exchange in some capillaries. At constant flow, eightfold total area gives one-eighth average velocity. Barrier type and gradients still matter.

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2. Arteries carry away; veins return. Ratios: $0.24/0.96 = 0.25$ and $0.08/1.60 = 0.05$. A fits an artery's thicker muscular/elastic wall. Compliant veins permit muscle compression; valves in many veins limit backflow. Compare matched sizes and account for section angle/collapse.
3. Rates $23(3) = 69$, $24(3) = 72$, $25(3) = 75$ beats per minute; mean 72. One missed beat alters a 20-second estimate by 3 beats per minute. Repeats from one person measure within-person variation and repeatability, not independent population replicates.
4. Risks $24/400 = 6\%$ and $8/400 = 2\%$; difference 4 percentage points; ratio 3. Coronary narrowing or a thrombus reduces myocardium supply, limiting aerobic ATP and potentially causing cell death. Confounding such as age/smoking and biased exposure/outcome measurement can affect association; correlation alone does not demonstrate causation.
5. Digestion produces absorbable products; absorption crosses an epithelium; bulk transport distributes material. Sugars/amino acids enter blood; much packaged lipid initially enters intestinal lymph. Food tests detect components, not causal health outcomes. Bile assists fat processing but is not an enzyme.
6. Evaporation generates wall-pore tension, pulling water from xylem through cohesive sap. Hollow dead elements remove obstructions; open/perforated ends permit longitudinal flow; lignin resists collapse; pits permit lateral exchange. Adhesion supports wall contact. Evaporation, not ATP pumping within vessels, drives the pull.
7. Stem: peripheral bundles, xylem inward/phloem outward, cortex and central pith. Root: central often star-shaped xylem, phloem between arms, surrounding cortex/epidermis. Magnification $18/0.90 = 20$; actual width $4/20 = 0.20$ mm. Use the actual section because species and age alter patterns.
8. The -0.9 MPa region has lower pressure; the difference has magnitude 0.6 MPa. Under the stated conditions pressure favours flow toward it. Whole-plant movement also depends on height, solute conditions, resistance and continuity of the pathway.
9. Many veins have valves but not all. Pulmonary arteries are relatively oxygen-poor; direction defines an artery. Each capillary is narrow, but many parallel branches have large combined area, lowering average velocity at the same total flow.
10. Match vessel size, compare wall/lumen and tissues, calibrate and note section distortions. For pulse standardize rest, posture, timing and method; repeat and compare a sensor. Neither method alone gives oxygen content, stroke volume or blood pressure. Separate within-person repeats from independent participants.
11. Blood flows in bulk down pressure gradients. Membrane crossing depends on permeability, gradients and transport proteins/energy coupling. Nutrients reaching capillaries still need tissue/cell exchange. Xylem bulk flow likewise differs from water entry across living root membranes.
12. **AHL:** Tissue fluid has many plasma small solutes but generally much less pro-

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tein and normally no red cells. Hydrostatic pressure promotes filtration; retained proteins oppose fluid loss. Venous-end reabsorption is not universal steady-state recovery: glycocalyx effects and lymphatic return matter. Thin lymphatic entry routes and one-way arrangements collect excess fluid, returning it to venous blood. Accumulation $5.0 - 0.6 - 3.7 = 0.7$ mL per hour.

13. **AHL:** Fish: heart → gills → body → heart. Mammal: right heart → lungs → left heart → body → right heart. Tricuspid: right atrium to ventricle; pulmonary: right ventricle to pulmonary artery; mitral: left atrium to ventricle; aortic: left ventricle to aorta. At 9/5/82, mitral open/aortic closed; at 7/110/95, mitral closed/aortic open. Output $72(80) = 5,760$ mL per minute $= 5.76$ L per minute per side, not double that systemic flow. 124/76 gives the higher/lower arterial pressures during the cycle.
14. **AHL:** Ion accumulation in roots lowers solute potential, drawing water osmotically and generating positive pressure. Dead hollow lignified xylem conducts water; living enucleate sieve elements conduct sap through sieve plates; nucleated companion cells with mitochondria support metabolism and loading/unloading through connected pathways. Source loading and sink unloading sustain pressure-driven bulk phloem flow. Water follows water-potential gradients rather than direct ATP pumping. A storage organ can shift from receiving reserves to mobilizing them for growth.

Exam tips

Name vessel direction and tissue function. Compare like-for-like measurements; separate sample repeats from independent participants and bulk flow from membrane crossing.

- **AHL:** Infer valves from adjacent pressures. Distinguish each side's output from double-counted flow. State the limits of tissue-fluid models and separate ion/solute handling from bulk water movement.

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Muscle and motility

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

B3.3 Muscle and motility is a Higher Level-only unit. It covers movement in motile and sessile organisms, sliding filaments, titin and antagonists, motor units, skeletal levers, the human hip and joint-angle measurement, intercostal action, reasons for locomotion and swimming adaptations of marine mammals.

Prerequisites: protein structure, ATP, cell membranes, differentiated muscle fibres and ventilation. Numerical examples and datasets are invented teaching models. Scope follows the IB Biology guide, first assessment 2025.

AHL: Movement does not always mean locomotion

A rooted tree stays in one place, but its growing shoots can change direction and materials move within its cells. A swimming protist can relocate its whole cell. Both illustrate biological movement at different scales. Locomotion is movement of an organism from one location to another; it is one form of movement rather than a requirement for every organism to walk or swim.

The motile protist Paramecium caudatum swims by coordinated beating of cilia. A sessile English oak, Quercus robur, remains rooted while shoots can bend during growth toward light. Growth, intracellular transport and chromosome movement do not require skeletal muscle. Sessile does not mean motionless at every scale.

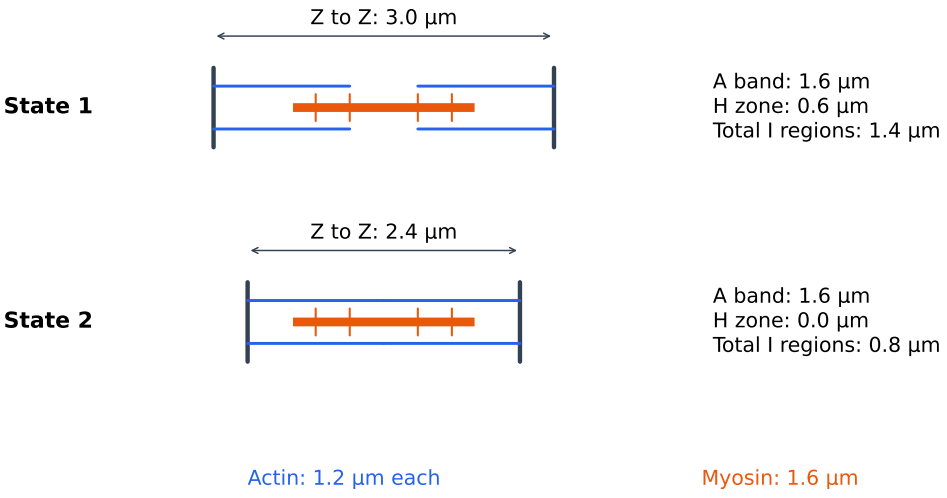
Explain the task and mechanism: cilia push against water; muscle fibres develop tension; joints permit relative bone movement. “For movement” alone explains none of these mechanisms.

AHL: Sarcomeres shorten by sliding constant-length filaments

A **sarcomere** is a repeating contractile unit between adjacent Z lines in a myofibril. Thin actin filaments attach toward the Z lines and overlap a central arrangement of thick myosin filaments. Myosin heads can interact with actin, forming a **cross-bridge** . Repeated interactions can move thin filaments relative to thick ones, bringing the Z lines closer during shortening.

The A band corresponds to the thick-filament length and stays approximately constant in the sliding model. The I band contains thin filaments without thick-filament overlap; it narrows as overlap increases. The H zone is the central part of the A band without thin-filament overlap; it also narrows and may disappear. Increased overlap does not mean actin becomes a thicker molecule or that every myosin filament is shortened.

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Compare filament ends and Z-line positions separately. The model illustrates shortening, not every possible state of a contracting muscle.

In the invented model, the sarcomere changes from 3.0 to 2.4 micrometres while each thin filament remains 1.2 micrometres and the thick filament remains 1.6 micrometres. Percentage shortening is $(3.0-2.4)/3.0\times100 = 20\%$. The thick-filament-length change is zero. The two quantities answer different questions.

Compare images at the same calibrated scale and identify Z lines and band boundaries. Uniform filament shortening would contradict sliding. Static images support structural comparison, not direct observation of the ATP cycle. Contraction can produce tension without overall shortening under a load.

AHL: Calcium and ATP control the cross-bridge cycle

In a resting skeletal muscle fibre, regulatory proteins restrict myosin access to binding sites on actin. Excitation leads to release of calcium ions from the sarcoplasmic reticulum. Calcium binding to troponin changes the position of tropomyosin, permitting actin–myosin interaction. Calcium does not supply the energy for the power stroke or become a permanent part of an actin filament.

A useful simplified sequence is:

1. An energized myosin head carrying ADP and phosphate can attach to exposed actin.
2. Strong binding and phosphate release are coupled to a conformational change, the power stroke; actin moves relative to the thick filament. ADP is released.
3. A new ATP molecule binds to myosin, allowing detachment from actin.
4. ATP hydrolysis re-energizes and repositions the head for another cycle when suitable sites are available.

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Distinguish ATP binding from ATP hydrolysis. Binding is needed for detachment; hydrolysis supports preparation for subsequent force generation. Calcium removal by energy-dependent transport into the sarcoplasmic reticulum allows regulatory blocking to be restored. Loss of a nerve signal does not instantly remove every calcium ion or mean that relaxation requires no energy-related processes.

Many heads cycle asynchronously, helping maintain tension. Load, filament geometry and regulation influence activity; the model does not place every head in the same stage simultaneously.

AHL: Titin and antagonists contribute different restoring forces

Titin is a very large elastic protein associated with sarcomeres. It helps stabilize thick-filament position, resists excessive stretch and contributes passive restoring force after extension. Its role differs from that of a myosin head cycling on actin. Elastic recoil depends on prior deformation; titin does not actively push two bones apart like a second motor.

Skeletal muscle develops tensile force. To reverse movement at many joints, an opposing muscle or muscle group can pull in the other direction. During elbow flexion, flexor activity can increase while extensors lengthen; reversing the pattern supports extension. Real movement often includes co-contraction for stability, so "one always completely switches off" is too simple.

When excitation declines and cross-bridge activation falls, a fibre can be lengthened by its load, antagonist activity or elastic forces. Distinguish loss of active tension from restoration of the whole joint position. A muscle does not normally push its tendon forward to reverse a previous pull.

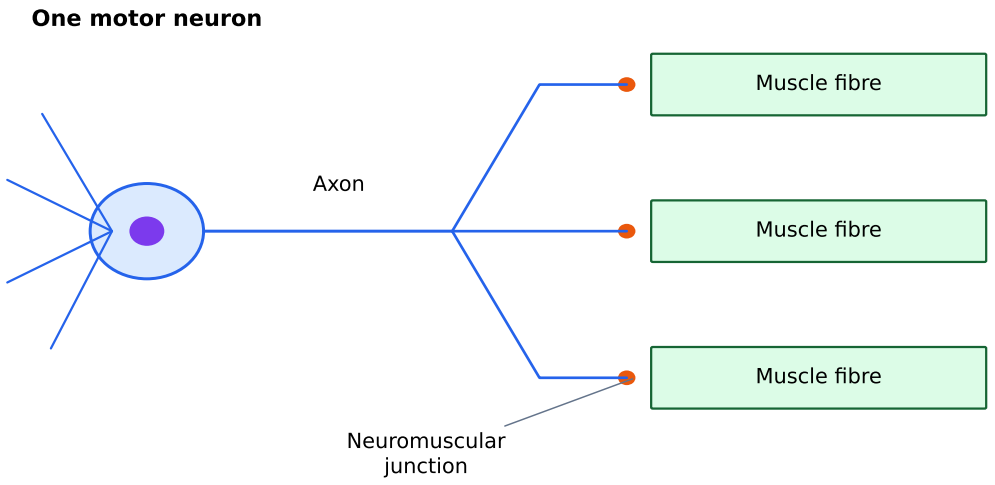
Exam tips

Name the changing structure, ATP step and angle convention. Use perpendicular moment arms. Distinguish relaxation from opposing active movement, and link adaptations to mechanisms and tasks.

AHL: A motor unit links one neuron to several fibres

A skeletal muscle motor unit consists of one motor neuron and all the muscle fibres it innervates. The neuron's axon branches to neuromuscular junctions on those fibres. A muscle fibre is a long multinucleate cell; a motor unit is a neuron-plus-fibres functional group, not another name for one sarcomere or one myofibril.

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The junctions belong to branches of the same neuron. Other motor units within the muscle are omitted.

At a neuromuscular junction, a nerve impulse promotes acetylcholine release. It crosses a synaptic gap and acts on muscle membrane receptors. Muscle excitation then triggers calcium release; the cell membranes do not merge.

Recruitment of additional motor units can increase whole-muscle force. Changing stimulation frequency also changes how twitches overlap and develop tension. Motor units with fewer fibres permit smaller increments and finer control, while larger units can add greater force increments, other conditions equal. Recruitment does not mean that each fibre must shorten by a larger percentage.

In an invented equal-force-per-fibre model, units A, B and C contain 4, 9 and 16 fibres. Activating A gives 4 force units; A plus B gives 13; all three give 29. The successive increments are 4, 9 and 16. Actual units can differ in fibre properties and activation, so counting fibres alone does not generally determine real force.

AHL: Skeletons anchor muscles and act as levers

A vertebrate endoskeleton lies within the body; an arthropod exoskeleton surrounds and supports it externally. Muscles attach to appropriate skeletal structures and generate forces across joints. Either arrangement can provide anchorage and lever systems; an internal skeleton is not the only way to transmit muscle force.

At a skeletal lever, a joint serves as a pivot, muscle tension supplies an effort and the limb or external object provides a load. Tendons transmit force from muscle to bone. The turning effect depends on force multiplied by the perpendicular distance from its line of action to the pivot. In a simplified static balance:

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$$F_{\text{muscle}} d_{\text{muscle}} = F_{\text{load}} d_{\text{load}}.$$

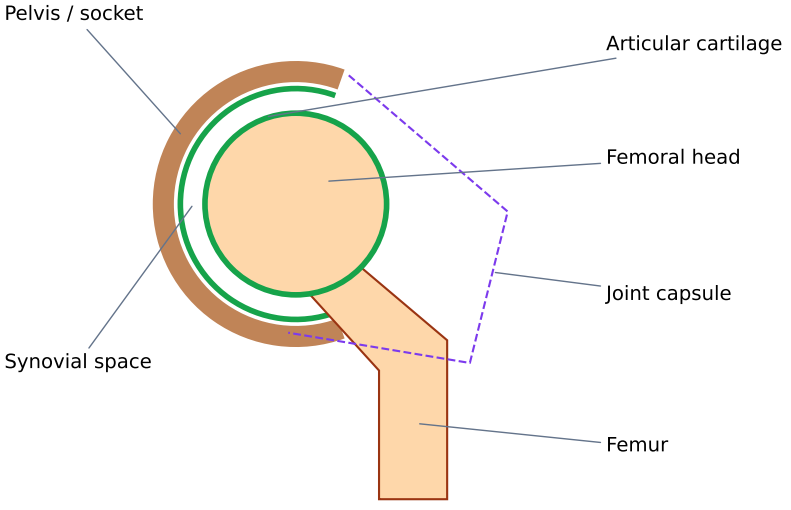
A model load of 20 N at 0.25 m creates a moment of 5.0 N metres. If the muscle's perpendicular moment arm is 0.025 m, its tension must be $5.0/0.025 = 200$ N when other moments are neglected. A small muscle moment arm can require much greater force than the load itself. This geometry can provide movement speed or displacement advantages, rather than demonstrating that the muscle is ineffective.

Use perpendicular moment arms, not automatically the full length of a bone or tendon. A real limb also has its own weight, changing geometry and dynamic forces. Tendons join muscles to bones; ligaments primarily connect bones across joints. Confusing these structures breaks the force pathway.

AHL: The human hip combines mobility with load support

The hip is a synovial ball-and-socket joint. The rounded head of the femur fits into the acetabulum of the pelvis. This arrangement permits motion in several planes while supporting loads. It does not mean the femur can rotate without restriction in every direction.

Articular cartilage covers the articulating bone surfaces, distributes contact loads and helps reduce friction. Synovial fluid lubricates the joint, and the surrounding capsule encloses the joint space. Ligaments connect skeletal parts and limit excessive movement; tendons transmit muscle tension to the skeleton. Muscles crossing the joint create movements when their lines of action produce appropriate turning effects.



The diagram separates tissues to show their roles. It is not a medical image or a measurement of a person's joint.

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Hip flexion/extension move the thigh forward/back relative to the pelvis; abduction/adduction move it away from/toward the midline. Internal/external rotation turn the femur around its long axis. Use the same anatomical reference position and explain the muscle groups' lines of action.

AHL: Measure joint range in named planes

A goniometer or computer analysis of images can estimate joint angles. Identify the joint centre and consistent landmarks along the adjoining segments. State the reference position and which included angle or anatomical movement angle is recorded. An included angle between segments can decrease while the named flexion angle increases, depending on the convention.

Use approved existing images, models or comfortable voluntary movements under supervision. Do not force maximal range or use painful/exhaustive tasks. Align the viewing plane with the movement plane, keep camera position and landmarks consistent, and repeat measurements. A two-dimensional photo can distort a three-dimensional joint position; clothing, soft-tissue landmarks and observer choices add uncertainty.

In an invented dataset, comfortable flexion from a defined neutral position reaches 105 degrees and extension reaches 15 degrees in the opposite direction. The total measured arc in that plane is 120 degrees. Abduction 35 degrees and adduction 20 degrees give a 55-degree arc in a different plane; these values should not be added to make one "175-degree hip range". Each describes a specified movement and method.

For a separate image example, the defined included angle changes from 150 to 95 degrees, a 55-degree change. Report the convention before calling this a final anatomical flexion angle. Repeated measurements 54, 56 and 55 degrees have mean 55 and range 2 degrees; this range describes repeat spread, not a proven absolute accuracy of plus or minus one degree.

AHL: Intercostal muscle orientation supports antagonistic actions

External and internal intercostal layers have different fibre orientations. In the simplified ventilation comparison, contraction of the external intercostals raises ribs up and out during inspiration; expiratory parts of the internal intercostal layer can draw ribs down during active expiration. Their geometry allows opposing movements. Relaxation and elastic recoil account for much quiet expiration, so do not claim forceful internal intercostal contraction is required for every resting out-breath.

Contraction of one layer can stretch the opposing muscle system, storing elastic energy associated with proteins such as titin. This links antagonistic action with passive recoil, while neural control and the diaphragm coordinate breathing. At the chest, skeletal muscles generate movement of ribs; the subsequent pressure difference moves air. Rib movement itself is not oxygen diffusion into the blood.

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AHL: Locomotion provides opportunities with costs

Foraging can move an animal to food: a dolphin can search for and pursue fish. Escape movement can reduce predation risk: a gazelle can run from a pursuing predator. Mate searching can bring reproductive partners together, as when male moths move toward pheromone sources. Migration can connect seasonally useful habitats: many humpback whale populations move between feeding and breeding regions. The NOAA humpback account describes this seasonal pattern; it is not a claim that every individual follows an identical journey.

Movement costs energy and may increase exposure to predators or hazardous conditions. Its benefits depend on context. A faster animal does not automatically obtain more food if extra travel costs exceed gains. In an invented comparison, route A yields 120 energy units of food at a travel cost of 35, leaving 85; route B yields 150 at a cost of 80, leaving 70. The route with the greater gross intake has the smaller net gain under this limited model. Handling costs, risk and nutrient composition are omitted.

AHL: Cetacean adaptations support swimming and periodic air breathing

A bottlenose dolphin provides a named marine mammal example. Its streamlined body reduces resistance to movement through water, while forelimbs modified as flippers support control and steering. Muscles in the posterior body drive movements of the horizontal tail flukes up and down, generating propulsion. Do not infer that a dolphin has fish gills or that every marine mammal has the same tail arrangement: seals and sea lions differ from cetaceans.

The blowhole provides an airway opening on the upper head, allowing breathing when it reaches the surface; it closes during submergence. A dolphin remains a lung-breathing mammal and must periodically surface for air. External access and control of the airway support the transition between swimming submerged and breathing at the surface. The opening is not extracting dissolved oxygen from water while closed.

Connect streamlining to drag, flukes to thrust, flippers to control and the airway to air breathing. These adaptations evolved across generations; an individual does not acquire them because it decides to live in water.

Practice

All numerical examples are invented. Use the stated models and units.

1. Contrast movement and locomotion in Paramecium and a sessile tree. Must movement involve skeletal muscle?
2. Define sarcomere, A band, I band and H zone. Predict changes during shortening and identify unchanged lengths.

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3. A sarcomere shortens from 2.5 to 2.0 micrometres; its thick filament stays 1.6 micrometres. Calculate percentage shortening of each. What makes an image comparison valid?
4. Explain a cycle of attachment, power stroke, ATP binding/detachment and hydrolysis/re-energization. Include calcium regulation and ATP's roles in relaxation.
5. Compare titin recoil with antagonist contraction. Why does removing excitation not necessarily restore a joint's original position?
6. Define motor unit and trace its signal to actin–myosin interaction. In an equal-force-per-fibre model, X, Y, Z contain 5, 8, 12 fibres. Calculate cumulative forces for X, X + Y, X + Y + Z, their increments and a model limit.
7. Compare arthropod and vertebrate muscle anchorage. A 30 N load acts at 0.20 m; muscle moment arm is 0.030 m. Calculate load moment and static tension; name an omitted force.
8. Explain the hip's bones, cartilage, synovial fluid, ligaments, tendons and muscles. Name three kinds of movement.
9. Flexion from neutral reaches 100 degrees; opposite extension reaches 20. Find the total arc. Why not add a separate 50-degree abduction/adduction arc? An included angle changes from 145 to 90 degrees: find the change and its interpretation limit.
10. Image readings are 53, 55, 57 degrees. Find mean and range. Plan camera/landmark controls; does repeat spread establish accuracy?
11. Explain intercostal orientation, antagonism and elastic energy. Distinguish active/quiet expiration and rib movement/gas diffusion.
12. Give named examples of foraging, escape, mate search and migration. Compare net gains: C intake 140/cost 45; D intake 180/cost 100. State two limitations.
13. Explain a bottlenose dolphin's streamlining, flippers, flukes and blowhole. Correct: "All marine mammals have identical tails and extract dissolved oxygen through their blowholes."

Worked answers

1. Paramecium relocates its whole cell using cilia: locomotion. A rooted tree can move parts during growth and has internal transport without relocating the whole organism. Movement ranges across scales and can use mechanisms other than muscle.
2. Sarcomere: Z-line to Z-line unit. A band spans thick filaments; I band has thin filaments without thick overlap; H zone has thick filaments without thin overlap. During shortening, Z lines approach, overlap increases, I bands/H zone narrow, and the A band and individual filament lengths remain approximately constant.

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3. Sarcomere shortening $(2.5 - 2.0)/2.5 \times 100 = 20\%$; thick-filament change zero. Calibrate scales and identify boundaries; band width is not filament length, and static images do not show ATP cycling.
4. Energized myosin attaches; force-generating conformational change moves actin; fresh ATP binds and permits detachment; ATP hydrolysis re-energizes the head. Calcium binds troponin, changing tropomyosin position and access to actin. Calcium reuptake and regulation restore resting conditions; detachment itself needs ATP binding. Heads cycle asynchronously.
5. Titin supplies passive recoil and resists overstretching; an antagonist actively pulls oppositely. Muscle pulls, not pushes. Joint position also depends on load, elasticity and coordination, including co-contraction.
6. One motor neuron plus its innervated fibres. Junction excitation leads to fibre excitation, calcium release and myosin access. Model forces: 5, 13, 25; increments: 5, 8, 12. Real force also depends on fibre properties, frequency and load.
7. Arthropod muscles act on an exoskeleton; vertebrate muscles act on an endoskeleton. Moment $30(0.20) = 6.0$ N metres; tension $6.0/0.030 = 200$ N. Use perpendicular distances. Limb weight, other muscle forces and acceleration are omitted.
8. Femoral head fits the pelvic acetabulum. Cartilage reduces friction/distributes contact loads; synovial fluid lubricates; ligaments connect bones and restrain excess motion; tendons transmit muscle force to bone. Muscles generate joint moments. Flexion/extension, abduction/adduction and axial rotation are distinct movements.
9. Flexion/extension arc $100 + 20 = 120$ degrees. The other plane's 50-degree arc is a separate measurement. Included-angle change $145 - 90 = 55$ degrees; final anatomical flexion still needs the reference/convention.
10. Mean 55 degrees; range $57-53 = 4$ degrees. Standardize viewing plane, landmarks, joint centre, reference and camera; repeat/compare observers. Agreement can coexist with systematic perspective or landmark error.
11. The layers' different fibre orientations allow opposing rib movements; contraction can stretch the opposing layer and store elastic energy including in titin. External intercostals support inspiration; expiratory internal portions can support active expiration. Quiet expiration is largely relaxation/recoil. Rib motion changes thoracic volume/pressure, causing bulk airflow; gases diffuse separately across exchange surfaces.
12. Dolphin food pursuit; gazelle escape; moth pheromone-guided mate search; seasonal humpback movement between feeding/breeding habitats. C gives $140 - 45 = 95$ units; D gives $180 - 100 = 80$. Greater intake does not ensure greater net gain. Risk, handling cost, nutrient quality and other demands are not represented.
13. Streamlining reduces drag; flippers help control; up-and-down movement of horizontal flukes generates propulsion. The blowhole gives surface access for air breathing and closes underwater. A dolphin uses lungs, not gills. Cetacean tail design should not be generalized to all marine mammals such as seals.

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B.4.1 · Adaptation to environment

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

Why can a dune grass survive drying wind while a mangrove tree grows in salty, oxygen-poor mud? Both need water and oxygen, but the obstacles to obtaining them differ. Explain each feature through a chain: environmental condition → effect on the organism → mechanism → benefit and possible cost.

This unit covers all B4.1 SL and HL understandings: habitats, adaptations, abiotic limits and transects, reef-building conditions, climate and biomes, convergence, and named desert/rainforest plants and animals. There is no additional higher-level content in B4.1. Prerequisites are water movement, gas exchange and natural selection. Numerical datasets and climate regions below are invented teaching models, not measurements of the named species. Scope follows the IB Biology guide.

Describe a habitat at an appropriate scale

A habitat is the place where an organism, population, species or community lives. A useful description can combine a geographical location, ecosystem type and physical position. "A coastal dune in northwest Europe" gives more information than "on land"; "the exposed sand near the seaward edge" distinguishes a microhabitat from the sheltered dune interior. A whole ecosystem includes the organisms and their interactions with the physical environment; habitat describes the place occupied.

Abiotic variables are non-living physical or chemical conditions, such as temperature, light, soil water, pH, salinity and dissolved oxygen. Biotic influences include competitors, food organisms and predators. The two interact: shade from a neighbouring plant is caused by a living organism but changes the abiotic light environment. Do not infer a single cause from a location label alone.

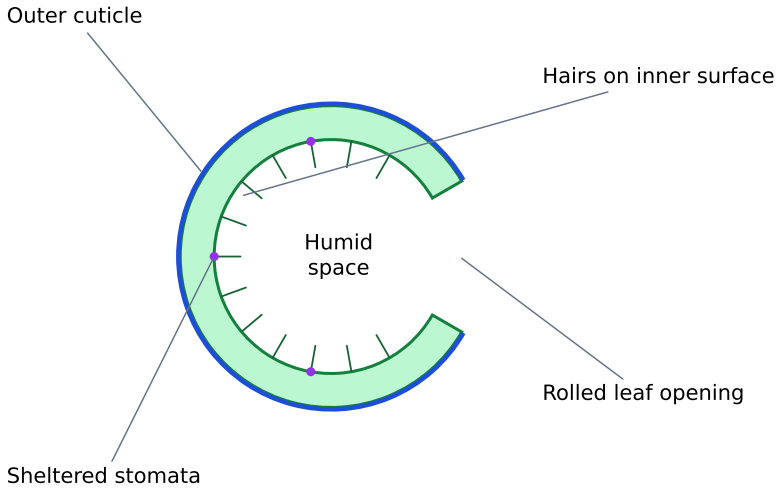
An **adaptation** is a heritable feature that contributes to survival or reproduction in a particular environment. Natural selection acts on variation across generations. Reversible changes within one individual, such as leaf rolling as water status changes, use an inherited response system; the plant does not evolve a new species-level adaptation during one dry afternoon. A convincing explanation identifies the inherited capacity and the immediate physiological response separately.

A dune grass reduces water loss while colonizing unstable sand

Marram grass, Ammophila arenaria, is a **xerophyte** adapted to dry conditions in coastal dunes. Sandy soil drains rapidly, exposed wind removes moist boundary air, and moving sand can bury shoots. These are several challenges, not one vague "harsh environment".

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Its leaf can roll so that the surface bearing stomata faces an enclosed space. Hairs and grooves on this inner surface restrict air movement near stomata; humid air is retained, reducing the water-vapour gradient and slowing water loss. A cuticle reduces loss across the outer surface. The published anatomical study describes rolling associated with water loss from bulliform cells; favourable water status can permit unrolling. Do not say that rolling makes stomata disappear or prevents all transpiration.



The section separates tissues to explain water-loss mechanisms; it is not a traced microscope specimen.

Rhizomes spread below the surface, help anchor the plant and allow new shoots to emerge as sand accumulates. These traits help the plant occupy a dynamic substrate. Conserving water also has costs: reduced stomatal conductance can restrict carbon dioxide entry and photosynthesis, and rolling can change illuminated area. A feature is advantageous in context, not universally superior to every broad flat leaf.

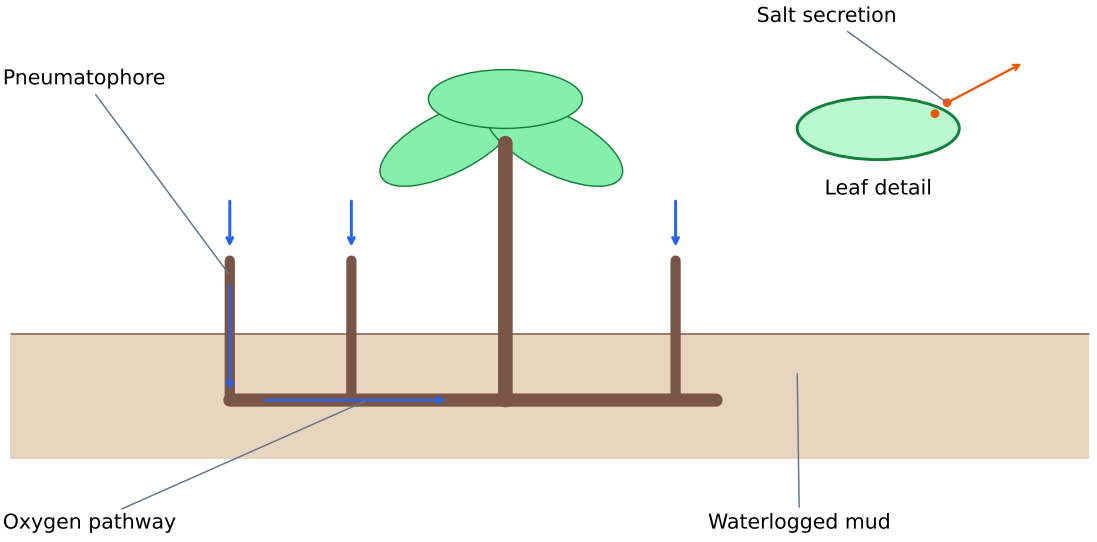
In an invented comparison, water loss changes from 10 to 4 milligrams per square centimetre per hour, while an assimilation index changes from 12 to 6 units. Water loss falls by 60%, but assimilation falls by 50%. Water conservation alone does not establish the best balance for growth or reproduction.

A mangrove separates salt management from root aeration

Grey mangrove, Avicennia marina, occupies intertidal coastal habitats. Saline surroundings lower external water potential, making water uptake difficult even when the ground is wet. Waterlogged mud also has restricted oxygen supply because gas diffusion through water is slow and organisms consume oxygen. Salinity stress and oxygen shortage need different explanations.

NParks describes its shallow lateral roots and upright pencil-like pneumatophores. When exposed above the mud and water, these breathing roots provide routes for atmospheric oxygen to enter through suitable surface pores and reach internal air spaces connected to below-ground tissues. Oxygen supports aerobic respiration. Pneumatophores do not photosynthesize enough oxygen to remove the need for gas exchange, and high tide can temporarily restrict atmospheric access.

Salt exclusion at roots limits entry, while salt glands on leaves can remove some salt that enters. The Smithsonian leaf account illustrates glands in A. marina. Salt handling requires selective transport and has limits; a mangrove is not freely drinking seawater without an osmotic challenge. Leaf structures that limit water loss complement salt management, rather than replacing the need to take up water.



Trace air entry above the mud separately from salt removal at leaves. Tide level changes which surfaces are exposed.

Different mangrove species use different root structures and proportions of exclusion/secretion. Do not attach every mangrove feature to every species. A prediction worth testing is that blocking exposed gas-entry surfaces reduces root oxygen availability under otherwise matched conditions; field observations alone would not isolate that mechanism from flooding duration and sediment differences.

Tolerance ranges constrain distribution without explaining it alone

A species can maintain survival and reproduction only within tolerable conditions. Below or above a limiting range, enzyme activity, membranes, water balance or other functions may fail. Within a range, performance can vary; the conditions permitting survival may be wider than those permitting successful reproduction. A limiting factor

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is a condition or resource that restricts a process under the circumstances, even if other requirements are suitable.

For a plant, soil moisture can restrict uptake and photosynthesis; salinity can restrict water uptake despite wet soil. For a fish such as brown trout, Salmo trutta, water temperature and dissolved oxygen affect the balance between oxygen demand and supply. These variables can covary: warm water generally holds less dissolved oxygen at equilibrium, while metabolic demand can rise within a tolerable temperature range. Do not claim that a temperature alone predicts every trout's distribution.

An observed absence does not prove physiological intolerance. Suitable sites may lack dispersing individuals, contain competitors or predators, or have a history of disturbance. Conversely, an adult surviving briefly does not prove that a self-sustaining population can reproduce there. Distinguish physiological tolerance from the actual occupied distribution.

Transects test associations along an environmental gradient

Lay a transect across a measured gradient in a permitted natural or semi-natural habitat. A semi-natural site is influenced by people but dominated by wild species, rather than a planted crop or garden bed. Use fixed intervals or a stated stratified design, sample equal quadrat areas, record organisms consistently and measure the abiotic variable at each position. Repeat independent transects rather than treating adjacent quadrats on one line as independent whole habitats.

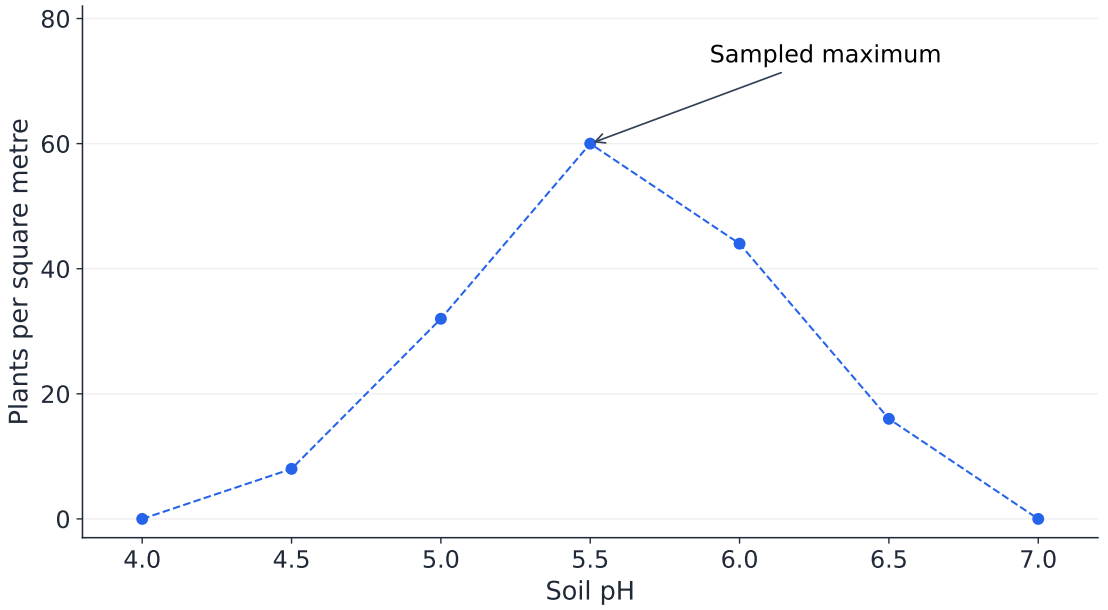
Invented teaching data for plant P are shown below. Each row represents one 0.25-square-metre quadrat; these are not real marram or mangrove measurements.

Distance along line / m	Soil pH	Plant P count
0	4.0	0
10	4.5	2
20	5.0	8
30	5.5	15
40	6.0	11
50	6.5	4
60	7.0	0

Exam tips

Name the species, variable and mechanism. Keep observed distribution separate from physiological limits, percentages from percentage points, and broad climate models from exact local predictions.

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Points show invented field observations. Connecting them is a visual guide, not proof of a physiological tolerance curve.

Density at pH 5.5 is $15/0.25 = 60$ plants per square metre. The species was recorded from pH 4.5 to 6.5 among sampled positions, with the highest count at 5.5. Do not report 4.5 and 6.5 as exact lethal limits: only seven positions were sampled, and other factors may vary along the line. The rise followed by decline also warns against fitting one straight trend and concluding that weak linear correlation means no association.

Calibrate a pH sensor with appropriate standards; use a consistent soil-water preparation and record sampling depth. Measure moisture, light and vegetation cover as possible confounders. For light sensors, standardize height and orientation; for temperature, state time and depth. Repeat seasons if the claim concerns year-round distribution. A controlled comparison across pH treatments could help separate pH from covarying field conditions, but its results would still depend on the life stage and other conditions tested.

Shallow reef formation requires several compatible conditions

A coral is an animal. Many shallow tropical reef-building corals contain photosynthetic symbionts that provide organic products. Clear, illuminated water supports this partnership; excessive depth or turbidity reduces light. Settling sediment can also smother surfaces. Depth therefore acts partly through light, rather than because a coral recognizes a depth number.

The NOAA reef-water account describes warm, clear, saline conditions. Many tropical reef builders grow well around 23–29 degrees Celsius, but species and exposure histories differ. This is not a universal survival interval for all corals: cold-water corals also exist. Sustained anomalous heat can disrupt symbiosis and lead to bleaching, which is

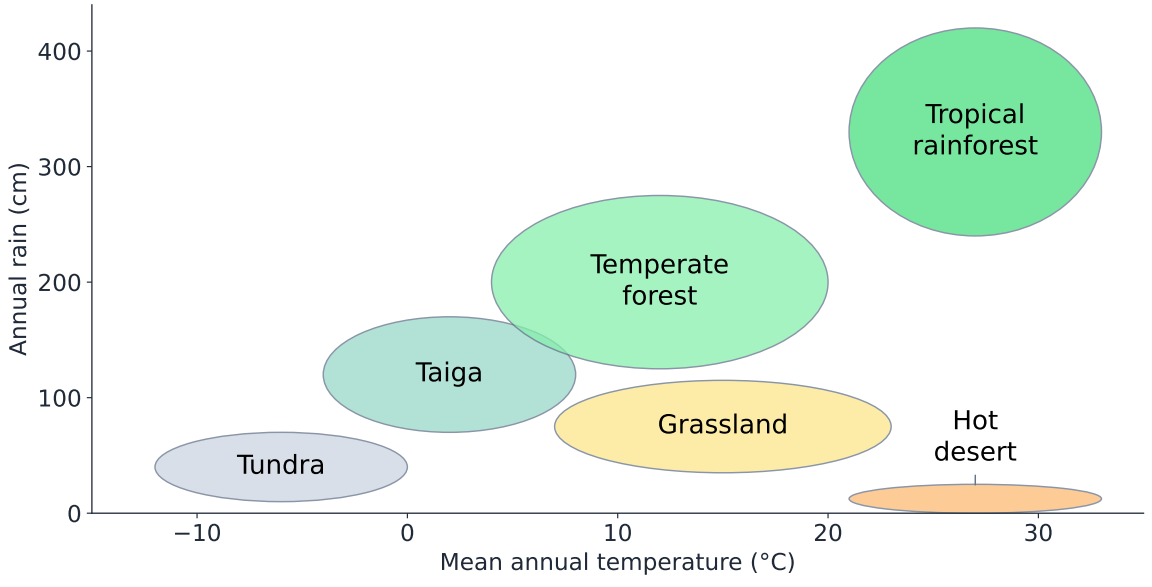
loss of symbionts or their pigments; bleaching does not mean the animal has already died in every case.

Salinity should remain within the species' tolerable marine range; strong freshwater inputs can stress colonies. Water chemistry also affects calcium carbonate skeleton formation. Lower pH reduces carbonate availability in seawater and can make calcification more difficult; "lower pH" need not mean below pH 7. Reef accretion requires skeletal construction to exceed losses through erosion and dissolution. Light, temperature, salinity, clarity and carbonate chemistry must be considered together, along with substrate and biological interactions.

In an invented monitoring example, live coral cover falls from 30% to 18%. This is a 12-percentage-point decrease and a $12/30 \times 100 = 40\%$ relative decrease. It is not evidence that 40% of every colony died, and coincidence with a warm period does not isolate warming from storms, disease or survey differences.

Temperature and precipitation shape broad terrestrial biome patterns

A biome is a broad grouping of ecosystems with characteristic communities and growth forms associated with similar abiotic conditions. Climate influences water supply, temperature limits and growing-season length. Similar climatic filters can favour similar functional traits in different evolutionary lineages: convergent evolution. Similar biome appearance does not require identical species on different continents or organisms with no shared ancestry at all.



Regions illustrate broad tendencies and overlap. They are not mapped boundaries or universal climate thresholds.

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Biome	Climate and characteristic vegetation
Tropical forest	Warm conditions and substantial rainfall support tall forest; tropical rainforest has high rainfall and usually a long growing season.
Temperate forest	Moderate temperatures, seasonal changes and adequate water support forests; deciduous and evergreen forms occur.
Taiga / boreal forest	Long cold winters and short growing seasons; conifers are prominent, with needle leaves and many evergreen species.
Grassland	Water availability and seasonality constrain tree cover; fire and grazing can also maintain grass-dominated vegetation.
Tundra	Cold, short growing season and low-growing vegetation; widespread permafrost is important in many Arctic regions.
Hot desert	Very low water supply relative to evaporative demand, often high daytime temperatures; sparse drought-tolerant vegetation.

Mean annual temperature and total precipitation are a useful first model. Seasonality, freezing, soil, altitude, water drainage, fire, grazing and human land use can produce different local communities at similar annual averages. A wet annual total concentrated into a short season is not equivalent to steady rain every month. A schematic diagram predicts broad possibilities, not the exact ecosystem at every coordinate.

Named desert and rainforest species illustrate different solutions

Saguaro, Carnegiea gigantea, is a Sonoran Desert cactus. Its succulent stem stores water; pleated ribs allow expansion as stored water increases. Reduced leaves represented by spines limit the broad leaf surface, and a photosynthetic stem carries carbon fixation. Widespread shallow lateral roots exploit brief surface wetting. The National Park Service account describes its root and pleat arrangements. Water storage bridges dry intervals but costs tissue investment and does not make seedlings immune to drought or freezing.

Fennec fox, Vulpes zerda, is a named desert animal. Large ears provide a high-area surface that can transfer heat when the temperature gradient permits; they also help detect prey. Nocturnal activity reduces exposure to peak daytime heat, and fur on the feet helps protect against hot sand. The Smithsonian species account describes these features. Large exposed surfaces do not guarantee cooling if surrounding conditions favour heat gain. Behaviour and insulation must be considered with geometry.

Kapok, Ceiba pentandra, provides a tropical forest tree example. A tall trunk and

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crown can place leaves in strong canopy light; large buttresses help support the trunk and distribute mechanical loads. NParks documents its mature buttresses. This is an explanation of support and light access, not a claim that every rainforest tree has the same roots or that a tall tree faces no wind risk.

Geoffroy's spider monkey, Ateles geoffroyi, uses long limbs and a prehensile tail during movement in trees. The tail can support gripping, leaving limbs available while feeding; the Los Angeles Zoo account describes its hairless gripping surface. Arboreal access helps exploit forest food and pathways. It does not establish that all primates have prehensile tails or that the species occurs only in one forest type.

Use surface-area models with explicit physical limits

Surface area to volume ratio compares exposed area with internal volume. For a cube of side s, area is $6s^2$, volume is s^3, and the ratio is $6/s$. A 1 cm cube has area 6 square centimetres, volume 1 cubic centimetre and ratio 6 per centimetre. A 3 cm cube has area 54, volume 27 and ratio 2 per centimetre. Tripling side length reduces the ratio to one third; it does not reduce total surface area.

For invented body models, $400/2000 = 0.20$ per centimetre is one tenth of 2.0 per centimetre. Under otherwise comparable conditions, lower exposed area per unit volume can reduce exchange per unit internal material. Actual heat and water exchange also depend on gradients, insulation, blood flow, shape and behaviour. A larger body is not universally better in a hot desert; reduced heat loss can be a disadvantage, and ears or specialized exchange surfaces can depart from simple cube scaling.

Practice

All numerical data are invented. Explain mechanisms and limits, not just feature names.

1. Describe a dune habitat at geographical and microhabitat scales. Distinguish abiotic and biotic influences, including shade from vegetation.
2. Explain three marram features and a water–carbon trade-off. Distinguish inherited adaptation from reversible rolling in one plant.
3. Compare the challenges of saline water and oxygen-poor mud for A. marina. Trace the separate roles of pneumatophores and leaf salt glands.
4. Define tolerance and limiting factor. Why do field absence and short-term adult survival not establish the limits for a reproducing population?
5. Use the plant P table to find density at pH 6.0, recorded pH range and the sampled maximum. Explain why neither exact lethal limits nor absence of association follows from these data.
6. Plan replicated transects across a soil-pH gradient, including quadrat area, sensor checks, two confounders and a limitation of one transect.

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7. Explain depth, clarity, temperature, salinity and pH in shallow tropical reef formation. Correct: "All corals require warm water, and lower pH always means acidic water below 7."
8. Reef cover falls from 40% to 28%. Find percentage-point and relative decreases. State two limits of a causal interpretation.
9. Compare the climates of all six listed biomes. Why can annual temperature/rainfall averages fail to identify a local community? Explain convergence without requiring identical species.
10. Give a named desert plant and animal and a named rainforest plant and animal. Explain two features of each, including at least one cost or condition-dependent effect.
11. Calculate area, volume and ratio for 2 cm and 6 cm cubes. State the changes when side length triples and explain two limits of applying cubes to animals.
12. A model body has area 300 square centimetres and volume 1500 cubic centimetres. Compare its ratio with 1.0 per centimetre. Does this alone prove a lower body temperature?
13. Two leaf samples each have area 10 square centimetres and are measured for 15 minutes. They lose 0.030 g and 0.012 g water. Calculate loss rates in milligrams per square centimetre per hour and percentage reduction. Assimilation indices are 20 and 12 units: evaluate the trade-off and name controls.

Worked answers

1. A coastal dune in a named region describes geographical/ecosystem location; exposed seaward sand versus a sheltered interior identifies microhabitats. Wind, water availability and temperature are abiotic. Neighbouring plants are biotic but can change light, an abiotic variable. State both cause and measured condition.
2. Rolling shelters the stomatal surface; hairs/grooves retain humid air and reduce vapour gradients; cuticle limits surface loss. Rhizomes anchor/spread and support emergence after sand deposition. Restricted gas exchange can reduce carbon gain. The inherited response system is an adaptation; one plant's immediate rolling is a physiological response, not evolution during that afternoon.
3. Salt lowers external water potential; flooded mud restricts oxygen. Exposed pneumatophores permit air entry and internal oxygen movement for root respiration. Leaf glands remove some salt; root exclusion limits entry. These mechanisms are distinct, energy/resource costs and limits remain, and tides affect exposure.
4. Tolerance is the conditions permitting a specified function such as survival or reproduction; a limiting factor restricts performance in context. Field absence can reflect dispersal, competition or disturbance. An adult surviving briefly does not show successful breeding or persistence through the life cycle.

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5. Density $11/0.25 = 44$ plants per square metre. Recorded pH range 4.5–6.5; sampled maximum pH 5.5, 60 per square metre. Sparse sampling and confounding prevent exact lethal-limit claims. The hump-shaped association need not have strong linear correlation.
6. Place multiple independent lines across the gradient with a stated selection method; sample equal areas at stated intervals, identify/count consistently and measure pH with calibrated equipment and consistent preparation/depth. Record moisture and light or other justified confounders. Adjacent positions on one line share conditions/history; repeat sites or seasons as the claim requires.
7. Depth and clarity affect light available to symbionts; sediment can smother. Suitable warmth supports tropical reef growth but excessive sustained heat stresses it. Salinity must be tolerable; lower pH can reduce carbonate availability/calcification even above 7. Cold-water corals exist. Reef growth requires skeletal construction to exceed losses; no single favourable factor guarantees it.
8. Decrease $40 - 28 = 12$ percentage points; relative decrease $12/40 \times 100 = 30\%$. Check comparable survey methods, disturbance/disease, exposure histories and replication. Cover is area fraction, not a percentage of every colony killed; association alone does not isolate a cause.
9. Tropical forest: warm/wet; temperate forest: moderate/seasonal with adequate water; taiga: long cold winter/short growing season; grassland: water/seasonality plus fire/grazing constrain forest; tundra: cold, short growing season; hot desert: low water relative to evaporative demand. Seasonality, soil, drainage, disturbance and land use modify annual-climate predictions. Similar selection pressures can favour similar functional traits in different lineages without identical species.
10. Saguaro: water-storing pleated stem and shallow spreading roots, with tissue investment/seedling limits. Fennec: ears for heat exchange/prey detection and nocturnal activity or protected feet; cooling depends on the gradient. Kapok: crown reaches light and buttresses support tall growth, without eliminating wind risk. Geoffroy's spider monkey: long limbs and a gripping tail facilitate arboreal movement/feeding; not every primate shares these features.
11. Side 2 cm: area 24 square centimetres, volume 8 cubic centimetres, ratio 3 per centimetre. Side 6 cm: area 216, volume 216, ratio 1 per centimetre. Tripling side multiplies area by 9 and volume by 27, reducing ratio to one third. Shape, insulation, gradients, circulation and behaviour alter actual animal exchange.
12. Ratio $300/1500 = 0.20$ per centimetre, one fifth of 1.0. It predicts less exposed area per unit volume in this model, not temperature by itself. Heat production, environmental temperature, insulation, evaporation and other fluxes also matter.
13. Time is 0.25 hour; masses are 30 and 12 mg. Rates $30/(10 \times 0.25) = 12$ and $12/(10 \times 0.25) = 4.8$ milligrams per square centimetre per hour. Water-loss reduction $(12 - 4.8)/12 \times 100 = 60\%$; assimilation reduction $(20 - 12)/20 \times 100 = 40\%$. Saving water costs carbon gain; test growth/reproduction. Match species/genotype, leaf age, light, temperature, humidity, airflow and time; replicate independently.

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Ecological niches

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

Two birds can feed in the same tree yet take different foods, use different branch sizes or forage at different times. Sharing a habitat does not establish identical roles, and different feeding observations do not by themselves prove that competition caused the difference. An explanation needs resources, conditions, interactions and evidence.

This unit covers all B4.2 SL and HL understandings: niches, oxygen requirements, photosynthetic, holozoic, mixotrophic and saprotrophic nutrition, archaeal nutritional diversity, hominid dentition, herbivory and predation adaptations, forest plant forms, fundamental/realized niches and competitive exclusion. There is no additional higher-level content in B4.2. Prerequisites are respiration, photosynthesis, digestion, adaptations and population sampling. All numerical examples are invented teaching models. Scope follows the IB Biology guide.

A niche includes resources, conditions and interactions

An **ecological niche** describes a species' role and its biotic and abiotic relationships affecting growth, survival and reproduction. It includes how the organism obtains food or other resources, which conditions it tolerates and how it interacts with competitors, consumers and partners. Habitat is the place occupied; niche describes how the species lives there. Trophic level describes one aspect of feeding position, not the entire niche.

For an insect-feeding bird, useful niche variables could include prey sizes, feeding times, branch positions, nesting sites and temperature tolerance. For a plant, light acquisition, water and mineral use, pollination, herbivory and competitive interactions matter. A species can change resource use with season, age or resource availability; a niche is not a single immutable food label.

Resource partitioning means differences in resource use that can reduce overlap among organisms. Different prey sizes, feeding heights or times may permit coexistence. However, observations of different use cannot alone distinguish competition from inherited preferences, unequal resource availability or other causes. Sample the alternatives rather than selecting only observations that fit a story.

Oxygen requirements are distinct from the carbon source

Obligate aerobes require oxygen for growth under the stated conditions. Facultative anaerobes can grow with oxygen and also without it, changing metabolic pathways as conditions permit. Obligate anaerobes grow in the absence of oxygen and cannot grow under sufficiently oxygenated conditions; oxygen can be toxic to them. This does not

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mean that every anaerobic cell dies instantly on contact with one oxygen molecule.

Category	Oxygen available	Oxygen absent
Obligate aerobe	Can grow if other requirements are met	Sustained growth requires oxygen
Facultative anaerobe	Can use aerobic metabolism	Can grow using suitable oxygen-independent pathways
Obligate anaerobe	Growth is inhibited under oxygenated conditions	Can grow if other requirements are met

"Oxygen-independent" does not mean "without any chemical reactions" or always "without respiration". Anaerobic respiration and fermentation are different metabolic possibilities; detailed classification is not needed here. Also, an organism can be heterotrophic and aerobic, or heterotrophic and anaerobic. Carbon source and oxygen requirement are separate questions.

In an invented supplied dataset, strains X, Y and Z have population increases of 80/0, 90/35 and 0/70 arbitrary units with/without oxygen. Under otherwise suitable matched conditions, X fits the obligate-aerobe model, Y the facultative model and Z the obligate-anaerobe model. A single negative treatment could instead reflect another missing requirement, so controls and repeat observations matter.

Photosynthesis builds organic material using light and inorganic carbon

Photosynthetic plants, algae and groups such as cyanobacteria use light energy to support synthesis of organic compounds from carbon dioxide. These compounds provide carbon skeletons and stored chemical energy; they can be used in respiration or biosynthesis. Mineral ions and water are required, but minerals are not the plant's main source of organic food carbon.

Keep energy and matter separate. Light supplies energy, while carbon dioxide supplies carbon. A plant does not convert sunlight atoms into sugar atoms or obtain most of its dry mass from mineral particles. Photosynthetic organisms also respire; darkness stops light-driven reactions rather than every metabolic process immediately.

Not every organism containing photosynthetic partners obtains nutrition in the same way. Reef corals remain animals that obtain organic material from their symbionts and from heterotrophic feeding. Avoid classifying all green-looking organisms by colour alone; investigate the actual carbon and energy pathways.

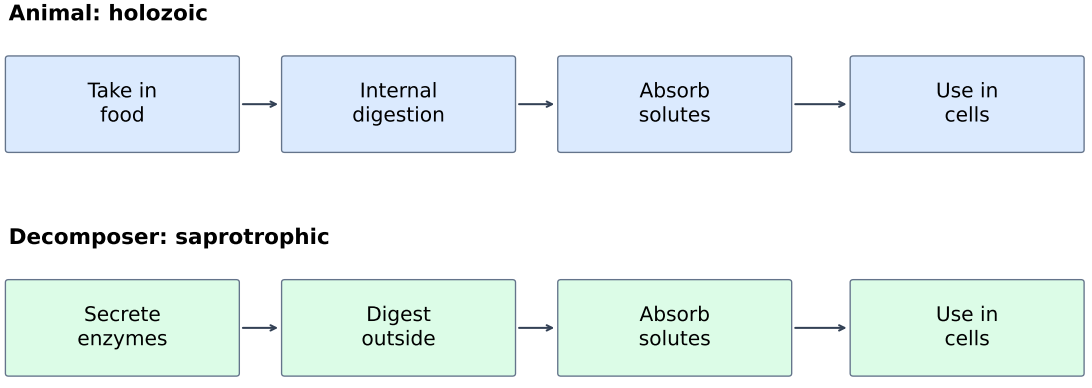
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Holozoic and saprotrophic nutrition place digestion differently

All animals are heterotrophic: they obtain organic carbon from other organisms or their products. In holozoic nutrition, food is ingested, digested inside the animal, absorbed and assimilated. Ingestion brings material into the feeding system; digestion breaks large molecules into absorbable forms; absorption crosses into internal tissues or transport; assimilation incorporates absorbed material into the organism's metabolism and structures. Egestion removes unabsorbed food, while excretion removes metabolic waste.

For example, protein in food is hydrolysed to smaller products; absorbed amino acids can be used to synthesize the animal's own proteins. Merely passing food through the mouth is not absorption. Animal feeding is diverse: heterotrophy does not require every animal to have an identical digestive tract or feeding mechanism.

Saprotrophic fungi and bacteria secrete enzymes onto dead organic material, digesting it extracellularly. Soluble products are then absorbed across cell surfaces and used in metabolism. A fungal hypha does not need to swallow a whole dead leaf before digestion begins. Saprotrophs contribute to decomposition and nutrient cycling, although not all fungi or bacteria are saprotrophs.



Trace where enzymes act and where soluble products cross a cell or tissue boundary.

In an invented animal budget, 100 units of organic food are ingested, 25 are egested and 5 of the absorbed units later leave in a specified metabolic-waste category. Absorption is 75 units; 70 remain after that stated waste loss. The remaining 70 are not automatically all new biomass: respiration, maintenance and other transfers still matter. State whether a budget concerns matter or energy before subtracting terms with different meanings.

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Mixotrophs combine nutritional capacities

Some protists can combine photosynthetic carbon acquisition with heterotrophic uptake. Euglena gracilis is a useful freshwater example: its chloroplasts permit photosynthesis, and it can take up dissolved organic substrates under appropriate conditions. Heterotrophy here need not mean engulfing visible prey. A growth study compared light and organic-carbon conditions; its treatment design supports distinguishing the two contributions rather than inferring nutrition from green colour alone.

Facultative mixotrophs can alter their reliance on modes, including growth using one mode when conditions allow. Obligate mixotrophs require contributions from both modes under the conditions defining that dependence. Do not assume every mixotroph can live indefinitely in darkness, or that "both" means exactly half its carbon comes from each route.

An invented factorial comparison starts each treatment with the same population: light/no added organic substrate gives an increase of 20 units; dark/organic gives 15; light/organic gives 32; dark/no organic gives 0. Growth under both single-source conditions is consistent with nutritional flexibility. The combined value need not equal $20 + 15$: light, substrate, respiration and other limits interact. Confirm that substrate is available, account for stored reserves or carryover, verify viability and match other nutrients and temperatures.

Archaea have diverse energy and carbon pathways

Archaea form a domain separate from Bacteria and Eukaryota. They are not all confined to extreme habitats, and the domain does not have one universal feeding mode. Different archaea obtain energy from light, oxidation of inorganic chemicals or oxidation of carbon compounds. This diversity matters in environmental cycling and ATP production.

Energy source does not by itself identify carbon source. An organism using light to help make ATP need not fix carbon dioxide like a green plant. Some archaea use organic carbon while benefiting from light-driven energy processes; other archaeal pathways couple inorganic chemical energy sources with carbon fixation. State what supplies energy and what supplies carbon as two pieces of information.

For invented organisms A, B and C: A uses light plus organic carbon; B obtains energy by oxidizing an inorganic substance and uses carbon dioxide as carbon source; C obtains both usable chemical energy and carbon from organic compounds. A is not demonstrated to be photoautotrophic merely because it uses light. Naming archaeal species or memorizing every biochemical pathway is unnecessary for this comparison.

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Dentition supports dietary inferences with limits

Hominidae is the great-ape family, including humans and extinct relatives. Human dentition illustrates an omnivorous pattern: incisors bite, canines contribute tearing, and premolars/molars crush and grind varied foods. Tooth size, shape, relief and enamel can indicate food-processing capabilities. They do not directly give the percentage of meat, fruit or leaves eaten by an individual.

Compare an attributed model or digital skull collection using consistent views and scale. Locate front teeth, canines, premolars and molars; distinguish an absent tooth from its preserved socket. The Smithsonian account of SK 46, a Paranthropus robustus specimen, describes large cheek teeth relative to front-tooth sockets. This supports substantial processing of plant material, but should not become a claim that the species never consumed any other food or that every meal required maximal chewing force.

Living predominantly plant-feeding great apes can still have conspicuous canines; canines can also serve social display or defence. Therefore "large canine equals obligate carnivore" is an invalid shortcut. Dietary reconstruction combines dentition with wear, isotopes and ecological evidence. Anatomy indicates capacity; wear records use over particular timescales, and each dataset has sampling limits.

For an invented planar model, total cheek-tooth area 120 square millimetres and front-tooth area 30 give a ratio of 4. A second model with 90 and 45 gives 2. These are area ratios, not species identifications or a direct four-to-one ratio of plants to meat in the diet. Compare like ages, preservation and measurement conventions.

Herbivores and plants influence each other's feeding success

Chewing mouthparts, such as grasshopper mandibles, cut or crush plant tissue. Piercing/sucking mouthparts, such as aphid stylets, reach fluids including phloem sap without removing a whole leaf piece. Both exploit plant resources, but the access mechanism and plant tissue affected differ. Mouthpart shape should be connected to the food's location and properties.

Plants resist feeding with physical structures such as thorns, tough tissues or hairs, and with toxic or deterrent secondary compounds in leaves or seeds. Some herbivores have metabolic pathways that detoxify particular compounds. This can permit feeding that would harm another species, but it is not universal immunity to every toxin or concentration. Defence and detoxification require resources and can involve trade-offs with growth or other functions.

Distinguish a proposed defensive function from evidence of its effect. In an invented supplied model, equally sized leaves with a tested compound suffer mean losses of 8% area versus 20% in a matched comparison. The difference is 12 percentage points, or 60% less relative loss. Attribution to the compound needs comparable leaf age, toughness, herbivore exposure and other factors; naturally different plant species do not isolate one chemical mechanism.

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Predation involves finding, capture, killing and resistance

Predators require ways to detect suitable prey and overcome it. Physical features can include sensory organs, claws, jaws or capture webs; chemical features can include venom; behavioural features include stealth, pursuit and ambush. Link a feature to a stage: detecting a signal is not yet capturing prey, and a claw's presence does not explain how prey is located.

Prey resistance can include camouflage, protective structures, chemical deterrents, vigilance, escape or group responses. These change probabilities, not guarantee survival in every encounter. Concealment can be useful against a visually hunting predator but less effective against another sensory system. A defence can also carry costs, such as reduced feeding time during vigilance.

In an invented sequence of 100 encounters, a predator detects 60, captures 30 and successfully kills 24. Detection success is 60% of encounters; capture conditional on detection is $30/60 = 50\%$; killing conditional on capture is $24/30 = 80\%$. Overall killing success is 24%, not 80%. Choose the denominator matching the claim.

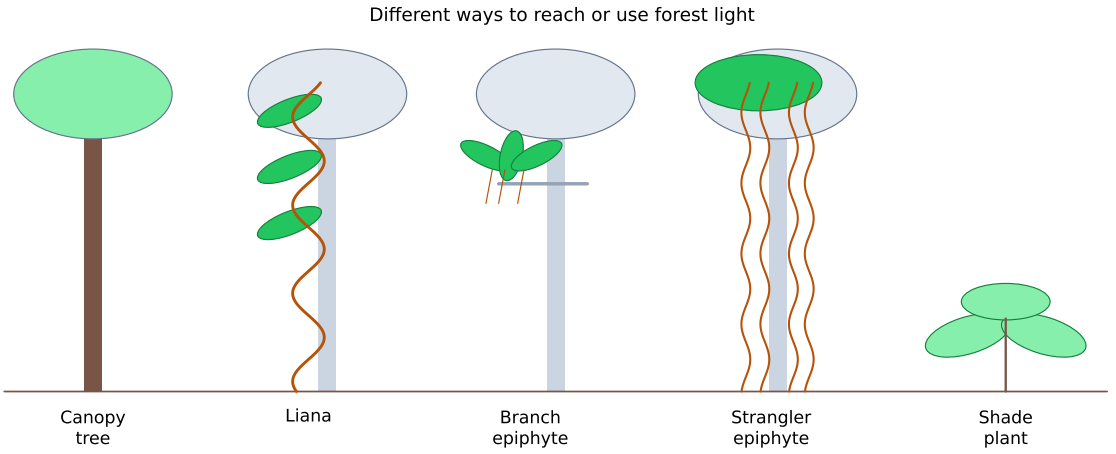
Forest plants reach or use light in several ways

Canopy trees invest in supporting trunks that raise crowns into strong light. Lianas climb existing supports, reducing the need for an equally massive self-supporting trunk while connecting foliage to resources obtained by their own roots. Epiphytes establish on branches; they use the host as support and need their own water/nutrient supply, rather than automatically parasitizing its transport tissues.

Strangler epiphytes, including some figs, establish high on a tree and send roots toward the ground. Growing roots and stems can surround the host and compete for light or below-ground resources; effects develop over time. Not every epiphyte is a strangler, and beginning on a branch does not by itself demonstrate parasitic uptake.

Shade-tolerant shrubs and herbs can persist near the forest floor by using lower light efficiently and having suitable photosynthetic and respiratory characteristics. They do not grow in zero light without any energy source. A broad leaf can intercept light but also costs material and loses water; architecture, physiology and light availability must be considered together.

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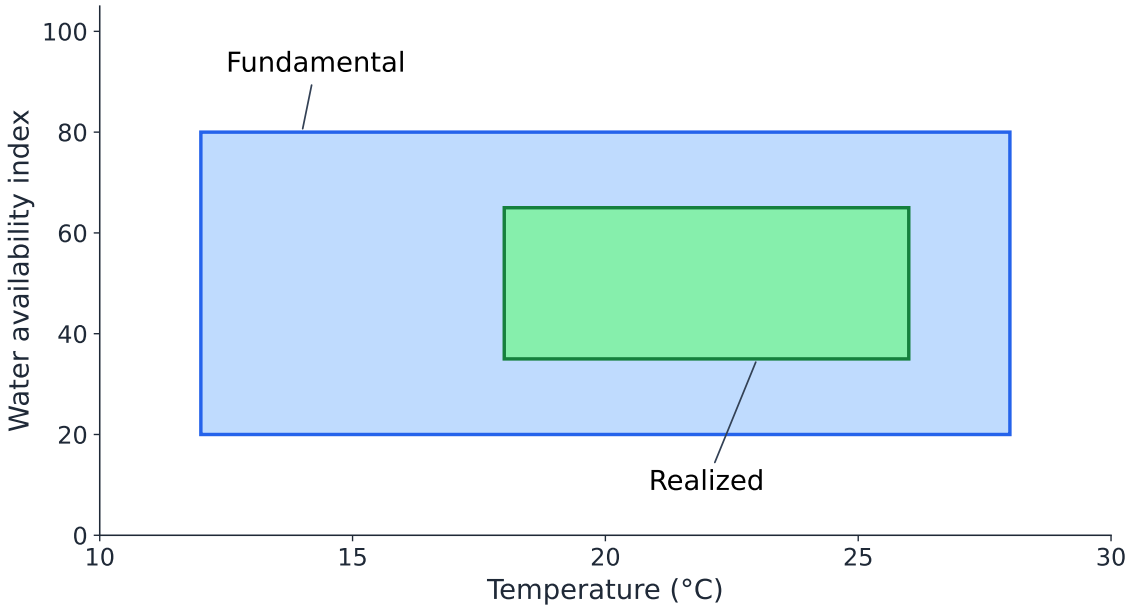
Trees, climbers, branch epiphytes, stranglers and shade plants represent different strategies, not five obligatory stages in one life cycle.

Fundamental and realized niches are explanatory models

A fundamental niche describes potential conditions and resources under which a species can maintain itself, based on its adaptations and tolerances in the stated model. In a competition-focused model, this means removing restrictive competition while retaining other essential requirements. It does not mean removing all organisms, including food or necessary partners.

The realized niche is the extent actually occupied or used under the interactions present. Competition may exclude a species from otherwise suitable sites or restrict both species to parts of their potential ranges. A diagram of a smaller realized region inside a fundamental region illustrates competitive restriction; it is not a proof that all interactions are harmful. Facilitation, dispersal, history and detection also influence field occupancy.

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The plotted regions are invented. The inner region represents a competition-focused model, not universal measured limits.

To test competitive restriction, use supplied simulations or an approved controlled comparison with and without the competitor, keeping abiotic conditions and resources comparable. If the focal species expands its use when competition is reduced, that supports a competition effect. Alternative mechanisms, indirect effects and time scale still need evaluation. Do not introduce competitors or remove native species merely to demonstrate the model.

Competitive exclusion depends on overlap and conditions

When two species depend on the same limiting resources in a stable, sufficiently uniform closed model, one can prevent the other from maintaining a population. Competitive exclusion describes this outcome; a finite observation of unequal numbers is not by itself proof of permanent elimination. Competition can also restrict both species' realized resource use, allowing coexistence where relevant differences or changing conditions matter.

Invented feeding observations are A: 18, 9, 3 across three categories, and B: 3, 9, 18. Both totals are 30. Their proportions are 60/30/10% and 10/30/60%. All three categories are shared, although preferences differ. If a comparison index is explicitly defined as the sum of the smaller proportional use in each category, its value is $10 + 30 + 10 = 50\%$. This describes observed proportional overlap, not a measured strength of competition.

Observed use of one category is $14/35 = 40\%$ versus $5/50 = 10\%$. This cannot rank overall specialization: other resource use, availability, sampling coverage and niche dimensions are unknown.

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Exam tips

State the mode, mechanism and denominator. Separate dietary capacity from actual diet, observed overlap from causal competition, and one food category's frequency from total niche breadth.

Practice

All numerical examples are invented. Use supplied data or non-destructive approved observations.

1. Distinguish niche, habitat and trophic level. Give four niche dimensions and explain why resource partitioning alone does not prove competition caused the difference.
2. Interpret growth increases with/without oxygen: X 70/0, Y 85/25, Z 0/60. Assign oxygen-requirement models and name a control and a limit.
3. State the carbon and energy sources in photosynthesis. Correct: "Plants eat soil minerals and stop all respiration at night."
4. Compare holozoic and saprotrophic pathways, distinguishing ingestion, digestion, absorption, assimilation, egestion and excretion. Of 120 food-matter units, 30 are egested and 6 of the absorbed units later leave as specified metabolic waste. Find absorption and the remaining stated balance; is it all new biomass?
5. Explain mixotrophy and obligate/facultative dependence. In matched treatments, growth occurs with light/no organic addition and dark/organic addition. What does this support, and why need combined growth not equal their sum?
6. Archaea A uses light/organic carbon, B inorganic oxidation/carbon dioxide, C organic oxidation/organic carbon. Separate carbon and energy sources; which are heterotrophic in this model?
7. Explain how hominid dentition can support but not uniquely determine dietary reconstruction. Model cheek/front areas are 150/30 and 100/50 square millimetres: calculate ratios and reject a diet-percentage inference.
8. Compare chewing and piercing/sucking herbivores and two plant defences. Mean area losses with/without a tested compound are 6%/15%. Find point and relative differences; discuss detoxification and a confounder.
9. Give physical, chemical and behavioural predator/prey adaptations. Of 200 encounters, 100 lead to detection, 40 to capture and 30 to killing. Find detection, conditional capture, conditional killing and overall killing percentages.
10. Compare canopy trees, lianas, epiphytes, strangler epiphytes and shade plants. Explain why neither every epiphyte is a parasite nor every forest-floor plant receives zero light.
11. Explain fundamental and realized niches in a competition model, including essential partners and two other causes of absence. What comparison could test competitive restriction without a destructive field manipulation?

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12. Supplied model populations end at A 80 and B 8 together, while each persists alone. Does this already establish permanent exclusion? Explain possible competitive outcomes and what further evidence is needed.
13. C uses resource counts 16, 8, 16; D uses 4, 12, 24. Find proportions and the defined overlap index (sum of category-wise minima). Can one category's 40% versus 10% identify the narrower-niche specialist?

Worked answers

1. Habitat is place, trophic level is feeding position, niche includes resource use/conditions/interactions. Examples: prey size, feeding time, branch height, nesting site or temperature tolerance. Differences can reflect resource supply, preferences or other factors; competition needs an appropriate comparison and repeated evidence.
2. X fits obligate aerobe; Y facultative anaerobe; Z obligate anaerobe under the model. Match nutrients, temperature, starting cells and measurement duration and verify oxygen conditions. Negative growth can reflect another unmet requirement, and categories do not imply every exposed cell instantly dies.
3. Light supplies energy; carbon dioxide supplies carbon for organic synthesis. Minerals provide essential inorganic nutrients, not most organic food carbon. Photosynthetic organisms respire in light and darkness; photosynthesis and respiration are different processes.
4. Holozoic feeding ingests before internal digestion and absorption; saprotrophs secrete extracellular enzymes then absorb soluble products. Assimilation uses absorbed material in metabolism/structures. Egestion removes unabsorbed food; excretion removes metabolic wastes. Absorption $120 - 30 = 90$ units; after the specified waste $90 - 6 = 84$. Respiration/maintenance and other transfers prevent calling all 84 new biomass; keep matter and energy terms distinct.
5. Mixotrophy combines photosynthetic and heterotrophic capacities; obligate dependence needs both under defining conditions, whereas facultative use is flexible. Growth in both single-source treatments supports flexibility if other requirements/viability match. Contributions interact and need not be additive; green colour alone is insufficient evidence.
6. A: light energy, organic carbon, heterotrophic. B: energy from inorganic oxidation, carbon dioxide carbon, autotrophic in the model. C: organic oxidation supplies energy and organic compounds supply carbon, heterotrophic. Archaeal diversity cannot be reduced to "all photosynthetic" or "all extreme-habitat anaerobes".
7. Tooth form/area, relief and enamel indicate processing capabilities; compare scaled, consistently oriented models and preservation. Ratios are $150/30 = 5$ and $100/50 = 2$. They do not give proportions of plant/meat in the diet. Combine anatomy with wear, isotopes and ecological context; conspicuous canines are not proof of carnivory.

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8. Mandibles cut/crush tissue; stylets pierce and withdraw fluids. Physical barriers and toxic/deterrent compounds can reduce feeding, while some animals detoxify particular compounds at a cost. Difference $15 - 6 = 9$ percentage points; relative reduction $9/15 \times 100 = 60\%$. Leaf age/toughness, herbivore exposure or other chemistry can confound an uncontrolled comparison.
9. Examples include sensory detection/claws or webs, venom, stalking/ambush; prey camouflage, armour, deterrent chemicals, vigilance or escape. Detection $100/200 = 50\%$; capture given detection $40/100 = 40\%$; killing given capture $30/40 = 75\%$; overall $30/200 = 15\%$. Conditional 75% is not overall success; each stage has a different denominator.
10. Trees support their crowns; lianas climb with their own soil connections; epiphytes use branches as support and obtain their own resources; strangler epiphytes develop ground-reaching roots and can compete with hosts; shade plants use low light efficiently. Support alone does not show parasitic uptake. Low light differs from none; traits have costs and conditions.
11. Fundamental niche is potential persistence under stated tolerances/resources without restrictive competition in this model; essential food/partners remain. Realized niche reflects interactions/actual use. Dispersal, disturbance, predators, facilitation or detection affect observed absence. Use supplied simulations or approved controlled comparisons with/without competitors and matched abiotic/resource conditions, with replication and sufficient time.
12. B remains at eight, so no permanent exclusion has yet been established. Both species persisting alone but B declining together supports competitive restriction if conditions match. Longer population trajectories, resource and demographic data are needed to distinguish eventual exclusion from stable coexistence or transient dynamics. Resource partitioning or changing conditions may allow coexistence; the model's assumptions matter.
13. Both totals are 40. C: 40/20/40%; D: 10/30/60%. Defined overlap $10 + 20 + 40 = 70\%$. This index describes the observations, not competition intensity. A single category's 40% versus 10% cannot rank overall specialization without the other resource distribution, availability, effort and niche dimensions.

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Enzymes and metabolism

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A starch mixture can stay blue-black with iodine for several minutes, then stop giving that colour after amylase is added. The enzyme changes how quickly starch is broken down; it does not provide the starch, supply unlimited energy or disappear after one reaction. Explaining that observation connects molecular structure to measurements and control of a whole metabolic network.

This unit covers C1.1 SL and HL understandings: catalysis, metabolism, anabolism and catabolism, active sites, induced fit, collisions, specificity, denaturation, rate factors, experimental measurement and activation energy. Sections marked **AHL** are for Higher Level only.

- **AHL** Additional content covers enzyme location, metabolic heat, pathway organization, reversible inhibition, feedback and mechanism-based inhibition.

Prerequisites are protein structure, diffusion, condensation/hydrolysis and graph gradients. All numerical data and plotted curves are invented teaching models, not measurements from real investigations. Scope follows the IB Biology guide.

Catalysts make a network of reactions fast enough for life

An **enzyme** is a biological catalyst: it increases reaction rate and is regenerated rather than consumed overall. Cells need reactions to proceed rapidly at temperatures compatible with their structures. Without suitable catalysts, many reactions would be too slow to sustain growth, transport or repair. An enzyme does not make an energetically impossible net process possible simply by being present.

Metabolism is the interacting network of chemical reactions in an organism. One product can become another enzyme's substrate; pathways branch, share intermediates and depend on supplies of matter and energy. Specificity means that cells require many different enzymes. Adjusting enzyme amount or activity helps regulate which reactions occur and how quickly resources flow through the network.

Anabolism builds larger or more complex molecules and requires an energy supply overall. Examples include joining amino acids during protein synthesis, forming glycogen from glucose units, and synthesizing organic material in photosynthesis. Condensation joins subunits with removal of water in a simplified net description; actual cellular mechanisms can involve activated intermediates and energy coupling. Photosynthesis is anabolic overall, not one single condensation reaction.

Catabolism breaks larger molecules into smaller ones. Digestion hydrolyses proteins or polysaccharides, using water to split bonds. Respiration oxidizes organic substrates through many steps and transfers some released energy to usable forms such as ATP.

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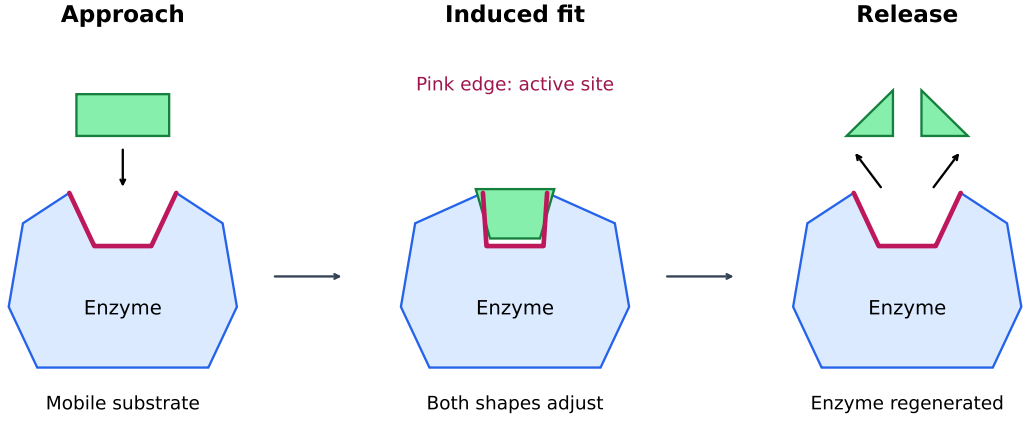
Do not replace this distinction with “breaking any bond releases energy”: bond breaking needs energy; the balance of bonds broken, bonds formed and other changes determines the net transfer.

An active site binds and changes with its substrate

The protein enzymes studied here are globular proteins. Folding brings a small number of amino-acid side chains together to form the active site, even when those amino acids lie far apart in the primary sequence. Interactions throughout the protein maintain that site’s three-dimensional shape and chemical environment. Not every amino acid directly touches the substrate, but a change elsewhere can still affect catalysis. Some biological catalysts are RNA; that exception does not alter the protein-enzyme model used here.

Specificity depends on complementary shape **and chemical properties**, such as charge and hydrogen-bonding possibilities. A molecule with a roughly similar outline need not bind productively. Binding brings substrate groups into useful positions and can strain bonds or stabilize a transition state, helping the reaction follow a lower-barrier route.

Induced fit describes changes in the shapes of both enzyme and substrate during binding. The active site is flexible rather than a permanently rigid hole. After reaction, products leave and the enzyme can participate again. Its temporary changes during a cycle are compatible with regeneration overall.



This schematic shows a flexible site around a substrate, followed by release of two products. It is not a scale drawing of a named enzyme.

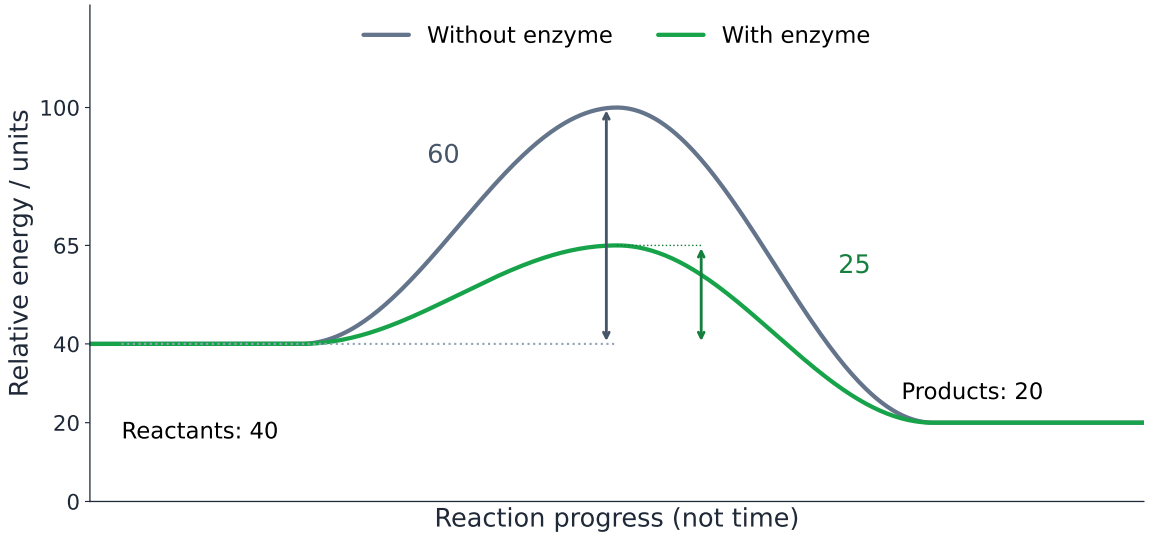
Random molecular motion brings substrate and active sites together. A productive encounter needs a suitable orientation and compatible chemistry, not just any collision. Both partners need not travel freely: a membrane-bound enzyme can encounter mobile substrate, while a large immobilized substrate can be reached by a mobile enzyme. “No enzyme movement” therefore does not necessarily mean “no reaction”.

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Activation energy is a barrier rather than the overall energy change

Reactants must reach a transition state before becoming products. The activation energy is the energy barrier from the reactant level to that transition state. Enzymes provide a pathway with a lower barrier, so a greater fraction of encounters can proceed at the same temperature. They do not heat each substrate molecule or lower the energy of the reactants to achieve this.

In the invented energy diagram, reactants are at 40 relative energy units and products at 20. The uncatalysed peak is 100; the catalysed peak is 65. Forward activation energies are therefore $100 - 40 = 60$ and $65 - 40 = 25$ units. The overall change is $20 - 40 = -20$ units in both cases. The horizontal axis is reaction progress, not elapsed time.



Compare each peak with the reactant level. A lower peak does not change the initial-to-final energy difference.

Breaking substrate bonds requires energy; forming product bonds releases energy. A net energy-releasing reaction can still be slow because it has an activation barrier. Conversely, a catalyst alone does not supply the energy needed to drive an energy-requiring net reaction. Enzymes accelerate forward and reverse reactions and do not change the equilibrium position.

Temperature, pH and substrate supply affect different parts of the model

As temperature rises over a suitable low range, molecular motion increases and productive collisions become more frequent. Rate often rises. At sufficiently high temperatures, disrupted interactions change the enzyme's folded structure and active site:

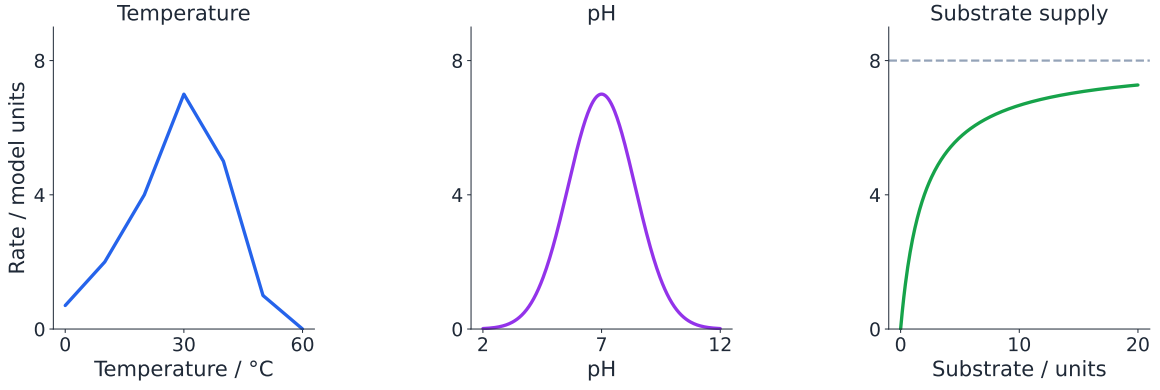
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denaturation reduces function. An observed optimum balances these effects under the particular assay conditions. It is not a universal temperature for all enzymes, and longer exposure can change the measured optimum.

Cooling usually reduces rate without permanently destroying the active site; warming can restore activity. A low reading alone cannot distinguish reversible slowing from denaturation. Compare activity after returning samples to the same suitable temperature. Severe heating may leave persistent loss, but recovery depends on the protein and treatment.

pH changes protonation and hence charges on amino-acid groups, affecting substrate binding and catalytic chemistry. More extreme changes can disrupt folding. Different enzymes have different suitable pH ranges: an acidic optimum for a digestive enzyme is not evidence that all human enzymes work best in acid. Not every pH-induced loss of rate proves permanent denaturation; retest under matched restored conditions.

At fixed enzyme concentration, increasing substrate concentration raises productive encounters when substrate is scarce. Rate then approaches a plateau because active sites spend most of their time occupied and turnover limits the rate. The plateau does not mean that substrate has denatured the enzyme or that every substrate molecule is bound. Adding more active enzyme can raise the plateau if substrate and other requirements are sufficient.



These are explanatory models. The temperature and pH peaks are condition-specific; the substrate plateau assumes a fixed amount of active enzyme.

For any graph, first describe the measured relationship and range, then explain a mechanism. An invented temperature series gives rates 2, 4, 7, 5 and 1 units at 10, 20, 30, 40 and 50 degrees Celsius. The greatest **tested** rate is at 30 degrees; the true peak could lie between tested values. Repeat measurements and smaller temperature intervals near the peak improve the estimate.

Exam tips: Label axes and units; justify the gradient or reciprocal time. Explain temperature responses with productive encounters and structural change, and substrate saturation with occupied sites. State the conditions: a tested maximum is not an exact optimum.

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Measure a rate that matches the reaction and the question

Rate can be product formed per unit time or substrate removed per unit time. For a product-time graph, gradient gives rate. An initial tangent estimates initial rate before major substrate depletion or product accumulation. A mean gradient over a long interval answers a different question and may be smaller as the reaction slows.

For invented gas volumes 0, 12, 21, 27 and 30 mL at 0, 20, 40, 60 and 80 s, the first-interval mean rate is $12/20 = 0.60$ mL/s. Across the full interval it is $30/80 = 0.375$ mL/s. Neither is automatically the exact tangent at time zero. Record the interval and units; do not label the final volume itself a rate.

For a fixed amount of change, $1/t$ is a useful **relative rate proxy**, with units per second if time is in seconds. Endpoint times 40 and 20 s give 0.025 and 0.050 per second, so the second proxy is twice the first. This comparison requires the same endpoint, starting concentrations and comparable detection. It is not a measured mass of product per second unless the amount represented by the endpoint is known.

For an amylase–starch investigation, equilibrate measured enzyme and starch solutions separately in the water bath, control pH with a suitable buffer, then mix and start timing consistently. Withdraw equal samples to separate iodine spots at fixed intervals. Do not put iodine into the reaction mixture: it can alter the conditions being tested. Use the same colour criterion, volumes and sampling method. Include an appropriate no-active-enzyme comparison and repeat each temperature using fresh mixtures.

If the final blue-black sample is at 50 s and the first negative sample at 60 s, disappearance occurred after 50 s and no later than 60 s. The endpoint is an interval, not exactly 60 s. Shorter sampling intervals improve time resolution but can change the reaction volume if repeated samples are large. Match starting amounts, minimize withdrawals and consider a separate-tube method. Follow the school's reagent and hot-water precautions.

Alternative assays include gas collection for catalase, substrate disappearance and calibrated colorimetry. A gas syringe avoids assuming equal bubble sizes; a colorimeter needs an appropriate blank and calibration if absorbance is converted to concentration. Hold enzyme amount, total volume and other rate factors constant when investigating one variable. Randomize treatment order where drift matters, use replicates, and separate instrument resolution from biological variation.

AHL: Enzyme location and metabolic heat

Intracellular reactions occur inside cells. Glycolysis occurs in the cytosol; in eukaryotes the Krebs cycle occurs mainly in the mitochondrial matrix. Their enzymes are not secreted into a digestive cavity. Extracellular enzymes act outside the cell that made them: digestive enzymes secreted into the gut lumen hydrolyse food before products are absorbed. "Inside the animal" is therefore not the same as "intracellular". Cells synthesize both types, but their destinations differ.

Metabolic transfers are not 100% efficient at retaining energy in immediately useful

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chemical forms or work; heat is generated. Respiration can transfer part of substrate energy to ATP while releasing heat, and later ATP-supported processes also dissipate energy. Mammals and birds use metabolic heat in maintaining body temperature, together with regulated heat loss. The catalyst does not change the net energy balance of a reaction simply by making it faster.

In a simplified invented budget, 200 kJ enters a process, 70 kJ is retained in the specified useful output and all the rest becomes heat within the stated boundary. Efficiency is $70/200 \times 100 = 35\%$ and heat is 130 kJ. This is an accounting model, not a universal ATP yield or proof that every organism releases the same fraction.

AHL: Linear and cyclical pathways connect enzymes

A linear pathway has a sequence such as $A \rightarrow B \rightarrow C \rightarrow D$, with a different enzyme catalysing each step. Glycolysis converts glucose to pyruvate through such a sequence. Inhibiting an early step can reduce downstream supply; a block later can lead to upstream accumulation if input continues. Branches, transport and other regulation can modify these predictions.

A cyclical pathway regenerates an acceptor or starting intermediate. In the Krebs cycle, an acetyl group enters, carbon dioxide leaves and oxaloacetate is regenerated. In the Calvin cycle, carbon dioxide is incorporated and some product leaves for synthesis while ribulose bisphosphate is regenerated using energy supplied by photosynthesis. "Cyclical" does not mean no net inputs/outputs, no energy requirement or that every entering carbon atom remains forever in the cycle.

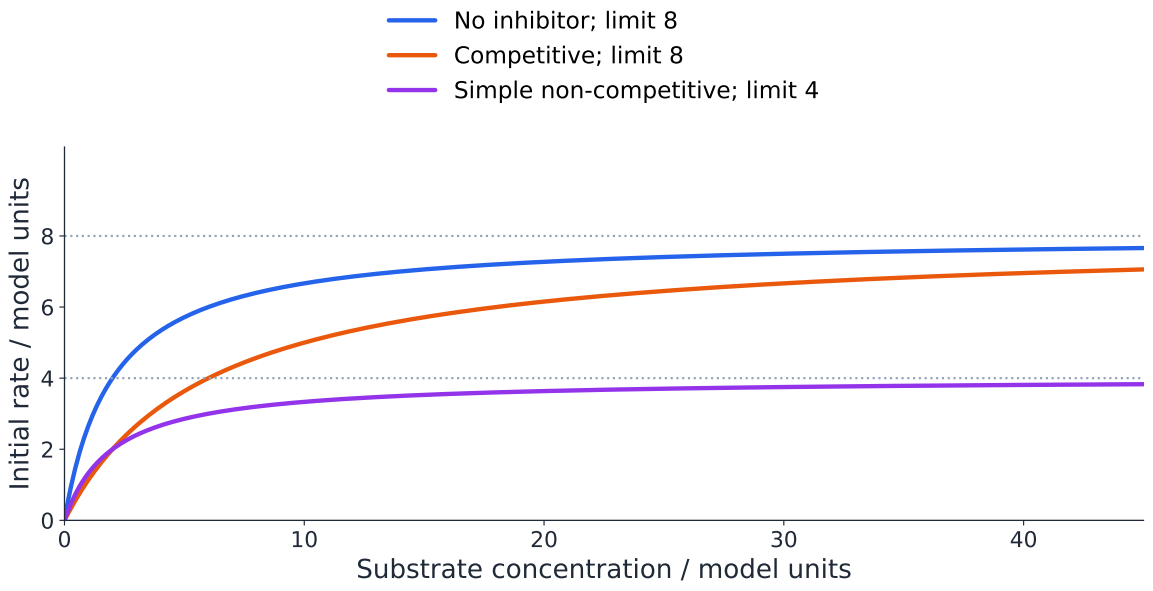
AHL: Reversible inhibitors act at different sites

A competitive inhibitor binds reversibly at the active site, reducing substrate access. At the same substrate concentration, rate falls. Increasing substrate concentration can reduce the inhibitor's effect by making substrate binding more likely; the simple model approaches the same maximum rate at very high substrate. This does not mean that a small increase always completely reverses inhibition.

Statins are a named example: they competitively inhibit HMG-CoA reductase, reducing a step that produces mevalonate in cholesterol synthesis. They do not digest cholesterol already in a blood sample. This is a biochemical example, not an instruction to change medicine. Structural research on enzyme–statin complexes shows occupation of the substrate-binding region.

An allosteric site is separate from the active site. Binding of a specific regulator can change protein conformation and thereby active-site function. In the reversible non-competitive model used here, an inhibitor reduces the amount of effective enzyme activity; increasing substrate cannot restore the original maximum. Allosteric binding need not always inhibit, and not every real allosteric system follows this simplest kinetic model.

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The competitive curve approaches the original maximum; the simple non-competitive curve approaches a lower maximum. These are model curves, not statin dose measurements.

At an invented substrate concentration of 2 units, rates without inhibitor, with competitive inhibitor and with simple non-competitive inhibitor are 4, 2 and 2. At 18 substrate units they are 7.2, 6 and 3.6. One low-substrate comparison cannot distinguish the mechanisms: investigate a concentration series while matching enzyme amount and other conditions. A reduced maximum alone also does not prove a binding site or reversibility; supporting experiments are needed.

AHL: End-product feedback regulates supply

In bacterial isoleucine synthesis, the end product isoleucine binds to a regulatory site on threonine deaminase, an early enzyme in its synthesis from threonine. More isoleucine reduces further pathway entry; when isoleucine is used and its concentration falls, reversible inhibition is relieved. This negative feedback limits unnecessary use of substrates and energy. The product is not simply competing with threonine for the same active site.

Draw threonine, successive intermediates and isoleucine connected by forward arrows. Then draw an inhibitory connection from isoleucine back to the early enzyme, not a matter-flow arrow converting isoleucine back into threonine. A pathway can have several control points; this example does not mean every end product inhibits every enzyme. Research on branched-chain amino-acid synthesis distinguishes the catalytic and regulatory sites.

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AHL: Mechanism-based inhibition changes an active site chemically

Penicillin targets bacterial transpeptidases, proteins involved in cross-linking peptidoglycan in the cell wall. Reaction with the antibiotic forms a stable covalent modification at the active-site serine, inactivating the target on the relevant timescale. This is mechanism-based inhibition rather than a freely reversible molecule simply competing for a site. Adding more substrate does not promptly remove the covalent modification.

Reduced cross-linking weakens construction of a growing wall; penicillin does not directly digest every membrane or inhibit a human cell-wall enzyme. Resistant bacteria can have altered transpeptidases with lower affinity for the drug while retaining enough normal function. Resistant target variants spread by selection or gene transfer; an individual enzyme does not learn to avoid the drug. Drug destruction by beta-lactamases is another resistance route, distinct from target alteration. Structural work on a resistant transpeptidase identifies reduced drug affinity and stable covalent target complexes.

Practice

All numerical data are invented.

- **AHL** Marked questions use the additional sections.

1. Define enzyme and metabolism. Explain why a living cell needs many enzymes and why regeneration does not mean an enzyme remains rigid throughout a reaction.
2. Classify protein synthesis, glycogen formation, photosynthesis, protein digestion and glucose oxidation as anabolic or catabolic. Correct “all bond breaking releases energy”.
3. Explain how folding, chemical properties, induced fit and molecular motion contribute to specificity. Can a membrane-bound enzyme work?
4. Reactants have relative energy 30, products 45, and peaks 95 without enzyme and 60 with enzyme. Calculate both forward activation energies and the overall change. Does the catalyst supply the net energy requirement or alter equilibrium?
5. Rates at 15, 25, 35, 45 and 55 degrees Celsius are 1, 3, 6, 4 and 0.5. Describe and explain the pattern. Give the greatest tested temperature-rate pair and an experiment distinguishing reversible cooling from lasting heat damage.
6. Explain how pH can change rate without every change causing irreversible denaturation. Why can a substrate-concentration curve plateau at fixed enzyme amount?
7. Product volumes at 0, 15, 30 and 60 s are 0, 9, 15 and 21 mL. Find first-interval and whole-period mean rates; distinguish an initial tangent.
8. Identical endpoints take 30 and 75 s. Calculate reciprocal-time proxies and their ratio. State two conditions needed to compare them and explain why their units are not mL/s.

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9. Plan an amylase–starch temperature investigation with controls and repeats. A final positive iodine result occurs at 40 s and the first negative result at 50 s. State what is known about the endpoint and one sampling limitation.
10. A learner counts 24 bubbles in a minute and concludes 24 mL/min. Evaluate this and propose an improved measurement. What extra work is needed to convert absorbance to product concentration?
11. A substrate series approaches 8 rate units with one enzyme dose and 16 with double the dose. Explain the plateaus and why this does not show denaturation. Name two controls needed for the comparison.
12. **AHL** Classify glycolysis, the Krebs cycle and gut-lumen digestion by location; compare glycolysis with the Krebs and Calvin cycles by pathway form. A process receives 300 kJ and retains 90 kJ in useful output, with the remainder becoming heat. Find efficiency and heat; give a limit of the model.
13. **AHL** At low substrate, no inhibitor, inhibitor A and inhibitor B give rates 4, 2 and 2; at high substrate they give 7.2, 6 and 3.6. Suggest simple inhibition models, explain sites/reversibility and why data alone do not prove them. Relate statins and isoleucine feedback to their targets.
14. **AHL** Explain penicillin's mechanism-based inhibition and how an altered transpeptidase can confer resistance. Distinguish target alteration from drug destruction and from an enzyme learning during exposure.

Worked answers

1. An enzyme catalyses a reaction and is regenerated overall; metabolism is the interacting reaction network. Different substrates/reactions require different specific catalysts. Enzyme and substrate can change shape on binding, then products leave and the enzyme returns to another catalytic cycle; regeneration does not imply no temporary conformational change.
2. Protein synthesis, glycogen formation and photosynthesis are anabolic overall; protein digestion and glucose oxidation are catabolic. Breaking bonds requires energy, forming bonds releases energy, and the net balance determines the transfer. Photosynthesis uses energy and is not merely one condensation step.
3. Folding positions active-site groups with suitable shape, charges and other chemical interactions. Induced fit changes both partners and supports catalysis; random motion brings them together with appropriate orientation. A fixed enzyme can encounter mobile substrate, so membrane attachment does not prevent activity.
4. Uncatalysed barrier $95 - 30 = 65$ units; catalysed barrier $60 - 30 = 30$. Overall change $45 - 30 = +15$ units for either route. The enzyme lowers the barrier but does not supply this net energy or change equilibrium; coupling or another energy input is still needed to drive the process.
5. Rate rises from 1 to 6 between 15 and 35 degrees, then falls to 0.5 at 55. Increasing productive encounters explains the rise; structural loss explains the high-

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temperature decline in this model. The greatest tested rate is 6 at 35 degrees, not the exact optimum. Retest cooled/heated samples at a common suitable temperature with fresh substrate, an untreated control and repeats. Recovery supports reversible slowing; persistent loss supports damage.

6. pH alters charges involved in binding/catalysis and may disrupt folding; restoring pH can reverse some effects. At high substrate, sites are occupied for most of the time and turnover becomes limiting. More active enzyme can raise this ceiling if substrate and other conditions permit; extra substrate alone cannot indefinitely increase rate.
7. First interval $9/15 = 0.60$ mL/s; whole period $21/60 = 0.35$ mL/s. The initial tangent estimates the gradient at time zero, not automatically either finite-interval average. Product accumulation slows in these data; report the interval used.
8. $1/30 \approx 0.0333$ per second and $1/75 \approx 0.0133$ per second; first:second $= 75/30 = 2.5$. Require the same endpoint amount/criterion and starting concentrations, with comparable detection and other factors. Reciprocal time is a proxy; no product volume has been measured by that calculation.
9. Equilibrate separate solutions, buffer pH, mix/start consistently and sample onto separate iodine spots. Match starting concentrations/volumes, endpoint criterion and sampling; use fresh mixtures, a no-active-enzyme control and replicates. The endpoint is after 40 s and no later than 50 s. Ten-second spacing limits resolution; minimize volume changes from withdrawals or use parallel tubes. Keep iodine out of the reaction.
10. Bubble number does not determine volume because sizes vary and gas can dissolve or escape. Collect a measured volume using an appropriate apparatus, account for leaks/background and divide by time. A colorimetric assay needs a suitable blank, calibration and a measured concentration-time change before a concentration rate is calculated.
11. At saturation, occupation/turnover limits fixed enzyme activity. Double the active enzyme provides twice the capacity: 16 rather than 8 in this model. Plateau alone does not show denaturation. Match pH, temperature, volume and sufficient substrate; verify enzyme doses and the initial-rate method.
12. **AHL** Glycolysis is intracellular in cytosol; the eukaryotic Krebs cycle is intracellular mainly in mitochondrial matrix; gut-lumen digestion is extracellular to secreting cells. Glycolysis is linear; Krebs and Calvin cycles regenerate acceptors while having net inputs and outputs. Useful fraction $90/300 \times 100 = 30\%$; heat $300 - 90 = 210$ kJ. This assumes the stated boundary and no other energy output; it is not a universal cellular efficiency. Metabolic heat contributes to temperature maintenance in birds and mammals.
13. **AHL** A fits a competitive model: reversible active-site binding and a high-substrate rate approaching the original maximum. B fits simple reversible non-competitive inhibition: regulatory-site binding changes effective activity and lowers the maximum. A concentration series and binding/recovery evidence

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are needed; two points do not locate sites or establish reversibility. Statins competitively inhibit HMG-CoA reductase in mevalonate/cholesterol synthesis. Isoleucine inhibits early threonine deaminase at a regulatory site; use of product relieves inhibition, limiting unnecessary pathway entry.

14. **AHL** Penicillin forms a stable covalent modification at a transpeptidase active site, reducing peptidoglycan cross-linking; extra substrate does not quickly restore that enzyme. An altered target can bind drug less effectively while retaining wall-building function. Beta-lactamase destroys drug instead. Resistant variants arise and spread through genetic change, selection or transfer; the same enzyme does not learn a new response during treatment.

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Cell respiration

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A germinating seed can use oxygen without breathing through lungs, and a muscle can make ATP when oxygen delivery cannot meet its immediate demand. Both observations concern chemical reactions in cells. To explain them, follow energy transfers, carbon atoms and electron carriers, rather than treating respiration as another word for moving air.

This unit covers C1.2 SL and HL: ATP, its uses and recycling, respiratory substrates, aerobic and anaerobic pathways in humans, and measuring respiration. Sections marked **AHL** are for Higher Level only.

- **AHL** Additional content covers NAD, glycolysis, lactate and yeast fermentation, the link reaction, Krebs cycle, electron transport, chemiosmosis, oxygen and differences between carbohydrate and lipid substrates.

Prerequisites are enzyme-controlled pathways, oxidation, membrane transport and rate calculations. All numerical examples and experimental data below are invented for teaching. Scope follows the IB Biology guide.

Exam tips: Write the quantity measured, its units, the control correction and the normalization. Separate ventilation, gas exchange, oxygen uptake and ATP production. ATP hydrolysis has an overall energy release; isolated bond breaking does not.

ATP connects energy release with cellular work

ATP stands for adenosine triphosphate. It is a nucleotide containing adenine, ribose and three phosphate groups. Adenosine is adenine plus ribose; ADP, adenosine diphosphate, has two phosphate groups. ATP is a transferable, short-term energy currency, not the main long-term energy store of a whole organism.

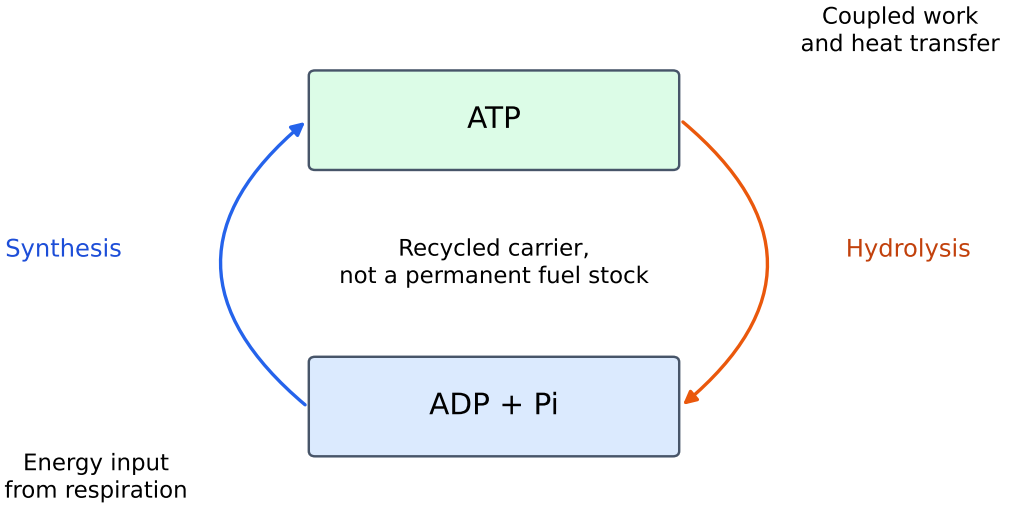
Hydrolysis of ATP to ADP and inorganic phosphate releases energy overall under cellular conditions. Cells couple this reaction to processes that need energy. Breaking a phosphate bond alone does **not** release energy: bond breaking requires energy, and the overall hydrolysis, including formation and interactions of the products, has a favourable net energy change.

Energy from respiration can drive phosphorylation of ADP, rebuilding ATP from ADP and inorganic phosphate. ATP synthesis requires energy; ATP hydrolysis can support a coupled process. A cycling ATP supply links where energy becomes available to where it is needed. ATP is rapidly regenerated and used; a cell cannot rely on one permanent stock that is never replenished.

ATP can support active transport against an electrochemical gradient, macromolecule synthesis, muscle movement and chromosome movement during cell division. Specific

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proteins couple ATP use to each process; ATP does not release a free energy packet that acts at a distance. Different processes need not use the same ATP amount.



Pi is inorganic phosphate. The arrows represent coupled transfers. ATP is rebuilt using an energy supply; its hydrolysis can support work, with some energy dissipated as heat.

An invented cell makes and uses 600 ATP molecules per second while its average ATP pool stays at 120 molecules. Its turnover corresponds to $600/120 = 5$ pool-equivalents per second. A constant ATP concentration therefore does not imply no ATP use. If use remains high while synthesis falls, the pool can decline and energy-dependent work becomes harder to sustain.

Respiration is cellular chemistry rather than ventilation

Respiration transfers energy from organic substrates through enzyme-controlled reactions and supports ATP production. Glucose and fatty acids are principal substrates; other organic compounds can also enter respiratory metabolism after suitable conversion. The cell does not create energy. It transfers energy from chemical substrates into usable chemical forms, work and heat.

Ventilation moves air into and out of an organ such as a lung. Gas exchange moves gases across a surface, often by diffusion. Cellular respiration consists of chemical reactions. A plant cell or a seed can respire without lungs, and lung ventilation alone is not ATP synthesis. Photosynthetic cells also respire, including when light is unavailable.

In aerobic respiration, oxygen is required for sustained complete oxidation of substrates through the aerobic system. For glucose, the simplified word equation is:

$$\text{glucose} + \text{oxygen} \rightarrow \text{carbon dioxide} + \text{water}$$

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Energy released by the overall process supports ATP production; some becomes heat. ATP is not a carbon-containing waste product in that word equation, and heat is not matter. The balanced overall chemical equation is $C_6H_{12}O_6 + 6O_2 \rightarrow 6CO_2 + 6H_2O$. It summarizes many reactions, not a single explosive step.

In human cells, glycolysis begins in the cytosol and aerobic stages depend on mitochondria. Oxygen-rich blood cannot make a cell with no functioning aerobic machinery carry out all those mitochondrial reactions. Conversely, the cytosol can still support a limited ATP-producing pathway without mitochondrial participation.

Human anaerobic pathways maintain a smaller ATP supply

When oxygen supply cannot support the required aerobic rate, human cells can obtain ATP through anaerobic metabolism of carbohydrate, with lactate as a final product. The simplified word equation is glucose → lactate. This pathway yields much less ATP per glucose than complete aerobic respiration; a larger fraction of the substrate's chemical energy remains in the organic product.

The anaerobic pathway occurs in the cytosol and does not require mitochondria. Aerobic respiration of glucose includes cytosolic and mitochondrial stages, requires oxygen for the system to continue, and has carbon dioxide and water as overall products. Fatty acids can support aerobic respiration but cannot replace glucose in the human lactate-producing pathway.

Feature	**Human aerobic glucose respiration**	**Human anaerobic glucose metabolism**
Oxygen requirement	Needed for sustained aerobic operation	Not required for the ATP-producing pathway
Location	Cytosol followed by mitochondrial stages	Cytosol
Overall carbon-containing product	Carbon dioxide	Lactate
ATP per glucose	Much larger yield	Small yield; two net ATP in the full pathway
Mitochondria	Required for aerobic stages	Not required

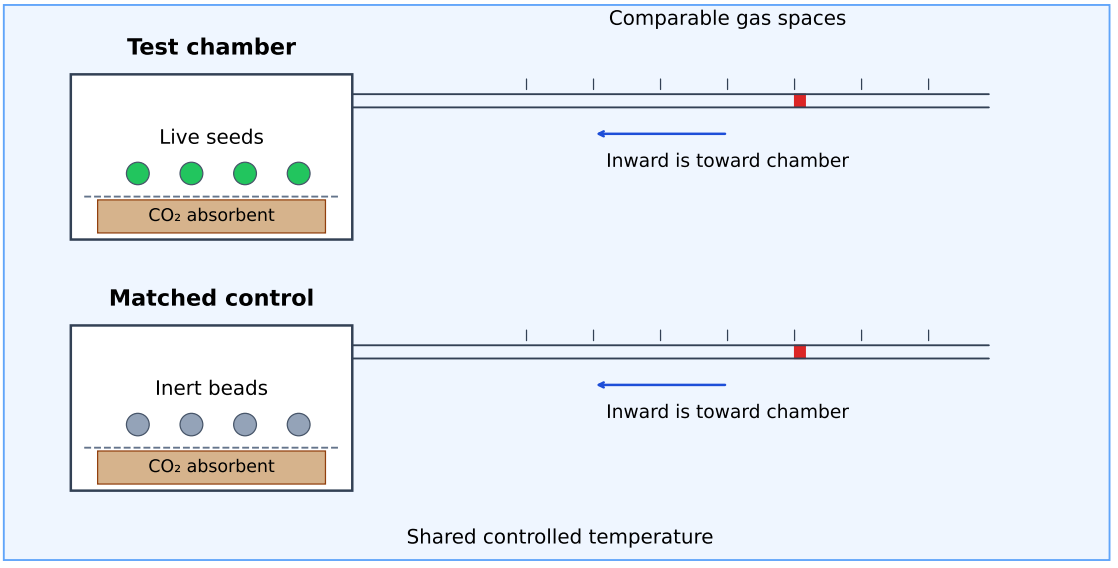
A cell can use aerobic and anaerobic routes at the same time; this is not necessarily an instantaneous all-or-nothing switch. Lactate can be transported and used in later metabolism, so calling it a permanently useless poison is misleading. Aerobic respiration can use carbohydrate or fatty acids, while the anaerobic comparison here specifically concerns human carbohydrate metabolism.

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A respirometer needs control of gas volume, temperature and pressure

A sealed respirometer can estimate oxygen uptake by a living sample. A suitable carbon dioxide absorbent removes carbon dioxide produced by respiration. As oxygen is removed from the gas phase, the resulting pressure/volume change moves a liquid marker in a capillary. Under controlled conditions this provides an oxygen-uptake estimate; it does not directly count ATP molecules or measure every anaerobic reaction.

Temperature changes alter gas volume and pressure. External pressure changes, leaks, unequal starting gas spaces and incomplete equilibration can also move a marker or distort the result. A matched control with non-respiring material helps estimate background drift. Use comparable occupied volume and gas space, the same absorbent arrangement and a common temperature-controlled bath. A control is not made equivalent simply by giving glass beads the same mass as seeds.



Keep absorbent physically separated from organisms. Compare matched gas spaces, allow equilibration and account for background drift before attributing movement to oxygen uptake.

Use germinating seeds or another approved sample under a school protocol. Keep corrosive absorbent separated from living material with a suitable barrier, and follow the risk assessment. Set the starting marker position after equilibration, seal as required, use a fixed interval and check leaks. Repeated measurements on the same sample describe repeatability; independent biological samples are needed to estimate variation between samples.

If a calibrated apparatus indicates 0.80 cubic centimetres of oxygen uptake in 4.0 minutes for 2.0 g of tissue, the mass-specific rate is

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$$\frac{0.80}{4.0 \times 2.0} = 0.10 \text{ cubic centimetres per minute per gram.}$$

Normalizing for time and mass supports comparison, but does not remove differences in species, developmental stage, water content or tissue condition. State the mass basis and use comparable samples. An oxygen-uptake rate alone is not a direct measure of useful energy retained as ATP because substrates, coupling and maintenance costs can differ.

Convert marker displacement into volume before calculating rate

For a uniform capillary, gas-volume change is cross-sectional area multiplied by displacement. If radius is r and corrected distance is d, the volume is $\pi r^2 d$. Keep all lengths in consistent units. A displacement in millimetres is not a volume in millilitres, and confusing radius with diameter changes the calculated area by a factor of four.

In an invented apparatus, radius is 1.0 mm, inward test displacement is 30.0 mm and matched inward control drift is 6.0 mm. Under the stated subtraction model, corrected displacement is $30.0 - 6.0 = 24.0$ mm. Volume is $\pi(1.0)^2(24.0) \approx 75.4$ cubic millimetres, or 0.0754 cubic centimetres. Over 4.0 minutes with 1.5 g of sample, rate is about $0.0754/(4.0 \times 1.5) = 0.0126$ cubic centimetres per minute per gram, or 0.013 to two significant figures.

Subtract drift with its sign: if the control moves outward while test moves inward, using both positive magnitudes and subtracting them can be wrong. Define a positive direction and justify that the control estimates the relevant shared drift. Large or unstable drift suggests the apparatus needs improvement, rather than that any subtraction automatically recovers a valid respiration rate.

For a temperature investigation, compare fresh equivalent samples at several controlled temperatures after equilibration. Hold sample type, germination stage, mass basis, gas space, oxygen availability and measurement interval suitably constant. Repeat and plot the rate against temperature with variation shown. A rate change can reflect enzyme activity and sample physiology; high temperature can damage the tissue. Do not infer an exact optimum from two temperatures or confuse three readings on one seed group with three independent groups.

AHL: NAD links substrate oxidation to another reduction

Oxidation is electron loss, while reduction is electron gain. Removing hydrogen with electrons from an organic substrate oxidizes it. Oxidized NAD accepts a pair of electrons and a proton to become reduced NAD, also called NADH. A simplified electron-accounting equation is:

$$\mathrm{NAD^+} + 2e^- + H^+ \rightarrow \mathrm{NADH}.$$

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The paired reaction oxidizes a substrate as NAD is reduced. Later NADH transfers electrons to another acceptor, becoming oxidized NAD again. NAD is therefore a recyclable carrier, not a permanent sink or a source that creates electrons. Regeneration matters: a pathway requiring oxidized NAD slows or stops if its carrier pool remains reduced.

AHL: Glycolysis invests ATP before making a net gain

In glycolysis, one six-carbon glucose is phosphorylated using two ATP in the investment stage. A six-carbon intermediate splits into two three-carbon phosphorylated molecules. Their subsequent oxidation reduces two NAD to NADH per original glucose, and substrate-level phosphorylation forms four ATP in total. The two three-carbon products are pyruvate.

Gross ATP production is four, but two were invested, giving a **net gain of two ATP per glucose**. Both the gross/net distinction and the duplication of reactions after splitting matter. Counting each later event once instead of twice loses half the products. Glycolysis itself does not release carbon dioxide and does not directly consume oxygen; whether it can continue depends on regeneration of the needed carriers and substrates.

In human anaerobic metabolism, pyruvate is reduced to lactate using NADH. This regenerates oxidized NAD so glycolysis can continue. Converting pyruvate to lactate does not add another ATP yield; the net two ATP came from glycolysis. Carbon remains in two three-carbon lactate molecules for each six-carbon glucose.

In yeast fermentation, pyruvate loses carbon dioxide to form ethanal; ethanal is reduced to ethanol using NADH, regenerating NAD. Per glucose, the products are two ethanol molecules and two carbon dioxide molecules, with the same net two ATP from glycolysis. Brewing uses ethanol production; baking uses carbon dioxide to expand dough. Oxygen conditions, temperature and substrate availability affect the process; yeast and human final products must not be swapped.

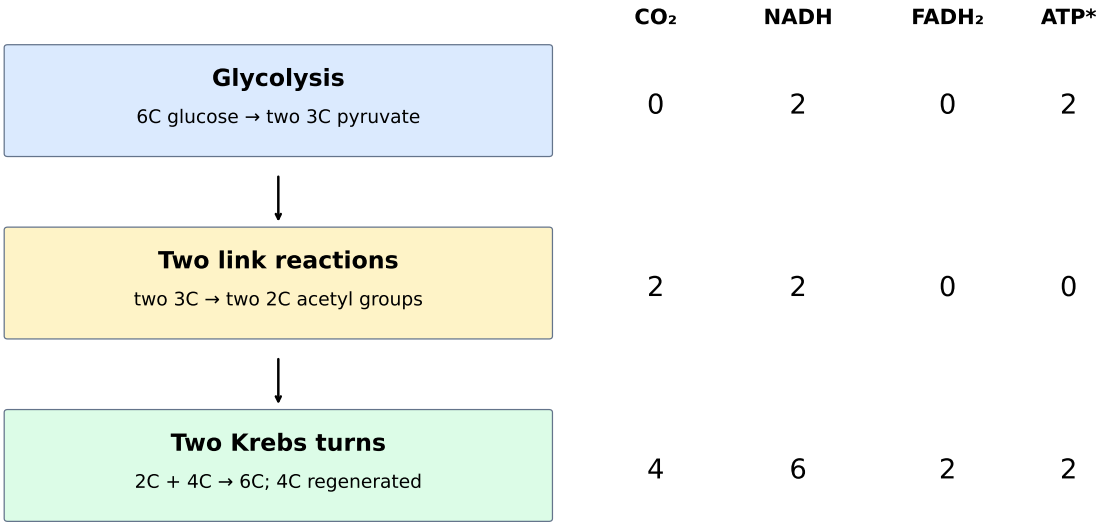
AHL: The link reaction and Krebs cycle account for carbon separately

In aerobic eukaryotic respiration, pyruvate enters the mitochondrial matrix. Each three-carbon pyruvate is oxidized and decarboxylated in the link reaction: one carbon leaves as carbon dioxide, NAD is reduced, and the remaining two-carbon acetyl group attaches to coenzyme A. One glucose gives two pyruvate, hence two acetyl groups, two carbon dioxide and two NADH at this stage. The link reaction does not directly produce ATP.

In the Krebs cycle, a two-carbon acetyl group joins four-carbon oxaloacetate to form six-carbon citrate. Subsequent reactions regenerate oxaloacetate. Two decarboxylations release two carbon dioxide per turn; four oxidations transfer electrons to carriers. In standard bookkeeping these yield three NADH and one reduced FAD per acetyl

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group, together with one ATP-equivalent by substrate-level phosphorylation. Four oxidations do **not** mean four NADH: the electron acceptor differs at one step.



Per glucose; *net substrate-level ATP-equivalents only

Count per original glucose. Oxaloacetate is regenerated, so it is not a new four-carbon net input on every turn. Electron transport is not included in the substrate-level ATP column.

Stage per glucose	Carbon dioxide	Reduced NAD	Reduced FAD	Net substrate-level ATP-equivalents
Glycolysis	0	2	0	2
Two link reactions	2	2	0	0
Two Krebs turns	4	6	2	2
Total	6	10	2	4

The carrier totals describe the standard complete-glucose model, not ATP molecules made directly by those carriers. Oxaloacetate regeneration and carbon counts explain cycle organization, but do not identify which particular carbon atom leaves in the first turn. Other pathways can draw off intermediates and need replenishment.

AHL: Electron transport builds a gradient across the inner membrane

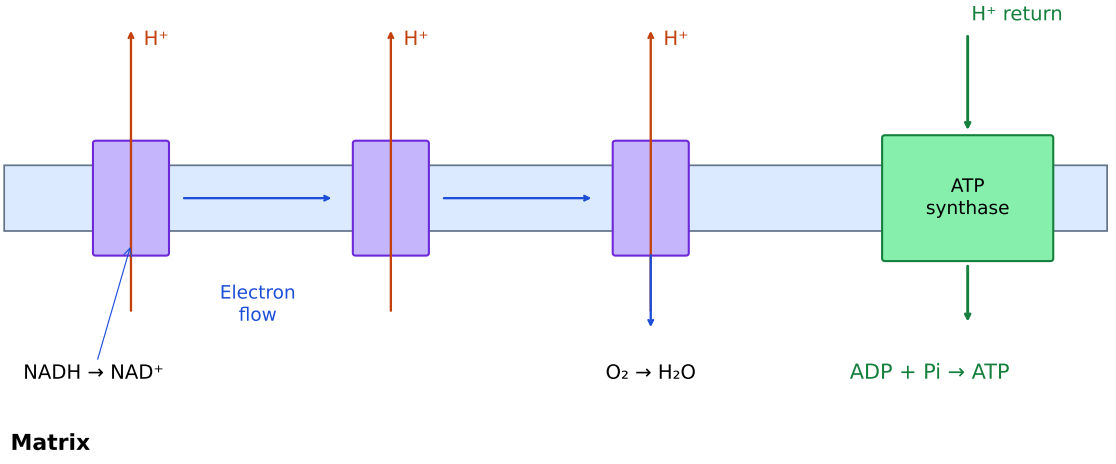
Reduced NAD supplies electrons from glycolysis, the link reaction and the Krebs cycle to the mitochondrial electron-transport system. Matrix NADH transfers a pair of electrons to the chain and is oxidized to NAD. Reducing equivalents from cytosolic

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NADH enter through shuttle systems; the NADH molecule does not simply diffuse freely across the inner membrane.

Energy transferred as electrons pass along carriers drives proton pumping from the matrix into the intermembrane space. This creates an electrochemical proton gradient: both concentration and electrical potential contribute. The inner membrane limits uncontrolled proton return; the gradient is across that membrane, not across the outer membrane or across the whole cell surface.

Oxygen is the terminal electron acceptor. It combines with electrons and protons to form water, allowing electron flow to continue. Oxygen is not converted into the carbon dioxide released by decarboxylation, and it does not directly join ADP to phosphate. A useful terminal step is $O_2 + 4e^- + 4H^+ \rightarrow 2H_2O$.



Electron flow pumps protons out of the matrix. Protons return through ATP synthase, coupling gradient energy to ATP formation on the matrix side.

AHL: Chemiosmosis couples proton return to ATP synthesis

Protons move from the intermembrane space toward the matrix through ATP synthase, down their electrochemical gradient. The enzyme couples that flow to phosphorylation of ADP using inorganic phosphate. This is chemiosmosis and supports oxidative phosphorylation. The return through ATP synthase is opposite to the proton-pumping direction driven by electron transport.

If electron flow stops, proton pumping falls; existing gradients can support ATP synthesis briefly but dissipate without continued maintenance. NADH oxidation is impaired, limiting regeneration of NAD and therefore upstream oxidation. If instead protons leak across the membrane without passing through ATP synthase, electron transport and oxygen uptake can continue while ATP yield falls. Oxygen consumption therefore does not by itself prove efficient ATP production.

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Do not insist on one universal total such as 36 or 38 ATP per glucose for every eukaryotic cell. Shuttle pathways, proton leak, transport and other costs affect the final yield. The robust comparisons are the small anaerobic net yield, the greater aerobic yield, the substrate-level contributions and the role of the gradient. A stated model yield can still be used for a calculation if its assumptions are given.

AHL: Lipids and carbohydrates enter the pathway differently

Fatty acids are broken down into acetyl groups, carried by coenzyme A into the Krebs cycle; the usual even-chain fatty-acid model enters downstream of glycolysis. These reactions also generate reduced carriers. Fatty acids contain relatively less oxygen and more oxidizable carbon/hydrogen per gram than carbohydrate, giving a higher energy yield per gram on complete oxidation.

For an invented comparison using 39 kJ/g for fat and 17 kJ/g for carbohydrate, 8.0 g gives 312 kJ versus 136 kJ, a ratio about 2.29. These are supplied overall energy values, not a claim that every gram yields this much ATP energy. Physiological conditions and the chemical substrate matter.

Carbohydrate can feed glycolysis and support the human lactate-producing anaerobic route. Fatty-acid oxidation cannot sustain that route: its reduced carriers require the functioning aerobic system for continued reoxidation. “Fat has more energy” does not imply it can replace carbohydrate in every oxygen-limited situation. Glycerol and other lipid components have their own entry routes, so distinguish a fatty acid from every molecule associated with a lipid.

Practice

All data are invented.

- **AHL** Marked questions use the additional sections.

1. State ATP’s full name and components. Explain how ATP synthesis and hydrolysis connect respiration with two named cellular processes, and correct “breaking the phosphate bond itself releases energy”.
2. A cell maintains a pool of 150 ATP molecules while synthesizing and using 750 per second. Calculate pool-equivalents used per second. Explain why a steady concentration does not mean no ATP turnover.
3. Distinguish ventilation, gas exchange and cellular respiration. Explain why a seed and a plant cell in darkness can respire.
4. Compare human aerobic and anaerobic glucose pathways by oxygen requirement, location, products, mitochondrial requirement and relative ATP yield. Write simple word equations and state whether fatty acids can replace glucose in the lactate pathway.

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5. Explain why respiration transfers rather than creates energy, why oxidation need not involve a flame, and why oxygen uptake alone does not give the exact ATP output.
6. Describe how a respirometer with carbon dioxide absorbent estimates oxygen uptake. Identify two non-respiratory causes of marker movement and the role of a matched inert-material control.
7. A sample of mass 1.5 g uses 0.96 cubic centimetres of oxygen in 4.0 minutes. Calculate its mass-specific uptake rate and give the units.
8. Capillary radius is 0.50 mm. Test and matched control move inward by 40.0 and 8.0 mm in 5.0 minutes. Sample mass is 0.80 g. Under the subtraction model, find corrected volume in cubic centimetres and mass-specific rate. What error follows from using the diameter as the radius?
9. Plan a temperature comparison using germinating seeds. Explain equilibration, comparable sample/gas-space conditions, repeats and absorbent separation.
10. A control moves outward by 2 mm while the test moves inward by 10 mm. Taking inward as positive, find corrected displacement. Why would large erratic control movement still undermine the experiment?
11. Three independent seed samples give mass-specific rates 0.09, 0.11 and 0.10 in the same units. Calculate the mean and range. State why normalizing by mass does not remove all differences between samples and why the range is not automatically a confidence interval.
12. **AHL** Account per glucose for glycolysis, the two link reactions and two Krebs turns: carbon products, carbon dioxide, NADH, reduced FAD and net substrate-level ATP. Explain NAD recycling, ATP investment versus gross production and oxaloacetate regeneration.
13. **AHL** Explain electron transport, proton-pumping direction, oxygen's terminal role and proton return through ATP synthase. Predict the effects of blocking electron flow versus allowing proton leakage; why can oxygen uptake persist with reduced ATP yield?
14. **AHL** Compare NAD regeneration and final carbon products in human lactate formation and yeast fermentation, including brewing/baking. Compare 6.0 g each of fat and carbohydrate using supplied values 39 and 17 kJ/g, and explain why the higher-energy substrate cannot replace carbohydrate in every anaerobic situation.

Worked answers

1. Adenosine triphosphate contains adenine, ribose and three phosphates; adenosine is adenine plus ribose. Respiration supports ATP synthesis from ADP and inorganic phosphate. Coupled hydrolysis supports active transport, macromolecule synthesis or movement of muscles/chromosomes. Hydrolysis releases energy overall, but isolated bond breaking requires energy; formation and interactions of products determine the net change.

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2. $750/150 = 5$ pool-equivalents per second. Equal synthesis and use maintain the pool while individual molecules cycle rapidly. If synthesis falls without a matching fall in use, ATP concentration can decline and ATP-dependent functions are limited.
3. Ventilation moves air; gas exchange transfers gases across a surface; respiration is cellular chemistry supporting ATP production. Seeds use stored substrates in living cells. Plant cells also respire in darkness: respiration differs from photosynthesis and does not require lungs.
4. Aerobic: glucose + oxygen → carbon dioxide + water; oxygen required, cytosolic glycolysis then mitochondrial stages, much greater ATP yield. Human anaerobic: glucose → lactate; cytosol, no mitochondria required, no oxygen required for that pathway, two net ATP per glucose. Both routes can operate simultaneously. Fatty acids do not replace glucose in the lactate-producing route.
5. Chemical energy in substrates is transferred to ATP, work and heat rather than created. Enzyme-controlled steps can proceed without combustion or a visible flame. Oxygen uptake measures one gas flux; substrates, energy coupling and other costs vary, so it is not an exact count of ATP made.
6. Absorbent removes respiratory carbon dioxide; oxygen uptake then reduces gas-phase amount and moves the marker under controlled conditions. Temperature/pressure drift, leaks or poor equilibration can distort movement. A non-respiring control with comparable gas space and apparatus estimates shared drift. Keep absorbent separated from organisms.
7. $0.96/(4.0 \times 1.5) = 0.16$ cubic centimetres per minute per gram. Divide by both duration and sample mass; retain the mass basis when comparing samples.
8. Corrected distance $40.0 - 8.0 = 32.0$ mm. Area $\pi(0.50)^2 \approx 0.7854$ square millimetres; volume $0.7854 \times 32.0 \approx 25.13$ cubic millimetres $= 0.02513$ cubic centimetres. Rate $0.02513/(5.0 \times 0.80) \approx 0.00628$, or 0.0063 cubic centimetres per minute per gram to two significant figures. Using diameter 1.0 mm as radius would multiply area, volume and rate by four.
9. Equilibrate equivalent samples in controlled baths before resetting/sealing markers as required and timing uptake. Match species/stage, mass basis, gas space, oxygen availability and duration; use matched controls and independent samples with repeats. Separate absorbent by a suitable barrier under the school protocol. Bath expansion or tissue damage can distort the comparison.
10. Test +10 mm; control −2 mm; corrected displacement $10 - (-2) = 12$ mm. The subtraction assumes control drift represents the test's shared background. Large unstable changes can indicate leaks or poor equilibration, and subtraction cannot guarantee a valid oxygen estimate.
11. Mean $(0.09 + 0.11 + 0.10)/3 = 0.10$; range $0.11 - 0.09 = 0.02$ in the stated units. Mass normalization leaves differences in tissue type, stage, water content and condition. Range describes observed spread; a confidence interval needs a defined statistical method and assumptions.

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12. **AHL** Glycolysis: 6C glucose → two 3C pyruvate, no carbon dioxide, two NADH, gross four ATP minus investment two = net two. Link: two 3C pyruvate → two 2C acetyl groups + two carbon dioxide, two NADH, no ATP directly. Krebs: two turns release four carbon dioxide, produce six NADH, two reduced FAD and two ATP-equivalents. Totals: six carbon dioxide, ten NADH, two reduced FAD and four net substrate-level ATP-equivalents before oxidative phosphorylation. Oxidized NAD accepts electrons/hydrogen and must be regenerated; oxaloacetate accepts acetyl groups and is regenerated, not consumed as a new net input each turn.
13. **AHL** Electrons from reduced carriers pass through the inner-membrane chain; released energy pumps protons from matrix to intermembrane space. Oxygen accepts electrons and protons to form water. Protons return to matrix through ATP synthase, coupling gradient energy to ADP phosphorylation. Blocking electron flow stops continued pumping; the gradient then dissipates, ATP synthesis falls and NADH oxidation is impaired. Proton leakage instead bypasses ATP synthase: oxygen uptake/electron transport can continue while less gradient energy is captured in ATP, so oxygen flux alone does not establish efficiency.
14. **AHL** In humans, pyruvate accepts reducing equivalents from NADH to form lactate, regenerating NAD; two 3C lactates per glucose and no extra ATP at that conversion. In yeast, pyruvate loses carbon dioxide, then ethanal is reduced to ethanol; two ethanols and two carbon dioxide per glucose, regenerating NAD. Both retain net two ATP from glycolysis. Brewing uses ethanol, baking carbon dioxide. Supplied energy comparison: $6.0 \times 39 = 234$ kJ versus $6.0 \times 17 = 102$ kJ; ratio $234/102 \approx 2.29$. Fatty acids enter mainly as acetyl groups and depend on aerobic carrier reoxidation; they cannot sustain the human glycolytic lactate route despite their larger energy per gram.

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Photosynthesis

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A brighter lamp can increase a plant's oxygen output, but only up to a point. Understanding the plateau needs both an energy pathway and a controlled experiment.

This handout covers full common C1.3 content: energy conversion, oxygen production, pigments and chromatography, absorption and action spectra, limiting factors and carbon-dioxide enrichment. Sections marked **AHL** are for Higher Level only.

- **AHL** adds photosystems, photolysis, cyclic and non-cyclic photophosphorylation, chloroplast compartments and the Calvin cycle.

Prerequisites: membranes, enzyme activity, ATP, oxidation and reduction. All numerical examples and datasets below are invented for teaching; pigment curves are illustrative models, not measured spectra.

Exam tips: State which rate was measured, keep carbon separate from energy, and name the controlled variable that makes a comparison valid. A plateau alone does not identify the next limiting factor.

Photosynthesis converts light energy into chemical energy

Photosynthesis uses light energy to build organic compounds from carbon dioxide. In oxygenic photosynthesis, water supplies hydrogen and electrons; oxygen is released as a by-product. Plants, algae and cyanobacteria perform oxygenic photosynthesis. Cyanobacteria do not need chloroplasts: their photosynthetic membranes are inside prokaryotic cells. Some other photosynthetic bacteria use different electron donors and do not release oxygen.

Word equation: carbon dioxide + water → glucose + oxygen, using light energy and photosynthetic pigments. A simplified net equation for producing one glucose equivalent is

$$6CO_2 + 6H_2O \xrightarrow{\text{light}} C_6H_{12}O_6 + 6O_2.$$

This balances atoms, but is not a single reaction or a map of atom movements. The oxygen released comes from water, not from splitting carbon dioxide. Carbon dioxide supplies the carbon incorporated into carbohydrate. Light supplies energy; it does not supply carbon atoms. Chlorophyll participates in absorption rather than being consumed as a reactant in this net equation.

Energy becomes stored in the chemical organization of organic compounds. These can support respiration, growth and food webs. Plants also respire, including in illuminated tissues. A gas measurement in light therefore often records a **net** exchange,

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after respiratory uptake or release has occurred alongside photosynthesis.

If an invented leaf produces oxygen photosynthetically at 15 units per hour and uses 3 units per hour in respiration, net oxygen release is 12 units per hour. Conversely, net 12 plus respiratory use 3 gives gross production 15, **if** the respiration estimate applies under the light conditions. A dark measurement is a useful estimate, but assuming that respiration is identical in darkness and light is a limitation to state.

Separate pigments before interpreting their absorption

Chloroplasts contain several pigments, including chlorophyll a, chlorophyll b and carotenoids. They absorb different ranges of visible wavelengths. Their combined absorption broadens the light that can support photosynthesis. Leaf colour reflects light that is reflected or transmitted as well as what is absorbed; a green leaf does not absorb no green light at all.

Chromatography separates an extract because pigments differ in their interactions with a stationary phase and a moving solvent. A pigment that spends relatively more time in the mobile phase travels farther under those conditions. Use an approved extraction solvent, small concentrated spots and a covered chamber under the school safety protocol. Keep the starting spot above the solvent level, or the pigment can dissolve directly into the solvent reservoir.

Draw the origin in pencil. Mark the solvent front promptly after removing the paper or plate; it may become invisible as solvent evaporates. Measure from the origin to the **centre of each pigment band**, and from the same origin to the solvent front:

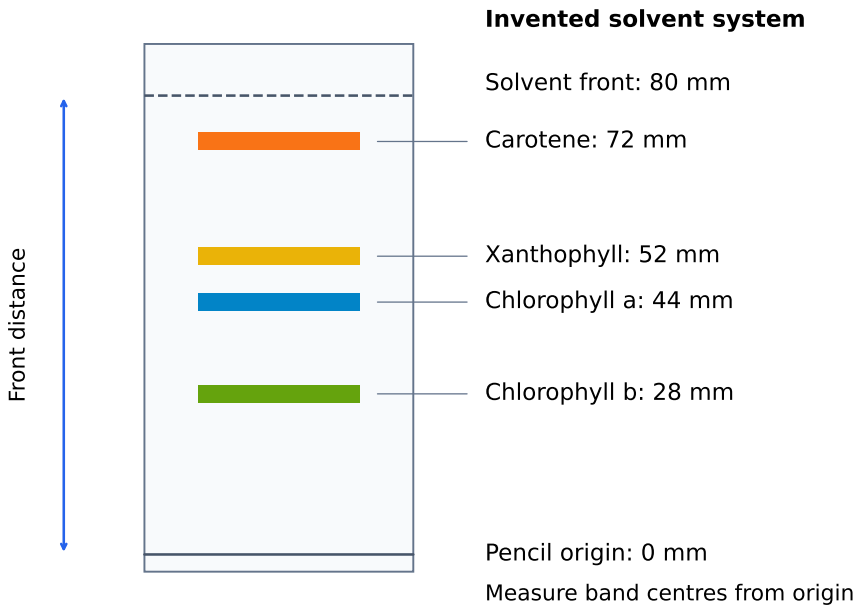
$$R_f = \frac{\text{distance travelled by pigment}}{\text{distance travelled by solvent front}}.$$

The ratio has no unit. For a resolved band between origin and front it is between zero and one. The paper's top edge is not automatically the solvent front. A streaked or overlapping band makes its centre uncertain and may need a better separation.

In this example the front travels 80 mm. Orange carotene travels 72 mm, a yellow xanthophyll 52 mm, blue-green chlorophyll a 44 mm, and yellow-green chlorophyll b 28 mm. Their ratios are 0.90, 0.65, 0.55 and 0.35. A separate unknown band travelling 56 mm has $R_f = 56/80 = 0.70$; it does not match any of those four standards exactly.

Compare unknown and reference pigments under the same solvent, stationary phase and other conditions. Colour and a matching ratio support an identification, but two compounds can migrate together. A ratio is not a universal molecular identity or an absorption wavelength. Changing the solvent can change both the ratios and the order of separation.

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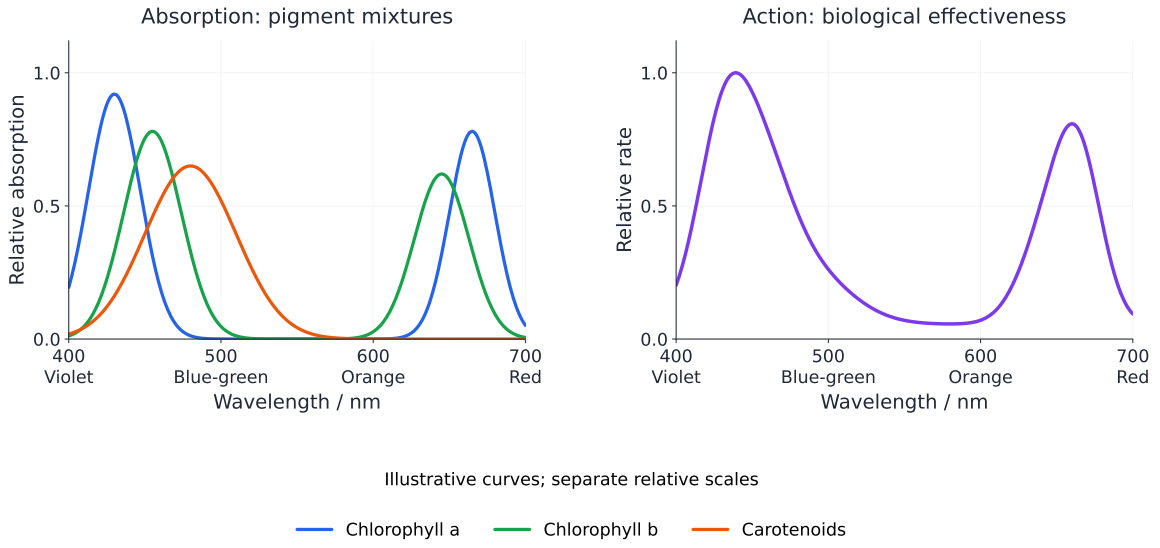
Invented separation: compare band centres with the same solvent front. Names and ratios apply to this teaching example, not every solvent system.

Absorption and action spectra answer different questions

Visible light spans approximately 400–700 nm for this topic. Violet/blue wavelengths are shorter than red wavelengths. A pigment absorbs a photon when the photon can cause an allowed change in electron energy. The electron becomes excited; the nucleus does not absorb light by changing into another element. Different pigments have different patterns of absorption because their molecular structures differ.

An **absorption spectrum** plots how strongly a pigment or pigment mixture absorbs light against wavelength. Chlorophylls absorb strongly in parts of the blue and red regions, and less strongly in much of the green region. Carotenoids absorb mainly in the blue/blue-green region. Exact peaks depend on the pigment and measurement conditions; the curves below are deliberately simplified.

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An absorption curve concerns absorbed light. An action curve concerns biological rate. Curves have separate relative scales and are not interchangeable measurements.

An **action spectrum** plots photosynthetic effectiveness or rate against wavelength, for example using oxygen production or carbon-dioxide uptake. Keep photon supply, temperature, carbon-dioxide availability and sample conditions comparable. Equal lamp power does not necessarily mean equal numbers of photons at each wavelength. State whether the measurement is net gas exchange or a gross rate corrected using an explicit assumption.

In a separate invented test, 450, 550 and 650 nm light each illuminates comparable samples for 2.0 minutes. Supplied oxygen volumes are 4.8, 1.2 and 3.6 cubic centimetres. Rates are 2.4, 0.60 and 1.8 cubic centimetres per minute. Plot wavelength horizontally and rate vertically to make an action spectrum; these three observations alone do not locate an exact peak.

The action spectrum often has relatively high rates in blue and red light, consistent with the absorption of photosynthetic pigments. It is not necessarily identical to the spectrum of purified chlorophyll a. Accessory pigments transfer excitation energy, intact tissues scatter light, and other rate limits can intervene. A green-light rate above zero is compatible with weaker absorption; it does not disprove pigment selectivity.

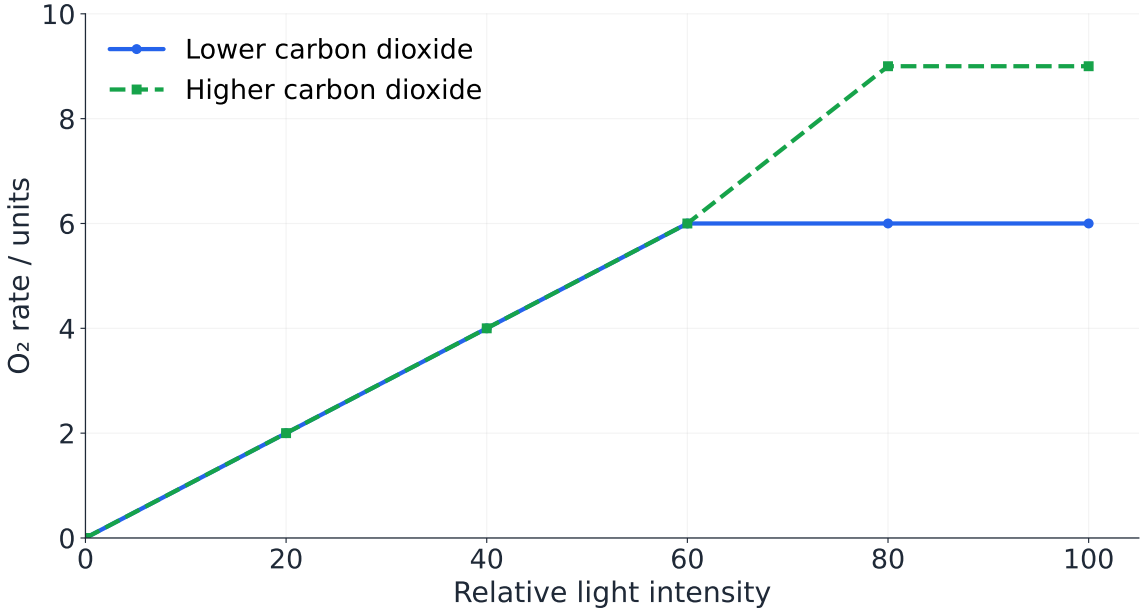
A limiting factor restricts the rate under stated conditions

A **limiting factor** is a factor whose availability or condition restricts rate in the situation tested. At low light, extra photons can increase photosynthesis. At higher light, another constraint can prevent a further increase. Raising light further does not remove insufficient carbon dioxide or a temperature constraint on enzyme-controlled reactions.

Temperature is not simply a material used up. Warming below a suitable range can

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accelerate enzyme-controlled steps. Excessive heat can impair enzymes, membranes and water balance. There is no single universal optimum for every plant and condition. Carbon dioxide is a substrate; greater supply can increase rate when carbon fixation is constrained by its availability and other requirements are met.



The higher-carbon-dioxide treatment raises the high-light plateau in this invented experiment. The original plateau alone would not have identified carbon dioxide as the constraint.

Suppose at fixed temperature and carbon dioxide, raising relative light from 20 to 40 raises oxygen release from 2 to 4 units per minute, while raising light from 80 to 100 leaves it at 6. The second comparison shows little response to added light in that interval. It does not establish whether carbon dioxide, temperature, water status or another factor caused the plateau. Increase carbon dioxide in a separate controlled comparison at the same high light to test one candidate explanation.

If rate then rises from 6 to 9 units per minute, the increase is $3/6 \times 100 = 50\%$. This supports carbon-dioxide limitation under the original high-light conditions. It does not show that further increases will always help, or that the same factor limits at low light. Several constraints can interact rather than acting as one fixed limit for the whole plant's life.

Measure a rate without introducing a second changing variable

A pondweed investigation can collect gas volume over a fixed time after acclimation. For 7.2 cubic centimetres collected in 4.0 minutes, rate is $7.2/4.0 = 1.8$ cubic centimetres per minute. Bubble counts are a rough proxy: bubble volumes vary, and dissolved gas need not immediately enter collected bubbles. Gas identity and collection conditions matter if the claim is specifically about oxygen production.

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Use a suitable light sensor at the sample rather than treating lamp distance as a direct intensity measurement. Moving a lamp also changes heating and sometimes the illuminated area. A controlled water bath, suitable low-heat lighting and temperature measurement help separate these effects. An inverse-square relationship is an approximation for a point source under suitable geometry, not a guaranteed calibration for a nearby extended lamp.

Control species, shoot size or an appropriate mass/area basis, health, temperature, carbon-dioxide supply, acclimation and measurement interval. Changing bicarbonate concentration can change pH as well as dissolved inorganic carbon, so a bicarbonate comparison needs attention to solution chemistry rather than being labelled a pure carbon-dioxide manipulation automatically. Check for leaks and gas-volume changes caused by temperature or pressure.

Use independent biological samples to estimate variation between shoots. Randomizing treatment order or using separate comparable samples helps reduce time/order effects. Repeated readings on one shoot measure repeatability, but do not create independent plants. Report means with a measure of variation and avoid claiming an exact optimum from two temperatures or a causal mechanism from one uncontrolled correlation.

Carbon-dioxide enrichment must be tested at a useful scale

Greenhouses can enrich carbon dioxide while controlling light, temperature, water and mineral supply. If carbon dioxide limits photosynthesis, enrichment can increase assimilation. That does not guarantee a proportional increase in harvested yield: respiration, allocation, water or nitrogen limitation, heat stress and development also affect the crop.

Free-air carbon-dioxide enrichment, or FACE, releases additional carbon dioxide around plants outdoors. It tests enrichment under a field environment with much less enclosure-induced change to wind, humidity and temperature. Maintaining the target concentration in moving air is difficult, however, and weather still varies. Use replicated treatment and control rings, monitor concentrations and compare comparable crops and management. Plants within one enriched ring share an experimental environment; many sampled leaves in one ring are not many independent enrichment treatments.

In invented data, control rings average 400 g of dry harvested product per square metre and enriched rings average 460 g. The yield increase is $60/400 \times 100 = 15\%$. Evaluate ring-to-ring variation and other conditions before attributing the difference to enrichment. A short-term leaf-rate increase alone does not establish whole-season yield, long-term carbon storage, or the response of all species. Predictions are conditional and can be tested rather than treated as guarantees.

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AHL: Photosystems collect excitation energy at reaction centres

Photosystems are membrane-embedded pigment–protein arrangements. They occur in cyanobacterial photosynthetic membranes and, in plants and algae, in chloroplast thylakoid membranes. Antenna pigments absorb photons across a broader range of wavelengths than a single pigment type. Excitation energy is transferred among pigments toward a reaction centre. This is energy transfer through an organized array, not every antenna pigment sending its own electron down the full electron-transport chain.

A special chlorophyll at the reaction centre can transfer an excited electron to an acceptor, initiating electron flow. The oxidized reaction centre must gain a replacement electron to continue. The array combines broad light capture with organized charge separation; an isolated pigment can absorb light but does not on its own carry out the coordinated membrane pathway.

In non-cyclic flow, photosystem II acts before photosystem I; their numbers reflect discovery history, not that order of operation. At photosystem II, light-driven water oxidation replaces electrons lost from the reaction centre:

$$2H_2O \rightarrow O_2 + 4H^+ + 4e^-.$$

This **photolysis** releases oxygen and contributes protons to the thylakoid lumen. Oxygen is a by-product here; it is not the terminal electron acceptor of this pathway as it is in aerobic respiratory electron transport.

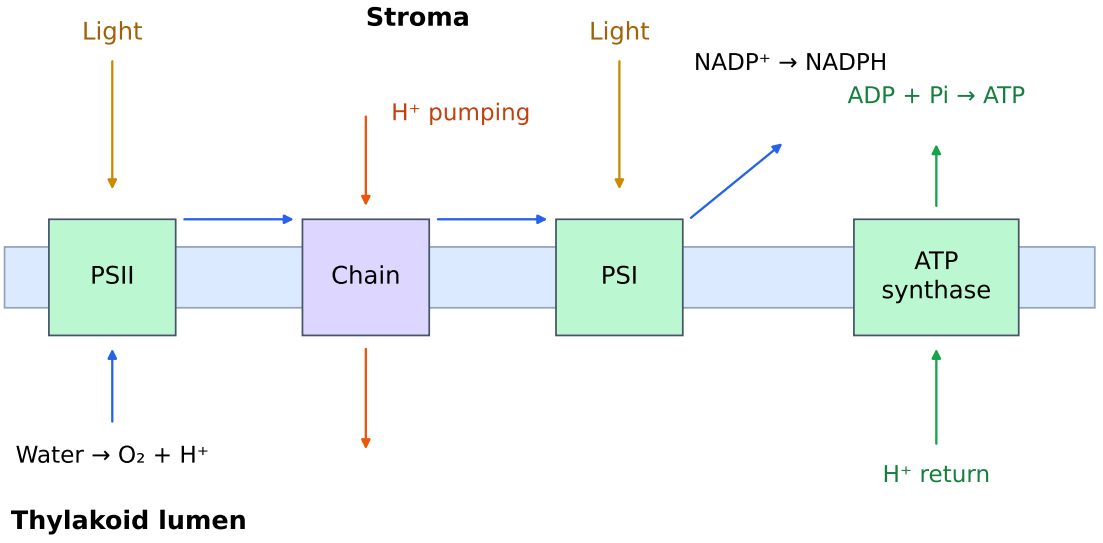
AHL: Thylakoid membranes couple electron flow to ATP and reduced NADP

Electrons pass from photosystem II through carriers toward photosystem I. Energy transfer through the chain supports movement of protons from the stroma into the lumen. Photosystem I absorbs further light energy, re-exciting electrons. They can then support reduction of NADP on the stromal side:

$$NADP^+ + 2e^- + H^+ \rightarrow NADPH.$$

Reduced NADP is NADPH; it carries reducing power for later carbon reactions. It is different from NADH, although both act as electron carriers. Water oxidation replenishes electrons at the start, and NADP accepts them at the end of this non-cyclic route.

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Electron flow is from water through photosystems II and I toward NADP. Proton return through ATP synthase is from lumen to stroma, opposite to pumping.

The membrane limits uncontrolled proton flow. Both proton concentration and electrical differences contribute to the electrochemical gradient. Protons returning from the lumen to the stroma through ATP synthase support ATP production from ADP and inorganic phosphate on the stromal side. This is chemiosmosis. ATP synthesis driven by light-supported electron flow is **photophosphorylation**.

Thylakoid membranes provide extensive area for photosystems, carriers and ATP synthase. The small enclosed lumen allows a proton gradient to develop. The surrounding stroma contains enzymes for carbon fixation. Chloroplast envelope membranes surround the organelle but are not the photosystem-bearing thylakoid membrane; distinguish these boundaries in a diagram.

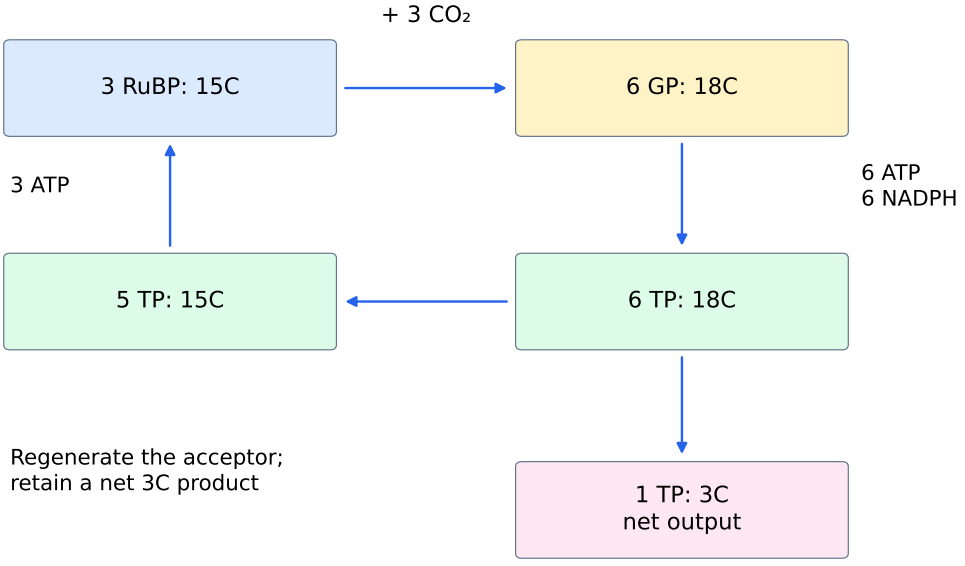
In **cyclic** photophosphorylation, electrons excited at photosystem I return through carriers instead of producing a net reduction of NADP. Their flow can support proton translocation and ATP production. This route involves photosystem I, produces no net NADPH, and does not split water or release oxygen as part of that cyclic route. Non-cyclic flow uses both photosystems and supplies ATP and NADPH while releasing oxygen. Cyclic flow helps adjust ATP supply relative to reducing power; it does not replace the need for carbon dioxide.

AHL: The Calvin cycle fixes carbon and regenerates its acceptor

In the stroma, Rubisco catalyses fixation of carbon dioxide by ribulose bisphosphate, RuBP, a five-carbon acceptor. The unstable six-carbon product forms two molecules of glycerate 3-phosphate, GP, each with three carbons. This is carboxylation, not the production of a complete glucose molecule from a single carbon-dioxide molecule.

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ATP and reduced NADP support conversion of GP into triose phosphate, TP. ATP supplies energy and phosphate-transfer capacity; NADPH supplies reducing power and becomes oxidized NADP. Most TP is used to regenerate RuBP, requiring further ATP. Some TP can leave the cycle for synthesis of other compounds. An enzyme catalyses a reaction rather than being consumed in the carbon ledger.



Three incoming carbon atoms permit a net three-carbon TP output while the original 15-carbon acceptor pool is regenerated. The 15 recycled carbons are not a new net input.

Follow three carbon-dioxide molecules. Three RuBP contain 15 carbons; adding three more produces six GP containing 18 carbons in total. Conversion forms six TP, also containing 18 carbons. Five TP, containing 15 carbons, regenerate three RuBP. One TP, containing three carbons, is the net output. Thus five-sixths of the TP molecules are used for regeneration in this model. This does not mean five-sixths of the newly fixed carbon dioxide is simply wasted or released.

For one net TP, reduction of six GP uses six ATP and six NADPH; regeneration uses three further ATP. The total is nine ATP and six NADPH per three carbon dioxide fixed. Two net TP supply a six-carbon carbohydrate equivalent, requiring six carbon dioxide, 18 ATP and 12 NADPH in this standard accounting. The cycle does not directly deliver a whole glucose at each turn.

TP-derived carbon can enter glucose, sucrose, starch, cellulose, lipid and amino-acid synthesis. Additional pathways and raw materials are needed: amino acids require a nitrogen source, while other compounds require minerals such as phosphorus or magnesium. Fixing carbon cannot create these elements. Rubisco is commonly described as the most abundant enzyme on Earth. High stromal concentrations help support carbon fixation despite relatively slow catalytic turnover and poor performance at

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low carbon-dioxide concentrations; enzyme presence alone does not guarantee a high rate when substrates or energy carriers are limited. Its global abundance has been investigated using biomass and leaf-protein estimates (Bar-On and Milo, 2019).

AHL: Light reactions and carbon reactions depend on each other

Light-dependent reactions provide ATP and reduced NADP. The Calvin cycle uses them and returns ADP, inorganic phosphate and oxidized NADP. Without enough light, ATP and NADPH supply falls, restricting GP reduction and RuBP regeneration. Calling carbon reactions "light-independent" means they do not directly absorb photons; it does not mean they can operate indefinitely in darkness without the products of light reactions.

If carbon dioxide becomes scarce while light remains high, carboxylation and downstream carbon processing are restricted. Use of NADPH decreases, reducing regeneration of NADP as an electron acceptor. Non-cyclic electron flow can then be constrained too. These responses involve transient pools and regulatory/alternative pathways; they do not imply that all photon absorption instantly stops or that the chloroplast is immediately destroyed. This interdependence explains why light alone cannot overcome all carbon-dioxide limitation.

Practice

Attempt each question before using the worked answers. All data are invented.

1. A student says the oxygen released by a leaf is removed from carbon dioxide, and that light supplies the carbon in sugar. Correct both claims and name three groups that carry out oxygenic photosynthesis.
2. A chromatogram's front moves 90 mm. Bands move 81, 54 and 36 mm from the same origin. Calculate their ratios and explain why measuring to the paper's top would be unreliable.
3. An unknown has the same colour and ratio as a reference pigment. Give one condition needed for a fair comparison and one reason identification is still provisional.
4. Distinguish absorption and action spectra. At 450, 550 and 650 nm, invented oxygen volumes in 3.0 minutes are 6.0, 1.5 and 4.5 cubic centimetres. Calculate rates and plot an action spectrum. Explain why it need not equal purified chlorophyll a's absorption spectrum.
5. A leaf's net oxygen release is 9 units per hour; estimated respiratory oxygen uptake is 2 units per hour. Estimate gross production and state the assumption.
6. At fixed temperature and carbon dioxide, photosynthetic rate reaches a plateau as light rises. Explain what the plateau shows, what it does not show, and how to test carbon-dioxide limitation.

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7. Adding carbon dioxide at fixed high light raises an invented rate from 8 to 10 units per minute. Calculate the percentage increase and explain why unlimited further increases are not implied.
8. A shoot produces 9.0 cubic centimetres of gas in 5.0 minutes. Calculate rate, give two controls for a light investigation, and explain one weakness of counting bubbles instead.
9. A student moves a lamp closer and sees a faster gas release. Identify a confounding variable and a way to address it. Explain why ten readings on this one shoot are not ten independent shoots.
10. Invented FACE control rings average 500 g dry yield per square metre; enriched rings average 560 g. Calculate the increase and explain why a higher leaf photosynthetic rate does not guarantee the same percentage yield increase.
11. Compare one advantage and one limitation of FACE with an enclosed greenhouse experiment. Identify the experimental replicate in a trial with four separate enriched rings.
12. **AHL** Trace electrons from water to reduced NADP in non-cyclic flow, distinguishing energy transfer in an antenna from reaction-centre electron transfer. Name the direction of ATP-synthase proton flow.
13. **AHL** Account for carbon, ATP and reduced NADP when six carbon-dioxide molecules supply two net TP. Explain why regenerating RuBP is necessary and compare cyclic flow with non-cyclic flow.
14. **AHL** Light stays high but carbon dioxide supply falls. Explain how restriction of the Calvin cycle can eventually restrict non-cyclic electron flow, and why "light-independent" does not mean independent of light-reaction products.

Worked answers

1. Released oxygen comes from water. Carbon dioxide supplies carbon; light supplies energy. Plants, algae and cyanobacteria carry out oxygenic photosynthesis. The net balanced equation alone does not trace every atom's route.
2. $81/90 = 0.90$, $54/90 = 0.60$, $36/90 = 0.40$. The ratios have no units. The paper top need not be where the solvent front reached, so it can give the wrong denominator.
3. Use the same solvent and stationary phase under comparable conditions. Distinct compounds can co-migrate; colour and ratio are supporting evidence rather than unique proof of identity.
4. Absorption measures absorbed light; action measures biological rate/effectiveness. Rates are $6.0/3.0 = 2.0$, $1.5/3.0 = 0.50$ and $4.5/3.0 = 1.5$ cubic centimetres per minute. Plot those rates vertically against 450, 550 and 650 nm horizontally. Accessory pigments, excitation transfer, tissue optics and other limits can make the curves differ; control photon supply.

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5. Gross estimate $9 + 2 = 11$ units per hour. This assumes the respiratory uptake estimate applies in the illuminated state and the gas quantities use comparable conditions.
6. Extra light produces little rate increase over the plateau interval. The graph alone does not identify the other constraint. Raise carbon dioxide at the same high light with other conditions controlled, replicate and measure variation; a rate rise supports the proposed limitation there.
7. $(10 - 8)/8 \times 100 = 25\%$. Another constraint can limit at a higher carbon-dioxide supply. The response at high light does not establish the response at low light or in a different plant.
8. $9.0/5.0 = 1.8$ cubic centimetres per minute. Control temperature and carbon-dioxide supply, with comparable shoot size and acclimation. Bubble volumes vary, so equal counts need not mean equal gas volumes.
9. Lamp heating can change temperature; use temperature measurement and a controlled bath or suitable lighting. One shoot's repeated readings estimate within-shoot repeatability, not biological variation between independent shoots. Also consider time/order effects.
10. $(560 - 500)/500 \times 100 = 12\%$. Yield depends on whole-season assimilation, respiration, allocation, water, nutrients and development. A short leaf measurement does not fix the final harvested amount; use variation between independent rings to evaluate the trial.
11. FACE retains a more realistic outdoor microclimate, but wind and weather make concentration control harder. A greenhouse allows tighter environmental control but the enclosure changes conditions. The independent ring is the treatment replicate; four separate enriched rings give four enriched treatment replicates, not one replicate per leaf.
12. **AHL** Antenna pigments transfer excitation energy to a reaction centre. Water oxidation replaces electrons lost at photosystem II; electrons pass through carriers to photosystem I, are re-excited and support NADP reduction to NADPH. Water splitting releases oxygen and lumen protons. Proton translocation builds a gradient; ATP-synthase return is lumen to stroma, supporting ATP formation on the stromal side.
13. **AHL** Six carbon dioxide add six carbons to six RuBP (30 carbons), producing 12 GP and then 12 TP (36 carbons). Ten TP regenerate six RuBP (30 carbons), leaving two TP (six carbons) net. The standard cost is 18 ATP and 12 NADPH. Regeneration maintains the carbon acceptor. Cyclic flow uses photosystem I to support ATP without net NADPH or oxygen production; non-cyclic flow uses both photosystems and supplies ATP and NADPH with oxygen release.
14. **AHL** Less carbon fixation reduces NADPH use and regeneration of oxidized NADP. Acceptor shortage can restrict non-cyclic flow despite available light. Conversely, low light restricts ATP/NADPH supply to carbon reactions. Existing pools permit transient activity, so neither shortage instantly stops every reaction.

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Chemical signalling

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A hormone can travel throughout the bloodstream while only certain cells change their behaviour. Delivery of a signal and recognition of that signal are different steps.

This handout covers full C2.1 chemical signalling, an HL-only topic: receptors and ligands, bacterial quorum sensing, animal signals, membrane and intracellular receptors, ion channels, G proteins, adrenaline, insulin, steroid effects and feedback. There is no SL counterpart.

Prerequisites: membrane proteins, diffusion, ATP, gene expression and enzyme activation. Numerical datasets and pathway counts are invented teaching models. They are not measurements from real investigations.

Exam tips: Name the ligand, receptor location, intracellular relay and response in order. Distinguish binding from activation and a final-to-initial ratio from a percentage increase. An extracellular hormone does not need to enter a cell to change its behaviour.

AHL: Receptors connect selective binding to a cellular response

A **receptor** is a protein that recognizes a compatible signalling chemical, called a ligand. Binding depends on the shape and chemical properties of the binding site, including suitable interactions between the ligand and amino-acid side chains. It is more selective than a cell responding equally to every molecule nearby. Related ligands can sometimes bind the same receptor, however; specificity does not mean one receptor can recognize only one molecule under all circumstances.

Ligand binding can change receptor conformation and initiate **signal transduction**: a sequence that connects reception with changes inside the cell. The final response might involve ion movement, enzyme activity, vesicle traffic or transcription. A molecule that occupies a binding site need not activate it. An antagonist can bind and reduce activation by a normal ligand, while an agonist activates the receptor.

Two cells exposed to the same ligand can differ because they express different receptors or different downstream proteins. Even cells with the same receptor can respond differently if their intracellular pathways differ. A lack of response is therefore not proof that the signal never reached the cell. Check receptor expression, signal availability, cell viability and downstream function before assigning a cause.

In an invented assay, untreated response is 12 units, ligand-treated response 60, and ligand plus a proposed receptor blocker 24. The ligand gives a fivefold final value, $60/12 = 5$, but a $48/12 \times 100 = 400\%$ increase. The blocked raw value is 60% below

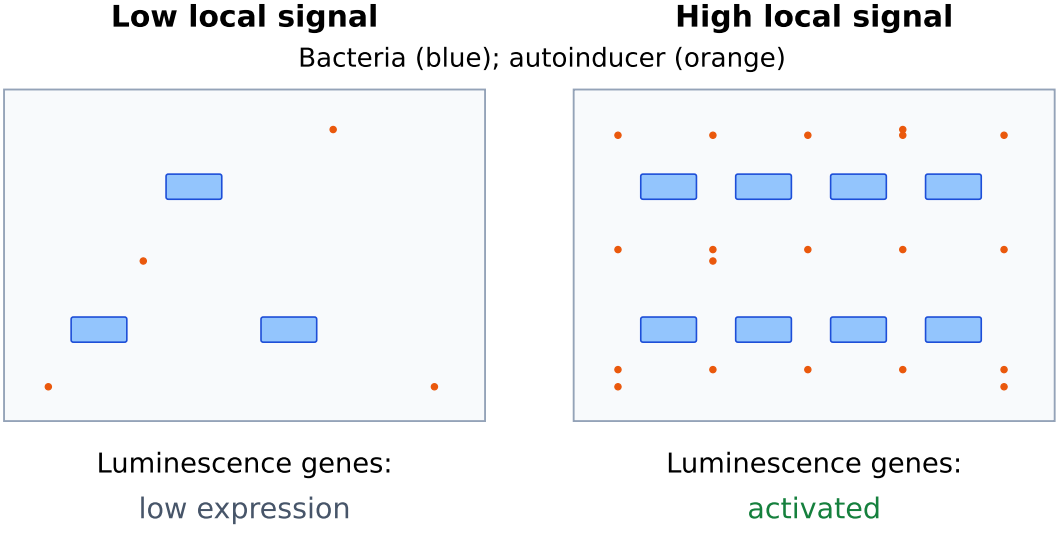
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60. After subtracting the common baseline, responses are 48 and 12; inhibition of the ligand-induced increase is $36/48 \times 100 = 75\%$. Name which comparison is being made. Neither calculation alone proves where the blocker acts.

AHL: Bacteria use chemical accumulation in quorum sensing

Bacteria can release small signal molecules into their surroundings and detect their local concentration. In **quorum sensing**, signal accumulation changes gene expression when it reaches a suitable range. Increasing cell density can increase signal supply per local volume, but diffusion, fluid flow, degradation and confinement also matter. The bacterium does not directly count every other cell or consciously decide to cooperate.

The marine bacterium *Vibrio fischeri* provides a bioluminescence example. Its LuxI enzyme makes an autoinducer. As the signal accumulates, binding to the regulator LuxR supports activation of luminescence genes. These encode machinery for light production; the signal itself is not a packet of light. Regulation can also increase autoinducer production, reinforcing the response. The detailed network has further controls, so the simplified LuxI–LuxR model should not be treated as its only input.



Local signal concentration depends on production and loss. Receptor-mediated regulation changes expression; cells do not measure population size with a counting organ.

For example, an invented culture emits 2 relative light units at a low cell density and 50 at a higher density. This is a 25-fold bulk light value, but it does not establish 25 times more light per cell. If cell number increases tenfold, light per cell rises by $25/10 = 2.5$ times. Normalize the measured quantity appropriately before comparing expression or output.

To test a diffusible-signal explanation, compare suitable low-density cultures receiving

fresh medium or cell-free conditioned medium under controlled nutrients, pH and growth conditions. A response to conditioned medium supports a soluble influence but not its chemical identity by itself. Complementary signal-synthesis or receptor evidence strengthens the mechanism. Original work comparing autoinducer analogues also shows that receptor binding and activation can differ (Schaefer and colleagues, 1996).

AHL: Signal categories describe roles as well as chemistry

Hormones are signals secreted by endocrine cells and transported to distant targets, commonly through blood. A neurotransmitter is released by a neuron and acts on compatible receptors at a target; in a chemical synapse it diffuses across a short gap. Cytokines are small signalling proteins used in communication such as immune-cell coordination. They often act locally, although some have wider effects. A calcium-ion signal commonly acts inside a cell: a rise in cytosolic calcium can change the activity of calcium-binding proteins.

These categories are not four mutually exclusive chemical families. Hormones include amines such as epinephrine, protein hormones such as insulin, and steroids such as oestradiol, progesterone and testosterone. Neurotransmitters include amino acids such as glutamate, amines such as dopamine, and peptides. Acetylcholine belongs to another chemical class. Nitric oxide, NO, is a gaseous neural signal; it is not nitrous oxide, N_2O.

Chemical diversity permits different solubilities, lifetimes, delivery routes and receptor mechanisms. Peptide hormones generally do not cross the lipid bilayer freely and act through membrane receptors. Steroids can enter cells and act through intracellular receptors. Avoid turning a useful pattern into an absolute rule: thyroid hormones are amino-acid-derived hormones with intracellular receptors, while epinephrine is an amine acting through membrane receptors.

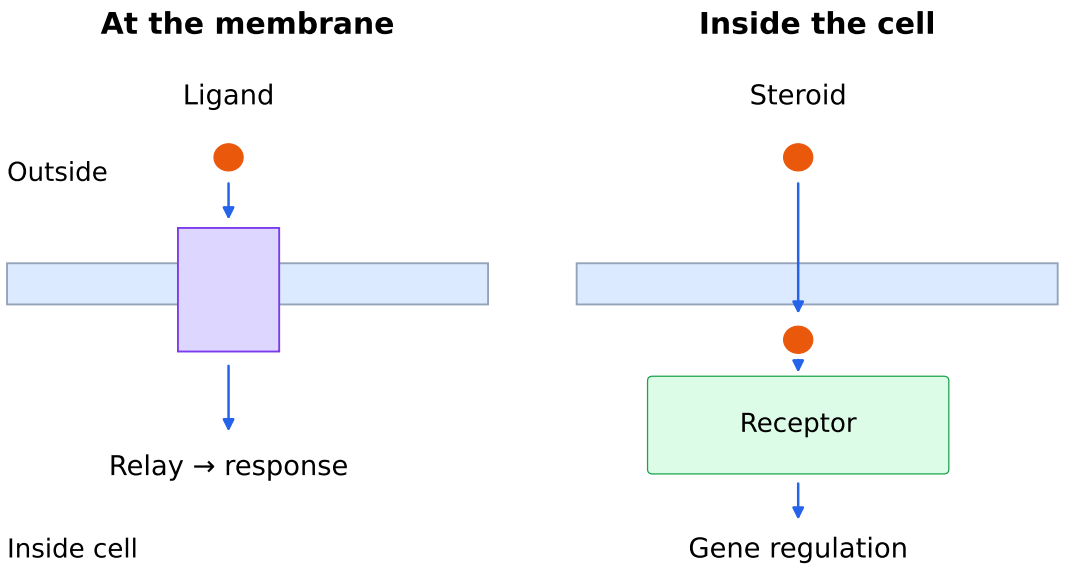
A hormone circulating widely still acts selectively where appropriate receptors and pathways are present. A synaptic signal is more localized by the release site, short diffusion distance and removal mechanisms. Local action does not imply that the chemical can never have another role elsewhere, and distant transport does not mean every cell responds.

AHL: Receptor location fits both protein structure and ligand access

A transmembrane receptor spans the plasma membrane. Hydrophobic amino-acid side chains are exposed to the lipid interior in the spanning region; regions facing extracellular fluid or cytosol have surfaces compatible with those aqueous environments. The extracellular binding site can recognize a ligand that remains outside, while a cytoplasmic region or associated protein initiates the response inside. The protein transmits a change; it does not necessarily carry the ligand through the membrane.

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Intracellular receptors are in the cytoplasm or nucleus. A steroid can cross the plasma membrane and bind a suitable receptor inside. The receptor's ligand-binding pocket accommodates the hormone, while other regions interact with cellular partners and DNA. A soluble protein can contain a hydrophobic binding pocket without its whole surface being hydrophobic and insoluble.



Membrane location determines which side receives the ligand. Both routes still require a compatible receptor and appropriate downstream machinery.

The principal routes below illustrate different mechanisms. Do not merge them into one universal chain: a ligand-gated ion channel is not automatically a G-protein-coupled receptor, and the insulin receptor is not a glucose transporter.

AHL: An acetylcholine receptor can directly gate an ion channel

The nicotinic acetylcholine receptor is a ligand-gated cation channel. Acetylcholine binding increases the probability that the channel opens. Near the usual resting potential, sodium entry contributes to a net inward positive current, making the inside less negative: depolarization. Potassium can also pass through this channel, so describing it as exclusively a sodium channel is too narrow.

Whether depolarization triggers further electrical activity depends on its size and the target membrane's other channels and threshold. Receptor opening does not guarantee an action potential from every binding event. Removing acetylcholine and channel closure help end the signal. Ion movement is down electrochemical gradients; the channel is not using one ATP to pump in every sodium ion, although energy-dependent transport maintains gradients over time.

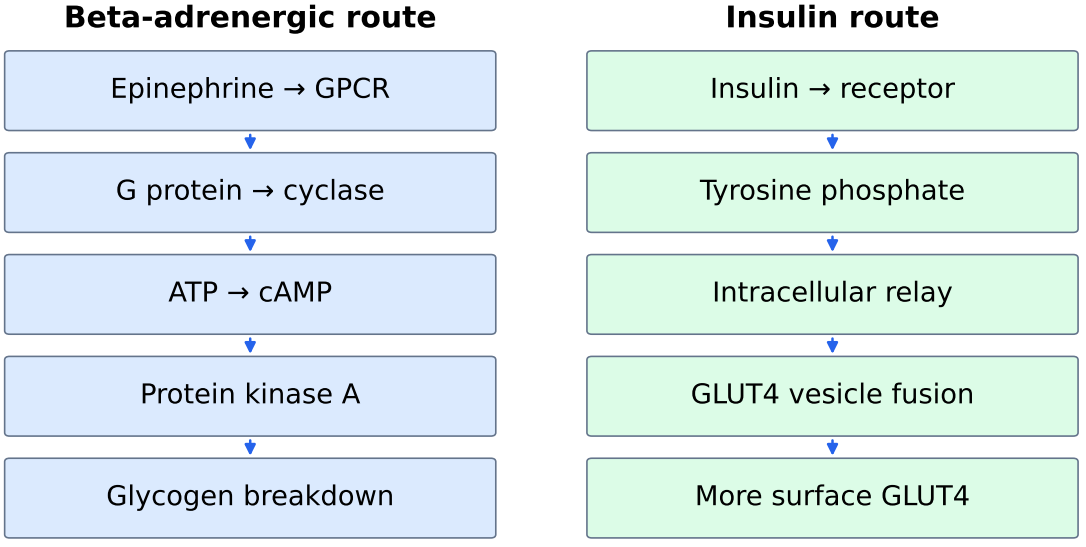
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Not all acetylcholine receptors are nicotinic channels. Muscarinic receptors act through G proteins. This is a concrete reason why a ligand's name alone does not determine the target's intracellular response.

AHL: Epinephrine can activate a G protein and a second messenger

A G-protein-coupled receptor, or GPCR, changes shape after ligand binding and activates an associated G protein on the cytoplasmic side. In a common model, the G protein exchanges bound GDP for GTP. The activated G protein regulates an effector protein. Later GTP hydrolysis helps switch the G protein off. Different GPCRs can stimulate or inhibit different effectors; not every G protein raises cyclic AMP.

In the beta-adrenergic pathway, epinephrine binding can activate a stimulatory G protein, which activates adenylyl cyclase. This enzyme converts ATP into cyclic AMP, cAMP. Epinephrine is the extracellular first messenger; cAMP is an intracellular second messenger. cAMP helps activate protein kinase A, which phosphorylates target proteins. In an appropriate target such as a liver cell, the cascade can promote glycogen breakdown and rapid fuel availability. Epinephrine does not enter the cell to become cAMP, and cAMP is not simply a second name for ATP.



The left route illustrates a glycogen-storing target; cyclase means adenylyl cyclase. The right route illustrates GLUT4 insertion in an insulin-responsive muscle/adipose cell.

Amplification occurs when one activated component affects many molecules at the next stage. In an invented ideal model, two activated receptors each activate four G proteins. Each activates one cyclase producing 50 cAMP molecules per second for 3 seconds. The calculated output is $2 \times 4 \times 50 \times 3 = 1200$ cAMP molecules. These

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are supplied model assumptions, not a fixed physiological ratio. ATP supplies the material for cAMP; information is amplified without creating matter or energy from nothing.

Phosphodiesterases break down cAMP, phosphatases can remove protein phosphates, and receptor/G-protein regulation helps terminate the response. Without signal removal or inactivation, a short ligand pulse could cause inappropriate prolonged activity. Reversible steps support controlled responses rather than unlimited irreversible amplification.

Epinephrine and adrenaline name the same hormone. The two names arose from Greek and Latin roots referring to the adrenal glands near the kidneys. International naming conventions usually help researchers identify the same substance; this familiar pair persists across different naming traditions. Neither term means a separate receptor pathway.

AHL: Insulin activates a receptor with tyrosine kinase activity

Insulin is a protein hormone that binds outside the cell to the insulin receptor. The receptor is an existing multi-subunit membrane complex. Binding changes its activity and promotes phosphorylation of tyrosine residues on its intracellular portions. Tyrosine kinase activity transfers phosphate from ATP to suitable protein sites, initiating downstream signalling.

In responsive skeletal-muscle and adipose cells, the pathway promotes movement of vesicles containing GLUT4 glucose transporters to the plasma membrane. Vesicle fusion increases the number of surface transporters, increasing the capacity for facilitated glucose entry when the glucose gradient supports it. The transporter carries glucose; the receptor receives insulin. Insulin is not itself an ion channel or the pore through which glucose moves.

Suppose an invented cell has 100 GLUT4 molecules in total, initially 20 at its surface and later 50. Surface fraction rises from 20% to 50%, an increase of 30 percentage points. Surface number is 2.5 times the initial value, a 150% increase. The observation can be explained by redistribution without synthesizing 30 new transporters. It does not establish that uptake rises exactly 2.5-fold: substrate availability and other constraints also matter.

Do not extend this particular mechanism to every body cell. Liver glucose handling has important insulin-regulated effects, but it is not explained by inserting GLUT4 into every liver-cell membrane. A response depends on the cell's transporter repertoire and intracellular machinery.

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AHL: Steroid-receptor complexes can change gene expression

Oestradiol, progesterone and testosterone are steroid hormones. After entering an appropriate target cell, each can bind its compatible intracellular receptor. Activation permits receptor interactions with specific DNA regulatory sequences and other transcriptional machinery. This can increase transcription of particular genes, producing mRNA and then proteins that alter cell function. Other genes can be repressed through context-dependent regulation. A hormone does not bind randomly to all DNA and turn on every gene.

The receptor provides specificity at ligand recognition and DNA regulation. Different receptor distribution, regulatory sequences and associated proteins help explain tissue-specific effects. A transcription-mediated response often develops more slowly than direct opening of an ion channel because RNA/protein production takes time. This is a mechanistic comparison, not a rule that every steroid effect is slow or every membrane-receptor effect is immediate.

Oestradiol affects hypothalamic regulation of gonadotropin-releasing hormone, GnRH, and therefore reproductive signalling. During much of the cycle it contributes to negative feedback; sustained high pre-ovulatory oestradiol supports positive feedback in the hypothalamic–pituitary system leading to the ovulatory surge. The sign depends on concentration, timing and physiological context. This involves a neuronal network, including oestradiol-sensitive inputs controlling GnRH neurons, rather than requiring every effect to be a direct action on a GnRH neuron's DNA (research on oestradiol-sensitive inputs).

Progesterone acts on compatible endometrial cells to support the secretory, maintained uterine lining after ovulation. Intracellular receptor-mediated changes in expression help alter their function. Progesterone does not simply duplicate oestradiol's proliferative effects or bind any receptor in every tissue. Testosterone provides another steroid-receptor example; target response depends on receptor and tissue context rather than the hormone changing the genetic code.

AHL: Feedback changes the activity of an earlier part of a system

In negative feedback, an effect opposes the initiating change. A rise in blood glucose can stimulate insulin secretion; increased uptake/storage and reduced glucose output help bring glucose down. Reduced stimulation then reduces further insulin secretion. The regulated quantity is moved toward a suitable range; the response need not be an exact return at every instant.

In positive feedback, an effect reinforces an initiating change. During labour, cervical stretch can stimulate oxytocin release, which increases uterine contractions and can increase cervical stretch. A later event such as delivery breaks the reinforcing loop. Positive does not mean beneficial, and negative does not mean harmful: the words describe the direction of regulation.

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Feedback differs from simple forward amplification. One activated receptor generating many second messengers is amplification even without any product returning to regulate an earlier step. To identify feedback, name the returning influence and explain whether it reinforces or opposes the original change.

Practice

All data are invented. Attempt each problem before checking the answers.

1. Explain how a ligand can change a cell without entering it, and give two reasons why another cell exposed to the same ligand might not respond.
2. A compatible molecule binds a receptor but does not activate it and reduces the normal ligand's effect. Explain why binding and activation must be distinguished.
3. Untreated response is 10 units, signal-treated response 50, and signal plus blocker response 20. Calculate the signal's fold value and percentage increase. Calculate suppression using both raw responses and baseline-subtracted responses.
4. Explain quorum sensing in *Vibrio fischeri*, linking local signal accumulation, a regulator and light-producing machinery. Explain why fluid flow can affect the result at unchanged cell density.
5. Bulk light increases 30-fold while cell number increases sixfold. Calculate the light-per-cell ratio and explain why the bulk ratio alone is insufficient.
6. Distinguish a hormone, a synaptic neurotransmitter, a cytokine and an intracellular calcium signal. Give amine, protein and steroid hormone examples.
7. Compare transmembrane and intracellular receptor locations, linking protein structure and ligand access. Explain why "all amines use membrane receptors" is unreliable.
8. Explain how nicotinic acetylcholine receptor opening can cause depolarization. Explain why opening does not guarantee an action potential and why it is not the same mechanism as every acetylcholine receptor.
9. Trace the beta-adrenergic epinephrine pathway from binding to kinase activation. Identify the first and second messengers and one termination mechanism.
10. In a supplied ideal cascade, four active receptors each activate three cyclases. Each makes 20 cAMP molecules per second for 2 seconds. Calculate total output and explain why amplification does not create matter from nothing.
11. An insulin-responsive cell has 120 GLUT4 molecules, initially 24 at the surface and later 60. Calculate the surface percentages, percentage-point change and percentage increase in surface number. Explain the receptor-to-transporter mechanism.
12. Explain a steroid receptor's route to altered protein production. Describe oestradiol's context-dependent effect on hypothalamic GnRH control and progesterone's effect on endometrial cells.

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13. Contrast negative feedback in glucose regulation with positive feedback in labour. Explain why a forward kinase cascade is not automatically a feedback loop. Suggest two controls before concluding that a proposed receptor blocker proves receptor involvement.

Worked answers

1. Binding to a membrane receptor can change its conformation and initiate intracellular relays without transporting the ligand through the membrane. Another cell may lack the receptor or appropriate downstream proteins; signal availability and viability also need checking.
2. Occupancy need not produce an activating conformational change. Such a molecule can act as an antagonist, reducing normal ligand activation while still binding; a binding assay and a response assay measure different things.
3. $50/10 = 5$-fold; $(50 - 10)/10 \times 100 = 400\%$ increase. Raw suppression is $(50 - 20)/50 \times 100 = 60\%$. Baseline-subtracted effects are 40 and 10, so suppression is $(40 - 10)/40 \times 100 = 75\%$. This assumes a comparable baseline and a response scale suitable for subtraction.
4. Bacteria release an autoinducer; sufficient local accumulation supports LuxR-mediated activation of luminescence genes. Their protein products enable light production. Flow can remove signal, lowering concentration without changing cell density. A density association alone does not prove the whole pathway.
5. Light per cell rises by $30/6 = 5$ times. Bulk output combines changes in cell number with changes in output per cell, so it cannot distinguish them alone.
6. Hormones commonly travel through blood to distant compatible targets; synaptic neurotransmitters cross a short gap; cytokines are signalling proteins often coordinating local immune responses; cytosolic calcium rises can relay signals inside a cell. Epinephrine is an amine, insulin a protein and progesterone a steroid. Roles and chemical classes are different classifications.
7. A spanning receptor has hydrophobic membrane-facing regions and aqueous-facing extracellular/cytoplasmic regions. It can receive a ligand outside and signal inside. An intracellular receptor binds a ligand that gains access to cytoplasm/nucleus, such as a steroid. Thyroid hormones show that an amino-acid-derived hormone can act through intracellular receptors.
8. Acetylcholine binding opens a cation channel; near rest, net positive charge enters and the membrane depolarizes. The response may be below threshold. Potassium as well as sodium can pass; ion flow is down electrochemical gradients. Muscarinic acetylcholine receptors instead use G proteins.
9. Epinephrine → beta-adrenergic GPCR → stimulatory G protein → adenylyl cyclase → cAMP → protein kinase A. Epinephrine is the extracellular first messenger; cAMP is the intracellular second messenger formed from ATP. GTP hydrolysis, cAMP breakdown or dephosphorylation can help end the response.

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10. $4 \times 3 \times 20 \times 2 = 480$ molecules. The assumed cyclase rate and duration determine this model output; real responses vary. ATP provides substrate for cAMP production, so a larger number of signalling molecules does not appear without material inputs.
11. $24/120 = 20\%$ and $60/120 = 50\%$: 30 percentage points. Surface number increases by $36/24 \times 100 = 150\%$ (2.5-fold final number). Insulin binding activates receptor tyrosine phosphorylation and downstream signalling; GLUT4-containing vesicles fuse with the membrane. Redistribution can occur without changing total transporter number; uptake need not rise proportionally.
12. Hormone enters → compatible intracellular receptor activation → interaction with DNA regulatory regions → altered transcription → mRNA/protein production → altered function. Oestradiol can inhibit or, in the sustained high pre-ovulatory context, stimulate the hypothalamic–pituitary pathway controlling GnRH and the ovulatory surge. Progesterone supports secretory maintenance of the endometrium. Receptor distribution and cellular context matter.
13. Insulin-linked effects oppose raised glucose, reducing the original stimulus. In labour, oxytocin-supported contractions can increase stretch and further oxytocin stimulation until delivery interrupts the loop. A cascade is feedback only if an influence returns to regulate an earlier stage. Include a vehicle/untreated control and a cell-viability or independent-response control; assess specificity and replication because a blocker might also act downstream or damage cells.

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Neural signalling

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

Pulling a hand away from a hot surface requires messages along cells and across junctions. Ion movements produce electrical signals; neurotransmitters communicate between cells. Follow both parts of the pathway.

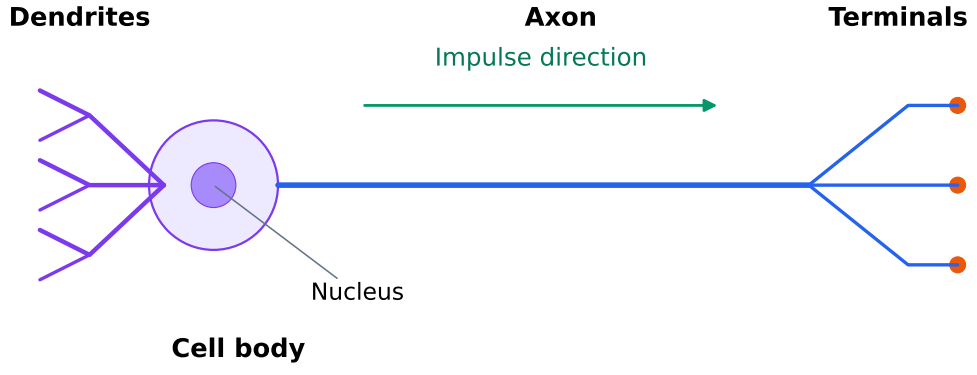
This handout covers full C2.2 common content: neurons, resting potentials, impulses, conduction speed, correlation skills and chemical synapses. Sections marked **AHL** are for Higher Level only.

- **AHL scope:** voltage-gated channels, local currents, voltage traces, saltatory conduction, external chemicals, inhibition, summation, pain and emergent consciousness.

Prerequisites: selective membrane permeability, diffusion, ion gradients and ATP-dependent transport. All numerical datasets and traces below are invented teaching examples, not experimental recordings. Use supplied data or simulations rather than electrical stimulation of people.

Neurons connect specialized receiving and conducting regions

A neuron has a plasma membrane, cytoplasm and nucleus. The cell body contains the nucleus and machinery for maintaining cell components. Usually several short, branching dendrites receive inputs, while one axon extends toward terminal branches. Length and arrangement vary; the diagram models a typical organization rather than every neuron's shape.



Receiving regions influence impulse initiation; impulses travel along the axon toward terminals. These processes are continuous with the cell body, not separate cells joined end to end.

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A long axon permits rapid communication between distant regions without releasing a hormone throughout the circulation. Branching can connect one neuron to several targets, while many inputs can converge on another neuron. A nerve is a bundle of nerve fibres with supporting tissues; it is not one unusually large neuron. Neurons depend on supporting cells as well as their own metabolism.

The membrane and its channels allow the electrical behaviour. A nerve impulse involves ion movement in aqueous solutions and across membranes. It is not an electron current flowing along a metal wire. The same individual sodium ion does not travel the whole axon length to carry one message.

Pumping and selective permeability maintain a resting potential

Membrane potential is the electrical potential difference between the cell interior and the surrounding fluid. A resting neuron commonly has a negative interior relative to the outside; −70 mV is a representative teaching value, not a universal fixed voltage. Saying "negative" needs this reference: it does not mean both fluids contain only negative particles.

Sodium–potassium pumps use ATP to move sodium ions out and potassium ions in, against their concentration gradients. In the standard model, one cycle exports three sodium ions and imports two potassium ions. Sodium becomes more concentrated outside, potassium inside. The unequal charge transfer contributes directly to voltage, while the maintained gradients support much of the voltage through selective ion movement.

At rest, potassium leak channels make the membrane relatively permeable to potassium. Potassium tends to diffuse outward down its concentration gradient, leaving a small charge separation. The increasingly negative interior opposes further loss of positive charge. Sodium permeability is lower at rest, and large intracellular negative ions do not freely leave. Resting voltage therefore depends on gradients and relative permeabilities, not just the pump moving "more positive ions out".

Feature	Contribution and limit
ATP-dependent pump	Maintains sodium and potassium gradients over time; three sodium out and two potassium in per standard cycle
Potassium leak channels	Permit passive movement; electrical and concentration effects oppose each other as voltage develops
Selective membrane	Prevents immediate unrestricted mixing of ions
Charge separation	Involves a small fraction of ions near the membrane; the bulk fluids remain close to electrically neutral

In an invented pump count, eight cycles export 24 sodium ions, import 16 potas-

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sium ions and use eight ATP molecules. The net outward transfer is eight positive charges. Do not interpret this bookkeeping as a calculation of −70 mV: membrane area, capacitance, ion concentrations and permeabilities would also be needed.

Rest means no impulse is occurring at that location, not that all transport has stopped. ATP supply and membrane integrity remain important. Conversely, switching off a pump does not instantly erase every gradient; the time course depends on continued ion movements and the cell's reserves.

An action potential propagates without fading along a healthy axon

An **action potential** is a brief, self-propagating change in membrane potential. A sufficiently strong local depolarization brings an excitable region to threshold, initiating the rapid change. Successive regions of membrane generate their own action potentials, so the impulse can cover a long axon without simply shrinking like a passive voltage change.

For a given healthy fibre under stable conditions, each action potential is approximately all-or-none: a subthreshold input does not produce a smaller travelling action potential, while a threshold-crossing input triggers a full one. This does not mean every possible neuron has exactly the same voltage trace. Temperature, ion gradients, channel types and physiological state affect the waveform.

A stronger sustained stimulus can increase impulse frequency and recruit additional sensory neurons. It need not make each action potential taller. Compare recordings from the same fibre and distinguish the number of active fibres from the frequency within one fibre. A larger compound signal from a nerve can reflect recruitment of more fibres rather than a larger individual action potential.

For a counted time window, frequency is count divided by elapsed time. Thirty impulses in 0.50 s give $f = 30/0.50 = 60$ Hz. Twenty-one in 0.30 s give 70 Hz. These numbers describe impulses per second, not metres travelled per second. Count only events in the stated window; do not divide the number of plotted peaks by the interval between the first and last peak without considering the sampling convention.

Conduction speed depends on fibre structure and conditions

Conduction speed is distance along a fibre divided by propagation time. An invented impulse covers 0.80 m in 0.016 s, giving $v = 0.80/0.016 = 50\ \mathrm{m\,s^{-1}}$. A whole reflex delay includes receptor processes, synaptic delays and muscle activation as well as axon conduction, so it cannot usually be inserted into this equation to isolate one axon's speed.

Larger axon diameter lowers internal electrical resistance and can allow faster propagation. Giant unmyelinated squid axons illustrate the benefit of diameter compared with smaller unmyelinated fibres under comparable conditions. A myelin sheath, pro-

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duced by supporting cells, electrically insulates portions of an axon and can greatly increase speed without requiring a giant diameter. Small myelinated fibres can therefore conduct faster than some larger unmyelinated ones.

Compare like with like: species, temperature, diameter and myelination can all differ. There is no sound universal rule that every larger animal must have slower impulses, or that body size alone determines conduction speed. A dataset covering one sampled group may show a pattern that does not generalize to all animals.

Correlation describes a dataset rather than proving a mechanism

A positive correlation means larger values of one variable tend to accompany larger values of another; a negative correlation means larger values tend to accompany smaller values. Pearson's correlation coefficient r describes the direction and strength of a linear association, from -1 to $+1$. A value near zero means little linear association, which does not rule out a curved relationship.

Consider these invented measurements from four comparable unmyelinated fibres at one temperature:

Diameter / micrometres	Conduction speed / metres per second
10	1
20	2
30	2
40	5

The diameter mean is 25 and the speed mean 2.5. In the Pearson calculation, sum each product of paired deviations from the means. Here $\sum(x-\bar{x})(y-\bar{y}) = 60$, $\sum(x-\bar{x})^2 = 500$ and $\sum(y-\bar{y})^2 = 9$. Thus:

$$r = \frac{60}{\sqrt{500 \times 9}} \approx 0.894.$$

This is a strong positive linear association in the four supplied pairs. In a simple linear regression with an intercept, $R^2 = r^2 = 0.80$: 80% of the observed variation in speed is accounted for by the fitted linear relationship with diameter in this dataset. It is not a claim that diameter causes 80% of every neuron's speed, nor that a new animal's speed can be predicted exactly.

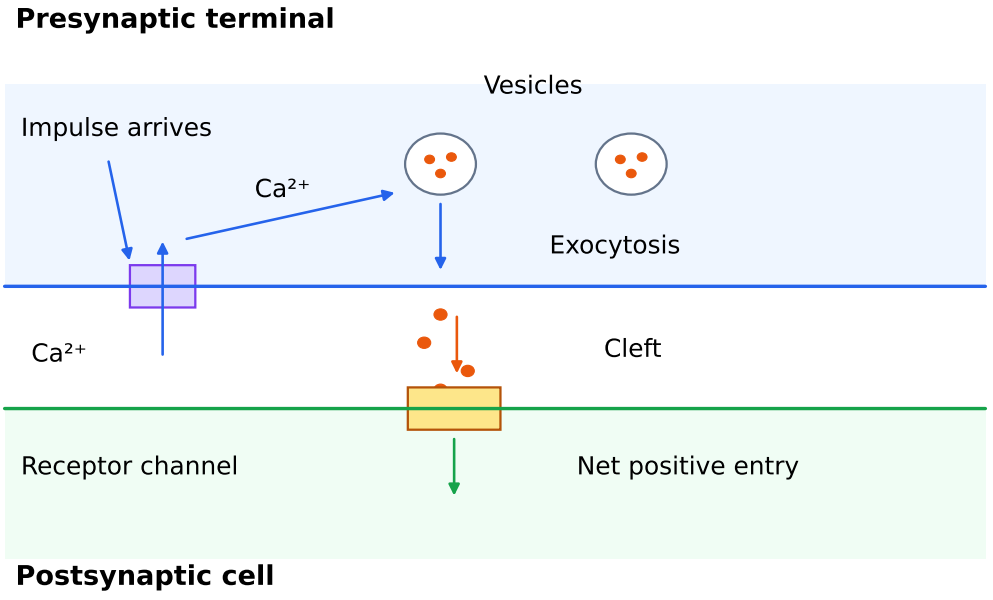
Small samples, measurement uncertainty, confounding and limited ranges restrict inference. Plot the pairs to inspect outliers and possible curvature, standardize conditions and use independent fibres. Repeated timings on one fibre estimate timing variation, not variation among four independent fibres. A negative $r = -0.70$ gives $R^2 = 0.49$; squaring removes the direction, so report the sign of r as well as R^2.

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Chemical synapses convert an arriving impulse into transmitter release

A **synapse** is a junction through which a neuron communicates with another neuron or an effector such as a muscle cell. This topic concerns chemical synapses. The presynaptic terminal and postsynaptic membrane are separated by a narrow fluid-filled synaptic cleft. Vesicles in the terminal contain neurotransmitter molecules.

When an action potential depolarizes the presynaptic membrane, voltage-gated calcium channels open. Calcium ions enter down their electrochemical gradient. Increased cytosolic calcium acts as an internal signal that promotes vesicle fusion with the presynaptic membrane. Exocytosis releases neurotransmitter into the cleft. The arriving electrical event therefore triggers a chemical release step; sodium does not simply jump across and become a neurotransmitter.



The model separates calcium entry into the terminal from transmitter binding at the target membrane. The two channel types have different triggers and locations.

Transmitter diffuses across the cleft and binds compatible transmembrane receptors on the postsynaptic cell. A typical synapse transmits in one direction because release vesicles and their machinery are presynaptic, while receptors for that signal are postsynaptic. This structural asymmetry explains direction better than saying the transmitter "knows which way to move". Diffusion itself is random; the release site and receptors organize the communication.

A chemical synapse introduces a delay because release, diffusion and receptor activation take time. Communication is selective: the target needs the appropriate receptors. One neuron may receive inputs from many presynaptic cells rather than passively copying one incoming impulse into one outgoing impulse.

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Acetylcholine can generate an excitatory postsynaptic potential

Acetylcholine is used at many synapses, including neuromuscular junctions. At a nicotinic receptor, binding opens a ligand-gated cation channel. Near resting voltage, the net movement of positive charge is inward, with sodium entry important. The postsynaptic membrane becomes less negative: an excitatory postsynaptic potential, or EPSP.

An EPSP is a local graded voltage change. It is not automatically a new action potential. If the resulting depolarization brings an excitable region to threshold, an action potential can be initiated in the target cell. At a neuromuscular junction the target is muscle membrane, not another neuron; its local depolarization is called an end-plate potential. Potassium can also pass through nicotinic channels; "a sodium-only channel at every acetylcholine synapse" is an oversimplification.

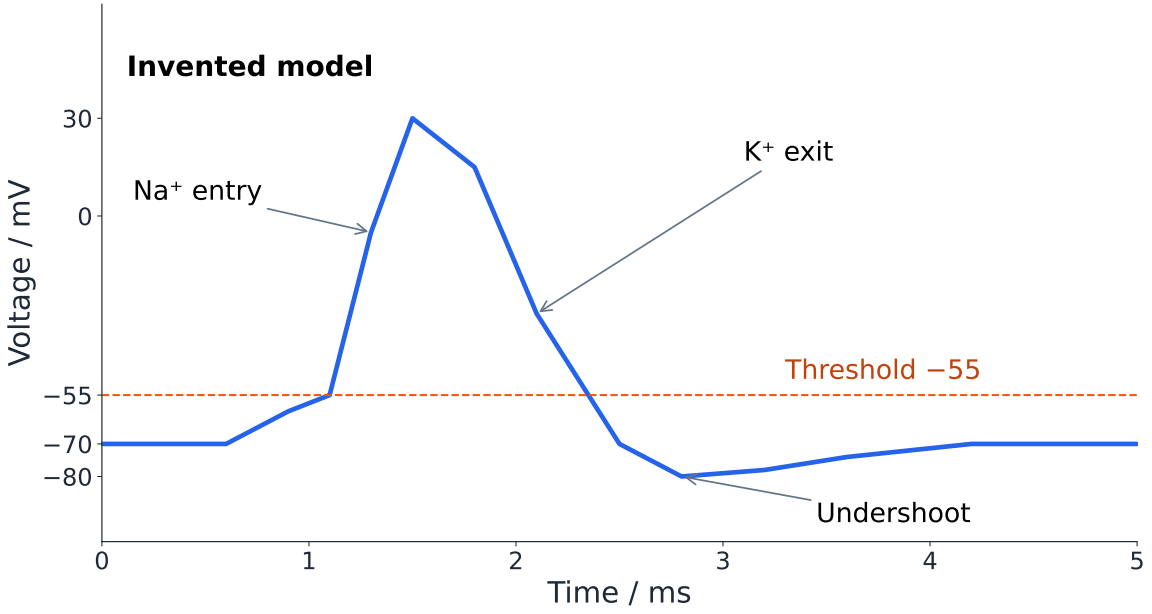
Acetylcholinesterase breaks acetylcholine into choline and acetate, helping end receptor stimulation. Choline can be taken back into the terminal for reuse. Distinguish this from claiming that all released acetylcholine is taken back intact. Other transmitters can have different removal mechanisms, including reuptake of the transmitter itself. Removing a signal allows a later message to be distinguished from a continuously present one.

Exam tips: Trace presynaptic depolarization → calcium entry → vesicle fusion → transmitter diffusion → receptor binding → postsynaptic voltage change. Keep voltage-gated calcium channels separate from ligand-gated target channels. State why a postsynaptic response may remain below threshold.

AHL: Voltage-gated channels explain the phases of an action potential

In the model trace, resting potential is −70 mV, threshold −55 mV, peak +30 mV and the temporary undershoot −80 mV. Depolarization initially makes the interior less negative. At threshold, rapid opening of voltage-gated sodium channels increases sodium entry, causing further depolarization and further channel opening: positive feedback. The inside can become positive relative to the outside.

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The voltage values illustrate a model, not universal measurements. During the rising phase sodium entry dominates; later sodium-channel inactivation and increased potassium exit permit repolarization.

Sodium channels then inactivate, reducing sodium entry. Delayed opening of voltage-gated potassium channels allows greater potassium exit, making the interior negative again: repolarization. Potassium conductance can remain elevated briefly, producing hyperpolarization below resting voltage. Channels recover toward their resting states and resting conductances again dominate.

The sodium–potassium pump maintains the underlying concentration gradients over time. It does not produce the rapid falling phase by suddenly pumping all sodium back out after each spike. Each action potential transfers only a small fraction of the available ions; channel opening changes voltage quickly without emptying the entire sodium or potassium pool.

During the absolute refractory period, many sodium channels are inactivated, preventing a second full action potential in that membrane region. This begins during the spike, not only after its peak. During subsequent relative refractoriness, recovery and elevated potassium conductance can mean a stronger stimulus is needed. Refractoriness helps limit maximum firing frequency and supports normal forward propagation.

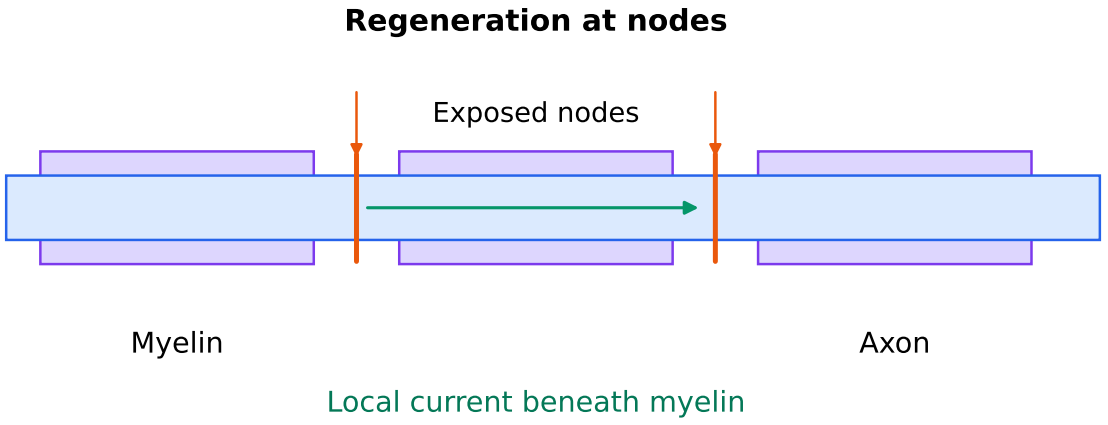
AHL: Local currents regenerate impulses and myelin changes their spread

Positive charge entering one active region creates local currents in nearby cytoplasm and extracellular fluid. These currents depolarize the adjacent membrane. If that region reaches threshold, its voltage-gated sodium channels open and a new action potential is generated. The signal is regenerated by local membrane events; it is not

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one fixed group of sodium ions diffusing from the cell body to the terminal.

Membrane just activated is refractory, favouring propagation into excitable membrane ahead. Under normal conditions initiation at a neuron's trigger region and its connections establish the functional route. Artificial stimulation in the middle of an axon can produce propagation both ways, so "an axon is physically incapable of carrying an impulse backward" is too absolute.



Myelin limits current loss across internodes. Local current spreads beneath it; action potentials are regenerated at exposed nodes of Ranvier.

Myelin increases insulation and reduces the membrane charging needed along internodes. Voltage-gated channels are concentrated at nodes of Ranvier, where ion exchange regenerates the impulse; ion pumps help maintain gradients. This node-to-node regeneration is saltatory conduction. "Jumping" describes the pattern of regeneration, not sodium ions flying across the myelin sheath or an empty space. Local current still travels continuously through conducting fluids.

On an oscilloscope trace with 0.50 ms per horizontal division, four divisions correspond to 2.0 ms. A regular peak-to-peak interval of 10 ms gives a frequency of $1/0.010 = 100$ Hz. An interval measures a period; a count in a longer defined window measures an average frequency. Neither calculation alone measures axon conduction speed.

AHL: External chemicals can disrupt different synaptic steps

Neonicotinoid insecticides act at insect nicotinic acetylcholine receptors. Some act as agonists: persistent receptor activation can disrupt normal signalling, including sustained depolarization and loss of normal action-potential firing. Blocking transmission therefore need not mean physically plugging the receptor without activating it. Effects vary with compound, receptor subtype and exposure.

For example, recordings from isolated honeybee brain cells showed that imidacloprid and clothianidin could cause depolarization block and inhibit nicotinic responses

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(Palmer and colleagues, 2013, research abstract). This establishes a cellular mechanism under the studied conditions; it does not by itself quantify population decline or make all pesticide effects identical.

Cocaine can bind dopamine transporters and inhibit dopamine reuptake. Dopamine then remains available longer outside cells, altering stimulation of its receptors. The transporter is a removal mechanism, not the dopamine receptor itself. This differs from acetylcholinesterase breakdown of acetylcholine and from an agonist activating a nicotinic receptor. Cocaine affects other targets too; the dopamine-reuptake example isolates one relevant mechanism (NIDA mechanism explanation).

AHL: Excitation and inhibition are integrated before an all-or-none output

An inhibitory postsynaptic potential, or IPSP, can make the postsynaptic membrane more negative, farther from threshold. Depending on the receptor and ion gradients, opening chloride channels can permit negative charge entry, or opening potassium channels can promote positive charge exit. Some inhibition also reduces the effect of excitation by increasing membrane conductance, without a large hyperpolarization. The simple hyperpolarizing model remains useful when the supplied data show it.

Temporal summation combines inputs close together in time from one source; spatial summation combines inputs from different locations. EPSPs and IPSPs influence the membrane potential at the impulse-initiation region. They are graded inputs, while the decision to generate an action potential is approximately all-or-none. A neuron integrates inputs rather than storing every incoming EPSP indefinitely.

In an explicitly additive teaching model, rest is −70 mV and threshold −55 mV. Two overlapping +8 mV EPSPs would give −54 mV, enough to reach threshold. Adding a −6 mV IPSP gives −60 mV, below threshold. Real voltage changes need not add exactly: their timing, locations, conductances and ion-driving forces matter. State the additive assumption before using arithmetic as a prediction.

AHL: Sensory transduction and interacting networks support perception

Some sensory neurons have free nerve endings in skin. Their membrane channels can respond to high temperature, acidity or chemicals such as capsaicin in chilli peppers. Opening channels permeable to positive ions can depolarize the ending. If excitation reaches threshold, action potentials travel along the sensory neuron and through pathways toward the brain.

The stimulus is converted into an electrical pattern; capsaicin does not have to travel along the axon to the brain. Pain is perceived through brain processing of sensory input and context, rather than being a conscious experience inside the skin ending alone. Detection of potentially damaging stimuli and the experience of pain are related but

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are not identical in every circumstance.

Consciousness is an emergent property associated with interactions among neurons and networks. Individual neurons can respond electrically, yet a single neuron's action potential is not itself a conscious thought. Integration across interacting systems permits properties that are not explained by simply listing isolated cell parts. The detailed neural basis of conscious experience remains an active research question; emergence is not a claim that every mechanism has already been solved.

Practice

All numerical data are invented. Explain mechanisms as well as calculating results.

1. Describe the cell body, dendrites, axon and terminal branches of a typical neuron. Distinguish a neuron from a nerve.
2. Explain how pumping and selective permeability contribute to a negative resting potential. Why is "the cytoplasm contains only negative ions" wrong?
3. A pump completes 12 cycles. Calculate sodium ions exported, potassium ions imported, ATP used and net positive charges moved outward. Explain why this does not determine a voltage by itself.
4. Explain all-or-none propagation. Give two ways a stronger stimulus can produce a larger nervous-system response without increasing each individual impulse's amplitude.
5. A fibre carries 24 impulses in 0.40 s. Calculate frequency and distinguish it from propagation speed.
6. A supplied recording shows an impulse travelling 1.20 m in 0.020 s. Calculate speed. Explain why using the full time from a skin stimulus to a muscle response could underestimate this axon's speed.
7. Compare giant unmyelinated squid axons, smaller unmyelinated fibres and myelinated fibres. Name two conditions to control in a diameter–speed investigation.
8. For the four-pair diameter–speed dataset, use the supplied deviation sums 60, 500 and 9 to calculate r and R^2. Interpret both without claiming causation.
9. Another dataset has $r = -0.70$. Calculate R^2 and interpret the direction, strength and limits of the linear association. Does $r = 0$ prove that two variables have no relationship of any kind?
10. Trace events from an arriving presynaptic action potential to transmitter binding. Explain the usual one-way transmission of a chemical synapse.
11. Explain how acetylcholine can produce an EPSP, why an EPSP may not trigger an action potential, and how acetylcholine signalling is ended.
12. **AHL:** Explain the ion-channel mechanisms at threshold, during depolarization, repolarization and undershoot. Identify the peak-minus-rest voltage change in the model trace. Explain local-current propagation, refractoriness and saltatory conduction without making the pump responsible for the rapid downstroke.

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13. **AHL:** A trace has 0.50 ms per division and regular peaks six divisions apart. Calculate period and frequency. Compare neonicotinoid disruption with cocaine's dopamine-transporter effect, identifying the target and affected signalling step.
14. **AHL:** In the additive model, rest is −70 mV, threshold −55 mV, and two EPSPs contribute +9 mV each. Predict threshold crossing with and without a −7 mV IPSP. Distinguish temporal and spatial summation. Trace a capsaicin response from skin ending to perception and explain why consciousness is described as emergent.

Worked answers

1. The cell body contains cytoplasm, nucleus and maintenance machinery. Usually several branching dendrites receive inputs; one axon conducts impulses toward terminal branches, which communicate with targets. A nerve bundles many nerve fibres with supporting tissue rather than being one cell.
2. ATP-dependent pumps maintain high external sodium and internal potassium. Greater resting potassium permeability allows potassium loss, producing a small charge separation opposed by electrical attraction. Trapped intracellular anions and low sodium permeability also contribute. Both bulk fluids contain positive and negative ions and remain nearly neutral away from the membrane.
3. $12 \times 3 = 36$ sodium out; $12 \times 2 = 24$ potassium in; 12 ATP used; net 12 positive charges outward. Voltage also depends on membrane capacitance/area, concentrations and permeability, so these counts alone do not specify it.
4. Threshold-crossing input initiates a full action potential that is regenerated at successive excitable regions. Subthreshold input does not produce a smaller travelling action potential. Stronger stimulation can increase firing frequency and recruit more neurons; those changes differ from increasing a single impulse's height.
5. $f = N/t = 24/0.40 = 60$ Hz. This is impulses per second. Speed needs distance divided by travel time and has units such as metres per second.
6. $v = d/t = 1.20/0.020 = 60\ \mathrm{m\,s^{-1}}$. Whole-response time includes receptor, synaptic and muscle delays; using that larger time as axon travel time would give too small a speed for the same distance.
7. Larger unmyelinated diameter reduces internal resistance, helping explain fast giant squid axons relative to smaller unmyelinated fibres. Myelin improves conduction without requiring giant diameter, so diameter alone cannot rank mixed fibre types. Control temperature and myelination/species, and use independent fibres with reliable distance/timing measurements.
8. $r = 60/\sqrt{500 \times 9} \approx 0.894$; $R^2 = 0.80$. The sample shows strong positive linear association. With a simple linear fit including an intercept, 80% of sample speed variation is accounted for by the fitted diameter relationship. Four pairs and possible confounders limit inference; the result is not a percentage causal contribution.
9. $R^2 = (-0.70)^2 = 0.49$. The association is negative and reasonably strong as a

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linear pattern, but strength labels depend on context. The corresponding simple linear fit accounts for 49% of sample variation. Zero Pearson correlation can coexist with a curved association; no coefficient alone proves causation.

10. Depolarization opens presynaptic voltage-gated calcium channels → calcium entry → increased cytosolic calcium → vesicle fusion/exocytosis → transmitter diffusion → binding to compatible postsynaptic receptors. Typical release machinery is presynaptic and target receptors postsynaptic, creating structural directionality. Transmitter does not carry an intact action potential across the cleft.
11. Nicotinic receptor binding opens cation channels; net inward positive current near rest depolarizes the target membrane. The graded EPSP may remain below threshold. Acetylcholinesterase hydrolyses acetylcholine; choline can be taken back for reuse. This differs from reuptake of intact transmitter at other synapses.
12. **AHL:** Threshold initiates regenerative sodium-channel opening and sodium entry. Sodium inactivation plus delayed potassium-channel opening and potassium exit permit repolarization; persisting potassium conductance contributes to undershoot. Peak minus rest is $30 - (-70) = 100$ mV. Local currents excite adjacent membrane; recent membrane is refractory. Under myelin, current spreads to nodes where action potentials regenerate. Pumps maintain gradients over time; channel currents cause the rapid phases.
13. **AHL:** Period is $6 \times 0.50 = 3.0$ ms $= 0.0030$ s; $f = 1/T \approx 333$ Hz, about 3.3×10^2 Hz to two significant figures. Neonicotinoids act at insect nicotinic receptors and persistent activation can disrupt normal firing. Cocaine inhibits dopamine-transporter-mediated reuptake, prolonging extracellular dopamine availability. These are different targets and steps, not two versions of acetylcholine breakdown.
14. **AHL:** Without inhibition, $-70 + 9 + 9 = -52$ mV reaches the -55 mV threshold. With inhibition, $-70 + 9 + 9 - 7 = -59$ mV does not. This assumes overlapping, linearly additive effects. Temporal summation combines closely timed inputs from one source; spatial summation combines different input locations. Capsaicin-sensitive positive-ion channels can depolarize a free ending, initiate impulses and engage brain processing where pain is perceived. Consciousness concerns interactions among neuronal networks, not a conscious property demonstrated by one spike.

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Integration of body systems

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A running animal coordinates contraction with gas exchange, circulation and fuel supply. A seedling likewise connects growth in distant parts through chemical signals.

This handout covers full C3.1 common content: organization and integration, nervous/endocrine coordination, reflexes, sleep rhythms, cardiovascular/ventilation feedback and gut control. Sections marked **AHL** are for Higher Level only.

- **AHL scope:** observing tropisms, shoot phototropism, phytohormones, polar auxin transport, cell elongation, auxin–cytokinin interactions and ethylene feedback.

Prerequisites: cell specialization, membrane signalling, neurons, circulation and gas exchange. All numerical datasets below are invented for teaching; they are not measurements from real investigations. Interpret supplied traces and use approved seedling observations rather than provoking pain, altering breathing or administering hormones.

Integration produces functions that isolated parts cannot perform

Integration means coordinating component parts so that they contribute to a combined function. Cells form tissues, tissues contribute to organs, and organs interact within and between body systems. An organ commonly contains several tissue types: a stomach has muscle, epithelium, connective tissue and nervous tissue, not one uniform cell type.

An emergent property depends on interactions among components. A cheetah's effective pursuit of prey combines sensory processing, coordinated skeletal-muscle contraction, supporting bones and joints, gas exchange, circulation, energy metabolism and temperature control. No isolated muscle cell has the whole animal's hunting behaviour. Listing the systems is only a start: explain how an output from one supports another.

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Connection during vigorous activity	Why the connection matters
Lungs → blood → active muscle	Oxygen supports aerobic ATP production; blood also removes carbon dioxide
Gut/stores → circulation → cells	Digestion, absorption and mobilization make respiratory substrates available
Nervous system → muscle groups	Timing and recruitment coordinate force and movement
Circulation → skin and other tissues	Material transport and heat transfer support suitable internal conditions

The blood system carries materials and heat; nerves and hormones provide information that changes target activity. These roles interact. A nervous command cannot replace oxygen supply, while oxygen-rich blood does not specify which opposing muscles should contract at each instant.

Nervous and hormonal signals coordinate different routes to a target

Nervous signalling uses impulses along neurons and neurotransmitter release at synapses. Connections can deliver signals to particular cells rapidly. Endocrine cells secrete hormones into the circulation; blood distributes them, and compatible receptors determine which cells respond. Widespread delivery is not the same as every cell responding.

Nervous effects are often rapid and localized, while hormonal responses can coordinate widespread and longer-lasting changes. These are useful comparisons, not absolutes: some hormones act within seconds, and neural signalling can initiate lasting changes in learning. A hormone's route, receptor mechanism and target context matter more than the slogan "hormones are always slow".

Homeostasis maintains internal conditions within suitable limits despite disturbances. Receptors detect relevant changes, integration centres coordinate information, and effectors change activity. In **negative feedback** , the response opposes the initiating change. A delay can produce temporary departures and oscillations; a control system need not keep a quantity perfectly constant at every instant.

As a familiar hormonal example, raised blood glucose can stimulate insulin-linked uptake/storage and reduced liver glucose output. Low glucose can stimulate glucagon-linked liver glycogen breakdown and glucose production. Both responses oppose the original disturbance, through different actions. Insulin does not insert GLUT4 into every liver cell, and all diabetes is not simply failure to produce insulin.

In an invented concentration trace, a value rises from 5.0 to 8.0 arbitrary units. The increase is $3.0/5.0 \times 100 = 60\%$. A return from 8.0 to 5.0 is a $3.0/8.0 \times 100 = 37.5\%$

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decrease. The denominators differ. A doubling from 2.0 to 4.0 is a 100% increase, not 200%. Such arithmetic describes change; an invented value is not a medical diagnosis.

The brain, spinal cord and connecting neurons integrate information

The brain combines information from several inputs, compares it with prior experience, and coordinates outputs. Learning changes how later information is processed; memory allows information from earlier events to influence present behaviour. These are interacting neural functions rather than a single "memory cell" holding every experience.

Sensory neurons carry input from receptors toward the central nervous system, or CNS. Signals can reach spinal-cord circuits and pathways leading to the cerebral hemispheres. Information processed in the cerebral hemispheres can influence motor-neuron output, through intervening circuits, so skeletal muscles contract in a coordinated way. The brain does not directly pull tendons; motor neurons communicate with muscle cells.

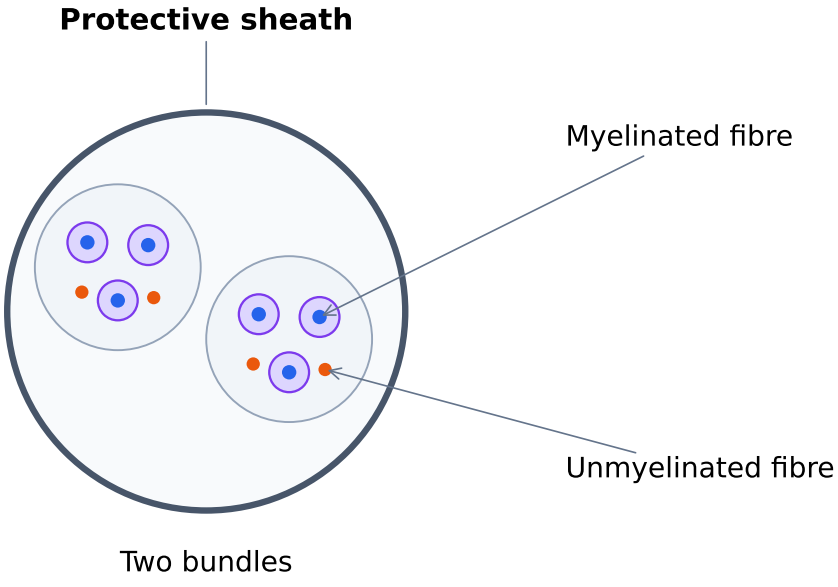
The spinal cord is also an integrating centre. It contains circuits for unconscious responses, rather than serving only as a cable carrying messages to and from the brain. An unconscious process does not require a deliberate decision, although brain activity can still influence it. Conscious awareness of an event and the first protective motor response need not begin at the same time.

The cerebellum contributes to coordination of skeletal-muscle contraction, posture and balance. It helps compare intended movement with sensory information and adjust timing and force. This supports smooth movement; it is not the sole origin of every motor command. Movement depends on interactions among cerebral, cerebellar, spinal and peripheral components.

A nerve bundles fibres while preserving separate cellular pathways

Many peripheral nerves contain both sensory and motor fibres. In a transverse section, individual axons appear as small profiles; some have surrounding myelin, others are unmyelinated. Connective tissues organize fibres into bundles and provide protective sheaths. Supporting cells are present; an unmyelinated fibre is not necessarily an axon floating without any cellular support.

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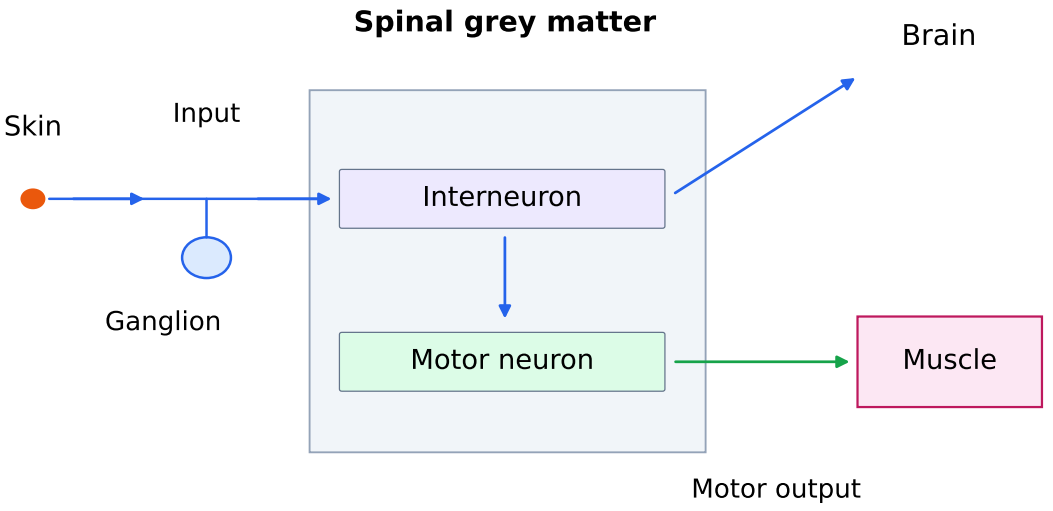
Rings represent myelin around selected axons; small solid profiles represent unmyelinated fibres in this simplified section. A transverse section does not reveal each fibre's sensory or motor direction by shape alone.

A sensory fibre carries impulses toward the CNS; a motor fibre carries them toward an effector. Their presence in one nerve does not make every individual axon switch between those functional roles. A bundle provides common routing and protection while many separate membrane pathways operate in parallel.

A withdrawal reflex links a receptor to skeletal-muscle contraction

Consider a free sensory ending in the hand detecting a potentially damaging hot stimulus. Ion-channel activation can initiate impulses in a sensory neuron. Its central process enters the spinal cord and synapses with an interneuron in grey matter. In this simplified reflex arc, that interneuron communicates with a motor neuron, whose axon carries impulses to a skeletal muscle that flexes the limb away.

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The illustrated arc has one interneuron and two neuronal synapses in the spinal cord. The brain arrow summarizes additional ascending pathways, permitting conscious processing without requiring a conscious decision to initiate withdrawal.

The sensory cell body is in a dorsal-root ganglion outside the spinal cord. The interneuron and motor-neuron cell bodies are in spinal grey matter in this model, and motor output leaves through a ventral root. Do not move the sensory cell body into the skin or make the interneuron run all the way to the muscle.

This is an involuntary response with skeletal muscle as the effector. It shows that skeletal muscle is not used only for voluntary movements. Other reflex circuits differ in their connections and number of interneurons, and withdrawal normally involves coordinated excitation and inhibition of muscle groups. The simple arc teaches the essential route without claiming it is the whole network.

Melatonin links the light–dark cycle to a circadian rhythm

Circadian rhythms are approximately 24-hour patterns generated within an organism and adjusted by environmental cues. In humans, information about light reaching the eyes helps synchronize brain timing pathways. The pineal gland normally secretes more melatonin during the biological night and less during the day; light at night can suppress secretion.

Melatonin helps signal biological night and modulate the sleep–wake pattern. Its daily pattern does not mean it instantly switches every person into unconsciousness. Sleep also depends on other neural processes and prior wakefulness. Light exposure, individual timing and environmental schedules influence how the internal cycle aligns with local day and night.

For a supplied time series, identify the rising phase, peak interval and fall, and compare

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them with darkness and sleep timing. Repeated days help distinguish a daily pattern from a single fluctuation. A phase shift means the timing moved; an amplitude change means the high–low difference changed. They are different descriptions of a rhythm.

Epinephrine and hypothalamus–pituitary control connect endocrine responses

The adrenal medulla secretes epinephrine, also called adrenaline, as part of preparation for vigorous activity. It can increase heart rate and contraction strength, promote rapid fuel mobilization and widen airways. Changes in vascular activity help redistribute flow toward active tissues. These coordinated effects support substrate and oxygen delivery as muscular ATP demand rises.

The response is not an order for every tissue to do the same thing. Receptor types and intracellular pathways differ; some vessels constrict while others can dilate. Epinephrine is a chemical signal, not the respiratory substrate that directly supplies all the energy for contraction. Glucose and other substrates must still undergo metabolic reactions to support ATP production.

The hypothalamus integrates neural and internal information and helps control the pituitary gland. Pituitary hormones can regulate other endocrine glands, whose hormones act on target tissues and can feed back on the control system. This creates coordinated pathways across several organs. Different pituitary hormones have different targets; "the pituitary controls every gland in exactly the same way" is too broad.

For example, hypothalamic signals can influence pituitary output that controls a peripheral endocrine gland. A rise in the gland's hormone can reduce earlier stimulation through negative feedback. Name the controlled pathway and direction of influence when interpreting such a diagram. Full details of anterior versus posterior pituitary mechanisms are not needed here.

Baroreceptors and chemoreceptors inform cardiovascular control

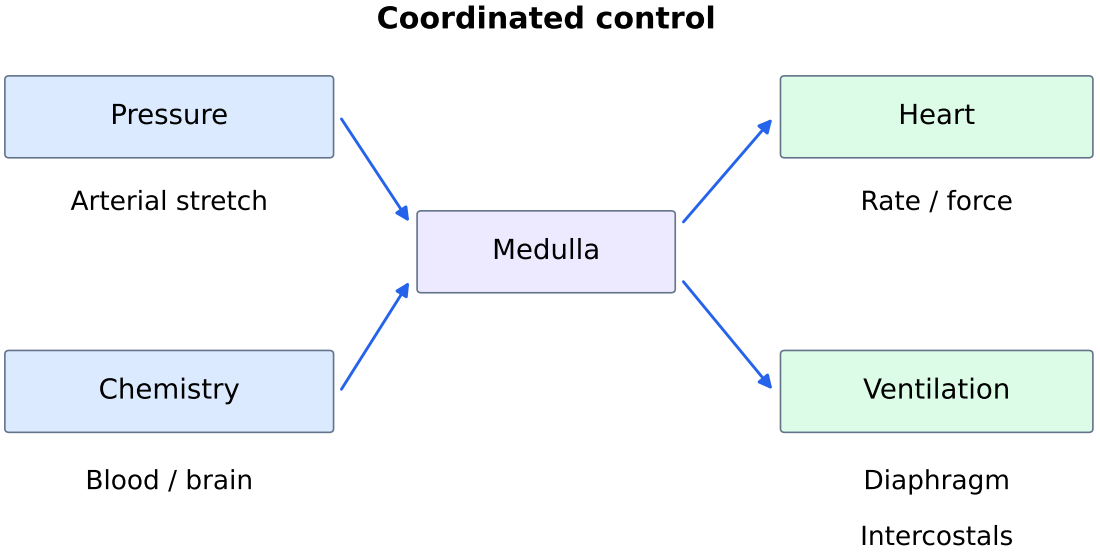
Baroreceptors respond to stretch in arterial walls, notably at the carotid sinuses and aortic arch. Stretch changes with blood pressure. Sensory input reaches cardiovascular control centres in the medulla, which adjust autonomic output to the heart and blood vessels. The heart's own pacemaker generates its basic rhythm; autonomic nerves modify it rather than supplying every heartbeat's initial trigger.

A fall in arterial pressure usually reduces baroreceptor firing. Increased sympathetic and reduced parasympathetic activity can raise heart rate and increase contractile force, supporting cardiac output and pressure. A pressure rise normally produces opposing adjustments. Cardiac output equals heart rate multiplied by stroke volume, but stroke volume also depends on filling and other conditions; it is not fixed by heart

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rate alone.

Chemoreceptors provide information about oxygen, carbon dioxide and acidity. Peripheral receptors in the carotid and aortic bodies monitor arterial conditions and help coordinate cardiovascular and respiratory responses. The carotid body is not the same structure as a carotid-sinus baroreceptor. Name the detected variable as well as the anatomical region.



Pressure receptors and chemical receptors provide different information. The medulla coordinates outputs; changes in heart activity or ventilation then influence the monitored conditions.

In an invented pressure disturbance, the recorded pressure falls from 110 to 90 arbitrary units, an $20/110 \times 100 \approx 18.2\%$ decrease. Heart rate then rises from 70 to 84 beats per minute, a 20% increase. If pressure subsequently recovers, the pattern is consistent with a compensating response. Timing and association alone do not prove that a single receptor pathway caused every change; other controls and repeat measurements matter.

Carbon dioxide and pH help regulate ventilation

Respiring tissues produce carbon dioxide, which is carried in blood. Carbon dioxide reacts with water through a reversible system involving carbonic acid, hydrogen ions and hydrogencarbonate. Increased carbon dioxide tends to increase hydrogen-ion concentration and lower pH, if other conditions are comparable. A lower pH means greater acidity, not “less carbon dioxide in every circumstance”.

Carbon dioxide can cross into brain extracellular fluid and cerebrospinal fluid, changing local pH. Central chemoreceptive pathways in the brainstem respond importantly to this carbon-dioxide-linked acidity. They are not simply oxygen meters directly exposed

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to arterial blood. Peripheral chemoreceptors add information about arterial oxygen, carbon dioxide and pH.

Respiratory control centres adjust motor output to the diaphragm and intercostal muscles. More frequent and/or deeper contractions can increase ventilation, removing more carbon dioxide and opposing the initiating rise. Appropriate gradients and functioning exchange surfaces and circulation remain necessary. Breathing rate alone is not the entire gas-exchange rate.

An invented comparison changes breathing frequency from 12 to 20 breaths per minute and tidal volume from 0.50 to 0.75 litres. Minute ventilation rises from $12 \times 0.50 = 6.0$ to $20 \times 0.75 = 15$ litres per minute, a 150% increase. This is total moved air; some remains in conducting airways, so it is not automatically alveolar ventilation. Do not infer an exact blood-gas change from that multiplication alone.

Exam tips: Name the disturbance, detected variable, receptor, coordinating centre, effector and returning influence. Distinguish carotid sinus from carotid body, and blood chemistry from brain-fluid chemistry. Feedback has a direction; a graph's recovery does not by itself identify every causal step.

Enteric circuits coordinate peristalsis with central control

The enteric nervous system, or ENS, contains networks within the digestive tract that coordinate gut movement, including peristalsis. Contraction behind a food bolus and relaxation ahead help propel contents. Local sensory information about contents and stretch can guide coordinated smooth-muscle activity, rather than each gut segment contracting independently at random.

The CNS influences this activity through autonomic pathways; the ENS also carries out substantial local processing. It is therefore misleading either to make the brain consciously direct every wave or to claim the gut never receives central influences. The contracting gut muscle is smooth muscle, unlike the skeletal-muscle effector in the hand-withdrawal example.

Beginning a swallow can be voluntary, followed by coordinated involuntary phases. Defecation combines reflex activity with voluntary control of appropriate external sphincter muscles when conditions permit. Saying "swallowing and egestion have voluntary control" does not mean every part of each process is a conscious action. Between these endpoints, peristaltic movement normally proceeds involuntarily.

AHL: Observe a tropism as a directional growth response

A tropism is a directional growth response to a directional stimulus. Positive shoot phototropism is growth toward lateral light. A shoot that bends gradually by unequal elongation differs from a whole seedling being pushed sideways. Identify the growing region, stimulus direction and change in orientation over time.

Record qualitative observations with labelled diagrams showing the light direction

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and seedling shape. Quantitative data can include the angle of the shoot relative to its original vertical direction, measured at stated intervals. Define the reference and measurement position consistently; a curved shoot does not have one unambiguous angle at every point.

Use comparable seedlings of one species and age, standardize watering, temperature, light intensity/direction and starting orientation, and include an appropriate uniform-light control. Photograph with a fixed camera and scale, randomize pot positions where suitable, and use independent seedlings. Repeated angle readings on one seedling are not multiple biological replicates. A lamp can also heat or dry one side, so control those influences before attributing every bend to light direction.

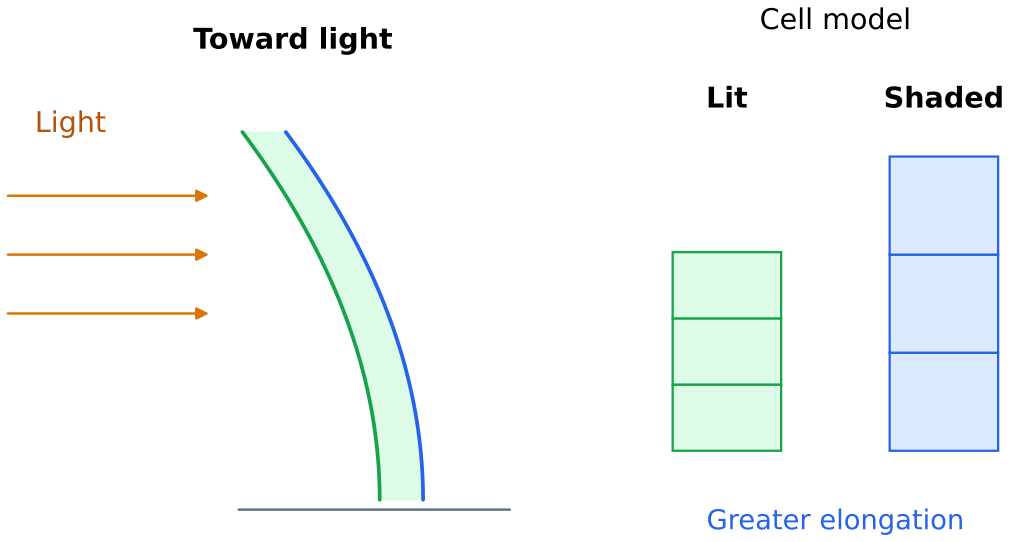
In invented repeat observations, three seedlings curve 10°, 12° and 11° from vertical after a fixed time. The mean is 11° and range 2°. A finer protractor scale can improve reading precision, but not remove an incorrectly placed reference line. Calibration, fixed geometry, suitable resolution, repeated readings and independent replicates address different sources of error and variation.

AHL: Polar auxin transport connects light reception with unequal growth

Phytohormones are plant signalling chemicals involved in growth, development and environmental responses. Examples include auxins, cytokinins and ethylene. Plants do not require animal-style endocrine glands for signals to be produced, transported and received. A response depends on tissue, developmental state and concentration, not just the hormone's name.

In a simplified shoot model under lateral light, redistribution produces more auxin action on the shaded side of the elongating region. Cells there elongate more than those on the lit side. The longer shaded side forms the outside of the curve, so the shoot bends toward the light. It is not the lit side growing longer and pushing the shoot away.

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The schematic is not a concentration measurement. Unequal growth across the shoot changes its curvature; cells do not crawl toward the lamp.

Polar auxin transport depends partly on efflux carriers positioned asymmetrically in cell membranes. Coordinated placement in neighbouring cells gives a preferred direction to cell-to-cell transport and can build unequal auxin distributions. Carrier location matters as well as how much auxin is produced.

The common auxin IAA is a weak acid. Its protonated form can diffuse into cells from the more acidic wall region; in the less acidic cytosol much is deprotonated and crosses the lipid bilayer poorly without appropriate transport. Efflux proteins provide a controlled exit route. Transport also uses influx routes and electrochemical gradients. Do not imagine every efflux carrier directly hydrolysing ATP once per auxin molecule: proton-pump activity helps establish the conditions supporting transport.

AHL: Wall acidification permits elongation and hormones integrate root and shoot

Auxin can promote hydrogen-ion pumping into the apoplast, which includes cell walls outside the plasma membrane. Lower wall pH favours wall-loosening processes, including expansin activity that permits rearrangement of interactions between wall components. Water uptake and turgor can then extend the loosened wall as the cell grows. The membrane and wall are different structures; elongation is not simply the cell wall dissolving.

Cellulose microfibrils are not cut into free glucose molecules each time a shoot elongates. Altered wall interactions permit extension while the wall remains a supporting network, and continuing growth also needs synthesis and organization of wall material. The effect of auxin depends on tissue and concentration; a shoot elongation model

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should not be turned into “auxin always accelerates every root and shoot equally”. Research using Arabidopsis hypocotyls supports the connection between auxin perception, acidification and growth (Fendrych and colleagues, 2016).

Root–shoot coordination also involves auxin and cytokinin. Shoot tips and young tissues are important auxin sources; signals can travel toward roots and influence root development. Roots, including root tips, produce cytokinins that can be transported toward shoots and influence shoot growth. Neither substance is made only in that one location. Local production, transport and sensitivity also matter.

Interactions between the hormones help adjust allocation and development across organs. A plant needs root uptake and shoot photosynthesis to support each other. Changing one hormone can alter the response to the other, rather than producing two independent, unlimited growth commands. Responses depend on the tissue and hormonal balance; the integration cannot be predicted from the amount of one hormone alone.

AHL: Ethylene can reinforce and synchronize fruit ripening

Ethylene, whose systematic name is ethene, is a gaseous plant hormone. In climacteric fruits such as tomato, increased ethylene signalling can promote ripening changes and further ethylene production. Ripening can include colour changes, softening and altered sugars and aromas. The return influence on signal production forms positive feedback.

Gas can spread between nearby fruits, helping coordinate a relatively rapid, synchronized transition. A mature-green tomato can respond differently from a very immature fruit because developmental competence matters. Not every fruit has the same ethylene-driven ripening pattern; do not make this model universal for all harvested produce. UC Davis tomato research and extension guidance describes the response of mature-green tomatoes to ethylene.

In negative feedback a response opposes an initiating change; in this ripening example the response reinforces it. Positive does not mean desirable, and feedback does not create an endless supply of matter or energy. Substrate availability, developmental changes, signal removal and eventual senescence still constrain the process.

Practice

All datasets are invented. Use causal links rather than lists of organs.

1. Explain the hierarchy from cells to body systems using an organ containing several tissues. Explain how interactions among at least three systems support a running animal’s performance as an emergent function.
2. Compare nervous and endocrine routes, target specificity and typical time courses. Explain why “every hormonal response is slow” is unreliable and why blood transport is still needed alongside information transfer.

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3. Distinguish brain and spinal-cord integration, sensory input, motor output and cerebellar coordination. Explain why an unconscious response can occur before conscious pain perception.
4. Describe what a transverse section of a mixed nerve shows, including its sheath and fibre types. Can one circular profile's shape alone identify sensory versus motor function?
5. Trace the simplified hand-withdrawal reflex with one interneuron. Locate the sensory cell body, spinal synapses and muscle effector. Explain why this does not make every reflex identical.
6. Describe the usual daily melatonin pattern and its relation to light and sleep. Distinguish a phase shift from an amplitude change and suggest why repeated days are useful.
7. Explain three effects of epinephrine that support vigorous activity. Outline hypothalamus–pituitary coordination and negative feedback from a peripheral endocrine gland.
8. Pressure falls from 120 to 96 arbitrary units, followed by heart rate rising from 60 to 78 beats per minute. Calculate both percentage changes and outline a baroreceptor–medulla–heart explanation, including receptor location and its limits.
9. Explain how raised carbon dioxide can affect brain-fluid pH and ventilation. Distinguish central and peripheral chemoreceptor information. Calculate minute ventilation for 10 breaths per minute at 0.60 litres and 18 breaths per minute at 0.80 litres, and its percentage increase.
10. Explain ENS coordination of peristalsis and CNS influence. Distinguish voluntary initiation or sphincter control from the involuntary phases of swallowing and gut movement.
11. Explain negative feedback in high and low blood-glucose control. Calculate the increase from 4.0 to 5.0 units, the increase from 5.0 to 8.0, the decrease back from 8.0 to 5.0, and the increase on doubling. Explain why none is a diagnosis by itself.
12. **AHL:** Seedling angles from the original vertical are 8°, 10° and 9° after fixed lateral illumination. Calculate mean and range. Design a controlled comparison and explain why finer angle resolution does not remove systematic camera error. Link the bend direction to unequal elongation.
13. **AHL:** Explain how carrier polarity and IAA's acid properties contribute to auxin distribution. Trace auxin → apoplast acidification → wall loosening → elongation, then explain how auxin and cytokinin connect root and shoot development without being produced exclusively in one organ.
14. **AHL:** Explain positive feedback in tomato ripening, including signal spread and synchronized change. Contrast it with baroreceptor negative feedback. Why should neither loop be described as an unlimited process applying identically to every tissue or fruit?

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Worked answers

1. Cells form tissues; several tissues contribute to organs, which interact in systems. A stomach includes epithelial, muscle, connective and nervous tissues. Running combines nervous coordination, skeletal support, respiratory exchange and circulatory delivery for ATP-producing metabolism. Their interactions produce whole-animal movement, an emergent function absent from an isolated cell.
2. Neurons carry impulses and release transmitter at connected targets; blood-borne hormones act through compatible receptors/pathways. Nervous effects are often rapid/local and endocrine effects broader/longer, but hormones can act quickly and neural changes persist. Blood also carries oxygen, substrates and heat; information does not replace material transport.
3. The brain combines current inputs with prior information and supports learning/memory. Sensory neurons provide CNS input; motor neurons carry effector output. Spinal circuits integrate unconscious responses before conscious processing of ascending information, although brain activity can modulate them. The cerebellum contributes movement timing/correction and balance.
4. Protective connective tissues organize bundles of axons, some myelinated and others unmyelinated with supporting cells. A mixed nerve contains sensory and motor fibres. A profile's shape alone does not identify functional direction; routing or other evidence is needed.
5. Free hand ending → sensory neuron → spinal interneuron → motor neuron → skeletal-muscle contraction withdrawing the limb. The sensory cell body lies in a dorsal-root ganglion; the illustrated synapses lie in spinal grey matter and output exits via a ventral root. Other reflex circuits differ; withdrawal coordinates several muscle groups.
6. Melatonin is generally higher at biological night, lower by day and suppressible by light at night. It helps align sleep–wake timing without instantly switching sleep on. A phase shift changes timing; amplitude changes the high–low difference. Repeated days reveal consistency and variation.
7. Higher heart rate/contractility supports output; wider airways support ventilation; mobilized fuels supply substrates for ATP-producing metabolism. Receptors permit different tissue effects. Hypothalamic control influences pituitary hormones that can regulate other glands; peripheral hormones can reduce earlier stimulation by negative feedback. Different targets require appropriate pathways.
8. Pressure decrease: $(120-96)/120\times100 = 20\%$. Heart-rate increase: $(78-60)/60\times100 = 30\%$. Reduced carotid-sinus/aortic-arch stretch lowers baroreceptor input; medullary autonomic adjustments can increase heart rate and contractile force, helping oppose the pressure fall. Other vascular, filling and control changes also matter; the trace alone cannot isolate their contributions.
9. Raised carbon dioxide can lower brain extracellular/CSF pH; central chemoreceptive pathways inform respiratory control. Carotid/aortic bodies also monitor

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arterial chemistry, including oxygen. Motor output adjusts diaphragm/intercostal activity to increase carbon-dioxide removal. Minute ventilation: $10 \times 0.60 = 6.0$ and $18 \times 0.80 = 14.4$ litres per minute; increase $8.4/6.0 \times 100 = 140\%$. Conducting-airway volume means this is not automatically alveolar ventilation.

10. Enteric networks coordinate contraction behind and relaxation ahead of contents, producing involuntary smooth-muscle propulsion. Autonomic CNS pathways modulate this local control. Swallow initiation and appropriate external-sphincter control can be voluntary, while later swallowing and defecation include reflex phases. The CNS does not consciously direct every gut contraction.
11. Insulin-linked responses oppose high glucose through appropriate uptake/storage and reduced liver output; glucagon-linked liver responses oppose low glucose. $(5-4)/4 \times 100 = 25\%$; $(8-5)/5 \times 100 = 60\%$; $(8-5)/8 \times 100 = 37.5\%$ decrease; doubling is a 100% increase. These describe relative changes and do not specify an individual's diagnosis or mechanism of disease.
12. **AHL:** Mean $(8 + 10 + 9)/3 = 9°$; range $10 - 8 = 2°$. Compare independent same-species/age seedlings under lateral versus uniform light with standardized temperature, watering, intensity, orientation and timing. Fix camera geometry and the angle reference, calibrate measurements and repeat readings. Finer resolution improves reading granularity without correcting a biased camera/reference. Greater shaded-side elongation places that side outside the curve, bending the shoot toward light.
13. **AHL:** Asymmetric efflux-carrier placement coordinates preferred cell-to-cell auxin transport. Protonated IAA can enter from acidic apoplast; much becomes anionic in cytosol and depends on transport for efficient exit. Auxin can promote hydrogen-ion secretion, lowering apoplast pH and favouring wall loosening; water uptake/turgor then support extension without cellulose dissolving into glucose. Shoot-derived auxin and root-derived cytokinins, alongside local sources and sensitivity, help coordinate organ development. Their locations are important sources, not exclusive factories.
14. **AHL:** Ethylene promotes suitable tomato ripening changes and additional ethylene production; gas spread can help synchronize competent fruits. The returning influence reinforces the initiating signal. Baroreceptor feedback instead supports adjustments opposing a pressure disturbance. Developmental state, receptor context, substrates and other controls constrain responses; not all fruits share the climacteric ethylene pattern and not all tissues have identical targets.

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C.3.2 · Defence against disease

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A small skin cut is usually sealed quickly, yet protection against a new virus can take days to develop. These responses solve different problems: closing an entry route, removing invaders and building a response that recognizes a particular molecular target.

This handout covers the full shared SL and HL content of C3.2: barriers, clotting, innate and adaptive responses, antibody production and memory, HIV, antibiotics and resistance, zoonoses, vaccination, herd immunity and infection data. There is no additional Higher-Level-only content in this topic. Prerequisites: cells, proteins, membrane receptors and mitosis.

Examples and numerical datasets are invented unless a published source is named. Use supplied data, safe simulations or approved school cultures; do not collect or grow unknown pathogens.

Pathogens must cross physical and chemical barriers

A **pathogen** is an agent that causes disease. Pathogenic bacteria are cellular prokaryotes; pathogenic fungi and protists are eukaryotes. Viruses are acellular and depend on host cells for replication. These groups contain human pathogens, but most members are not pathogenic. Archaea are another prokaryotic group; none is an established human pathogen. Association with illness alone does not demonstrate causation. Infection means an agent has entered and established itself; disease involves harmful disruption of normal function. Infection can occur without obvious symptoms.

Intact skin has closely packed cells and a tough outer layer that limits entry. Its surface conditions and secretions also inhibit many microorganisms. Damage makes an entry route available, so a protective surface is more than an outer covering.

Mucous membranes line passages such as the airways. Mucus traps particles and microbes; airway cilia move the mucus toward regions where it can be removed or swallowed. Mucus production and ciliary movement cooperate. Tears and saliva help wash surfaces and contain antimicrobial components, including lysozyme, which attacks bacterial cell-wall material. Stomach acid inhibits many swallowed organisms. These defences are effective barriers, not guarantees that every pathogen is destroyed.

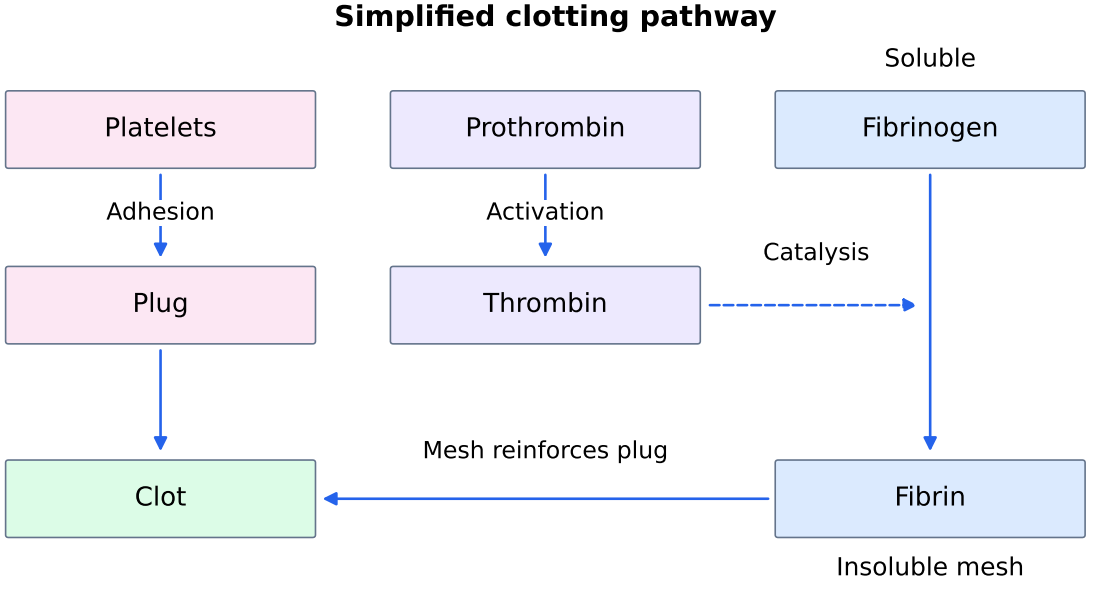
Historical observations helped test infection routes. Semmelweis compared childbed-fever deaths in Vienna and introduced hand disinfection; Snow compared cholera mortality among London households with different water supplies. These comparisons supported practical controls before every microbial mechanism was understood. Compare groups and alternative explanations, rather than treating a famous name as proof. See Semmelweis's translated account and Snow's water-supply study.

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Platelets and a protein cascade seal a damaged vessel

When a small vessel is damaged, platelets adhere at the damaged region, become activated and recruit more platelets. They form an initial plug and contribute signals and surfaces that support clotting reactions. Platelets are cell fragments, not small red blood cells and not the antibodies of the immune system.

Clotting factors activate other factors in a cascade. In the simplified final sequence, prothrombin is converted to the active enzyme thrombin. Thrombin converts soluble fibrinogen into fibrin, which forms an insoluble network. The fibrin mesh reinforces the platelet plug and traps cells, including red blood cells, producing a more stable clot. The sealed region reduces blood loss and closes an entry route for pathogens.



The arrows show activation and conversion, not the transformation of platelets or red cells into fibrin. The full cascade has more steps than this teaching model.

A cascade allows a small initiating event to activate many downstream molecules. The response must also remain controlled and localized: clot formation throughout normal circulation would obstruct flow. Related machinery protects at a cut but can obstruct an important vessel. Fibrin is a protein product; it is not an enzyme that digests the pathogen.

Innate responses recognize broad features and phagocytes engulf invaders

Innate defences are available without prior exposure to a particular antigen. Barriers and phagocytes are examples. They recognize broad features associated with microbes or tissue damage and respond rapidly. In contrast, adaptive responses select lymphocytes with receptors specific to particular targets and can generate long-lived memory.

The two systems cooperate; they are not separate armies that never communicate.

A phagocyte can leave small blood vessels and move through tissues by changing shape. It recognizes a particle, extends membrane around it and encloses it in an internal vesicle. Lysosomes supply digestive enzymes that break down the enclosed material. The membrane enclosure separates the digestion compartment from the rest of the cytoplasm. Some phagocytes also help activate adaptive responses by displaying fragments of ingested material.

This sequence is recognition → engulfment → enclosed digestion. It is not a red blood cell swallowing an antibody, and it is not a plasma cell releasing a digestive enzyme into every neighbouring cell. A phagocyte can attack different appropriate targets; an individual antibody does not bind every pathogen equally well.

Specific receptors select cooperating lymphocyte clones

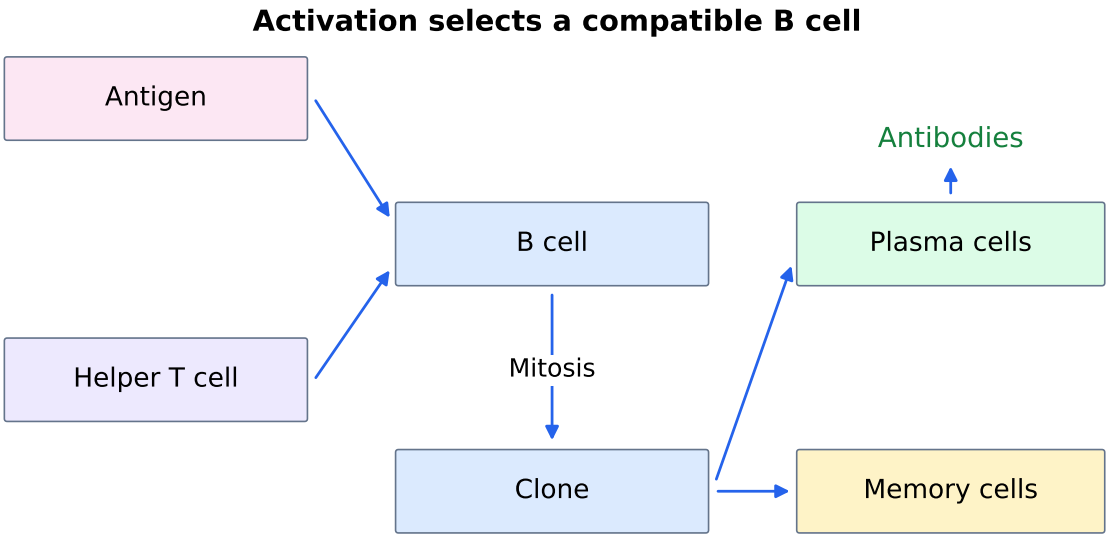
An **antigen** is a molecular structure recognized by a specific antibody or immune receptor. Antigens include proteins and parts of glycoproteins; they can come from the body itself or from outside it. An antigen need not be a whole organism, and recognition does not automatically mean that a substance is dangerous. Red-cell surface antigens, for example, are normal body components, but incompatible blood can provoke an immune response in a recipient.

B and T lymphocytes have different roles. Many different lymphocyte clones exist, with each clone carrying a particular receptor specificity. The appropriate receptor is already present before the encounter; the antigen does not instruct any random lymphocyte to manufacture a matching receptor from nothing. Cells circulate through blood and lymph and encounter antigen in lymphoid tissues, including lymph nodes.

For a typical response to a protein antigen, a B cell binds the target through its receptor and takes it in. It displays antigen-derived material that can be recognized by a compatible helper T cell. Activation requires appropriate interaction and signals. Antigen-presenting cells also help activate helper T cells. This cooperation supports B-cell activation rather than making every passing B cell respond. Not all B-cell responses use exactly the same activation route, but this helper-T-dependent model explains the cooperation required here.

The selected B cell divides repeatedly by mitosis: clonal expansion. Descendants retain the relevant specificity. Many differentiate into plasma cells, which secrete large quantities of **antibodies** , while others become **memory cells** . Helper T cells also form selected expanding populations and can contribute memory. Antibodies are proteins made by cells; they are not themselves cells that reproduce by mitosis.

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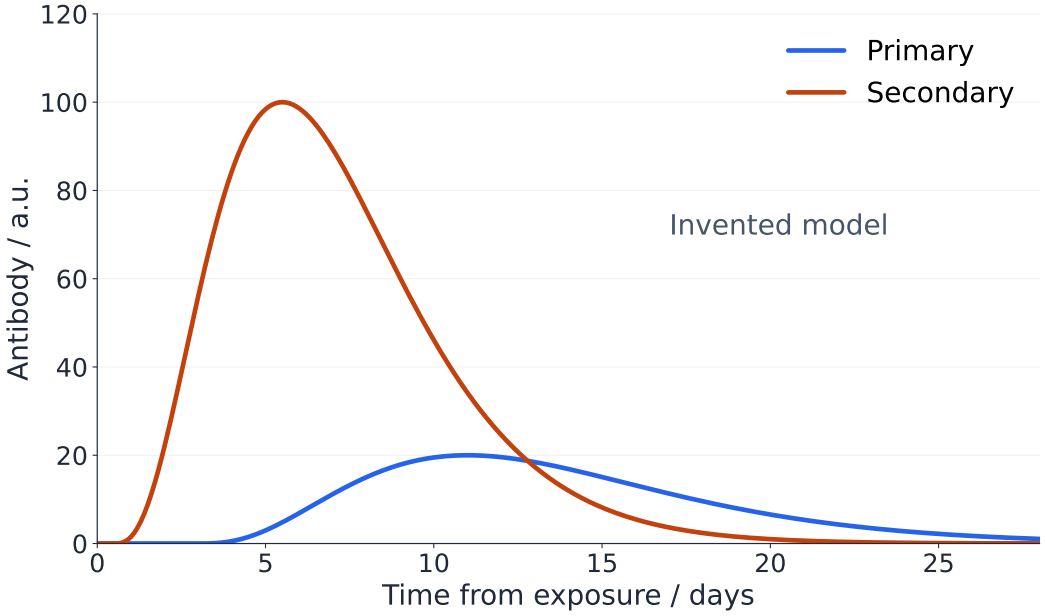
Selection and multiplication explain why the first large antibody response takes time. The diagram summarizes cooperation rather than showing every antigen-presentation step.

An antibody binds a compatible antigenic site through its binding region. Binding can neutralize a toxin or interfere with a pathogen's attachment; it can also help other immune mechanisms remove the marked material. Molecular fit determines binding; antigen change may reduce it without abolishing all protection.

Memory changes the timing and size of a later response

During a primary response, appropriate cells must be activated, expand and differentiate. The antibody concentration therefore rises after a delay. Many short-lived effector cells disappear after the antigen is cleared, but memory populations can persist. On later exposure to a sufficiently similar target, these populations can support a faster and larger secondary response.

In a separate invented comparison, the primary response first exceeds a stated detection level after six days and reaches 20 arbitrary units. A later exposure produces a response above that same detection level after two days, with a peak of 100 units. The peak is five times as large, a 400% increase; the detection delay is four days shorter, a decrease of about 66.7%. The graph must state its detection rule and time reference. A curve's first visible point is not necessarily the instant the first antibody molecule was made.



Both curves use time since their own exposure. Peaks, delays and persistence are schematic, not measurements for a named vaccine or disease.

Memory does not guarantee that infection is impossible or that protection is lifelong for every pathogen. Antigen change, waning protection and the site of infection matter. Many different viruses cause colds, so a later cold need not be the same antigenic challenge as an earlier one. A positive antibody result also needs context: sampling time, assay specificity and the measured target affect interpretation.

HIV disrupts helper-T-cell coordination

HIV is a virus that infects CD4 helper T cells and some other susceptible cells. Progressive damage to the helper-T-cell population can weaken the coordination of immune responses, including effective B-cell responses. AIDS is the advanced syndrome associated with severe immune damage and vulnerability to particular opportunistic infections and other illnesses. HIV infection and AIDS are not interchangeable terms, and infection does not mean a person already has AIDS.

Transmission requires an appropriate route involving infectious body fluids, including blood, semen, pre-seminal fluid, vaginal or rectal fluids, and breast milk. Examples include sexual exposure, sharing contaminated injection equipment and transmission during pregnancy, birth or breastfeeding. A fluid must reach susceptible tissue or the bloodstream; simply being near a person does not provide such a route. HIV is not transmitted by ordinary touching, sharing classroom space or a mosquito transferring it between people. This biological distinction prevents stigma as well as incorrect explanations.

Effective antiretroviral treatment can suppress viral replication and preserve immune function; HIV does not inevitably progress rapidly to AIDS. CDC's transmission expla-

nation and NIH's HIV and AIDS overview explain the CD4-cell target and transmission routes.

Antibiotics act on bacterial targets and select resistant variants

Antibiotics can inhibit bacterial processes such as cell-wall synthesis, bacterial ribosome function or particular metabolic pathways. Selectivity depends on differences between bacterial and human cells. Viruses do not have their own bacterial cell wall or bacterial ribosomes, so an antibiotic directed at those targets does not stop a viral infection by the same mechanism. Some viral illnesses can be complicated by bacterial infection; that does not mean the antibiotic directly kills the virus.

Resistance is a heritable ability of a microorganism to survive exposure that would inhibit susceptible organisms under specified conditions. Resistant variants can arise through mutation, and bacteria can also acquire resistance genes by gene transfer. Drug exposure then changes survival and reproduction: susceptible bacteria are removed more effectively, while resistant survivors leave more descendants. Exposure selects variation; it does not make an individual bacterium decide to mutate because it needs protection.

Consider an invented population of 10,000 bacteria containing 100 resistant and 9,900 susceptible cells. After treatment, 50 resistant and 99 susceptible cells remain. The resistant fraction rises from 1% to $50/149 \times 100 \approx 33.6\%$, although the number of resistant cells has actually fallen. A higher proportion does not necessarily mean an immediate increase in the absolute resistant count. Subsequent reproduction can expand the survivor population.

Resistance to several antibiotics can accumulate through multiple mechanisms or acquired genes, producing multidrug-resistant strains. Appropriate prescribing, infection prevention and careful use can reduce selection and spread; they do not guarantee that resistance will never evolve. Research can screen chemical libraries for compounds that inhibit bacterial growth, then test mechanism, toxicity and effectiveness. A clear inhibition zone in an approved school test is evidence of growth inhibition under those conditions, not proof of a safe medicine for humans. Standardize the organism, inoculum, medium, disc dose, incubation and measurement, with suitable controls and repeats.

Zoonotic infections connect animal hosts, vectors and humans

A zoonosis is an infection that can pass naturally between other vertebrate animals and humans. Distinguish the reservoir that maintains an agent from a vector that carries it between hosts. A pathogen's successful entry into a new species also depends on compatible cells and subsequent transmission; contact alone does not guarantee a sustained human outbreak.

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Example	Relevant host and route
Bovine tuberculosis	Mycobacterium bovis is associated with cattle and can infect people through contaminated unpasteurized dairy products or close exposure; it is distinct from the mainly human-associated M. tuberculosis
Rabies	Virus in an infected mammal's saliva can enter through a bite; the bite introduces the pathogen rather than manufacturing it
Japanese encephalitis	Culex mosquitoes transmit virus maintained among pigs and water birds; people usually do not develop enough virus in blood to continue the mosquito cycle
COVID-19 emergence	SARS-CoV-2 has animal-virus relatives; evidence supports zoonotic emergence, while the precise original spillover route remains unresolved

CDC's bovine-TB account distinguishes M. bovis from M. tuberculosis. WHO's Japanese-encephalitis account explains the mosquito–animal cycle. Calling Japanese encephalitis zoonotic does not mean that ordinary contact with a patient transmits it. Likewise, an animal origin for a virus does not mean that every later human case comes directly from an animal.

For COVID-19, keep the uncertainty specific: an unresolved original route is not uncertainty about whether later human-to-human transmission occurred. WHO's SAGO assessment evaluates origins evidence and remaining gaps.

Vaccination prepares memory without requiring the natural disease

Vaccination introduces a vaccine that presents chosen antigens, or instructions that allow cells to produce an antigen, and stimulates a specific immune response. Vaccine types include inactivated pathogens, weakened live pathogens, purified components and nucleic-acid approaches. These differ in how the target is delivered; an mRNA vaccine does not contain a whole reproducing virus simply because cells make one antigen.

Antigen recognition and lymphocyte activation lead to clonal expansion, effector activity and memory. Later exposure can then produce a faster protective response. Booster doses can strengthen or renew responses. Protection may be measured against infection, severe disease or another defined outcome; these are different endpoints. A vaccine that reduces severe disease does not automatically prevent all infection or all transmission.

Vaccine evaluation tests benefits and harms, with safety monitoring continuing after introduction. Low risk does not mean zero side effects. Early media reports may appear

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before scientific evaluation is complete: examine study design, outcome, denominator and uncertainty. WHO's vaccine-safety account explains testing and monitoring.

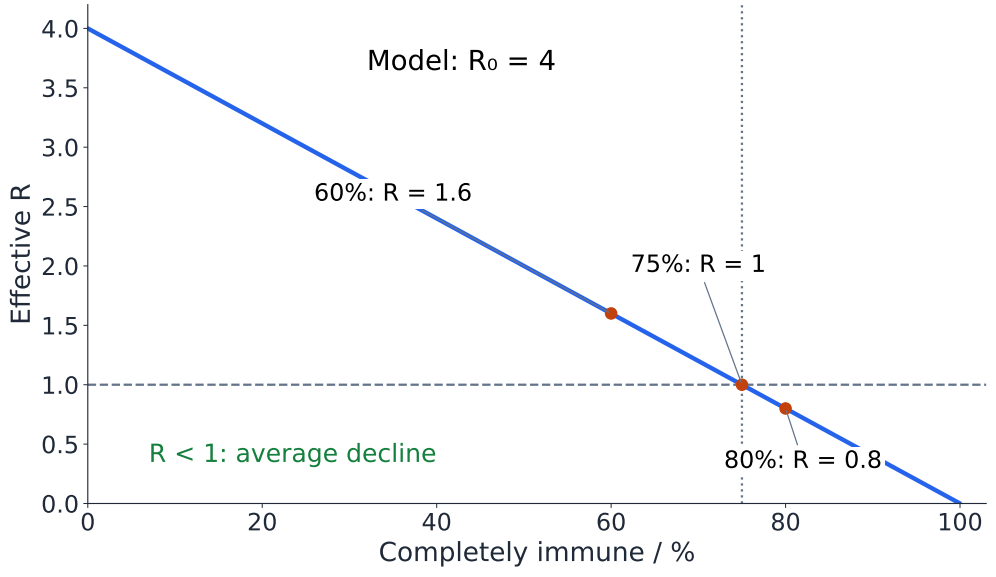
In an invented comparison with equal follow-up, 80 of 1,000 unvaccinated participants and 20 of 1,000 vaccinated participants develop the defined outcome. Risks are 8% and 2%. Relative risk is $2/8 = 0.25$, and the relative risk reduction is $1 - 0.25 = 75\%$. The absolute risk difference is six percentage points. These data alone do not show whether groups were comparable or whether the outcome was infection rather than severe illness. Randomization, complete follow-up, clear case definitions and uncertainty estimates strengthen the inference.

Herd immunity depends on reduced transmission and model assumptions

Herd immunity means indirect population protection when immunity reduces transmission. In a simple uniformly mixing population, let R_0 be the average secondary cases caused by one infectious case in an otherwise fully susceptible population under the stated conditions. Let p be the proportion completely protected against contributing to transmission. The model gives:

$$R = R_0(1 - p).$$

Transmission declines on average when $R < 1$. Solving gives $p > 1 - 1/R_0$ for $R_0 > 1$. At exactly $p = 1 - 1/R_0$, the model gives $R = 1$, the boundary rather than a declining average chain. With $R_0 = 4$, that boundary is 75%; 80% immune gives $R = 0.8$, while 60% gives $R = 1.6$. A value below one does not mean that no new case can ever occur.



The homogeneous model treats protection as complete and lasting. Exactly 75% is the boundary; values above it give average decline under these assumptions.

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Vaccination coverage is not automatically the immune fraction. In a further simplified model, if a fraction v is vaccinated and protection against contributing to transmission is e, use $p = ve$. At $R_0 = 4$ and $e = 0.90$, the coverage boundary is $0.75/0.90 \approx 83.3\%$; decline requires more than that boundary. This assumes an otherwise susceptible population, uniform exposure and no other controls. Clustered contacts, waning protection, variants and uneven uptake can change outcomes. Disease severity and infectiousness are different properties; a milder illness need not have a smaller R_0.

Infection data need a denominator, a time window and a case definition

Prevalence describes existing cases at a specified time or over a defined period. Incidence concerns new cases arising over a stated period, with a suitable population-at-risk or person-time denominator. A cumulative count of all reported cases since the start is not the number currently ill. Always identify whether a dataset measures detected infections, symptomatic cases, admissions or another outcome.

WHO's historical situation report 1, published on 21 January 2020 with data reported by 20 January, gives 282 globally confirmed cases. Report 2, published on 22 January with data reported by 21 January, gives 314. The difference is 32 reported cases, and the increase is $32/282 \times 100 \approx 11.3\%$. These are early historical reported totals, not present-day pandemic totals or a complete count of every infection. The subtraction does not establish that exactly 32 people acquired infection during that day: testing, reporting delays and case definitions affect the count. Sources: WHO report 1 and WHO report 2.

Percentage change uses the initial value as denominator. A symmetric percentage difference, when that convention is requested, uses the mean of the two values: $32/298 \times 100 \approx 10.7\%$. State the formula because courses and questions can use different meanings of "percentage difference".

For a published equal-population comparison, CDC's historical TB table gives annual US incidence of 2.5 cases per 100,000 people in 2022 and 2.9 in 2023. Multiplying each by ten gives 25 and 29 per million: the displayed difference is four cases per million per year. These are rounded surveillance rates for TB disease, not all latent infections or a snapshot of existing cases. Compare equivalent time windows, case definitions and detection, and consider age and exposure before claiming a biological cause.

Exam tips

- Trace the actual material or signal: platelets recruit, thrombin catalyses fibrin formation, plasma cells secrete antibodies and memory cells support later responses.
- Use antigen specificity and selected clones to explain the delay and secondary response. Do not say an antigen is always foreign or that all lymphocytes secrete antibodies.

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- Separate the pathogen, host, vector and transmission route. HIV does not spread by casual classroom contact; antibiotics do not target viruses as though they were bacteria.
- Name the denominator and outcome. Distinguish percentage change, percentage points, relative risk, cumulative reported cases and current prevalence.
- State the assumptions of a herd-immunity calculation and distinguish its boundary from $R < 1$.

Practice

All datasets below are invented. Explain mechanisms as well as calculating answers.

1. Compare one physical and one chemical barrier. Explain why damage to skin can increase infection risk, and distinguish a pathogen from a harmless microorganism.
2. Trace platelet activity and the simplified clotting cascade through prothrombin, thrombin, fibrinogen and fibrin. State how the clot reduces both blood loss and pathogen entry.
3. Describe phagocytosis from movement to digestion. Compare its broad recognition with antigen-specific adaptive recognition, including one way the systems cooperate.
4. Define antigen, antibody and lymphocyte clone. Explain why an antigen can be a body component and why a B cell is not the same thing as its secreted antibody.
5. Explain helper-T-cell cooperation, B-cell activation, clonal expansion and differentiation into plasma and memory cells. Include the role of lymph nodes and the source of response specificity.
6. An antibody response first exceeds a fixed detection level after eight days and peaks at 15 units. A later response exceeds it after two days and peaks at 90 units. Calculate the peak ratio, peak percentage increase and percentage decrease in the delay. Explain why memory need not prevent every future infection.
7. Explain how HIV can weaken an antibody response without directly destroying every plasma cell. Name two transmission routes and reject the claim that sitting beside an infected person transmits HIV.
8. A population initially contains 200 resistant and 9,800 susceptible bacteria. After exposure, 100 resistant and 98 susceptible bacteria remain. Calculate the initial and final resistant percentages. Explain why selection can raise the fraction while lowering the resistant count, and why the same antibiotic need not act on a virus.
9. Distinguish the host and route for bovine tuberculosis, rabies and Japanese encephalitis. Explain why a zoonotic origin does not imply that every later human infection is acquired directly from an animal. State one limitation on claims about the first SARS-CoV-2 spillover.
10. In comparable groups followed for the same period, 60 of 1,200 unvaccinated and 15 of 1,200 vaccinated people develop the stated outcome. Calculate both risks,

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the risk ratio, relative risk reduction and absolute difference in percentage points. Give two design details needed to interpret protection.

11. For $R_0 = 5$, calculate the immune boundary and R at 75% and 85% complete immunity. If protection against transmission is 90%, calculate the vaccination-coverage boundary using $p = ve$. Explain why severity alone cannot determine these quantities.
12. Two successive cumulative reported totals are 400 and 500. Calculate the percentage increase and the symmetric percentage difference using their mean. Explain why the difference is not necessarily the number of infections acquired between the report dates.
13. Over one month, group A has 24 new cases among 800 initially at-risk people, and B has 36 among 1,600. Calculate each risk and cases per 1,000, the risk ratio A/B and the difference in percentage points. Distinguish these new-case measures from a snapshot of existing cases, and propose a safe school investigation of a related question.

Worked answers

1. Intact skin is a physical barrier; tear/saliva lysozyme attacks bacterial cell-wall material, or stomach acidity inhibits many swallowed organisms. A cut bypasses the surface. Pathogens cause disease; many microorganisms are harmless, and infection need not immediately cause visible disease.
2. Platelets adhere and activate at damage, recruiting a plug and supporting clotting reactions. The cascade activates thrombin from prothrombin. Thrombin converts soluble fibrinogen to insoluble fibrin; its mesh reinforces the plug and traps blood cells. Sealing limits leakage and pathogen entry. Platelets do not become fibrin, and red cells do not produce the clotting enzyme.
3. A phagocyte can leave vessels and move through tissue, recognize a target, surround it with membrane and enclose it. Lysosomal enzymes digest the enclosed material. Broad innate recognition differs from the selected receptor specificity of adaptive clones. Antigen presentation by appropriate cells helps connect engulfment with helper-T-cell activation.
4. An antigen is a structure recognized by a specific antibody or immune receptor; self and external molecules can both qualify. An antibody is a binding protein secreted by a plasma cell. A clone contains descendants of a selected lymphocyte retaining relevant specificity. B cells are living cells; antibodies are protein products and do not divide.
5. A pre-existing compatible B-cell receptor binds antigen; uptake and presentation can permit recognition and activating signals from an appropriate helper T cell. Selected cells divide by mitosis, producing a clone. Plasma descendants secrete matching antibodies; memory descendants persist. Blood and lymph transport cells, and lymph nodes provide sites for antigen–lymphocyte encounters. The

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antigen selects a compatible clone rather than teaching an arbitrary receptor to change into a matching one.

6. Peak ratio $90/15 = 6$; increase $(90 - 15)/15 \times 100 = 500\%$. Delay falls by six days, so $6/8 \times 100 = 75\%$. Memory supports faster expansion and effector production, but antigen changes, waning responses and infection-site differences can limit protection. Six times the original peak is not a 600% increase.
7. HIV damages CD4 helper T cells, weakening coordination and appropriate B-cell help. Two routes are sexual exposure to infectious fluids and contaminated injection equipment; pregnancy, birth or breastfeeding can also transmit the virus. Sitting nearby, touching and shared classroom air do not provide a transmission route. HIV infection is not automatically AIDS.
8. Initially $200/10{,}000 \times 100 = 2\%$; finally $100/(100 + 98) \times 100 \approx 50.5\%$. Resistant cells fell from 200 to 100, but susceptible cells fell much more strongly. Differential survival changes the composition; survivors can then reproduce. Mutation or acquired genes supply heritable variation, not conscious adaptation. A virus lacks the bacterial cell wall or bacterial ribosomes targeted by many antibiotics.
9. Cattle-associated M. bovis can reach people through unpasteurized dairy products or close exposure; rabies virus can enter through an infected mammal's bite; infected Culex mosquitoes transmit Japanese-encephalitis virus from a pig/waterbird cycle. Human-to-human transmission can continue after an initial species jump. SARS-CoV-2 evidence supports zoonotic emergence, but its precise first route is unresolved; do not invent a proven intermediate animal.
10. Risks: $60/1200 = 5\%$ and $15/1200 = 1.25\%$. Risk ratio $1.25/5 = 0.25$; relative reduction 75%; absolute difference $5 - 1.25 = 3.75$ percentage points. Specify the measured outcome and equivalent follow-up; randomization or appropriate adjustment, comparable exposure, reliable case detection and uncertainty estimates also matter. The result does not automatically describe protection against transmission.
11. Boundary $1 - 1/5 = 0.80 = 80\%$. At 75% immunity, $R = 5(0.25) = 1.25$; at 85%, $R = 5(0.15) = 0.75$. At 90% protection, coverage boundary $0.80/0.90 \approx 88.9\%$; greater coverage is required for average decline under the assumptions. At the exact boundary $R = 1$. Severity is harm to the host; R_0 concerns transmission under stated conditions. Uniform mixing, susceptibility and stable protection are model assumptions.
12. Increase $(500 - 400)/400 \times 100 = 25\%$. Mean 450, so symmetric difference $100/450 \times 100 \approx 22.2\%$. These are reported cumulative cases; testing changes, reporting delays and revisions can affect the difference. Neither cumulative value measures the number currently ill, and report dates need not equal infection dates.
13. A: $24/800 = 3\% = 30$ per 1,000. B: $36/1600 = 2.25\% = 22.5$ per 1,000. Risk ratio $3/2.25 \approx 1.33$; difference 0.75 percentage points. A has fewer cases but higher risk. These are new cases over a month, not prevalence. Use supplied data or a repeated contact-network simulation with matched conditions; discuss limits.

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Populations and communities

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A field edge appears crowded with flowers, but the centre looks almost bare. Is the population genuinely large, or did the observer choose the patches that support that conclusion? A population estimate needs a sampling design, while an explanation of its size also needs resources, reproduction and interactions with other organisms.

This handout covers all shared C4.1 content: population sampling, mark–recapture, carrying capacity and growth, community interactions, mutualism, invasive competitors, tests of association, predator–prey regulation and chemical interference. The content is common to SL and HL. Prerequisites: arithmetic, percentages, graphs, cell division and ecological niches.

Numerical examples and all teaching figures are invented unless a published study is explicitly named. They are not real exam questions. Use approved organisms, permissions and low-impact methods; use supplied data or simulations when capture or manipulation would harm organisms or habitats.

Define the population and choose a representative sample

A **population** is an interacting group of organisms of the same species living in a defined area. Members of a sexually reproducing population normally breed with one another; reproductive separation helps distinguish neighbouring populations of the same species. A boundary must suit the question. "All daisies in this 100-square-metre lawn this month" specifies a species, area and period. "All plants near the school" mixes species and leaves the area uncertain. Immigration and emigration can connect populations, so a drawn boundary is not necessarily a biological wall.

Counting every individual may be impractical because an area is large, organisms are numerous or hidden, or repeated disturbance is undesirable. A sample is used to estimate the whole. Random selection gives eligible locations a known, non-preferential chance of selection; choosing the greenest or easiest patches creates bias. Random does not mean placing a frame wherever it happens to be convenient.

Define the accessible survey area before generating coordinates. Include sparse and empty locations, use the same counting rule throughout, and record locations that cannot be sampled safely. Do not silently move an inconvenient coordinate to a nearby rich patch. If clearly different habitats form part of the target area, random samples within each stratum can be combined using their actual areas; equal numbers of quadrats from unequal habitat areas do not automatically give the correct whole-area mean.

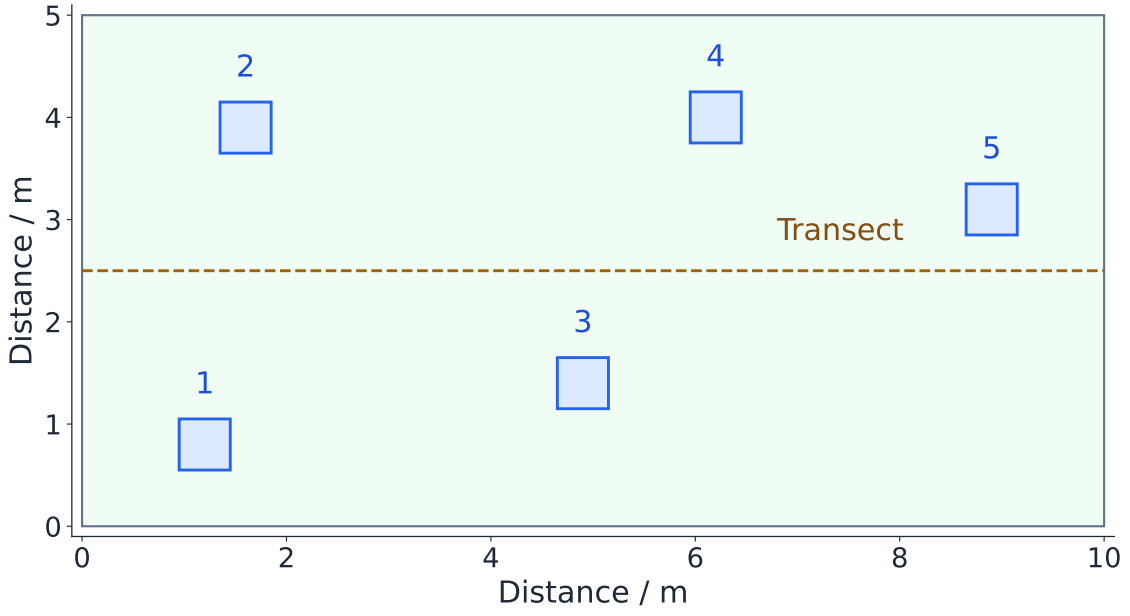
Sampling error is the difference between an estimate and the unknown true population value. Even a fair random sample usually differs from a census. Bias is a systematic

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tendency caused by the method. More independent random samples generally improve precision, but repeating a biased method more often does not remove its bias.

Quadrat counts connect mean abundance, density and total size

A **quadrat** defines a known sampling area. It suits sessile organisms, including rooted plants and attached animals, rather than rapidly moving animals that enter or leave while being counted. A square frame is convenient, but its area must be measured, not guessed. Decide in advance how to count individuals touching the boundary, for example including two specified edges and excluding the opposite two. For clonal plants, define whether the count concerns separate shoots or genetically distinct individuals. Cover may be a better measure when individuals cannot be separated reliably.



The positions are an invented survey plan, not evidence that the field has one particular density. A transect samples a gradient; random quadrats estimate an area's population.

In a separate invented survey, five 0.25-square-metre quadrats contain 3, 4, 6, 5 and 2 plants. The mean count is $20/5 = 4$ plants per quadrat. Dividing by quadrat area gives $4/0.25 = 16$ plants per square metre. Multiplying by the target area's 100 square metres gives an estimated 1,600 plants. Equivalently, 20 plants in a total sampled area of 1.25 square metres gives the same density. Multiplying the mean count directly by 100 would forget that a quadrat is smaller than one square metre.

The estimate assumes the sampled locations represent the target area and detection is reliable. A narrow field-edge strip may have a different density from the centre. For

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a gradient investigation, place a line transect along the stated direction and sample at pre-decided intervals, recording an abiotic variable such as light or soil moisture. A belt transect samples a strip rather than only organisms touching a line. Systematic sampling can reveal change along the gradient, but one conveniently placed transect does not automatically represent the entire field.

Variation between quadrats differs from uncertainty in their mean

Counts can vary because organisms are clumped or because habitat conditions vary. A sample standard deviation, s, describes spread among the sampled quadrat counts in the same units as those counts. For 3, 4, 6, 5 and 2, a calculator gives $s \approx 1.58$ plants per quadrat. It is not 1.58 plants per square metre unless the area conversion is also applied. Large spread suggests uneven counts, although a spatial map helps reveal the pattern.

The standard error of the mean, $s/\sqrt{n}$ for independent samples under the usual assumptions, describes estimated uncertainty in the sample mean. Here it is about $1.58/\sqrt{5} \approx 0.71$ plants per quadrat. Standard deviation and standard error are different quantities; neither is automatically a 95% confidence interval. More independent samples tend to reduce standard error, while the underlying between-quadrat variation can remain large. Adjacent quadrats in the same patch may be correlated, so treating all of them as independent exaggerates precision.

For equal-area counts 2, 4 and 6, mean 4 and sample standard deviation 2 describe a variable sample; counts 4, 4 and 4 have the same mean but zero observed spread. Zero spread in three observations does not prove the entire habitat is uniform. State the number of samples and inspect the actual distribution rather than presenting one error bar as a complete explanation.

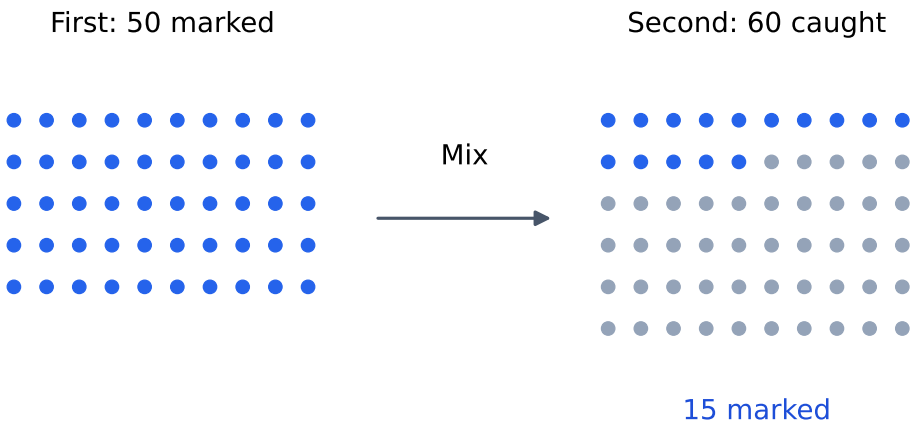
Mark–recapture estimates mobile populations from a marked fraction

For suitable mobile animals, capture a first sample, mark individuals using an approved method, release them and allow mixing. Capture a second sample and count both its total size and how many bear the original mark. Let M be the number originally marked, C the total second capture and R the marked individuals within that second capture. The Lincoln estimate is:

$$\widehat{P} = \frac{MC}{R}.$$

The reasoning is a proportion: if marked animals mix representatively, the marked fraction in the second catch, R/C, approximates the marked fraction in the population, M/P. Rearranging gives the estimate. The second catch includes marked and unmarked animals; it is not just the marked recaptures.

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Estimate: 50 × 60 ÷ 15 = 200

The highlighted subset is part of the second catch. Marks are identifiers, not a different species, and the two catches are not added to estimate the whole population.

Here $M = 50$, $C = 60$ and $R = 15$, so $\widehat{P} = 50 \times 60/15 = 200$. The second marked fraction is $15/60 = 0.25$, implying that the 50 marked animals represent about one-quarter of the population.

The model assumes little population change between samples, sufficient mixing, retained and recognized marks, and comparable capture probabilities for marked and unmarked individuals. Marking should not alter survival or behaviour. If marks are lost, observed R is too small and the estimate tends to be too large. Trap avoidance can have the same direction; attraction to traps can increase R and make the estimate too small. With very few recaptures, chance has a large effect; $R = 0$ makes this simple expression undefined, not evidence of an infinite population. Use repeated appropriate sampling or a more suitable model rather than claiming exactness.

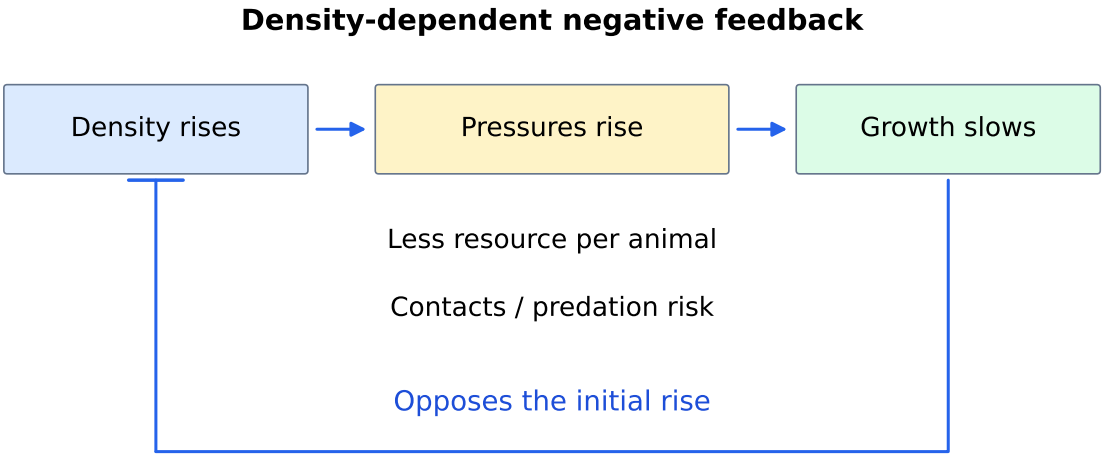
Limited resources and density-dependent feedback affect population size

Carrying capacity , K, is the population size an environment can sustain over time under stated conditions. Food, water, light, mineral nutrients, nesting sites and space can limit different populations. It is not a permanent number stamped on a habitat: seasons, habitat change and resource renewal can alter it.

Competition occurs when organisms share a limiting resource. Within one species, this is intraspecific competition. Greater density can reduce food per individual, lowering reproduction or survival. Close contact can increase transmission of some pathogens or pests, and **predation** can increase when predators find dense prey more easily. These are possible density-dependent effects: their per-individual

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impact changes with population density. They do not behave identically for every pathogen or predator.



The returning blue bar shows opposition to the initial rise. Delays, changing resources and disturbances can produce fluctuations or collapse rather than perfect constancy.

For a closed population, net change is births minus deaths. With movement, add immigration and subtract emigration. An invented population of 100 gains 30 births and loses 10 deaths in one interval, increasing by 20. At 200 individuals, 35 births and 35 deaths give zero net change. This is consistent with increased limiting pressures; it does not prove which resource caused them. Balanced births and deaths mean continuing turnover, not that every individual has stopped reproducing.

A density-independent disturbance can affect populations without its per-individual effect being controlled by density, for example a severe frost damaging exposed plants. It can still kill more individuals where more were present. Weather and resource limitation can interact, so explanations need not force every event into a single exclusive category.

Exponential and sigmoid models predict different growth patterns

When resources are abundant and average reproduction exceeds losses, a roughly constant per-capita growth rate can produce exponential increase. Larger populations then add more individuals in the same time even though the proportional increase is unchanged. In a doubling model,

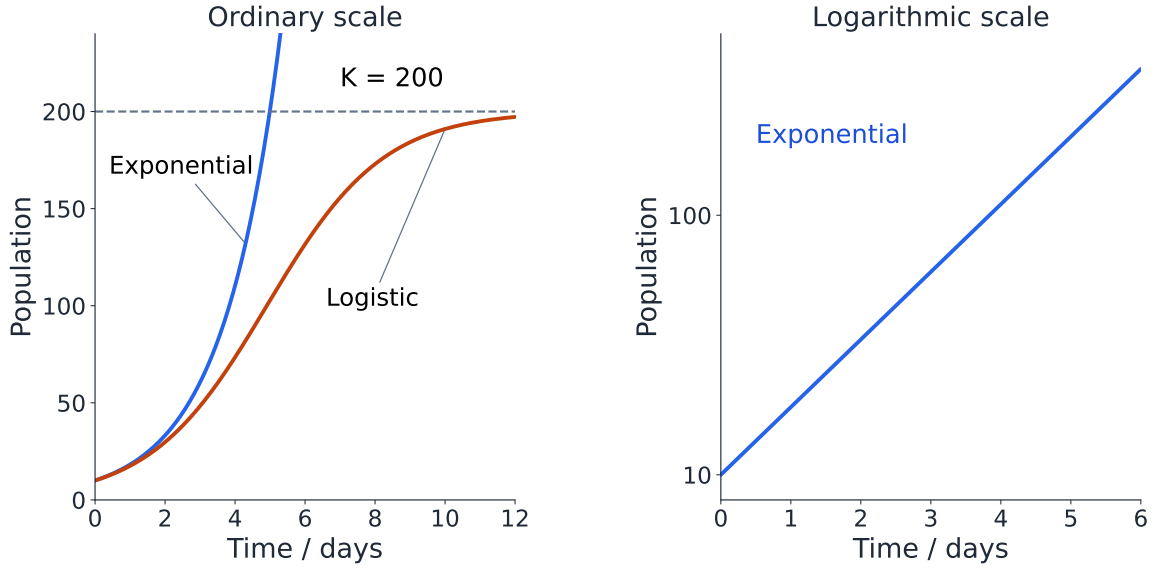
$$P_t = P_0\, 2^{t/T_d},$$

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where T_d is the doubling time. Starting at 10 individuals with one doubling per day gives 10, 20, 40, 80 and 160 at days 0–4. The increases are 10, 20, 40 and 80, not a fixed addition of ten every day.

Plot population size on a logarithmic vertical axis against ordinary time to test for an exponential phase. Equal multiplicative increases become equal vertical steps; a constant exponential rate gives an approximately straight line. With natural logarithms, $\ln P = \ln P_0 + rt$, whose slope is the continuous per-capita rate r. Logarithms of zero or negative counts are undefined. A short straight-looking segment supports a local model, not indefinite exponential growth.

As limiting pressures strengthen, growth can slow toward carrying capacity, giving an ideal sigmoid curve. Its middle is steep; its upper region approaches a balance of gains and losses. The continuous logistic model has $dP/dt = rP(1-P/K)$: per-capita growth decreases as P approaches K, and for positive r it is zero at $P = K$. A flat lag phase is not a required part of an ecological sigmoid growth curve. A culture may have an acclimation period for other reasons, but do not add one to every population model.



The logistic curve has an initially increasing population without a compulsory flat lag phase. The dashed carrying-capacity line is a model condition, not a measured universal limit.

An approved duckweed investigation can count fronds at fixed times in separate replicate vessels. Standardize starting number, light, temperature, nutrient medium, vessel area and counting rule; record new daughter fronds consistently. Replicate vessels are the biological replicates, not repeated counts of one vessel. Plot the data before choosing a model, examine the early logarithmic plot and later slowing, and consider shading, nutrients and space. Measure actual growth rather than forcing every observation onto an ideal S shape.

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Real populations can overshoot and decline sharply. Klein's published abstract on St Matthew Island reports 29 introduced reindeer in 1944, about 6,000 in 1963 and 42 survivors in 1966. Food depletion interacted with extreme winter conditions; the crash occurred in the winter of 1963–64, rather than being an event first caused by the 1966 count. This is a case of rapid growth and failure to stabilize smoothly, not proof that carrying capacity was exactly 6,000. Source: Klein's population study.

Members of one population can cooperate as well as compete

Individuals compete when they share a limiting resource, but cooperation can increase their success in other tasks. Male red deer compete for mating opportunities; crowded Scots pine trees compete for light, water and nutrients. Wolves can cooperate in hunting large prey, and penguin huddles reduce heat loss. These examples involve different mechanisms, rather than a single rule that social animals never compete.

Cooperation can improve food acquisition, defence or survival while still involving costs and unequal benefits. Members of a wolf pack can cooperate in hunting and compete over food or reproduction. The relationship depends on the activity and circumstances. Neither "all within-species relationships are harmful" nor "members of one species always help one another" is a useful generalization.

Communities contain interacting populations of different species

A **community** contains all the interacting populations in an ecosystem, including plants, animals, fungi and microorganisms. The ecosystem also includes the abiotic environment and its exchanges. A community is not just the conspicuous animals in a photograph.

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Interaction	Example and consequence
Herbivory	A caterpillar eats plant leaves; the animal gains food and the plant loses tissue
Predation	A lynx kills and eats a hare; prey supply and predation can affect population growth
Interspecific competition	Two plant species share limiting light, nutrients or space, reducing performance relative to suitable controls
Mutualism	Compatible legume roots and nitrogen-fixing bacteria exchange benefits
Parasitism	A tick feeds on a mammal, benefiting while imposing a cost on a living host
Pathogenicity	A disease-causing bacterium disrupts normal host function; disease can alter survival and reproduction

These categories describe effects and mechanisms. A parasite commonly maintains a living host for some time, whereas predation typically kills the prey during the feeding event; exceptions and overlap exist. Pathogenicity emphasizes disease, and a parasite can also cause disease. Classifying an interaction does not demonstrate how strongly it controls the whole community.

Mutualism requires a benefit to both partners in the stated context

In legume root nodules, compatible nitrogen-fixing bacteria convert atmospheric nitrogen into biologically usable nitrogen compounds. The plant gains a nitrogen supply; the bacteria receive plant carbon compounds for energy and growth and a suitable protected environment. The bacteria do not simply turn nitrogen gas straight into plant glucose, and not every bacterium around a root forms an effective nodule.

Orchid mycorrhiza needs careful qualification. Fungal partners supply nutrients and often carbon to orchids; tiny seedlings can depend on fungal carbon before they photosynthesize. For a demonstrated reciprocal example, adult green Goodyera repens can transfer photosynthetically fixed carbon to its fungus while receiving fungal nitrogen. Cameron and colleagues used labelled carbon and nitrogen to demonstrate these transfers. Some orchids remain dependent on fungal carbon, so "every orchid always gives sugar to its fungus" is false. Source: the Goodyera transfer study.

In many reef-building hard corals, symbiotic photosynthetic dinoflagellates live within coral tissues. The coral receives organic products of photosynthesis; the symbionts receive a habitat and access to carbon dioxide and inorganic nutrients from the host's metabolism. Adequate light and suitable conditions matter. Stress can disrupt the partnership, producing bleaching; the partnership is not a guarantee against all en-

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vironmental damage. In every example, state what each partner receives instead of writing only "they help each other".

Introduced competitors can alter native populations

Native species occur naturally in a region; endemic species are restricted to a particular geographical area. An introduced species has been moved outside its natural range. It becomes invasive when it establishes and spreads with harmful effects; introduction alone does not guarantee invasiveness. Native and endemic do not mean the same thing.

A regional example is introduced Spartina alterniflora competing with native Scirpus mariqueter in the Yangtze estuary. A historical study combined Dongtan field observations with potted competition experiments and found greater relative competitive ability in Spartina. Its growth traits, including greater height and biomass, are consistent with advantages in capturing light and occupying space. The resident Scirpus should be described as native to the regional system, rather than automatically called endemic to one sampling plot. Salinity, elevation, disturbance and propagule supply can also affect invasion. Source: Chen and colleagues' growth and competition study.

To test competition, compare a focal species with and without the suspected competitor while controlling other conditions. In an invented pot experiment, focal plants average 12 grams alone and 8 grams with the other species: a reduction of $4/12 \times 100 \approx 33.3\%$. Replicate and randomize pots; keep focal starting density, soil, water, light and pot size comparable. A replacement series and an additive design answer different density questions. In a field removal, include a sham-disturbance control because removal can change soil or light as well as eliminate the competitor. Field co-occurrence or separation alone is weaker evidence for a specific competitive mechanism.

A chi-squared association test does not identify the cause

A **chi-squared test** can assess association between presence/absence categories. Record A and B in randomly selected independent quadrats. Each quadrat belongs to one of four categories; it is counted once. An invented survey gives:

Species A	B present	B absent	Row total
Present	25	15	40
Absent	15	25	40
Column total	40	40	80

The null hypothesis is that the species' presence/absence is independent in the sampled habitat. Under independence, each expected count is its row total multiplied by its column total and divided by the grand total: $40 \times 40/80 = 20$. Use the four observed category counts, not the row and column totals again, in:

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$$\chi^2 = \sum \frac{(O-E)^2}{E}.$$

Each contribution is $5^2/20 = 1.25$, so $\chi^2 = 5.00$. For a two-by-two table, degrees of freedom are $(2-1)(2-1) = 1$. Using the supplied 5% critical value 3.84, the statistic exceeds the critical value, so reject independence at that significance level. The species occur together more often than the null predicts: this is positive association, not evidence that they must compete.

A shared habitat preference could cause association without either species directly benefiting the other. Negative association could arise from competition or from different abiotic preferences. Check adequate expected counts, independent sampling units and a preselected significance level. Repeated adjacent subsamples of one patch are not automatically independent sites. Failure to reject independence is not proof that no interaction exists; sample size and detection affect power. When assumptions fail, seek a more appropriate method instead of presenting an unsupported chi-squared conclusion.

Predators, resources and other consumers can regulate populations together

In a simple predator–prey model, abundant prey supports greater predator feeding, survival or reproduction. Increased predation then reduces prey growth; later prey shortage can reduce predator survival or reproduction. Predator peaks may follow prey peaks because population responses take time. Low prey density can reduce encounters and permit recovery. These links can generate cycles, but neither perfectly regular cycles nor one fixed delay are guaranteed.

A real example is the snowshoe-hare system in the Yukon boreal forest. Hares interact with lynx and other predators, and the system shows roughly decadal fluctuations. Krebs and colleagues manipulated food supply and mammalian predator access on large forest blocks. Predator exclusion and food addition each raised hare density, while the combined treatment had a larger effect than either alone. This supports interacting food and predation effects; a hare–lynx graph alone does not establish that lynx are the only cause. Source: the Kluane field experiment.

Top-down control starts with consumers influencing the populations below them, for example more effective predators reducing herbivores and thereby allowing plants to increase. Bottom-up control starts with resources or producers, for example increased limiting nutrients increasing plant production and potentially supporting more herbivores. Energy and material still enter through the actual food web; “top-down” does not reverse a feeding arrow. Both routes can operate, with one more influential in a particular community or period. Food addition is not the same treatment as nutrient addition to soil, and organisms do not always respond identically to the two.

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Released chemicals can inhibit competitors

Allelopathy involves chemicals released by an organism affecting other plants; inhibitory effects can reduce potential competitors. Sorghum bicolor root hairs release sorgoleone into the surrounding soil. It can inhibit competing plant growth, including effects on photosynthetic processes. Soil persistence, concentration, degradation and contact influence the outcome. An extract inhibiting seedlings in a dish does not by itself establish how much field suppression was caused by that chemical rather than shading or nutrient competition. A sorgoleone soil study demonstrates why chemical fate matters.

Antibiotic release can also inhibit potential microbial competitors. Penicillin-producing Penicillium rubens is a specific fungal example; penicillin interferes with wall synthesis in susceptible growing bacteria. The producer is a fungus, the target is a bacterium and the affected process is not a human cell wall. Not every surrounding species is susceptible, and laboratory inhibition is not proof that the same concentration occurs everywhere in soil. The modern identity of important penicillin-producing strains is documented by Houbraken and colleagues.

Exam tips

- Define the target area, counting unit, period and sampling method before calculating an abundance estimate.
- Keep quadrat count, count per square metre and total population distinct; distinguish standard deviation from standard error.
- Explain the marked-fraction model and the direction of a violated mark–recapture assumption.
- Distinguish a growth curve from an explanation of its cause. Carrying capacity can change, and an ecological sigmoid does not require a flat lag phase.
- For chi-squared work, show observed and expected counts, the statistic, degrees of freedom and the supplied decision rule; association does not prove competition.

Practice

All numerical datasets below are invented; no real-study result is being reproduced.

1. Define a population and distinguish population, community and ecosystem. Explain why a study of one lawn should specify species, area, period and what counts as an individual.
2. Six 0.50-square-metre quadrats contain 2, 4, 6, 3, 5 and 4 plants in a 300-square-metre habitat. Calculate mean count, density and estimated total population. Explain random-coordinate selection, boundary rules, empty quadrats and two limitations.
3. Two equal-area samples give counts 2, 4, 6 and 4, 4, 4. Both means are 4. Given sample standard deviations 2 and 0, interpret their difference. For the first sample

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calculate $s/\sqrt{3}$ and explain why this is not the standard deviation itself or automatically a 95% interval. Design a transect to investigate light-related change.

4. Eighty animals are marked. A later catch contains 50 animals, including 20 with marks. Estimate population size. If five marked recaptures lose their visible marks, calculate the estimate using the observed marked count and explain the bias. State three further assumptions.
5. A population starts at 150. During one interval there are 40 births, 18 deaths, 12 immigrants and 9 emigrants. Find the final population. Explain how competition and pathogen transfer can produce density-dependent feedback, and contrast a density-independent disturbance.
6. A model population starts at 8 and doubles every two days. Predict its sizes at days 2, 4 and 6. Explain the expected appearance on a logarithmic population axis. Contrast later sigmoid slowing, turnover at carrying capacity and the claim that every S curve must begin with a flat lag phase.
7. Plan a replicated duckweed growth investigation, including a suitable count, conditions to control, independent replicates and model testing. Explain why rapid growth followed by a crash in a real animal population does not demonstrate smooth logistic regulation or reveal an exact permanent carrying capacity.
8. Give real examples of intraspecific competition and cooperation, then explain why they can occur within the same population. Classify caterpillar–plant, lynx–hare, tick–mammal and disease-causing bacterium–host interactions, stating their effects.
9. Explain the benefit to each partner in legume nodules, a demonstrated adult green orchid–fungus association and a hard-coral photosymbiosis. Explain why the orchid example should not become a claim that all orchid life stages supply carbon to fungi.
10. Distinguish native, endemic, introduced and invasive. Explain the Yangtze-estuary example. A focal species averages 15 grams alone and 9 grams with a second species; calculate the reduction and design laboratory and field comparisons that can test competition.
11. In 100 independent quadrats, counts are: both A and B present 15; A only 35; B only 35; neither 15. Build the two-by-two table and calculate all expected counts under independence. Calculate χ^2, degrees of freedom and the decision at the supplied 5% critical value 3.84. State the direction of association and why the result does not prove competition.
12. Explain a possible delayed predator–prey feedback. Use the Yukon hare experiment to distinguish evidence for food and predation effects from a claim that one predator alone causes every cycle. Contrast top-down and bottom-up control with a causal chain for each.
13. Compare sorgoleone release by sorghum with penicillin release by Penicillium. Identify producer, potential competitor, chemical and effect, and propose controls needed before attributing a field pattern to chemical interference.

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Worked answers

1. A population is an interacting same-species group in a defined area; sexually reproducing members normally breed. A community includes interacting populations of different species; an ecosystem includes the community and abiotic environment. Specify species, space and time to define the target, and distinguish shoots from genetic individuals where needed. Movement can connect nearby populations, so a survey boundary is not necessarily reproductive isolation.
2. Total 24; mean $24/6 = 4$ per quadrat; density $4/0.50 = 8$ plants per square metre; abundance estimate $8 \times 300 = 2{,}400$. Generate coordinates before inspecting abundance, include zero counts and use a fixed rule for boundary individuals. Patchiness, inadequate sample size, missed organisms and unrepresentative excluded areas can affect the estimate. The total sampled area is three square metres, not six square metres.
3. Counts 2, 4, 6 vary around their mean, while the three equal counts show no observed spread; neither sample proves whole-habitat uniformity. First-sample standard error $2/\sqrt{3} \approx 1.15$ plants per quadrat estimates uncertainty in the mean under independence. It differs from between-quadrat standard deviation 2 and needs further assumptions and an appropriate multiplier for an interval. Place a transect along a defined light gradient, sample fixed intervals, measure light consistently and use additional independent transects.
4. $\widehat{P} = 80 \times 50/20 = 200$. With only 15 visible marked animals, $80 \times 50/15 \approx 267$, an overestimate from the smaller denominator. Require little demographic change, adequate mixing and comparable capture probabilities; marks should be retained, recognized and harmless. A sample contains marked plus unmarked animals, and the estimate is not exact simply because it is an integer.
5. Net change $40 - 18 + 12 - 9 = 25$; final $150 + 25 = 175$. Greater density can intensify resource competition and contacts transmitting some pathogens, reducing survival or reproduction and opposing further growth. Lower density can ease these pressures. A severe frost can act independently of density, although its total deaths still depend on how many individuals were present. Delays and changing conditions can prevent smooth regulation.
6. Days 2, 4 and 6: $8 \times 2 = 16$, $8 \times 4 = 32$, $8 \times 8 = 64$. Equal ratios at equal intervals give a straight line on a logarithmic size axis with ordinary time. Increasing limitations slow net growth toward a conditional carrying capacity; births and deaths can continue while net change is zero. A flat lag phase is not required by the ecological sigmoid model, and indefinite exponential growth is not justified.
7. Use approved duckweed in several independent matched vessels; count defined fronds at fixed times with starting number, medium, light, temperature and vessel area controlled. Repeated counts of one vessel are not independent vessel replicates. Plot observed growth and an early logarithmic graph, then assess later departures and plausible resource limits. Real animal crashes can involve resource depletion, weather and response delays; a peak is not an exact permanent carrying

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capacity.

8. Male red deer can compete for mates and trees for light; wolves can cooperate in hunting or penguins in huddling. Shared hunting and competition over food can coexist. Caterpillar–plant is herbivory, lynx–hare predation, tick–mammal parasitism and a disease-causing bacterium–host pathogenicity. The consumer or parasite benefits while imposing a cost on the plant, prey or host; categories can overlap, especially parasitism and disease.
9. Nodulating bacteria supply fixed nitrogen compounds; the legume supplies carbon/energy and a suitable habitat. In adult green Goodyera, fungal nutrient transfer supports the orchid while plant photosynthetic carbon reaches the fungus. Orchid seedlings and some adults depend on fungal carbon, so reciprocal carbon supply is not universal. Coral hosts receive photosynthetic organic products; their symbionts gain a tissue habitat, carbon dioxide and inorganic nutrients. Name both directions rather than calling every association beneficial by definition.
10. Native means naturally present regionally; endemic means geographically restricted; introduced means moved beyond natural range; invasive adds establishment/spread with harm. Introduced Spartina competes with native Scirpus in the regional example. Reduction $(15-9)/15 \times 100 = 40\%$. Randomize replicated pots, standardize focal density and conditions and include focal plants alone. Field removals need sham-disturbance controls and suitable replication; habitat differences or density changes can otherwise confound the conclusion.
11. The table is $15, 35$ in the A-present row and $35, 15$ in the A-absent row. Row and column totals are each 50; grand total 100. Every expectation is $50 \times 50/100 = 25$. Four contributions of $10^2/25 = 4$ give $\chi^2 = 16$. Degrees of freedom 1; $16 > 3.84$, so reject independence at the stated level. Co-occurrence is less common than expected: negative association. Competition is one possible explanation; different habitat preferences or detection differences can also cause it. Check independent sampling and adequate expectations.
12. More prey can support predator feeding/reproduction; delayed predator increase can reduce prey growth, and later prey scarcity can reduce predators. Low predation may then permit recovery. Yukon food and predator manipulations support interacting effects rather than a single lynx-only explanation. Top-down: more effective predators → fewer herbivores → reduced plant consumption. Bottom-up: more of a limiting resource → greater plant production → potentially greater herbivore support. The dominant route depends on community and conditions.
13. Sorghum releases sorgoleone from root hairs, potentially inhibiting competing plants; Penicillium rubens releases penicillin, inhibiting cell-wall synthesis in susceptible bacteria. Both can affect competitors chemically, but their producers, targets and mechanisms differ. Compare suitable chemical-present/absent treatments with solvent, nutrient, pH, light, density and disturbance controls, and measure realistic environmental concentrations. A strong extract effect alone does not establish the cause of a field distribution.

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Transfer of energy and matter

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A fallen leaf disappears into the soil, but the tree does not recover the sunlight that originally helped make it. Its carbon atoms can return to new leaves. The energy supporting the organisms that break it down eventually spreads into the surroundings as heat. Following the atoms and following usable energy therefore require different diagrams.

This handout covers all shared C4.2 content: open systems, energy sources, feeding relationships, nutrition, respiration, production and energy pyramids, carbon transfers, carbon sinks and sources, combustion and the Keeling Curve. The content is common to SL and HL. Prerequisites: photosynthesis, respiration, organic molecules, percentages and graph scales.

Numerical examples are invented unless a published dataset is explicitly identified. They are original teaching examples, not real examination questions. Draw a boundary and state the period before comparing any ecological budget.

Ecosystems exchange energy and matter across their boundaries

An ecosystem includes a community and its abiotic environment. A pond can receive sunlight, rain, mineral ions, wind-blown leaves and incoming animals. Heat, water, dissolved substances and migrating organisms can leave. It is an open system because both energy and matter can cross the chosen boundary. A drawn boundary makes a budget possible; it does not make the ecosystem physically sealed.

A closed system exchanges energy but not matter with its surroundings. A sealed transparent vessel can approach this model while still receiving light and transferring heat. A lid alone does not stop energy exchange. An isolated system exchanges neither energy nor matter; that is a different idealization. Decide whether the boundary concerns one organism, a pond, a catchment or the whole biosphere.

For an invented pond carbon budget, annual inputs are 120 kilograms of carbon and outputs 95 kilograms. With all transfers included, the stored amount increases by 25 kilograms. A large internal transfer from algae to grazers does not itself add carbon to the whole pond: it moves carbon between stores already inside the boundary. Do not add internal arrows again as external inputs.

Sunlight sustains most ecosystems by supplying energy to photosynthetic producers. A dark cave may instead receive leaves, wood or animal waste whose energy was originally captured outside it. A deep-sea community can receive sinking organic matter or depend on local chemosynthetic production. Darkness does not by itself

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prove that an ecosystem is independent of sunlight. Identify the actual input rather than assuming that every dark habitat has the same energy source.

Autotrophs and heterotrophs obtain carbon in different ways

Autotrophs build organic carbon compounds from simple inorganic carbon sources, such as carbon dioxide. Carbon fixation and the anabolic construction of larger molecules require energy. Photoautotrophs use light; green plants, algae and cyanobacteria are examples. Plants also need water and mineral nutrients: light is an energy source, not a replacement for matter.

Chemoautotrophs obtain energy from chemical oxidation reactions and use it to build organic carbon compounds. Iron-oxidizing bacteria provide a concrete example: oxidation of reduced iron, Fe(II), to Fe(III) can supply energy for carbon fixation. The iron is an energy-linked electron donor, not the carbon atoms of the resulting sugar. Not every iron-associated bacterium is necessarily autotrophic; identify the organism's carbon and energy sources.

Heterotrophs obtain organic carbon compounds made by other organisms. A consumer may digest food internally; many fungi release enzymes outside the body and absorb the smaller products. **Assimilation** incorporates absorbed material into the organism's own usable compounds and tissues. Amino acids from food can be used to build different proteins; a consumer does not simply preserve every ingested molecule unchanged.

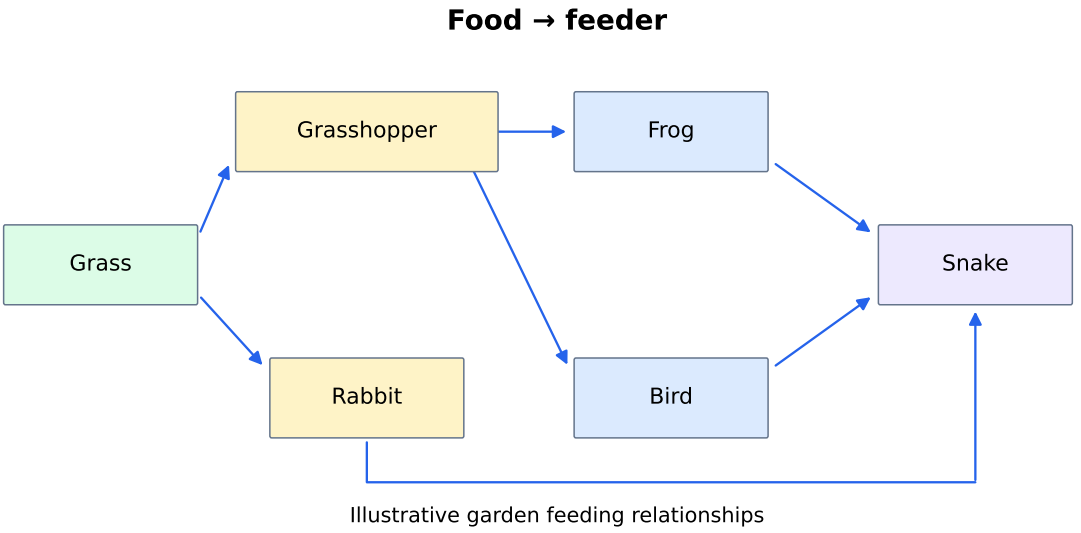
Both autotrophs and heterotrophs respire. Oxidation of organic carbon compounds releases energy that can be transferred to ATP and then used for cellular work. A plant uses some of its photosynthetic products in its own respiration; it is not a producer that only photosynthesizes and never uses energy. Neither respiration nor ATP use is completely efficient at retaining energy for further biological work, so heat is released during these transformations.

Food-chain arrows follow energy and biomass toward the feeder

A food chain is one feeding route through a community. In an illustrative school-garden chain, grass → grasshopper → frog → snake means that grasshopper feeding transfers grass material to the grasshopper, and frog feeding transfers grasshopper material to the frog. The arrow points toward the receiver of energy and biomass. It does not point toward what the predator wishes to attack.

A producer makes organic material from inorganic carbon using an external energy source. A primary consumer feeds on producers, a secondary consumer on primary consumers and a tertiary consumer on secondary consumers. These are positions in a particular chain. An omnivorous bird eating seeds is a primary consumer on that route but a secondary consumer when it eats a herbivorous insect. A species does not always have one fixed trophic position in a food web.

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Each arrow means transfer from food to feeder. This is an invented set of feeding relationships to practise interpretation, not a measured local survey.

To construct a local web, first identify organisms and reliable evidence of their diets. Draw only supported feeding links; shared habitat alone does not establish feeding. Connect several chains while keeping the same arrow convention. A missing link may reflect incomplete observation. A web shows possible routes, not the amount or rate of every transfer unless quantities are supplied.

Detrital pathways receive material from every trophic level

Dead whole organisms, fallen leaves, shed parts and faeces contain organic carbon compounds with chemical energy. Detritus feeders ingest dead organic material; decomposers such as many bacteria and fungi digest material and absorb products. These roles can overlap in a decomposition community, but an earthworm feeding on litter is not identical to a fungus secreting enzymes onto it.

The dead material can come from producers, herbivores and predators. Decomposers therefore should not be drawn only after the final predator as if everything must pass through the same long chain. A large part of primary production can enter detrital routes without ever being eaten by a herbivore.

Decomposers use absorbed organic compounds for respiration, growth and reproduction. Their respiration transfers energy as heat and releases carbon dioxide. Breakdown and subsequent mineralization can return nutrients in forms producers can take up. Decomposers recycle matter; they do not recycle the heat back into sunlight or manufacture an unlimited energy supply.

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Production is a rate of new biomass, not a standing stock

Primary production concerns organic carbon compounds made by autotrophs. Gross primary production, GPP, is the total rate of fixation into organic compounds. Producers use some of that material in respiration. Net primary production, NPP, is what remains after their own respiratory use:

$$\mathrm{NPP} = \mathrm{GPP} - R_{\mathrm{producers}}.$$

For invented annual energy equivalents of 18,000 kilojoules fixed per square metre and 8,000 used in producer respiration, NPP is 10,000 kilojoules per square metre per year. Use NPP when the question concerns new producer biomass potentially supporting consumers and detrital routes. Do not subtract consumer respiration from GPP and call the result NPP.

Secondary production is the formation of new biomass by heterotrophs through growth and reproduction. It is not all the energy a consumer ingests or all the organic material it absorbs. Production includes new tissue that may later be eaten, exported or die during the measuring period; a start-to-end standing-stock difference alone can underestimate it.

Carbon-based production can be stated in grams of carbon per square metre per year. Energy-based production can be stated in kilojoules per square metre per year. A standing biomass value, such as grams per square metre on one date, lacks the time denominator. Mass of carbon, total dry mass and energy are also different quantities. Converting between them requires a stated carbon fraction or energy content, not merely changing the unit label.

In an invented algal population, standing biomass is 20 grams of carbon per square metre, while annual production is 400 grams of carbon per square metre per year. The production-to-stock ratio is 20 per year. Rapid replacement can support high annual production even when little biomass is present at any instant. A small standing stock is not evidence of negligible energy flow.

An energy budget separates ingestion, assimilation and production

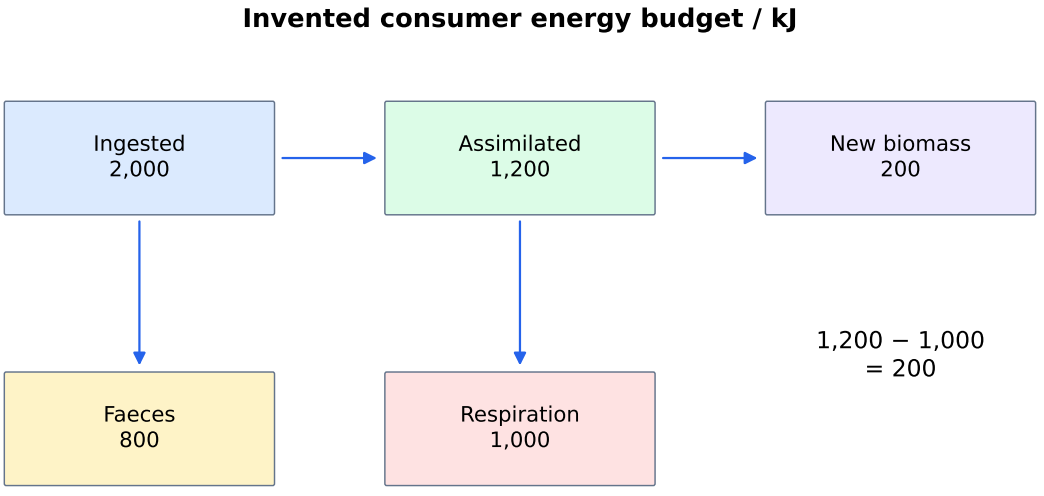
Consider one invented herbivore budget over a fixed area and year. Ingestion is 2,000 kilojoules. Faeces contain 800 kilojoules of unassimilated material, leaving 1,200 assimilated. Respiration accounts for 1,000, leaving 200 as new biomass. For this deliberately simplified budget, other organic losses are taken as zero:

$$A = I - F, \qquad P = A - R.$$

Here I is ingestion, F unassimilated faecal loss, A assimilation, R respiratory use and P secondary production. If a real budget includes energy-containing excretory losses,

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include those in the appropriate balance as well. Egestion and excretion are different processes.



All figures concern the same area and year. Arrows show a balance, not widths proportional to energy. Faecal material retains chemical energy for detrital pathways.

The assimilation efficiency is $1200/2000 \times 100 = 60\%$. Production as a percentage of assimilation is $200/1200 \times 100 \approx 16.7\%$. Production as a percentage of ingestion is $200/2000 \times 100 = 10\%$. These different percentages answer different questions. Always name the numerator and denominator.

If the producer NPP supporting this herbivore pathway is 10,000 kilojoules in the same area and year, the herbivore's 200 kilojoules of production is only 2% of it. The 10% production-to-ingestion value is not a 10% producer-to-herbivore transfer efficiency. Uneaten producer material can remain, die, be exported or support other routes; it was not automatically all respired by this herbivore.

Energy pyramids compare matched production flows

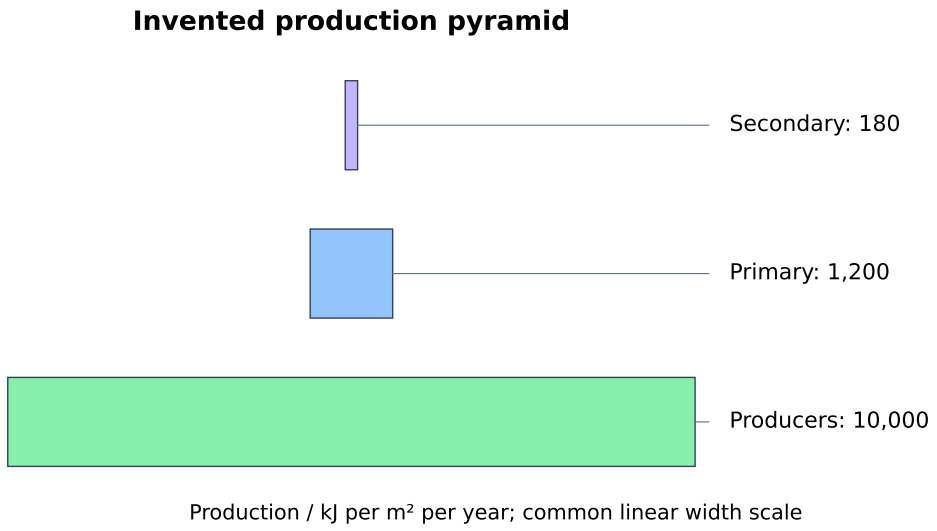
Trophic transfer efficiency compares production at one trophic level with production at the preceding level for a matched area and interval:

$$\text{transfer efficiency} = \frac{\text{production at the receiving level}}{\text{production at the preceding level}} \times 100\%.$$

In a separate invented chain, annual production is 10,000 kilojoules per square metre for producers, 1,200 for primary consumers and 180 for secondary consumers. The first transfer is 12%; the next is 15%. Across both steps, the secondary-consumer

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production is $180/10000 \times 100 = 1.8\%$ of producer production. Multiply the fractions, $0.12 \times 0.15 = 0.018$; do not add the percentages.



The narrow upper bars follow a common linear width scale. Labels are placed outside so that low production remains readable. All values are kilojoules per square metre per year.

Construct a pyramid with producers at the base and successively higher consumers above. Use a common scale, consistent height and width proportional to the production value. Label trophic groups, values, units and interval. A logarithmic width scale would change the meaning of the rectangle areas; do not secretly use one to make the upper bars wider.

Research data need their original definitions. In Odum's historical Silver Springs study, Figure 7 and its notes report rough annual production estimates for the Florida spring community. Three consumer groups have production estimates of 1,478, 67 and 6 kilocalories per square metre per year, respectively for herbivores, carnivores and top carnivores. Plot these three labelled levels on a common linear scale: each successive value is smaller. The latter two production ratios are about 4.53% and 8.96%, not a fixed 10%. These are estimates in a complex open food web, not measurements of one isolated three-species chain. The paper distinguishes production from consumer intake; its larger intake values should not be relabelled as new biomass production.

Energy transformations limit the support for higher consumers

Not all organisms or body parts are eaten. Some consumed material is not digested or assimilated, and organisms use assimilated compounds in respiration rather than retaining all of them as new biomass. Heat is released in ATP production and use. Dead material and faeces can still support decomposers, so energy absent from the next grazing level is not necessarily already heat.

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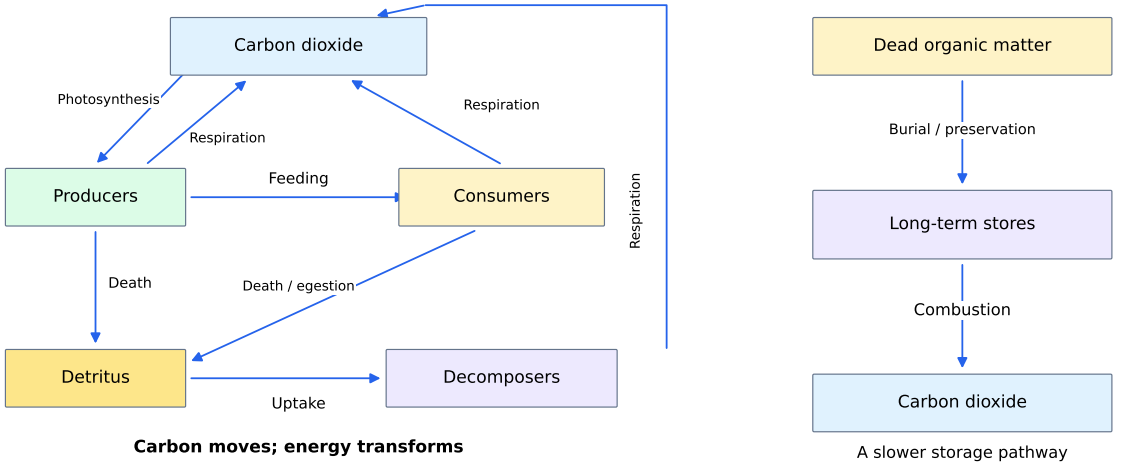
Energy is conserved through these transfers. Calling it “lost” means lost from a specified biological production pathway or dispersed as heat unavailable to drive that pathway under normal ecosystem conditions. It does not mean that energy has been destroyed. Nutrient atoms can be reused, but continued biological work requires a continuing supply of usable energy.

These transfers generally leave less production available to support successively higher trophic levels. Food chains therefore cannot usually support unlimited levels. In a hypothetical chain starting with 10,000 energy units and retaining 10% as next-level production at each step, successive values are 1,000, 100, 10 and 1. This is a model illustrating compounding, not a universal measured efficiency.

Do not infer that every predator is smaller than its prey or that its tissue must contain less energy per gram. A few large predators may be supported by many smaller prey. Biomass snapshots can be inverted, for example when rapidly replacing phytoplankton support a larger standing consumer stock. A matched production-energy pyramid for the same supporting pathway remains upright; imported food or mismatched intervals require a wider boundary before comparison.

Carbon-cycle arrows connect stores through named processes

A carbon-cycle diagram follows carbon atoms, rather than treating carbon and energy as the same substance. Photosynthesis transfers carbon from atmospheric or dissolved carbon dioxide into organic compounds in producers. Feeding transfers organic carbon into consumers. Death and egestion transfer it to detritus. Decomposer uptake builds decomposer biomass, and respiration by producers, consumers and decomposers returns carbon dioxide.



Boxes are carbon stores and arrows are transfers. Detritus is organic matter, while carbon dioxide is an inorganic carbon source. Decomposition includes both incorporation into decomposers and respiratory release.

Aerobic respiration depends on oxygen released by oxygenic photosynthesis. In turn,

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respiration supplies carbon dioxide that can be fixed again by photosynthesis. These large exchanges link autotrophs and heterotrophs. Oxygenic photosynthesis releases oxygen from water; it does not simply remove carbon from carbon dioxide and release the remaining oxygen atoms as its oxygen gas.

Some dead organic matter accumulates when production exceeds decomposition, especially under conditions restricting breakdown. Peat can accumulate in waterlogged, oxygen-poor conditions. Over geological times, buried organic matter contributed to fossil carbon stores, including coal, oil and natural gas; their origins are not all the same organisms or formation conditions. Fossil fuels are not formed from the previous year's ordinary leaf fall on a human timescale.

Combustion of biomass, peat and fossil fuels oxidizes carbon compounds, releasing carbon dioxide and transferring energy, including heat. Lightning can start natural fires. Human activities greatly increase combustion and can release old carbon stores much faster than their replacement. Burning recent biomass also releases carbon dioxide immediately; whether regrowth later balances that release depends on the boundary, time interval and actual subsequent growth.

A carbon sink is defined by net transfer over a stated interval

A carbon store is an amount present. A carbon sink takes up more carbon than it releases over the stated interval; a carbon source releases more than it takes up. A large forest carbon stock is not automatically a large current sink. Fire, drought, tree growth, decomposition and exports can change the balance.

For a simplified ecosystem exchanging carbon dioxide through photosynthesis and respiration only, compare total photosynthetic fixation with respiration by the whole community. If fixation is 900 grams of carbon per square metre per year and producer respiration is 350 while heterotrophic respiration is 400, net uptake is 150. NPP is $900 - 350 = 550$, but the whole-community balance is $900 - 350 - 400 = 150$. Using producer respiration alone would exaggerate the ecosystem sink.

If community respiration instead totals 980 with the same fixation of 900, the ecosystem is a net source of 80 grams of carbon per square metre per year. In a more complete open-system budget, include fire, dissolved carbon, harvest, imports and other transfers. A photosynthesis-minus-respiration result alone does not prove that the total carbon stock increased by the same amount when these additional fluxes are important.

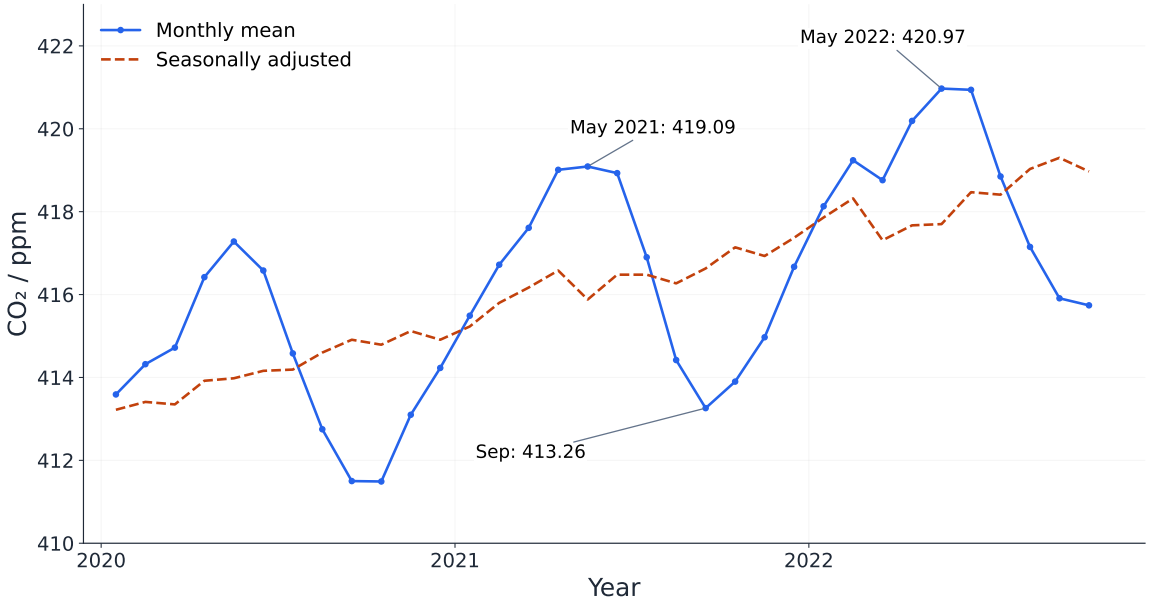
Recycling applies to every chemical element organisms need, not just carbon. Decomposers help return mineral nutrients; producers incorporate them into new biomass, and feeding moves them onward. Local recycling need not be complete: runoff, harvest and movement can remove elements, while weathering or external inputs replace them. Detailed nitrogen-cycle pathways are not required for this topic, but energy must never be listed as another chemical element being recycled.

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The Keeling Curve contains a seasonal cycle and a long-term rise

The atmospheric carbon-dioxide record at Mauna Loa shows repeated within-year changes superimposed on a longer rise. The curve is a concentration record, not a graph of the number of factories or a direct measurement of one forest's photosynthesis. Parts per million, ppm, expresses the atmospheric mole fraction here: about 419 parts of carbon dioxide per million parts of dry air means roughly 0.0419%, not 419%.

NOAA's monthly dataset gives Mauna Loa monthly means of 419.09 ppm in May 2021 and 413.26 ppm in September 2021. The fall is 5.83 ppm. May 2022 is 420.97 ppm, 1.88 ppm above May 2021. The seasonal decline and year-to-year rise coexist; one summer's fall does not demonstrate reversal of the long-term trend.



Blue values are published monthly means, not invented measurements. The dashed orange series has the average seasonal cycle removed by NOAA. The record spans part of three historical years, not the present-day concentration.

Strong Northern Hemisphere land-plant growth in spring and summer increases carbon uptake through photosynthesis. Later reduced photosynthesis, together with continuing plant and decomposer respiration, contributes to the seasonal return. The Northern Hemisphere's large land area and vegetation help explain the strong seasonal influence on this record. Plants do not stop respiring during the growing season; the balance changes. Scripps explains the seasonal pattern and fossil-carbon evidence.

The long-term rise reflects carbon inputs exceeding removals from the atmosphere, with fossil-fuel combustion a major driver and land-use changes also important. Natural respiration is a huge flux, but a large two-way natural exchange is not the same as the extra net input from releasing fossil carbon. Compare equivalent months or

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use an appropriately adjusted trend before describing the underlying rate. A graph's shape alone does not identify every cause: combine it with measurements of emissions, isotope composition and ecosystem/ocean exchanges.

Scientific laws or useful generalizations describe reproducible patterns and can support predictions; explanatory theories account for mechanisms and evidence. Neither is just an untested guess, and a theory does not become a law by collecting enough approval. Energy conservation is a robust principle; a simple 10% trophic rule is a rough model whose usefulness depends on the question and data. State assumptions, scope and exceptions, then compare predictions with observations.

Exam tips

- Draw the boundary, identify each store and distinguish external inputs from internal transfers.
- Name carbon and energy sources separately; producers respire and decomposers receive material from many levels.
- Draw food-web arrows from food toward feeder and carbon-cycle arrows with a named process.
- Distinguish standing biomass from production, and ingestion from assimilation and new biomass.
- Write the efficiency ratio with matched units, area and period. Do not assume every transfer is 10%.
- Compare total community respiration with fixation for a simplified ecosystem carbon balance; include other fluxes when relevant.
- Explain seasonal and long-term carbon-dioxide patterns separately, using dates, units and qualified causal evidence.

Practice

All numerical datasets below are invented, except the explicitly identified NOAA comparison in question 12.

1. A pond receives 180 kilograms of carbon and exports 145 in a year. Assuming all external transfers are included, calculate its storage change. Explain why transfer of 60 kilograms from algae to grazers must not be counted as another external input. Distinguish open, closed and isolated systems.
2. Compare the carbon and energy sources of a green alga, an iron-oxidizing chemoautotrophic bacterium and a fungus. Explain why an unlit cave can still depend on sunlight captured elsewhere and why producers must respire.
3. Construct a food web for these stated links: grass feeds grasshoppers and seed-eating birds; grasshoppers feed frogs and birds; frogs and birds feed snakes. State the bird's trophic levels on two routes, identify a tertiary-consumer route for the snake and explain what the arrows mean.

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4. Explain how dead leaves, faeces and a dead predator can each supply decomposers. Distinguish a detritus feeder from an externally digesting fungus, and explain why this is matter recycling without energy recycling.
5. Annual GPP is 24,000 kilojoules per square metre, and producer respiration is 9,000 in the same rate units. Calculate NPP. A producer stock is 30 grams of carbon per square metre and annual production 450 grams of carbon per square metre per year. Calculate production divided by stock and explain its units.
6. A consumer ingests 3,000 kilojoules in a stated period, egests 1,200 and uses 1,500 in respiration. Other organic losses are zero. Calculate assimilation, production, assimilation efficiency and production as a percentage of both assimilation and ingestion. If supporting producer NPP is 15,000 in the same area and period, calculate the production transfer percentage.
7. Production is 12,000, 1,800 and 180 kilojoules per square metre per year at three successive trophic levels. Draw a proportional energy pyramid. Calculate both step efficiencies and the producer-to-final-level percentage. Explain why adding step percentages is wrong.
8. A separate hypothetical chain begins with 50,000 energy units of producer production and has four successive transfers of 10%. Calculate production after each step. Explain how this model helps explain restricted chain length without proving that every real transfer is 10% or every predator is smaller than its prey.
9. Explain why a low phytoplankton standing biomass can support a larger zooplankton standing biomass without violating energy conservation. Identify the measurements needed for a valid production-energy comparison and two causes of energy failing to reach the next grazing level.
10. Draw a carbon cycle containing atmospheric carbon dioxide, producers, consumers, detritus and decomposers, with photosynthesis, feeding, death/egestion, assimilation and respiration correctly directed. Add a long-term organic store and combustion. Explain the oxygen–carbon-dioxide link between photosynthesis and aerobic respiration.
11. Annual carbon fixation is 1,200 grams of carbon per square metre. Producer respiration is 450 and heterotrophic respiration 600 in the same annual area units. Calculate NPP and the simplified net ecosystem carbon uptake. Then include an additional fire loss of 220 in the full budget. Explain why the whole system's sink/source conclusion changes.
12. From NOAA's historical monthly Mauna Loa data, May 2021 is 419.09 ppm, September 2021 is 413.26 ppm and May 2022 is 420.97 ppm. Calculate the within-2021 fall, the corresponding-May increase and the May 2021 concentration as a percentage of dry air. Explain the two patterns without claiming that a seasonal fall disproves a long-term rise.
13. Evaluate these claims: "Decomposers recycle energy"; "all dark ecosystems use chemosynthesis"; "burning biomass is instantly carbon neutral"; "a scientific theory becomes a law when proved". State a better explanation for each. Propose

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how a class could compare energy production data from a published ecosystem study without confusing intake, biomass stock and production.

Worked answers

1. Storage change = inputs − outputs = 180 − 145 = 35 kilograms of carbon gained. Algae-to-grazer transfer is inside the pond boundary, so adding it again double-counts existing carbon. Open systems exchange matter and energy; closed systems exchange energy but not matter; isolated systems exchange neither.
2. The alga uses inorganic carbon and light energy. The chemoautotroph uses inorganic carbon and energy associated with oxidation of reduced iron. The fungus obtains organic carbon and chemical energy from material made by other organisms, often after external digestion. Cave food can arrive as organic material made by photosynthesis elsewhere. Producers respire organic compounds to supply ATP for cellular work; autotrophy concerns carbon acquisition, not exemption from respiration.
3. Draw grass → grasshopper, grass → bird, grasshopper → frog, grasshopper → bird, frog → snake and bird → snake. The bird is primary consumer on grass → bird and secondary on grass → grasshopper → bird. The snake is tertiary on grass → grasshopper → frog → snake or the insect–bird route, but secondary on grass → bird → snake. Arrows show energy and biomass transfer toward the feeder, not predator pursuit.
4. All three materials contain organic carbon and chemical energy. Detritus feeders ingest dead material; the fungus secretes digestive enzymes and absorbs smaller products. Decomposers build biomass and respire, returning carbon dioxide and helping return mineral nutrients. Carbon and other elements can be reincorporated by producers; heat dispersed during energy transformations is not a renewable food input to the same pathway. Detrital routes receive material from every trophic level.
5. NPP = GPP − R = 24000 − 9000 = 15000 kilojoules per square metre per year. Production-to-stock ratio 450/30 = 15 per year. The time unit remains because production is a rate while stock is an amount per area. Rapid replacement means a small stock can sustain substantial annual production.
6. $A = I - F = 3000 - 1200 = 1800$ kilojoules; $P = A - R = 1800 - 1500 = 300$ kilojoules. Assimilation efficiency $1800/3000 \times 100 = 60\%$. Production/assimilation $300/1800 \times 100 \approx 16.7\%$; production/ingestion $300/3000 \times 100 = 10\%$. Trophic production transfer $300/15000 \times 100 = 2\%$. The denominators describe different stages. The simplified balance assumes no additional organic loss or unmatched interval.
7. Equal-height centred rectangles have widths in the ratio 12000 : 1800 : 180 = 1 : 0.15 : 0.015, producers at the base. First efficiency $1800/12000 \times 100 = 15\%$; second $180/1800 \times 100 = 10\%$; overall $180/12000 \times 100 = 1.5\%$. Multiplying

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fractions $0.15 \times 0.10 = 0.015$ gives the overall result. Adding 15% and 10% would combine fractions of different starting amounts. Label all levels and rate units.

8. After transfers one to four: 5,000, 500, 50 and 5 energy units. Each is the preceding value multiplied by 0.10. Rapid reduction leaves limited production to support further consumers, but efficiencies differ with organisms and conditions. A few large predators can be supported by many smaller prey; decreasing total production is not a rule about every individual's size or energy per gram.
9. Rapid phytoplankton growth and replacement can sustain large annual production despite low stock on a sampling date. Measure production for matched boundaries, intervals and units, accounting for imports and relevant feeding routes. Uneaten material, unassimilated faeces and respiratory heat all reduce transfer to next-level grazing production in different ways. Energy in faeces or dead tissue may still support detrital organisms; heat is not destroyed energy.
10. Carbon dioxide → producer organic carbon by photosynthesis; producers → consumers by feeding; producers/consumers → detritus by death or egestion; detritus → decomposers by uptake/assimilation after breakdown. Respiration from every living group returns carbon dioxide. Long-term storage receives some buried organic carbon; combustion sends stored carbon toward carbon dioxide. Oxygenic photosynthesis supplies oxygen from water for aerobic respiration; respiration supplies carbon dioxide for carbon fixation. The elements and usable-energy routes must not be confused.
11. NPP $= 1200 - 450 = 750$ grams of carbon per square metre per year. Simplified net ecosystem uptake $= 1200 - 450 - 600 = 150$ in the same rate units: a sink under that restricted budget. Including fire, net storage change rate $= 150 - 220 = -70$: a net source of 70 grams of carbon per square metre per year, assuming no other external fluxes. NPP alone does not include heterotrophic respiration or fire. A store's size and its net change are different properties.
12. Fall $419.09 - 413.26 = 5.83$ ppm; corresponding-May increase $420.97 - 419.09 = 1.88$ ppm. Percentage $419.09/1000000 \times 100 = 0.041909\%$. Strong growing-season uptake by Northern Hemisphere vegetation helps explain the seasonal fall; continued respiration and reduced photosynthesis help the later return. Net atmospheric additions, including fossil-carbon release, explain the longer rise alongside exchanges with land and oceans. Compare equivalent months or adjusted values, and use other evidence to investigate causes.
13. Decomposers recycle elements; energy is transformed and dispersed as heat. Dark ecosystems may import photosynthetic material, use chemosynthesis or receive both. Biomass combustion releases carbon dioxide now; later offsets require actual regrowth over a stated boundary and time. Laws describe patterns and theories explain; theories are not promoted to laws. For published ecosystem data, read methods and definitions; record units, area, period and uncertainty. Distinguish intake, stock and new biomass production. Plot matched production on a common scale and cite the study.

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DNA replication

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A tiny biological trace may contain too little DNA to compare directly. A laboratory can copy selected DNA regions and separate the products, but neither a visible band nor a matching profile automatically identifies the person who committed an offence. The explanation begins with how a cell copies its own DNA.

This handout covers D1.1: faithful copying, semi-conservative replication, helicase and DNA polymerase, PCR, gel electrophoresis and DNA profiling. Sections marked **AHL** are for Higher Level only.

Prerequisites: DNA nucleotides and complementary bases, hydrogen and covalent bonds, powers of two and percentages. All numerical examples, gel patterns and profiles below are invented teaching examples. The linked historical experiment is published research.

DNA copying supports continuity between cells and generations

During growth, an organism needs additional cells. Tissue replacement supplies new cells as others are lost. Reproduction also requires genetic information to pass to descendants. Before a dividing cell distributes its genome, DNA must be copied so the resulting cells can receive the required sequences.

Replication produces DNA from a DNA template. The intended result is matching base sequences, not a new sequence adapted to the cell's needs. Very high accuracy preserves instructions, but copying is not infallible: errors that escape correction can contribute to mutations. Do not claim that every copied genome is guaranteed error-free.

Replication is different from transcription. Transcription makes RNA from a DNA template as part of gene expression; it does not duplicate the whole DNA molecule into two DNA molecules. Replication is also different from chromosome separation. Copying creates the DNA that will later be distributed; movement during cell division does not itself manufacture new strands.

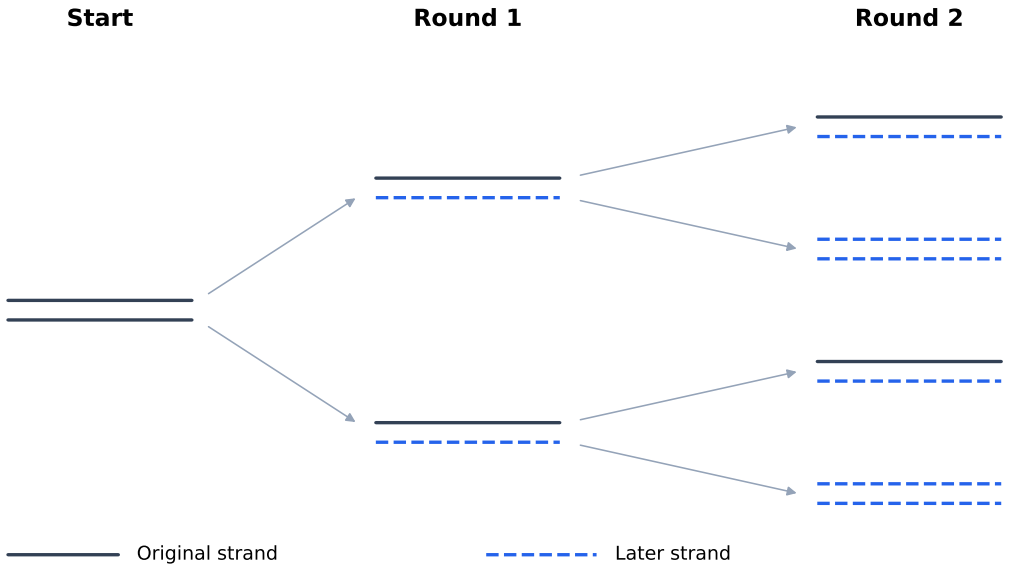
Complementary pairing makes each parental strand a template

When a double helix opens, each exposed strand supplies an ordered sequence of bases. An adenine on the template pairs with thymine in the new strand; thymine specifies adenine, cytosine specifies guanine, and guanine specifies cytosine. Free DNA nucleotides are selected through these complementary relationships and incorporated into a new strand.

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For an aligned template segment A–C–G–T–T–A, the new paired segment is T–G–C–A–A–T. These two aligned strands are complementary, not identical. A second copying event using the complementary strand can reconstruct the original sequence. Do not replace thymine with uracil: these are DNA products.

Semi-conservative replication means that each daughter double helix contains one strand from its parent molecule and one newly synthesized strand. The original double helix does not remain together beside an entirely new double helix. Nor is every daughter strand a patchwork of old and new sections in this model.



Each pair of parallel lines is one double-stranded molecule. Solid dark lines are the two original strands; dashed blue lines are later strands. Colour is not the only code.

Count molecules separately from original strands

Start with one double helix containing two labelled original strands. All subsequent nucleotides are unlabelled. Assume complete rounds, no strand destruction, no exchange of labelled material and no extra DNA entering the population.

After one round there are two molecules. Each contains one original strand. After two rounds there are four molecules: two still contain an original strand, while two contain only newly made strands. After three rounds there are eight molecules, but still only two contain an original strand.

The fraction of molecules containing an original strand after three rounds is $2/8 = 25\%$. However, eight double helices contain sixteen individual strands, of which only two are original: $2/16 = 12.5\%$. The different percentages have different denominators. A strand made in round one becomes a template in round two, but it does not thereby become an original labelled strand.

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■ Distinguish a model from experimental evidence

Meselson and Stahl grew bacteria using heavy nitrogen, then transferred them to a light-nitrogen supply. Their 1958 study separated extracted DNA by buoyant density in a caesium chloride gradient. After one generation the DNA had intermediate density; later intermediate and light components appeared, supporting conserved parental contributions to daughter molecules. Heavy nitrogen here was a stable isotope, not a radioactive tracer. This was density-gradient centrifugation, not separation of fragment lengths in a gel.

The authors explicitly discussed the limits of what their measurements established about molecular subunits. A classroom old/new-strand drawing expresses a mechanism and predicts observations; the drawing itself is not a photograph of enzyme action. See the original research paper, especially Figures 4–6 and the associated discussion.

Helicase opens the template and polymerase builds new DNA

Helicase unwinds and separates the paired DNA strands by disrupting hydrogen bonds between complementary bases. It does not normally cut each sugar–phosphate backbone into nucleotides. Keeping those covalent backbones intact preserves the ordered templates.

DNA polymerase DNA catalyses synthesis of a new DNA strand using a template and available DNA nucleotides. Complementary pairing guides which base is incorporated; the enzyme joins nucleotides into the new covalent sugar–phosphate backbone. Hydrogen bonding between paired bases and covalent bonding along a strand are different relationships.

A clear basic explanation therefore has a sequence: open the double helix, expose each template, pair appropriate nucleotides and build new strands. Saying only "the DNA splits and copies itself" omits both the template mechanism and the different enzyme roles.

PCR amplifies a selected DNA region through temperature cycles

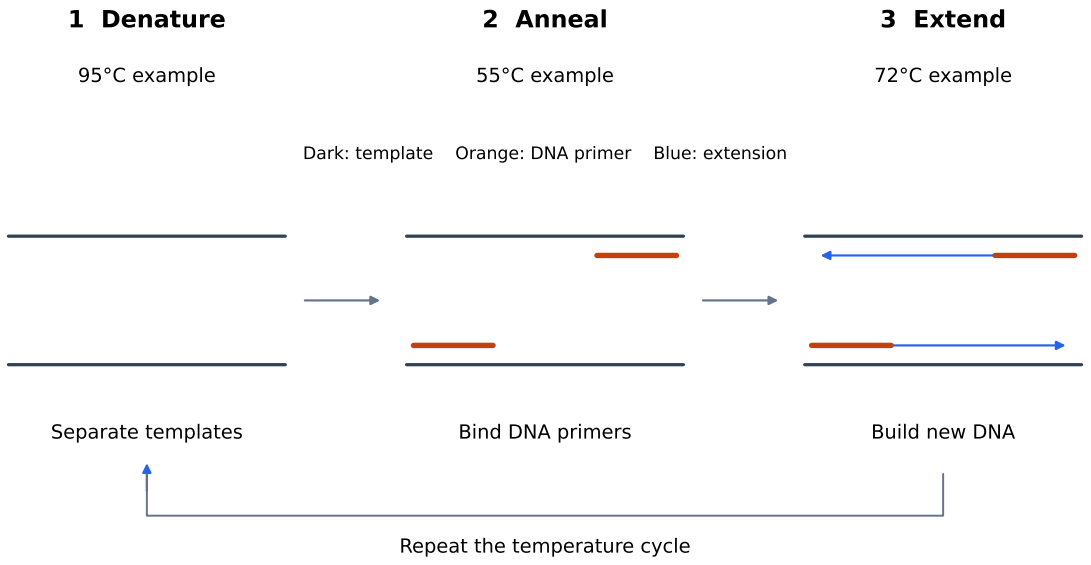
The polymerase chain reaction, PCR, makes many copies of a selected region outside living cells. It can provide sufficient material for later analysis when the starting quantity is small. PCR is amplification; it is not itself a method for sorting fragments by size or identifying a person's complete genome.

A reaction needs template DNA, two suitable short DNA primers, free DNA nucleotides, a thermostable DNA polymerase such as Taq, and an appropriate buffer including required ions. The primers pair with opposite templates at sites flanking the chosen region. Their sequences help determine what is amplified, so the same primer pair does not automatically copy every gene in a sample.

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Stage	Main event
Denaturation	Heating separates paired DNA strands
Annealing	Cooling permits primers to pair with matching sites
Extension	Polymerase extends from the bound primers

An illustrative cycle uses about 95°C for denaturation, 55°C for annealing and 72°C for extension with a suitable Taq-based protocol. These are examples, not settings guaranteed to work for every primer and enzyme. Annealing depends on primer sequences; extension conditions depend on enzyme and product length. Manufacturer optimization guidance gives ranges and protocol-specific differences.



The temperatures illustrate one suitable protocol. Heating separates the templates in PCR; the reaction does not need helicase to perform that step.

Taq is useful because it tolerates repeated high-temperature stages. This does not mean its main extension stage occurs at the denaturation temperature, or that the enzyme must be added afresh after every cycle. Primers provide the starting points; free nucleotides provide material for the new DNA. They are not interchangeable ingredients.

■ Use an ideal doubling model with stated limits

Suppose five defined double-stranded target molecules are initially present and every target is copied once per cycle. Under this simplified ideal model, the target population doubles each cycle:

$$N = N_0 \times 2^n$$

Here N_0 is the starting number, n the number of complete cycles and N the resulting

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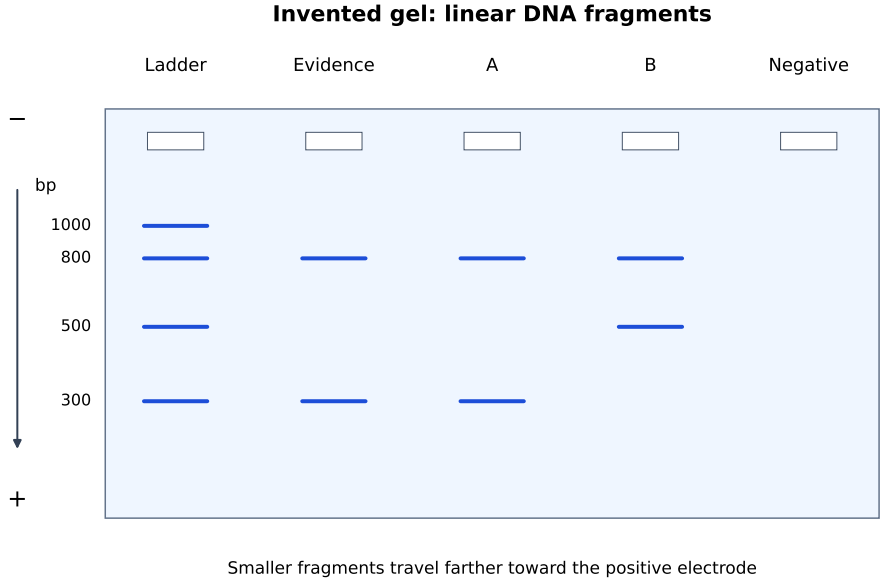
number. After ten cycles, $N = 5 \times 2^{10} = 5120$ target copies. Five additional ideal cycles multiply the current population by $2^5 = 32$, rather than adding five copies.

Real PCR does not double indefinitely. Reagents become limiting, products can interfere with the reaction, some templates fail to amplify and incorrect products can form. In the earliest cycles from longer genomic templates, not every new strand has both ends defined by the primer sites. Treat the equation as the stated ideal target-copy model, not an exact inventory of every early molecular product or a guaranteed experimental yield.

Gel electrophoresis separates DNA fragments mainly by size

DNA's phosphate backbone carries negative charge. In a suitable agarose gel and buffer, DNA moves from wells near the negative electrode toward the positive electrode. The gel acts as a molecular sieve: smaller linear double-stranded DNA fragments generally travel farther than larger ones during the same run under comparable conditions.

Load a size ladder containing fragments of known lengths beside the samples. Compare sample-band positions with the ladder to estimate fragment lengths in base pairs. A band contains many fragments of similar size, made detectable by an appropriate stain or label; it is not one visible DNA molecule or one base.



All lanes share the same run. The evidence pattern matches comparison A at these illustrated fragment sizes; comparison B differs. The negative-control lane has no visible bands.

In this invented example, the evidence and comparison A have fragments corresponding to 800 and 300 base pairs. Comparison B has 800 and 500. The shared 800-base-pair

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band alone cannot establish that B matches the full pattern. Smaller fragments appear farther from the wells; 300 base pairs is not a longer fragment merely because its band travelled farther.

Distance travelled is not generally a simple linear measure of fragment length in base pairs. Use the ladder and a suitable calibration, often involving the logarithm of fragment size, rather than declaring that twice the distance means half the base pairs. DNA conformation also matters: comparisons among supercoiled plasmids, nicked circles and linear DNA require extra care beyond this linear-fragment model.

■ Controls help distinguish a result from a failed test

A negative amplification control contains the reaction materials but no intended template. A target-sized band in it raises concern about contamination or unintended amplification. It is not an extra matching person. A positive control tests whether a known suitable template can produce the expected product under the procedure.

If every sample and the positive control lack bands, "none of these people has DNA" is not a valid conclusion. Reagents, amplification, loading or detection may have failed. If the negative control contains product, investigate before treating the sample profiles as reliable. Clean controls support interpretation but cannot eliminate every possible sampling or handling error.

DNA profiles compare inherited markers rather than whole identities

DNA profiling compares selected variable regions. At a simple autosomal marker, a person normally has two inherited alleles, one from each biological parent. Length differences at suitable markers can be amplified and compared. Actual profiling uses validated multi-marker procedures; the small examples here teach the logic, not an operational forensic protocol.

PCR supplies copies of selected marker regions. Separation reveals product sizes. The analysis then compares alleles across several markers. Equal fragment size does not prove identical base sequence throughout a fragment, and agreement at one marker does not prove that two samples came from the same person.

■ A paternity example tests inheritance at every marker

Person	Marker 1	Marker 2
Mother	12, 14	7, 9
Child	14, 16	9, 12
Candidate A	16, 18	10, 12
Candidate B	20, 22	10, 12

These numbers are invented allele labels, not the gel's base-pair lengths. At marker 1 the child's 14 can come from the mother; 16 must come from the other parent under

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the stated simple inheritance model. At marker 2 the corresponding required paternal allele is 12. A can supply both required alleles and is consistent at these markers. B cannot supply 16 and is inconsistent at marker 1, assuming correct samples, accurate measurements and no relevant mutation.

Consistency at two markers does not prove paternity. More suitable, sufficiently independent variable markers reduce the probability of an unrelated person matching by chance. Related people can share more alleles than unrelated people. An exclusion or match also depends on quality controls, mixtures, mutation and the statistical model. Do not invent a universal false-match probability from the number of bands alone.

■ A forensic match is evidence, not a complete account of events

A matching profile can support the proposition that a person contributed DNA to a trace. It does not by itself establish when the material arrived, how it was transferred or whether the person committed an offence. Mixed contributors, contamination, degraded DNA and missing alleles can complicate interpretation.

The NIST explanation of DNA mixtures shows why trace evidence needs careful interpretation. Document sample provenance and handling, compare appropriate controls and use other evidence. Increasing reliable marker measurements strengthens discrimination, but repeating the same contaminated sample is not equivalent to obtaining independent corroboration. Identical twins also show why DNA sequence similarity cannot be equated automatically with one unique identity in every context.

AHL: New DNA grows from its 5′ end toward its 3′ end

The numbers 5′ and 3′ refer to carbon positions in the deoxyribose sugar. The two strands in a double helix are antiparallel: alongside a strand running 5′ to 3′, its partner runs 3′ to 5′. These labels describe chemical orientation, not the top and bottom of the paper.

DNA polymerase extends an existing strand at its free 3′ end. An incoming DNA nucleotide triphosphate contributes its 5′ phosphate to a bond with the growing strand's 3′ hydroxyl group. Consequently, every newly synthesized DNA segment grows 5′ to 3′ while its template is followed in the opposite direction. Polymerase does not switch to building a new strand 3′ to 5′ on the other side of a fork.

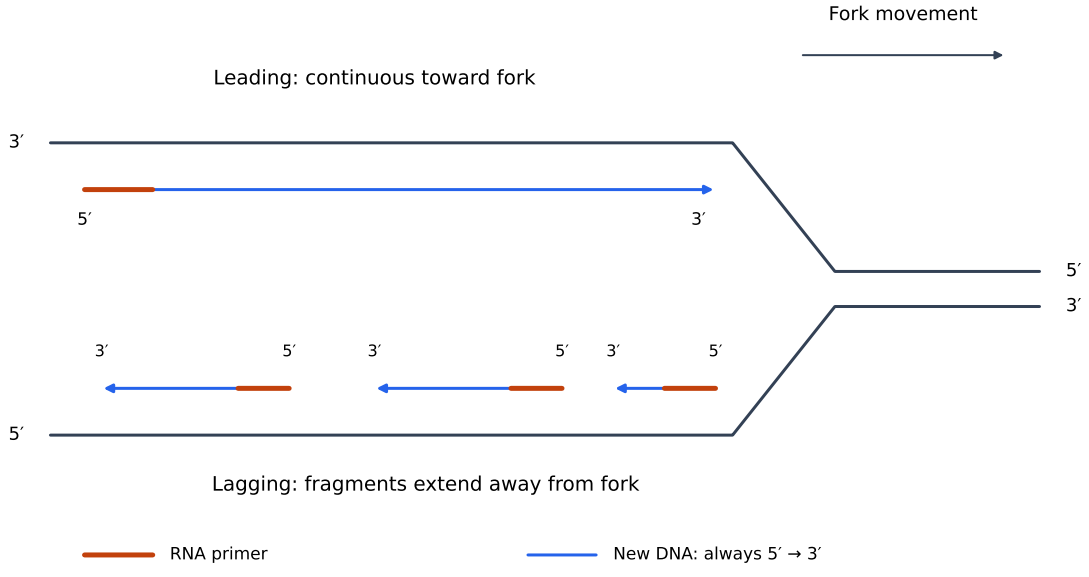
For a template written 3′-ACGTTA-5′, the aligned new strand is 5′-TGCAAT-3′. If asked to write the complement in the opposite left-to-right orientation, reverse the sequence as well as the end labels. Simply swapping 5′ and 3′ labels on unchanged letters produces a different answer.

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AHL: Fork geometry produces continuous and discontinuous synthesis

A replication fork is the region where the parental strands separate and new strands are assembled. Consider one fork moving to the right. On the parental template running 3′ to 5′ toward that fork, polymerase can extend the new leading strand continuously toward the opening region.

The opposite parental template runs 5′ to 3′ toward the fork. Polymerase must still synthesize 5′ to 3′, so it builds short sections away from the advancing fork. As more template is exposed, another section begins nearer the fork. These sections are Okazaki fragments, later joined to make a continuous daughter strand.



The diagram follows one fork, not an entire chromosome. Arrowheads on new strands show their synthesis direction; the separate fork arrow shows fork movement. Short orange segments are RNA primers.

DNA polymerase cannot start the new strand without an appropriate starting end. Primase makes a short RNA primer. In the simplified model, the leading strand needs initiation once for continuous synthesis at this fork, whereas each Okazaki fragment needs another primer. The later initiation sites lie closer to the advancing fork; each fragment still grows away from it.

The final lagging daughter is not permanently a set of disconnected pieces. Primer processing and ligation complete a continuous backbone. “Leading” and “lagging” are relative to the chosen fork and template, not permanent names for the upper and lower strands in every diagram.

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AHL: Prokaryotic enzymes perform distinct steps

The named polymerase I and III roles here describe the prokaryotic system used in this topic. Do not assign these exact enzyme names universally to eukaryotic replication.

Enzyme	Main role in this model
Helicase	Opens the paired parental strands
Primase	Makes short RNA primers
DNA polymerase III	Extends new DNA from primers
DNA polymerase I	Removes RNA primer material and replaces it with DNA
DNA ligase	Seals remaining nicks in the covalent backbone

After an Okazaki fragment is extended, its RNA primer must be replaced by DNA. Replacement nucleotides fill the primer region; ligase then seals the remaining nick between adjacent DNA sections. Ligase does not supply all the missing nucleotides, copy the template instead of polymerase, or reattach the two parental strands to each other.

Cellular primers in this explanation are RNA. Standard PCR uses supplied DNA primers. Cells use helicase to open templates, whereas PCR uses heating. Both processes exploit complementary pairing and polymerase activity, but they do not require identical ingredients or conditions.

AHL: Proofreading corrects a terminal mismatch

Complementary pairing gives high accuracy, but an incorrect nucleotide can occasionally be incorporated. DNA polymerase III can detect a mismatched base at the growing strand's 3′ terminal, remove that terminal nucleotide through proofreading and replace it with the correctly matched nucleotide before extension continues.

For example, if a template C has been paired with a newly added terminal A, the incorrect A must be removed and G incorporated in its place. Do not change the template C merely to agree with the error. Removing a wrong nucleotide is distinct from separating complementary strands with helicase or joining a backbone nick with ligase.

Proofreading reduces error frequency; it does not make mutation impossible. Other cellular repair processes also exist, but this objective concerns the terminal removal-and-replacement action of polymerase III. The direction of the removal activity does not change the 5′-to-3′ direction of subsequent DNA synthesis.

Practice

1. Explain why DNA replication is required in growth, tissue replacement and reproduction. Distinguish it from transcription and chromosome separation, and qualify the claim that copied DNA is always perfect.

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2. An aligned template segment is C–A–T–G–G–A. Write the complementary DNA segment. Explain how pairing and semi-conservative replication preserve sequence information.
3. One double helix has two labelled original strands. After five complete rounds using unlabelled material, calculate the number of molecules, the number containing an original strand, and the percentages of molecules and of individual strands carrying original material. State assumptions.
4. A student says helicase breaks covalent backbones and polymerase supplies hydrogen bonds instead of making a new backbone. Correct both statements and identify the templates and building material.
5. List the essential PCR materials. Explain the purpose of a cycle at illustrative temperatures of 95°C, 55°C and 72°C, and why primers and Taq are needed. Explain why the temperatures are not universal constants.
6. An ideal PCR model starts with two defined double-stranded targets. Calculate the number after twelve complete doubling cycles and the factor produced by five more cycles. Give two reasons a real reaction may differ.
7. In the illustrated gel, identify the electrode toward which DNA moves, the smaller of the 800- and 300-base-pair fragments, and the comparison lane matching the evidence pattern. Explain why one equal-sized band cannot establish identity.
8. A sample has no visible bands and its positive control also fails. In another run the negative control has a target-sized band. Explain the separate problems and an appropriate response before interpreting profiles.
9. In the paternity table, identify the required paternal allele at each marker. Decide which candidate is inconsistent with the simple model and explain why the other is not thereby proven to be the father.
10. A trace and a reference match at one variable marker. Explain why more markers improve discrimination and identify two reasons a multi-marker match still does not establish guilt.
11. Compare the old/new-strand model, the historical heavy-nitrogen experiment, PCR and gel electrophoresis. For each state its purpose and what it cannot establish by itself.
12. **AHL:** A template runs 3′-GACTTA-5′ from left to right. Write its aligned new DNA with end labels, then write that new strand from 3′ to 5′ left to right. Explain where a further nucleotide can be added.
13. **AHL:** Draw a right-moving fork with the upper parental template running 3′ to 5′ toward it. Label template ends, leading and lagging synthesis, RNA primers and Okazaki fragments. Explain repeated priming and distinguish primase, polymerase III, polymerase I and ligase.
14. **AHL:** Polymerase III has just added terminal T opposite a template G. Explain proofreading and replacement, distinguish this from ligation, and evaluate “the lagging strand is synthesized 3′ to 5′ and proofreading eliminates every mutation”.

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Worked answers

1. New cells and descendants need appropriate DNA sequences. Replication makes DNA from DNA before distribution; transcription makes RNA for expression, and chromosome separation distributes existing copies. Complementary pairing and correction support high fidelity, but errors can escape, so perfect copying is not guaranteed.
2. G–T–A–C–C–T. Each exposed parental strand specifies its complementary bases; polymerase builds the new strand. Each daughter molecule retains one parental strand with one newly made partner. Complementary paired sequences are not identical left-to-right sequences.
3. Molecules $= 1 \times 2^5 = 32$. Two retain one original strand, so $2/32 \times 100 = 6.25\%$ of molecules contain original material. There are 64 individual strands, of which two are original: $2/64 \times 100 = 3.125\%$. Assume complete rounds, intact original strands and no label exchange or additional labelled input.
4. Helicase disrupts hydrogen bonding between paired bases and separates the strands; their covalent backbones remain intact as templates. Polymerase incorporates complementary free DNA nucleotides into a new covalent backbone. Base pairing across strands and backbone bonding along strands have different roles.
5. Template, two suitable DNA primers, free DNA nucleotides, thermostable polymerase and suitable buffer/ions. Heating separates strands; cooling permits primer pairing; extension builds new DNA from those starting points. Taq tolerates repeated heating. Primer sequences and enzyme/product properties determine suitable conditions; 95/55/72°C is an example protocol.
6. $N = N_0 \times 2^n = 2 \times 2^{12} = 8192$ target copies. Five more ideal cycles multiply this by $2^5 = 32$. Incomplete amplification, reagent limitation, interfering products or incorrect priming can change the yield. The model assumes defined targets and complete doubling, not exact counting of all early genomic-template products.
7. Toward the positive electrode because DNA is negatively charged. The 300-base-pair fragment is smaller and travels farther in this comparable linear-fragment run. A matches the illustrated 800/300 pattern; B's 800/500 pattern differs. One band represents size agreement, not necessarily the same sequence or a unique person.
8. A failed positive control means the procedure has not demonstrated successful amplification/detection; the sample's blank cannot safely be treated as a valid negative. Product in the negative control raises contamination or unintended-amplification concerns. Investigate reagents, handling and controls and repeat appropriately before accepting the profiles.
9. The required paternal alleles are 16 at marker 1 and 12 at marker 2. B lacks 16 and is inconsistent under the stated error-free, no-mutation model. A can supply both, but other people may also have those alleles. More markers, appropriate statistics and reliable sampling are required for a strong inference.

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10. More sufficiently independent variable markers reduce a chance match because agreement must extend across more inherited variation. A match still does not establish the time or mechanism of deposition or an offence. Contamination, transfer, mixtures or sample errors can matter; profile evidence must be interpreted with controls and the case's other evidence.
11. A strand model predicts semi-conservative inheritance but is not an observation of enzymes. The historical study tested parental contributions through DNA density, not gel fragment length; its authors discussed molecular interpretation limits. PCR amplifies selected regions without proving identity. A gel separates comparable fragments by size without reading their complete sequences or proving paternity/guilt.
12. **AHL:** Aligned new DNA is 5′-CTGAAT-3′. Written in the opposite orientation it is 3′-TAAGTC-5′. Extension adds a nucleotide to the free 3′ end, joining the incoming nucleotide's 5′ phosphate to that growing end. The new strand still grows 5′ to 3′; changing the written orientation does not change its chemistry.
13. **AHL:** Upper template is 3′ left to 5′ right; its leading daughter is 5′ left to 3′ right and extends toward the fork. Lower template is 5′ left to 3′ right; lagging fragments begin with primers nearer the fork and extend 5′ to 3′ leftward. Primase supplies RNA primers, once for leading initiation and repeatedly for fragments. Polymerase III extends DNA; polymerase I replaces RNA primer material with DNA; ligase seals the remaining backbone nicks. The completed lagging daughter is continuous.
14. **AHL:** Remove the incorrect terminal T from the new strand's 3′ end and replace it with C complementary to template G, then resume synthesis. Proofreading changes the mismatched terminal nucleotide; ligase seals a nick between existing DNA sections. Every new segment grows 5′ to 3′, including Okazaki fragments. Proofreading reduces errors but cannot guarantee that no mutation ever occurs.

Exam tips

- Name the molecule and denominator: parent strand, new strand, double helix and individual strand are different counting units.
- Explain each PCR temperature change, each ingredient and each control. Amplification, separation and profile interpretation are separate steps.
- Read a gel from the wells and electrode labels; smaller comparable fragments travel farther. A marker match is not a whole-genome identity or proof of guilt.
- **AHL:** Label both strand ends before deciding leading and lagging. Keep RNA priming, DNA synthesis, primer replacement, ligation and proofreading distinct.

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D.1.2 · Protein synthesis

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

Two cells can contain the same gene but produce different amounts of its protein. Even when both make a polypeptide, only one may process it into an active product. To explain the difference, follow the information from DNA to RNA, then follow the amino acids through synthesis and maturation.

This handout covers D1.2: transcription, stable templates and gene expression; mRNA, ribosomes and tRNA; the genetic code, elongation and mutation effects. Sections marked **AHL** are for Higher Level only.

Prerequisites: DNA and RNA structure, complementary bases, amino acids, enzymes and peptide bonds. Unless identified as a named biological example, sequences, lengths and numerical data below are original, invented teaching examples.

Protein synthesis connects a gene with a polypeptide

Protein synthesis uses a base sequence to specify the order of amino acids in a polypeptide. Two processes must be distinguished. **Transcription** makes RNA using a DNA template. **Translation** uses an mRNA sequence at a ribosome to assemble amino acids into a polypeptide. DNA does not become protein, and RNA bases are not converted into amino acids.

For a protein-coding gene, transcription supplies an RNA copy of relevant information; translation interprets the coding region of that message. Other genes produce functional RNAs, such as rRNA or tRNA, rather than polypeptides. A polypeptide is a chain of amino acids; a functional protein may require folding, processing or assembly of several chains.

In eukaryotes, nuclear genes are transcribed in the nucleus and their mRNAs can be translated by cytoplasmic ribosomes, including ribosomes attached to rough endoplasmic reticulum. Prokaryotes have no nucleus, so do not draw a nuclear membrane around bacterial transcription. The two-stage information relationship remains useful in both cases.

Transcription copies one DNA template into RNA

RNA polymerase exposes a short region of DNA and uses one strand as the template for the particular transcript. Complementary RNA nucleotides pair with exposed DNA bases. DNA A pairs with RNA U, DNA T with RNA A, DNA C with RNA G and DNA G with RNA C. RNA polymerase catalyses covalent joining of RNA nucleotides into the new RNA backbone.

Hydrogen bonding helps select complementary bases across template and product. It

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is different from the covalent bonds joining adjacent nucleotides within the RNA. The RNA separates as transcription proceeds, and DNA strands can pair again. The whole chromosome need not remain permanently open.

For an aligned DNA template segment T–A–C–C–G–A–A–A–G, the complementary RNA segment is A–U–G–G–C–U–U–U–C. When this is part of the stated reading frame, its codons are AUG, GCU and UUC. Do not copy the template unchanged, substitute T into the RNA, or read DNA template triplets directly from an mRNA codon table.



Invented aligned segment; complementary RNA, then codon meanings

The invented segment is aligned for pairing. RNA bases are copied from the template relationship; amino acids are selected through the genetic code. The arrows represent information use, not conversion of DNA material into protein.

A reusable template is not consumed by transcription

Transcribing a sequence does not normally change the DNA base sequence. The nucleotides in the RNA come from the cell's available RNA-nucleotide supply; they are not pieces cut out of the DNA template. A gene can therefore be transcribed repeatedly while retaining its information.

Long-lived non-dividing somatic cells still need to maintain their DNA sequences. They can continue making RNA and proteins without duplicating the whole genome for cell division. "This cell does not divide" does not mean "this cell never expresses genes". Equally, template stability is not a guarantee that DNA damage or mutation can never occur.

Regulating transcription helps switch gene expression on and off

Cells do not use every gene at the same time. Whether a protein-coding gene is transcribed affects whether its mRNA is available for translation. A cell making more of one mRNA can often make more of the associated product, although later stages also influence protein abundance.

A pancreas beta cell and a nerve cell can share most of their genome while expressing different sets of genes. Different gene activity helps cells perform different functions; it does not require every specialized cell to possess a completely different set of protein-coding genes.

Imagine two otherwise comparable cells. Both retain an intact gene, but only cell A has a detectable transcript and product under the measured conditions. This is consistent with different gene expression, not proof that B lacks the gene. Check DNA presence, RNA detection and protein detection separately. The absence of a signal can also reflect a method's sensitivity or the sampling time.

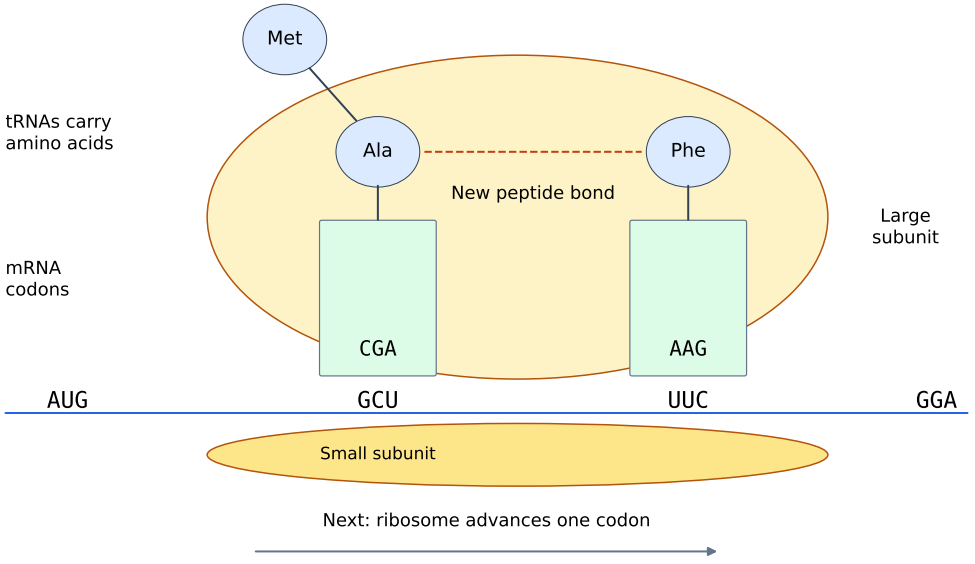
Ribosomes and tRNA connect codons with amino acids

Messenger RNA, mRNA, carries the sequence used during translation. The small ribosomal subunit binds the mRNA and helps position it for reading. The large subunit participates in the machinery that links amino acids. Two tRNAs involved in peptide-chain transfer can be bound at the same time; a ribosome does not have to release the growing chain into solution after each amino acid.

A **codon** is a group of three mRNA bases read together in a specified reading frame. A tRNA has an anticodon that pairs with a complementary codon and carries a particular amino acid when charged. The anticodon is RNA, so it also uses U rather than T. Correct matching of amino acid to tRNA is essential: the ribosome reads the pairing relationship, not a printed name tag on the amino acid.

For an mRNA codon GCU, an aligned anticodon is CGA. For UUC it is AAG. These are aligned complements; direction labels are developed in the Higher Level section. The tRNA's amino-acid attachment site is separate from its anticodon. A codon does not directly attach to a free amino acid without the adaptor relationship.

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The growing Met–Ala chain is attached to one tRNA; the next tRNA brings Phe. Chain transfer forms the Ala–Phe peptide bond. The diagram shows a stage in elongation, not a whole ribosome at molecular scale.

A triplet code offers enough combinations for amino acids

There are four kinds of RNA base. A one-base code gives only $4^1 = 4$ different signals; a two-base code gives $4^2 = 16$. A triplet code gives $4^3 = 64$, enough combinations to specify twenty standard amino acids and termination signals. "Three" refers to three bases in one codon, not three codons per amino acid.

The genetic code is degenerate: several different codons can specify the same amino acid. For example, GCU and GCC both specify alanine. This does not mean that one codon has an arbitrary choice of amino acids in the standard code. Keep several-codons-to-one-amino-acid distinct from one-codon-to-several-amino-acids.

The code is nearly universal: most organisms use the same codon meanings, reflecting shared evolutionary history. Important exceptions exist, for example in some mitochondrial codes. "Universal" in this course describes the broad shared system, not a claim that no organism or organelle ever differs.

A **stop codon** ends translation and specifies no amino acid. In the standard code the stop signals are UAA, UAG and UGA. AUG specifies methionine and commonly serves as the start signal in the appropriate initiation context. Not every AUG anywhere in an mRNA automatically begins an independent protein.

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■ Use the mRNA table with the correct strand and frame

The following excerpt contains the codons needed in this handout's examples. Read codons as mRNA, not anticodons. Use a complete supplied table when a question contains other codons. The NCBI genetic-code tables identify the standard code and documented alternatives; their DNA-style T corresponds to U in mRNA.

mRNA codon or codons	Amino acid or signal
AUG	Methionine, Met
GCU, GCC, GCA, GCG	Alanine, Ala
UUU, UUC	Phenylalanine, Phe
GGU, GGC, GGA, GGG	Glycine, Gly
GAA, GAG	Glutamic acid, Glu
GUU, GUC, GUA, GUG	Valine, Val
UAU, UAC	Tyrosine, Tyr
CUU	Leucine, Leu; other Leu codons omitted
UCG	Serine, Ser; other Ser codons omitted
GAU, GAC	Aspartic acid, Asp
UAA, UAG, UGA	Stop; no amino acid

For the invented coding sequence AUG GCU UUC GGA UAA, begin at the stated start and group successive triplets. The product is Met–Ala–Phe–Gly, then translation stops. There are five codons but four amino acids. UAA does not add an amino acid named "stop".

■ Count only the translated coding region

Suppose a coding region contains 63 nucleotides, including its single terminal stop codon, with no untranslated sequence or introns in the stated count. The number of codons is $63/3 = 21$. One is stop, so the initial polypeptide contains $21 - 1 = 20$ amino acids. A linear chain of twenty amino acids has nineteen peptide bonds before any processing.

The model can be written as $L = B/3 - 1$, where B is the coding-region nucleotide count including the terminal stop and L the initial amino-acid count. It cannot be applied to an entire gene or full mRNA without checking the included regions. Nor does it necessarily give the length of a mature protein after processing. A 90-nucleotide coding region under the same conditions gives $90/3 - 1 = 29$ amino acids.

Elongation alternates matching, peptide bonding and movement

An incoming charged tRNA pairs its anticodon with the next mRNA codon. The growing polypeptide is initially attached to another tRNA. A peptide bond forms between the chain's terminal amino-acid residue and the incoming amino acid, transferring the

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chain onto the incoming tRNA.

The ribosome then advances by one codon relative to the mRNA. The tRNA now carrying the chain occupies the position used for the next transfer; the uncharged tRNA can leave and be recharged. Repetition extends the chain in the codon-specified order. The mRNA is not consumed one codon at a time or permanently turned into the polypeptide.

For the sequence Met–Ala–Phe–Gly, forming the four-residue chain requires three peptide bonds. Adding another amino acid increases both the residue count and the number of peptide bonds by one. The precise initiation and binding-site sequence is treated in the Higher Level sections; the shared explanation focuses on how elongation links base order to amino-acid order.

Mutations can alter a protein through changes in its codons

A base substitution replaces one base. Because the code is degenerate, some substitutions leave the amino-acid sequence unchanged: GCU to GCC still specifies alanine. Other substitutions change an amino acid, such as UUC to UAC, changing phenylalanine to tyrosine. A substitution that creates a premature stop can shorten the product. The effect depends on the sequence and position; "one base change always destroys the protein" is false.

A real example is the sickle-cell variant of human beta-globin. The coding-DNA triplet GAG changes to GTG, corresponding to mRNA GAG changing to GUG and a glutamic-acid-to-valine substitution. It is commonly numbered position 6 in the mature beta chain. The altered side-chain properties favour association of deoxygenated haemoglobin S molecules into polymers, which can distort red cells. This connects a sequence change with protein behaviour rather than treating "mutation" as a complete explanation. See the NIH description of the molecular change.

An insertion or deletion not divisible by three shifts the grouping of downstream bases within a coding region. In the invented sequence AUG GCU UUC GGA UAA, deleting base 4 gives AUG CUU UCG GAU AA when regrouped from the same start. The displayed complete codons specify Met–Leu–Ser–Asp; two bases remain at the end of this displayed segment. No stop appears among those four complete codons, so the eventual full product cannot be inferred without downstream sequence.

The reading frame changes, but do not claim every downstream amino acid must differ or that a premature stop must occur immediately. Degeneracy and the particular sequence matter. Removing three bases can remove one amino acid without a sustained frame shift, depending on where the deletion lies; a deletion crossing a codon boundary can also alter the codon at its junction.

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AHL: Transcription and translation have defined directions

RNA polymerase follows a DNA template 3′ to 5′ and extends the RNA 5′ to 3′ by adding nucleotides at its growing 3′ end. The coding DNA strand has the same sequence as the corresponding RNA when both are written 5′ to 3′, except DNA has T where RNA has U. “Coding strand” and “template strand” are not interchangeable names.

For example, template 3′-TAC CGA AAG-5′ specifies RNA 5′-AUG GCU UUC-3′. The corresponding coding DNA is 5′-ATG GCT TTC-3′. If instead the template is supplied 5′ to 3′, construct its complementary RNA antiparallel, then reverse the written RNA to report it 5′ to 3′. Copying unchanged letters with new end labels is incorrect.

During translation the ribosome reads the mRNA 5′ to 3′. This is movement along the message; polypeptides do not have 5′ and 3′ sugar ends. Amino-acid addition extends a chain at its carboxyl end, giving amino-to-carboxyl synthesis. Sequence orientation and the chosen start establish which triplets are read.

AHL: Promoters and non-coding DNA have distinct roles

Transcription begins at an appropriate initiation region associated with a promoter. Proteins including transcription factors recognize specific DNA sequences and help recruit or position transcription machinery. Their binding can affect whether transcription begins. The promoter is not an amino-acid sequence and does not need to be translated to perform its role.

Non-coding DNA means DNA that does not specify a polypeptide sequence. It does not mean all such DNA is useless or never transcribed. Regulatory sequences can control gene activity; introns are transcribed into primary RNA but removed during processing; telomeres help protect chromosome ends. Genes for rRNA and tRNA produce functional RNA products that are not translated into polypeptides.

Also distinguish a non-coding DNA region from the “non-coding” or template strand of a protein-coding gene. That strand name describes its relationship to the RNA sequence, not a claim that the whole strand has no biological information. Some retained exonic sequence is untranslated, so “exon” is not a universal synonym for “amino-acid-coding segment”.

AHL: RNA processing produces mature eukaryotic mRNA

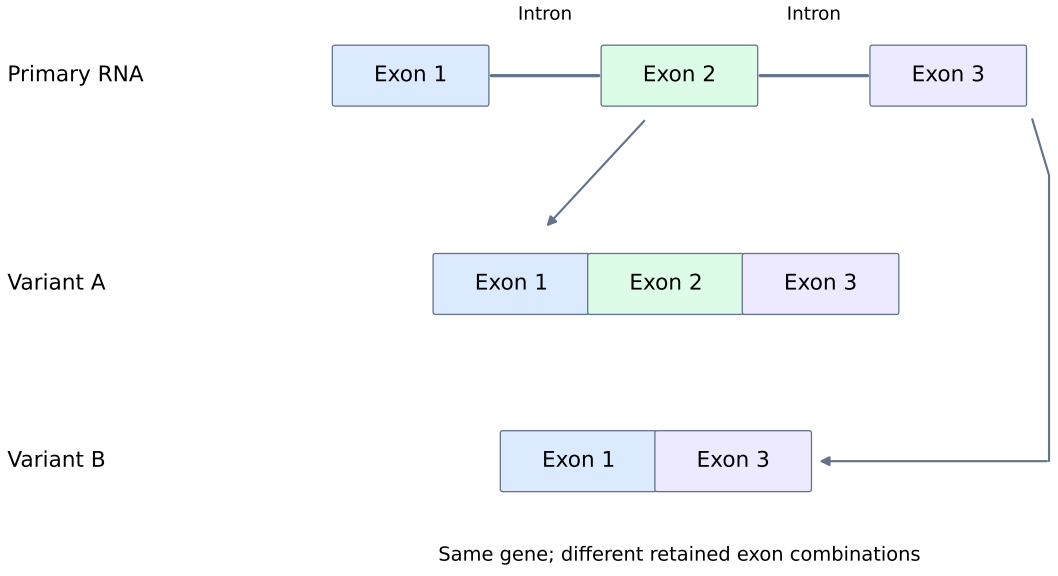
A primary transcript of a typical eukaryotic protein-coding gene includes exons and introns. Splicing removes intron sequences and joins the retained exon sequences. The result is a continuous mature RNA sequence; introns are not translated and then removed from the finished polypeptide by splicing.

A modified 5′ cap and a 3′ polyA tail contribute to stability and other aspects of mRNA handling. The tail is added during processing; it is not simply a long DNA-

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encoded run copied from the gene in this model. These modifications do not mean that every nucleotide in mature mRNA becomes an amino acid: untranslated regions remain, and the cap and tail are not part of the translated coding region.

Alternative splicing joins different combinations of exons from the same gene, allowing different polypeptide variants. Exons retain an appropriate order rather than being shuffled at random. Whether an alternative produces a functional product depends on its sequence, reading frame and regulation; do not infer 2^n functional proteins merely from counting exons.



This illustrative exon-skipping model assumes frame-compatible coding sequences. Exons 1 and 3 are retained in both variants. Introns and the skipped exon are absent from the second mature message; the gene's DNA is unchanged.

In an invented model, exons have 120, 90 and 180 coding nucleotides and the two introns contain 210 and 150. The primary transcript segment contains $120+90+180+210+150=750$ nucleotides. Retaining all three exons leaves 390 coding nucleotides; skipping the middle exon leaves 300. If each retained sequence begins at the same start, remains in frame and includes one terminal stop, their products contain $390/3-1=129$ and $300/3-1=99$ amino acids. Caps, tails and untranslated sequences are excluded from these stated counts.

AHL: Eukaryotic initiation establishes the frame and binding sites

In the simplified cap-dependent model, the small ribosomal subunit associates with the 5′ end of the mRNA and an initiator tRNA, then scans to an appropriate start codon. The initiator tRNA pairs with that start, establishing the reading frame. The large subunit joins to complete the ribosome, and an incoming tRNA can occupy the

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next codon position. This is an ordered assembly, not two arbitrary tRNAs choosing any adjacent triplets.

The ribosome has A, P and E tRNA-binding sites. During elongation the A site receives the incoming charged tRNA; the P site holds the tRNA carrying the chain before transfer. Peptide-bond formation transfers the chain onto the A-site amino acid. Movement by one codon shifts that chain-carrying tRNA into P and the uncharged tRNA toward E, from which it exits.

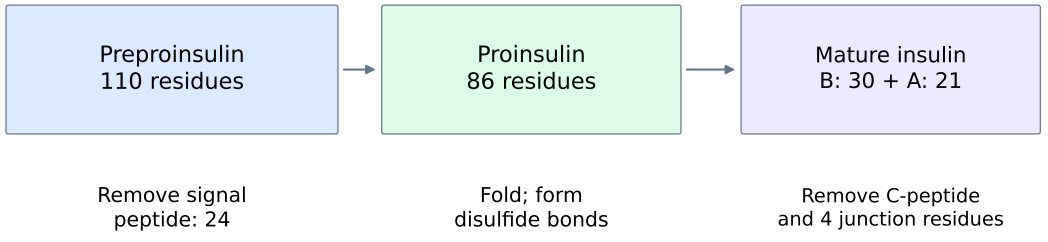
The initiator tRNA is positioned in P at initiation; do not apply "every tRNA first enters A" to it. Two tRNAs can participate simultaneously in chain transfer even though three binding-site names are used to describe the cycle. A stop codon is recognized by termination machinery, not by a tRNA carrying an amino acid called stop.

AHL: Processing makes many polypeptides functional

A newly translated chain may need folding, cleavage, chemical modification or assembly with other chains before it functions. Examples of modification include addition of carbohydrate groups or phosphate groups where appropriate. These are regulated processes; not every protein receives every possible modification.

Human insulin illustrates two stages of precursor processing. Preproinsulin includes a signal peptide that directs entry into the secretory pathway. Removal of the signal peptide during entry into the endoplasmic reticulum yields proinsulin. Proinsulin folds and forms disulfide bonds; later processing removes the connecting C-peptide and adjacent basic residues, leaving the A and B chains joined by disulfide bonds.

Human insulin: two stages of precursor processing



Human precursor lengths are biological values: 110 residues in preproinsulin, 86 in proinsulin and 51 in mature insulin. The boxes show processing steps, not molecular structures or lengths to scale.

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Human preproinsulin has 110 amino-acid residues; removing the 24-residue signal gives proinsulin with 86. Mature insulin retains 30 residues in B and 21 in A, a total of 51. The removed 35 residues comprise the 31-residue C-peptide plus four basic junction residues; calling all 35 "the C-peptide" would be inaccurate. The PDBe structural account describes the precursor and folding stages; the Reactome processing pathway distinguishes protease cleavage and additional residue removal.

RNA splicing and insulin processing therefore operate on different substrates. Splicing changes an RNA transcript's retained sequence. Proteolytic processing cuts a polypeptide precursor. Neither process rewrites the underlying DNA gene into a shorter gene each time a product is made.

AHL: Proteasomes support protein turnover and amino-acid recycling

A functional proteome requires both protein synthesis and protein breakdown. Damaged, misfolded or no-longer-needed proteins can be targeted for degradation. Proteasomes break appropriate proteins into smaller peptides; further breakdown can release amino acids for reuse in new synthesis. Other degradation pathways also exist, so do not assign all cellular protein breakdown exclusively to proteasomes.

Recycling recovers building blocks, not the original protein's function unchanged. The cell still needs an mRNA sequence, ribosomes and energy to construct new polypeptides in the required order. Proteasomes degrade proteins; they do not splice introns out of RNA or proofread DNA templates.

For an invented cellular pool, synthesis of 120 protein molecules per hour and degradation of 80 gives a net increase of 40 per hour, assuming no other gains or losses. A constant pool could instead have synthesis and degradation both at 200 per hour. Constant abundance therefore does not demonstrate that its molecules never change.

Practice

1. Distinguish replication, transcription and translation by template, product and role. Explain why a non-dividing somatic cell can still need a stable DNA template and active protein synthesis.
2. Write the complementary RNA aligned with DNA template T–A–C–C–C–G–A–A–A. Explain the roles of RNA polymerase, hydrogen bonds and covalent bonds, and why the template is reusable.
3. Two cells have an intact gene but only one has detectable transcript and protein. Explain how transcription can regulate expression and why the observation does not establish gene absence in the other cell.
4. Explain the separate roles of mRNA, tRNA and the ribosomal subunits. For codons GCU and UUC write aligned anticodons and identify the carried amino acids using the table.

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5. Show why a triplet code has enough combinations for twenty amino acids. Explain degeneracy, near universality and stop signals without claiming that one standard codon can arbitrarily specify several amino acids.
6. Translate AUG GCC UAC GAG UGA using the table, stating the number of codons and amino acids. Explain why using anticodons in the table would give the wrong interpretation.
7. A coding region has 156 nucleotides including one terminal stop and no other included sequence. Calculate codons, initial polypeptide length and peptide bonds in that single chain. Explain why a whole gene's length cannot normally be used in the same calculation.
8. Explain an elongation step from incoming charged tRNA to peptide-bond formation and ribosome movement. For an existing six-residue chain, state the effect of adding one amino acid on residue and peptide-bond counts.
9. Classify the changes GCU to GCC, UUC to UAC and UAU to UAA. Relate the sickle-cell coding-DNA change GAG to GTG to its RNA codon and amino-acid consequence. Explain why a substitution need not destroy a protein.
10. Delete base 4 of AUG GCU UUC GGA UAA, regroup from the original start and use the excerpt to interpret the complete codons shown. Explain what additional information is needed to know the full product and why "every later amino acid must change" is too strong.
11. Compare removal of three consecutive coding bases with removal of one. Explain the effect on frame, why the junction can matter, and why the actual sequence and position are needed before predicting function.
12. **AHL:** A template is supplied as 5′-AGC CAT-3′. Report the corresponding RNA 5′ to 3′ and its coding DNA in that direction. Explain polymerase and ribosome direction, promoter/transcription-factor roles, and why introns, telomeres and rRNA/tRNA genes are not all useless DNA.
13. **AHL:** An invented primary transcript contains coding exons of 150, 120 and 210 nucleotides and introns of 180 and 240. Calculate the primary length and the coding lengths/products with all exons or with the middle exon skipped. Assume frame compatibility, one terminal stop and no other included sequence. Explain cap/tail roles and A/P/E-site events during initiation and one elongation cycle.
14. **AHL:** Human preproinsulin contains 110 residues, its signal peptide 24, and mature insulin B/A chains 30/21. Calculate proinsulin length, total subsequent removal and the residues removed in addition to a 31-residue C-peptide. Distinguish RNA splicing from these steps. Explain how proteasomes support recycling, and calculate pool change if synthesis is 150 and degradation 110 molecules per hour.

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Worked answers

1. Replication uses DNA to make DNA copies for genetic continuity; transcription uses DNA to make RNA; translation uses mRNA to specify a polypeptide at ribosomes. A non-dividing cell still maintains DNA instructions and can repeatedly express genes to replace proteins and support its functions without copying the whole genome for division.
2. A–U–G–G–G–C–U–U–U. Complementary RNA nucleotides pair with DNA bases through hydrogen bonding; RNA polymerase catalyses the new RNA's covalent backbone. DNA is not consumed or normally changed in base sequence by the copying event, so it can be reused.
3. Different transcription activity can produce different mRNA availability despite a shared gene. Protein amount also depends on later stages. Check DNA directly and consider timing and detection sensitivity; a missing transcript/protein signal does not by itself prove a gene deletion.
4. mRNA supplies ordered codons. A charged tRNA carries an amino acid and has a matching anticodon; the attachment and anticodon sites differ. The small ribosomal subunit binds/positions mRNA; the large subunit supports peptide bonding. Aligned anticodons are CGA for GCU, carrying Ala, and AAG for UUC, carrying Phe. Two tRNAs can take part together in chain transfer.
5. Four bases give 4, 16 and 64 combinations for one-, two- and three-base signals respectively. Sixty-four can represent twenty standard amino acids plus stop signals. Degeneracy means several codons can specify the same amino acid; near universality means broad sharing with known exceptions. UAA/UAG/UGA specify termination, not an added amino acid.
6. Met–Ala–Tyr–Glu, then stop. Five codons specify four residues. The table keys are mRNA codons; a complementary tRNA anticodon is a different sequence, not the sequence to look up as the message.
7. Codons $= 156/3 = 52$; residues $= 52 - 1 = 51$; peptide bonds $= 51 - 1 = 50$ in the stated initial single chain. Whole genes can contain regulatory, untranslated and intron regions; full mRNA also includes untranslated sequence. Later protein processing may change chain length or number.
8. An incoming charged tRNA pairs with the next codon while another tRNA carries the chain. Peptide bonding transfers the chain to the incoming amino acid; movement by one codon repositions the tRNAs and permits exit/recharging. Six residues with five peptide bonds become seven residues with six peptide bonds. The mRNA is reused, not consumed as amino-acid material.
9. GCU to GCC is synonymous for Ala; UUC to UAC changes Phe to Tyr; UAU to UAA creates a stop. Sickle-cell coding DNA GTG yields mRNA GUG and Val instead of the Glu specified by GAG. Effects depend on sequence, position, side-chain properties and protein structure; a base substitution may leave the amino-acid sequence unchanged or have varied functional effects.

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10. Deleting G at base 4 gives AUG CUU UCG GAU AA. Complete codons specify Met–Leu–Ser–Asp; AA is incomplete and no full stop is shown. More downstream sequence is needed for the full product. Grouping changes, but degeneracy or chance agreement can preserve some amino acids.
11. A one-base deletion shifts downstream grouping; three bases preserve the downstream frame. A boundary-crossing deletion can also change the joined codon. Function depends on sequence, position and affected residues; preserving a frame does not guarantee function.
12. **AHL:** Pair antiparallel to obtain RNA 3′-UCG GUA-5′, then reverse: 5′-AUG GCU-3′. Coding DNA is 5′-ATG GCT-3′. RNA polymerase reads template 3′ to 5′ and builds RNA 5′ to 3′; a ribosome reads mRNA 5′ to 3′. Promoter-bound factors help regulate initiation. Introns can be transcribed then removed, telomeres protect chromosome ends, and rRNA/tRNA genes provide functional RNAs without polypeptide coding.
13. **AHL:** Primary length $= 150 + 120 + 210 + 180 + 240 = 900$ nucleotides. All exons give 480 coding nucleotides and $480/3 - 1 = 159$ residues; skipping exon 2 gives 360 and $360/3 - 1 = 119$, forty fewer. Cap and polyA tail support stability/handling and are excluded from these counts. During cap-dependent initiation, the small subunit with initiator tRNA scans to the start; the initiator occupies P, then the large subunit joins. Charged tRNA enters A; peptide bonding transfers the chain from P to A; one-codon movement shifts the chain-carrying tRNA to P and uncharged tRNA toward E for exit.
14. **AHL:** Proinsulin $= 110 - 24 = 86$ residues; mature insulin $= 30 + 21 = 51$. Subsequent removal $= 86 - 51 = 35$, including $35 - 31 = 4$ basic junction residues as well as C-peptide. Signal removal and later cleavage/folding process a protein precursor; splicing removes/joins RNA regions. Proteasomal breakdown and subsequent peptide breakdown can recover amino acids for new, sequence-directed synthesis. Pool increase $= 150 - 110 = 40$ molecules per hour under the stated closed balance. Equal synthesis and breakdown could maintain a constant abundance despite continual turnover.

Exam tips

- Identify the molecule before using a code table. Pair DNA with RNA correctly and read the message in the stated frame; a stop adds no amino acid.
- Give a mechanism: template pairing and polymerase action for transcription; tRNA matching, peptide transfer and one-codon movement for elongation.
- Check whether a length includes untranslated sequence or a terminal stop. A shifted frame does not prove that every later amino acid differs.
- **AHL:** Keep strand direction, promoter control, RNA splicing/cap/tail, translation initiation, protein cleavage and proteasomal recycling as distinct steps.

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D.1.3 · Mutations and gene editing

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This handout covers D1.3: molecular changes in genes, their causes, inheritance and role in variation. Sections marked **AHL** are for Higher Level only.

Prerequisites: complementary DNA bases, gene expression, mRNA codons and the distinction between body cells and gametes. Sequence examples and numerical datasets are invented unless a published study is named.

A changed DNA sequence can have several different consequences

Two cells can have different DNA sequences yet make the same amino-acid sequence. Another single-base change can introduce an early stop. The size of a DNA change alone therefore does not tell us the size of its effect.

A **mutation** is a change in the DNA base sequence. Once copied, a sequence change can pass to descendant cells. DNA damage is not automatically a lasting mutation: repair may restore the original sequence before it becomes established. A change in gene activity without a change in DNA sequence is also a different process.

Deliberately changing a chosen DNA sequence is **gene editing** . Both spontaneous mutations and targeted changes must be understood through their actual sequence and biological context. The Higher Level sections examine how targeted editing can test gene function.

A gene mutation changes sequence within a gene; larger chromosome changes can involve many genes. Always identify the scale in the question. Here the main molecular changes are base substitutions, insertions and deletions. A change may affect a protein-coding region, RNA processing or a sequence controlling expression.

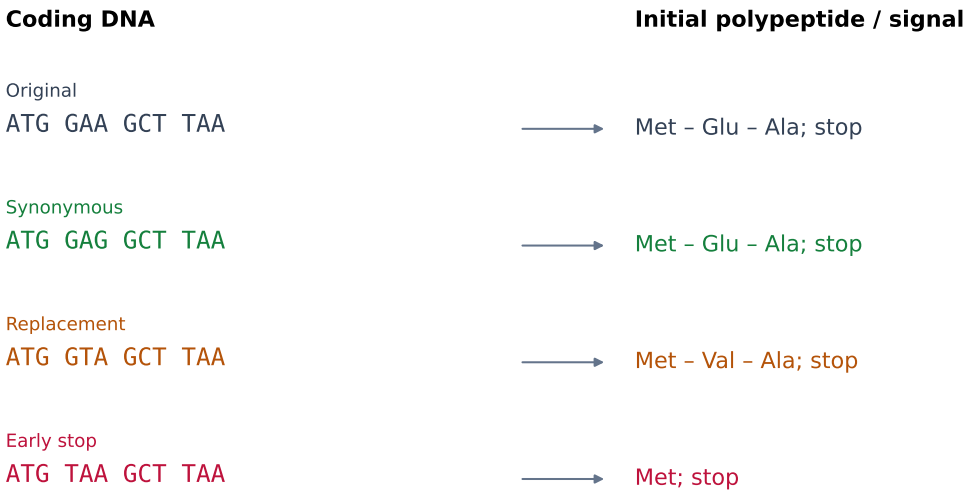
Substitutions can preserve, replace or stop an amino-acid instruction

A **substitution** replaces one base with another. DNA still contains the same number of bases, so a simple substitution inside a coding region does not by itself shift subsequent codon boundaries. However, the new triplet may have a different meaning.

For the following invented example, the coding DNA strand is written in the same direction as its mRNA; replace T with U to obtain the RNA. The original coding DNA is ATG GAA GCT TAA. Its mRNA is AUG GAA GCU UAA, specifying Met–Glu–Ala, followed by a stop. The stop specifies no amino acid.

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Coding DNA change	Corresponding mRNA	Consequence in this example
GAA becomes GAG	GAA becomes GAG	Both specify Glu: synonymous
GAA becomes GTA	GAA becomes GUA	Glu becomes Val: amino-acid replacement
GAA becomes TAA	GAA becomes UAA	A stop replaces Glu: early termination



Each row begins in the same stated frame. DNA changes are interpreted through mRNA codons; the stop is not an amino acid.

The genetic code is degenerate: several codons can specify one amino acid. A synonymous change leaves that amino acid unchanged. This is narrower than proving that every aspect of gene expression and cell function is unaffected. Sequence context can also influence processing or expression.

An amino-acid replacement may alter charge, bonding, folding, a binding site or stability. Its effect depends on the residue and its role. Some replacements have little detectable effect; others have large effects. An early stop often shortens the translated chain, but the observed amount of product may also be affected by RNA quality-control mechanisms. Do not assume every altered gene produces an abundant shortened protein.

A single-nucleotide polymorphism, or SNP, is variation at one nucleotide position among individuals. Such variation can arise through base substitution mutations. An observed base difference is not automatically disease-causing. Population variation, molecular consequence and effect on a person's traits are separate questions.

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Insertions and deletions can change the reading frame

The **reading frame** is the grouping of successive bases into codons from a defined starting position. An insertion adds bases; a deletion removes them. Within a translated region, a change whose length is not a multiple of three shifts the grouping downstream of the change.

Delete base 4, the first G after AUG, from the invented mRNA sequence AUGGAAGCUUAA. The sequence becomes AUGAAGCUUAA. Regrouping from the same start gives AUG AAG CUU AA: Met–Lys–Leu and an incomplete final group. The original stop is no longer a complete stop codon in this displayed frame. More downstream sequence is needed to predict where translation ends.

This is not evidence that every later amino acid must differ or that translation stops immediately. Chance agreement and degeneracy can preserve some residues. The frame changes at the edited position; the actual new codons determine the consequences.

Deleting the complete GAA codon instead gives AUG GCU UAA: Met–Ala, then stop. The downstream frame is preserved and one amino acid is lost. A three-base deletion that crosses a codon boundary can also change the newly joined codon. Preserving the downstream frame does not guarantee normal protein function. A large insertion or deletion can disrupt a whole functional domain, including a binding or catalytic region, even when its length is a multiple of three.

Change inside a coding region	Downstream frame	What still needs checking?
Add 1 base	Shifted	New codons and location of a stop
Remove 2 bases	Shifted	New sequence and product
Remove 3 bases	Preserved downstream	Missing residue(s), joined codon and function
Add 6 bases	Preserved downstream	Added sequence, junctions and function

Mutations outside translated sequence can also matter. A promoter change can alter transcription; a change at a splice-related sequence can alter which RNA sequence is retained. A non-coding change is therefore not automatically harmless, and it need not act by directly replacing an amino acid in a codon.

Mutagens raise the chance of changes; errors can also arise without exposure

DNA replication and repair are accurate but not perfect. An incorrectly incorporated base or an error during repair can establish a new sequence. A spontaneous mutation

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does not require a known external mutagen.

A mutagen increases the frequency of mutations under stated conditions. Examples include some chemicals and radiation. Ultraviolet radiation can damage neighbouring pyrimidine bases; ionizing radiation can damage DNA directly or through reactive products. Some chemicals alter bases or interfere with normal copying. For example, metabolic products of benzo[a]pyrene, found in tobacco smoke, can bind to DNA and interfere with accurate copying or repair. Damage may be repaired, cause cell death or become a lasting sequence change; exposure does not guarantee one particular result.

In a fair comparison, control the starting population, growth conditions, exposure measurement, number of generations and detection method. A larger fraction of mutant colonies after treatment is evidence to investigate. It is not automatically a measured mutation rate: survival, selection and multiplication of an early mutant can change that fraction.

For example, an invented sample contains 24 resistant colonies among 12,000 viable colonies. The observed resistant fraction is $24/12000 = 0.002$, or 0.20%. This counts a phenotype in the final sample. It does not tell us that 0.20% of DNA-copying events produced resistance.

Mutations are random with respect to an organism's needs

A new mutation is not normally directed to the exact useful base because an organism needs a particular adaptation. Changes can occur in many genome regions, including changes with no benefit. However, random with respect to need does not mean every position, base change or environment has the same mutation probability. Sequence hotspots, copying processes, repair and exposure can produce biases.

Consider bacteria exposed to an antibiotic. Resistant variants may already exist before exposure. The antibiotic removes susceptible competitors and resistant lineages become more common. This is selection acting on variation. Observing more resistant cells after exposure does not by itself show that the antibiotic instructed susceptible cells to make the needed mutation.

Independent cultures started before selection can produce very different resistant counts if mutations arise at different times: an early event leaves many descendants, a late event few. Such a pattern is consistent with pre-existing mutation and clonal growth. Interpretation still requires suitable controls, repeated cultures and a model accounting for growth and survival.

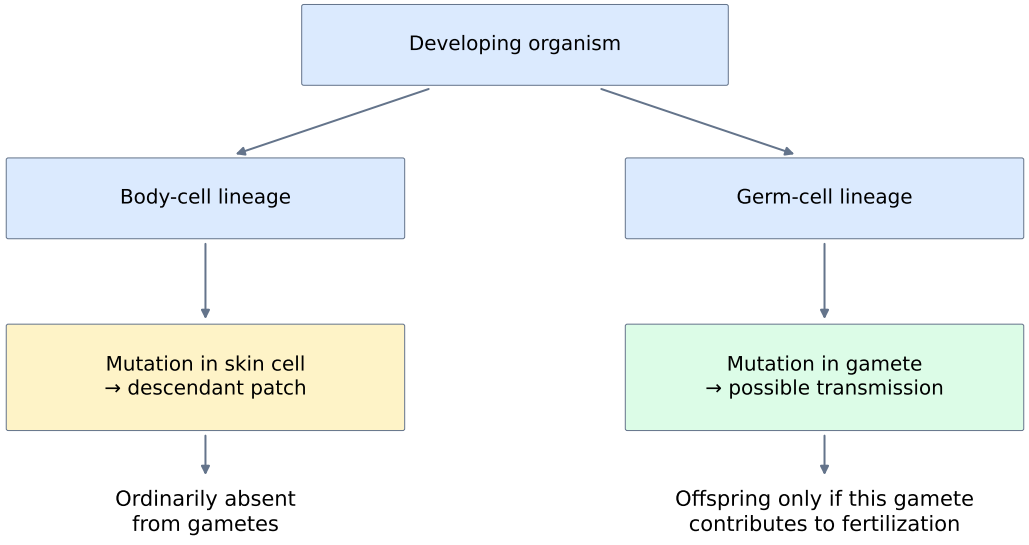
Mutation creates new alleles. Recombination and sexual reproduction can rearrange existing alleles into new combinations. Both contribute to variation, but shuffling existing alleles is different from the origin of a new sequence variant.

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The cell lineage determines whether a change can be inherited

A somatic mutation occurs in a body-cell lineage. It can pass through mitosis to a patch of descendant cells, producing genetic mosaicism. In ordinary sexual reproduction, a mutation confined to skin cells does not enter the gametes and is not inherited by offspring.

A mutation in a germ-cell lineage can reach a gamete. It can then enter an offspring if that gamete contributes to fertilization. Not every germline mutation is transmitted: many gametes do not contribute, and survival and development also matter. A change inherited at fertilization can be present throughout many descendant tissues; a later embryonic change may be present only in some lineages.



Arrows show cell ancestry. A somatic change can spread within the body without entering an offspring; transmission through a gamete is conditional on fertilization.

Some somatic mutations affect control of cell division, DNA repair or cell survival. Descendant cells may gain a growth advantage, contributing to cancer development. One mutation is not automatically sufficient to cause cancer: several changes and tissue conditions can matter. A mutation affecting a protein unrelated to growth is not automatically a cancer mutation either.

Keep inheritance and effect separate. A harmful mutation can be somatic or germline. A germline mutation can be neutral. The word inherited does not mean beneficial, and a mutation arising during one person's life is not necessarily somatic if it occurs in a germ-cell lineage.

Variation supplies material for evolution, with effects that depend on context

Many mutations have no detectable effect on reproductive success in a particular environment; many that affect important functions are harmful. A small proportion can provide an advantage in a given context. No fixed percentage applies to every gene, organism and environment.

A change affecting an enzyme may be harmful when its normal substrate is essential, yet useful under another condition. A variant allowing growth during antibiotic exposure may also carry a cost without the antibiotic. Benefit describes a relationship between a variant and its conditions, not a permanent label on the DNA change.

For mutation to contribute to evolution through sexual generations, it must enter the inherited variation of a population. Natural selection can then change allele frequencies when variants differ in reproductive success. Neutral variants can also change frequency through genetic drift. Mutation is essential over evolutionary time because it is the ultimate source of new alleles; that does not mean an individual needs a damaging mutation to stay healthy.

■ Evaluate a genetic-test claim

A commercial report identifying a variant is not, by itself, a diagnosis or a guarantee about a future trait. Ask which variants were tested, whether the association is supported in an appropriate population, how large the effect is, and what environmental or other genetic influences remain. A negative result for a small panel does not exclude every relevant variant.

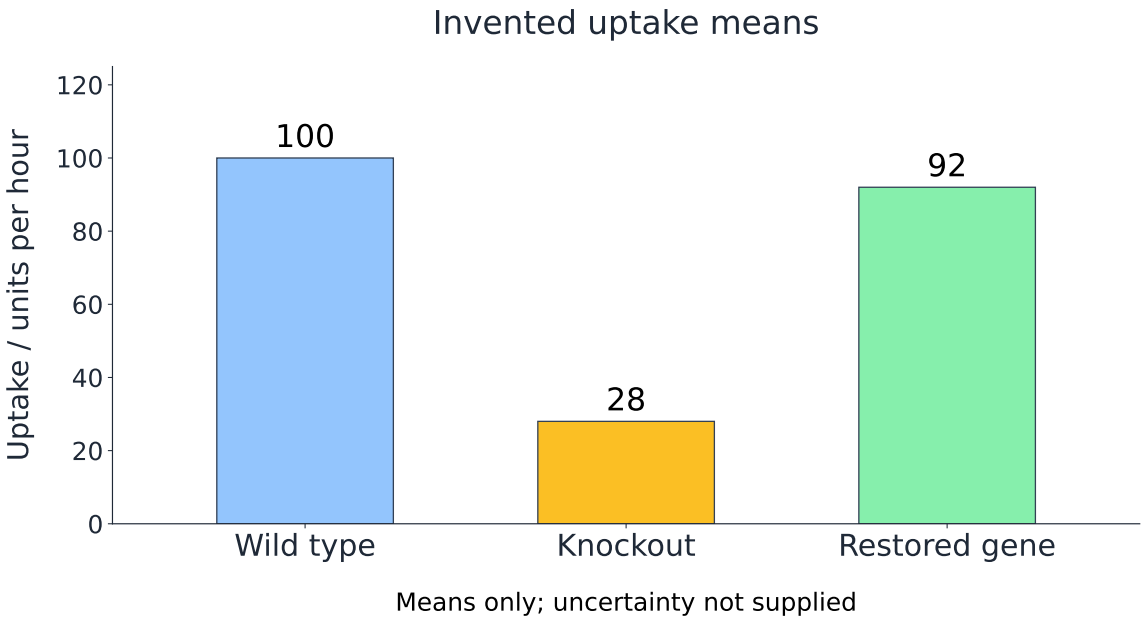
Use absolute quantities when possible. In an invented comparison, a risk rises from 2 in 1,000 to 4 in 1,000. It doubles, but the absolute increase is 2 in 1,000, or 0.2 percentage points. Neither description means every carrier develops the condition. Expert interpretation matters because sequence detection, statistical association and individual prediction are different levels of evidence.

AHL: Gene knockouts test contributions to function

A gene knockout makes a gene inoperative so that its contribution can be investigated. Compare organisms or cells with a functional gene against otherwise comparable knockout material. Knockout libraries allow many genes to be studied systematically in a model organism.

Suppose a gene may contribute to uptake of a nutrient. An invented experiment measures mean uptake in matched conditions: wild type 100 units per hour, knockout 28, and knockout with a restored functional copy 92. The reduction is $100 - 28 = 72$ units per hour, or 72% of wild type. Restoration towards the original value supports a contribution of the gene to the process.

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Bars show invented group means, not uncertainty intervals. Replicate counts and variation would be needed to assess precision and statistical support.

The experiment does not yet show that the gene's protein binds the nutrient directly. It might regulate another gene, build a membrane structure or supply energy. A rescue strengthens causal interpretation, but controls for background differences and unintended changes remain necessary. Use independent lines, matched environments and measurements of the proposed mechanism.

No visible phenotype does not prove that a gene has no function. Another gene may compensate, the function may appear only under another condition, or the measured outcome may miss the relevant process. Distinguish a complete knockout from a partial reduction in expression.

AHL: CRISPR-Cas9 links targeting, cutting and cellular repair

In a simplified CRISPR-Cas9 model, guide RNA helps recognize a complementary DNA target next to an appropriate short recognition sequence, the PAM. Cas9 cuts the DNA strands. Cellular repair determines the resulting sequence: a cut is not itself a guarantee of the intended final edit.

Repair can create small insertions or deletions that disrupt a coding frame. Other editing outcomes can involve a specified sequence change under suitable conditions. Not every cut disables a gene, and a non-coding target can change expression without deleting a protein-coding sequence. The target, actual repaired sequence and measured function must all be checked.

A useful evaluation asks whether the intended site changed, whether all relevant cells or alleles changed, whether other similar sites were affected, and whether the biological result persisted. Target specificity does not establish complete safety. A successful

molecular edit and a useful clinical outcome are related but separate endpoints.

A published example and its limits

In a 2024 single-group study of exagamglogene autotemcel for severe sickle-cell disease, patients' blood stem cells were edited outside the body and returned after conditioning. Editing targeted an erythroid-specific enhancer of BCL11A, reducing repression of fetal haemoglobin; it did not directly repair the sickle-cell HBB substitution. Of 44 treated patients, 30 had sufficient follow-up for the primary endpoint: 29/30, approximately 97%, were free of severe vaso-occlusive crises for at least 12 consecutive months. This is a defined observed benefit, not proof of lifelong cure or zero risk. Frangoul and colleagues, 2024.

The denominator must match the outcome being discussed. Patients without enough follow-up are not automatically treatment failures or successes. A study without a randomized comparison group also requires care when judging causes and comparing treatments. Long-term follow-up and adverse-event assessment remain important.

Ethics needs a named application and affected people

Editing a person's somatic cells and editing a germline that may affect future generations raise different issues. Consider expected benefit, uncertain harm, consent, fair access, effects on future offspring and the distinction between treating illness and selecting preferred traits. Animal research also requires attention to welfare and alternatives.

A useful argument states who may benefit, who bears risk and what evidence or safeguards would change the judgement. Countries can regulate uses differently, creating challenges for international standards. International cooperation seeks to harmonize regulation while addressing these ethical differences. Do not assume that technical possibility gives permission for every application, or that the same rules apply everywhere.

AHL: Conserved sequences can indicate functional constraints

A conserved sequence remains similar across compared species or lineages. One explanation is purifying selection: variants disrupting an important function leave fewer descendants, so functional sequence is retained. Conservation can help identify regions worth investigating, including regulatory DNA as well as coding DNA.

However, conservation is not proof that mutations never occurred there. Another hypothesis is a lower local mutation rate. Closely related species can also share sequences simply because little time has passed. Compare equivalent sequences, divergence times and nearby or suitably matched neutral regions. Then test function rather than relying on similarity alone.

In an invented eight-position alignment, ACGTACGT and ACGTTCGT match at seven positions. Identity is $7/8 \times 100 = 87.5\%$, with no gaps included in this model. A difference at one position does not identify the trait it affects. Conversely, 100%

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identity in a short sample does not show that change is impossible.

Coding DNA can change while amino-acid sequence is conserved because of degeneracy. Specify whether conservation is measured in DNA or protein. Evidence from a knockout, altered expression or a biochemical test can complement comparative sequence evidence.

Practice

1. Distinguish a lasting mutation from repaired DNA damage and from a change in transcription activity.
2. For coding DNA ATG GAA GCT TAA, give the mRNA and initial amino-acid sequence, including the meaning of the final triplet.
3. Compare changes of the second coding-DNA triplet from GAA to GAG, GTA and TAA. Which shifts the downstream frame?
4. Delete coding base 4 from the corresponding mRNA AUGGAAGCUUAA. Regroup it and explain what can and cannot be concluded about the final peptide.
5. Explain the difference between a complete-codon deletion and a two-base deletion within a translated region. Is either guaranteed harmless?
6. A promoter change leaves the coding DNA unchanged but lowers protein abundance. Explain a plausible mechanism and one useful measurement.
7. An invented exposure experiment finds 36 resistant colonies among 18,000 viable colonies. Calculate the observed fraction as a decimal and percentage. Why is this not automatically a mutation rate?
8. Explain why increased resistance after antibiotic exposure does not show mutations occurred because bacteria needed them. Does random mean equal probability at all bases?
9. Compare a mutation restricted to skin cells with a mutation entering a gamete. State the condition needed for transmission to an offspring and one possible consequence within a body-cell lineage.
10. Explain how mutation, recombination, selection and drift make different contributions to population variation. Why can the same variant have different effects on fitness in different conditions?
11. An invented report changes estimated risk from 3 in 1,000 to 6 in 1,000. Give the relative change and absolute change in percentage points. Explain why a positive or negative panel test is not a complete prediction.
12. **AHL:** Invented uptake means are 80 for wild type, 20 for knockout and 74 for restored gene, in units per hour. Calculate the knockout reduction as a percentage of wild type. Give a supported conclusion, a control and a limit on the inferred mechanism.

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13. **AHL:** Explain guide recognition, Cas9 cutting and repair as distinct stages. Why does a cut not prove a precise, safe, successful edit? Distinguish somatic and potentially inherited uses when evaluating ethics.
14. **AHL:** Two invented ungapped DNA sequences, AACCGGTTAA and AACCGATTAA, differ at one position. Calculate identity and evaluate the claim that a highly conserved sequence proves mutations never occur there. Name evidence that could separate functional constraint from a low mutation rate.

Worked answers

1. A lasting mutation changes the established DNA sequence. Repair may remove damage and restore the original sequence, leaving no lasting mutation. Transcription can increase or decrease without a DNA-sequence change.
2. mRNA AUG GAA GCU UAA; Met–Glu–Ala, then stop. UAA is a termination signal and adds no amino acid: four codons here produce three residues before processing.
3. GAG gives mRNA GAG, still Glu; GTA gives GUA, Val; TAA gives UAA, an early stop. None changes sequence length or shifts the downstream frame by itself.
4. Removing base 4 gives AUGAAGCUUAA, grouped AUG AAG CUU AA: Met–Lys–Leu and two remaining bases. The frame changes after AUG and no complete stop is displayed. Further downstream sequence is required to predict the full product or termination position.
5. A complete-codon deletion removes one amino acid while preserving the downstream frame. A two-base deletion shifts subsequent grouping. Either can affect function; the location, new sequence and biological role determine the outcome.
6. The promoter change could reduce transcription initiation, lowering mRNA supply and therefore protein production. Measure transcript abundance as well as protein, with appropriate controls; protein breakdown and other regulation can also affect abundance.
7. $36/18000 = 0.002 = 0.20\%$. This is a final resistant fraction. Differential survival, generations and expansion of early mutants can affect it; it is not a count of new sequence changes per copying event.
8. Selection can enrich resistant variants that arose before exposure. A useful mutation need not arise in response to need. Random with respect to need does not require equal rates at all genome positions or identical probabilities for all base changes.
9. A skin-cell mutation can pass to descendant skin cells but ordinarily not to gametes. An offspring can inherit a mutation if the affected gamete contributes to fertilization. Somatic changes may produce mosaic patches or contribute to cancer through altered growth control; a single mutation need not cause cancer.
10. Mutation creates new alleles; recombination shuffles existing alleles. Selection changes frequencies through differences in reproductive success; drift changes fre-

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quencies through chance sampling. Environmental conditions can change the benefits and costs of a variant, so its effect on fitness is context-dependent.

11. Risk doubles, a 100% relative increase. It rises from 0.3% to 0.6%, an absolute increase of 0.3 percentage points. A tested variant may alter probability without determining outcome; an untested variant, other genes or the environment may matter. A negative limited panel does not exclude every relevant change.
12. **AHL:** Reduction $= 80 - 20 = 60$; $60/80 \times 100 = 75\%$. Knockout reduces uptake and restoration towards 80 supports a contribution of the gene. Use matched genetic background and conditions, independent lines and replication. The gene might act indirectly; these means alone neither prove direct nutrient binding nor establish statistical significance.
13. **AHL:** Guide pairing and PAM recognition help locate the target; Cas9 cuts DNA; cellular repair generates the final sequence. Unintended repair, incomplete editing, off-target effects or loss of function at an important site can limit success. Verify sequence and biological outcome. Somatic treatment primarily affects the treated person; germline changes may affect future descendants, adding consent and inheritance concerns alongside benefit, risk and access.
14. **AHL:** Nine matches out of ten give 90% identity. Conservation can reflect purifying selection, a lower mutation rate or limited divergence time; it does not prove mutation is impossible. Compare suitably matched neutral regions and lineages, and experimentally test the conserved region's function. Distinguish DNA conservation from protein conservation.

Exam tips

- Name the altered molecule and region before predicting an effect. A non-coding change can affect expression or processing.
- Translate mRNA codons in the specified frame. Give the new sequence before claiming a frameshift, stop or residue change.
- Separate a new mutation, its inheritance and its frequency after selection. A final mutant fraction is not automatically a mutation rate.
- **AHL:** Separate targeting, cutting, repaired sequence and functional outcome. Explain what a knockout or conserved sequence supports and which alternatives remain.

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Cell and nuclear division

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This handout covers D2.1: cell division, mitosis, meiosis and variation. Sections marked **AHL** are for Higher Level only.

Prerequisites: DNA replication, chromosomes and the distinction between a gene and an allele. Numerical examples and cell diagrams are invented teaching models, not measurements from a real investigation.

New cells come from existing cells

A growing root needs more cells; damaged skin needs replacement cells; a sexually reproducing organism needs cells with half its usual chromosome number. These problems all involve division, but they do not require the same nuclear outcome.

In living organisms, new cells arise by division of existing cells. A parent cell divides to produce daughter cells. Unicellular organisms can reproduce in this way, while multicellular organisms also use division to build and maintain tissues. A cell does not normally assemble a whole new living daughter cell from non-living material outside itself.

In a nucleated eukaryotic cell, nuclear division distributes genetic material before the cytoplasm is fully separated. Without suitable nuclear division, one product could lack a nucleus or receive an incomplete chromosome set. Mitosis maintains chromosome number in daughter nuclei; meiosis reduces it and generates new combinations of inherited information.

Nuclear division and cytokinesis are distinct. One separates nuclei and chromosomes; the other separates the cytoplasm into cells. They usually work together, but their timing can overlap. Nuclear division without cytokinesis can produce a cell containing more than one nucleus.

Cytokinesis separates cytoplasm, with equal or unequal shares

In animal cells, a contractile ring containing actin and myosin tightens beneath the cell membrane. A cleavage furrow deepens and the membrane is pinched into two daughter cells. A rigid wall would prevent this same inward-pinching mechanism in plant cells.

In a dividing plant cell, vesicles gather near the centre and contribute membranes and wall materials to a growing cell plate. It expands outward and connects with the existing cell boundary, separating the daughter cytoplasms and building a new wall between them. Do not describe a plant cell as forming an animal-style cleavage furrow through its rigid wall.

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The shares of cytoplasm are often similar but need not be equal. During human oogenesis, unequal division retains most cytoplasm in the developing egg, while polar bodies receive much smaller amounts. This helps retain resources in the cell that can support early development. It does not mean each nucleus should receive an arbitrary chromosome number.

In budding yeast, a smaller bud receives a daughter nucleus and cytoplasm before separating. The products differ in size. Both daughters must also receive at least one mitochondrion and other organelles required for their continued life. Mitochondria, and chloroplasts where present, grow and divide from existing organelles; daughter cells cannot replace their inheritance merely by receiving nuclear DNA.

Replication changes DNA amount before it changes the chromosome count

A **chromatid** is one of the DNA-containing copies of a replicated chromosome before the copies separate. Each sister chromatid contains a long DNA molecule associated with proteins. Sister chromatids are joined until their separation; homologous chromosomes are the corresponding maternal and paternal members of a pair, not the two sisters of one chromosome.

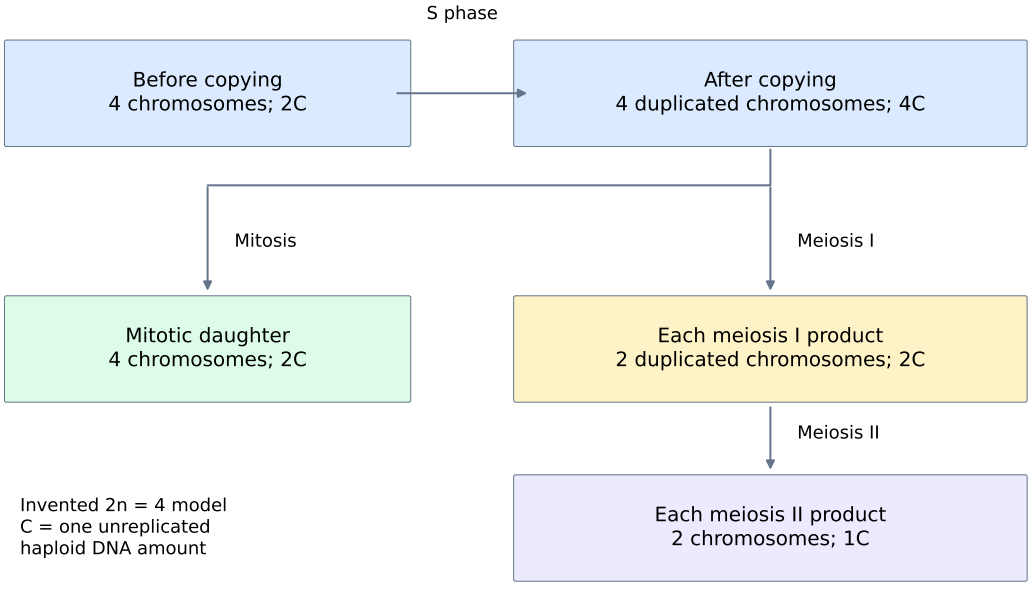
DNA replication precedes mitosis and also precedes meiosis I. One round of replication makes sister chromatids. There is no second DNA-replication round between meiosis I and meiosis II. Without the preceding replication, the usual segregation processes could not distribute the expected copies to both sets of products.

Use the stated counting convention. A replicated chromosome with two attached sister chromatids counts as one chromosome. When its sisters separate, each becomes a daughter chromosome. The chromosome count briefly doubles within the still-undivided anaphase cell, but each future daughter nucleus receives the original number.

For an invented species with $2n = 4$, the following table distinguishes chromosome sets from DNA amount. Here C means the DNA amount in one unreplicated haploid set; it is not a count of chromosomes.

Stage, with boundary specified	Chromosomes	DNA amount
One diploid cell before replication	4	2C
Same cell after replication, sisters attached	4	4C
Each daughter after mitosis	4	2C
Each product after meiosis I	2, still duplicated	2C
Each product after meiosis II	2, unduplicated	1C

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Counts refer to one cell or one daughter product as labelled. Replication doubles DNA amount; meiosis I halves the chromosome-set number.

A four-chromosome metaphase cell contains eight chromatids and eight DNA molecules. An anaphase cell before cytokinesis contains eight daughter chromosomes in total, four moving towards each pole. Saying only "there are eight chromosomes" without naming the cell boundary can therefore mislead.

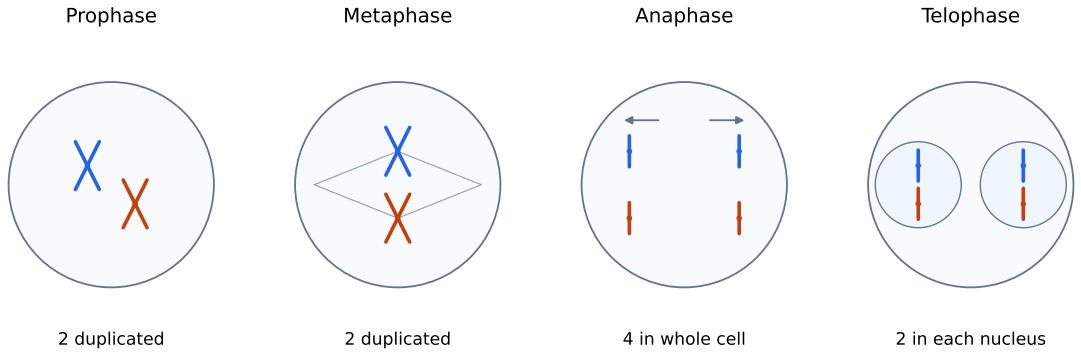
Mitosis packages, aligns and separates the copied chromosomes

During condensation, DNA wrapped around histone proteins is further coiled and packaged into compact, visible chromosomes. Condensation changes arrangement, not the number of DNA copies. A replicated chromosome drawn as an X is not two homologous chromosomes: it represents two attached sister chromatids.

Microtubules form the spindle and attach at chromosome-associated structures. Together with motor proteins and changes in microtubule length, they move chromosomes and help distribute them. Chromosomes do not swim independently to the poles, and the cell does not sort them by reading a printed X shape.

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Phase	Evidence and mechanism
Prophase	Chromosomes condense; spindle develops; nuclear-envelope breakdown allows spindle access
Metaphase	Duplicated chromosomes align at the equatorial region with sisters connected towards opposite poles
Anaphase	Sister chromatids separate; daughter chromosomes move towards opposite poles
Telophase	Chromosomes reach poles, nuclear envelopes form and chromosomes become less condensed



The model tracks two replicated chromosomes into two nuclei, each receiving two daughter chromosomes. Cell shapes and spindle lines are schematic.

For the normal model, mitosis gives two daughter nuclei with the same chromosome number and equivalent genetic information as the parent before replication. New mutation or segregation errors are possible; "identical" describes the normal distribution model, not a guarantee that every real division is error-free.

When identifying a phase in a diagram or micrograph, use chromosome arrangement. A central line of condensed duplicated chromosomes supports metaphase. Two groups of separating chromosomes support anaphase. Two forming nuclei support telophase. A round cell alone is not enough evidence. Interphase cells usually show less distinct chromosome outlines; interphase is not one of the four mitotic phases.

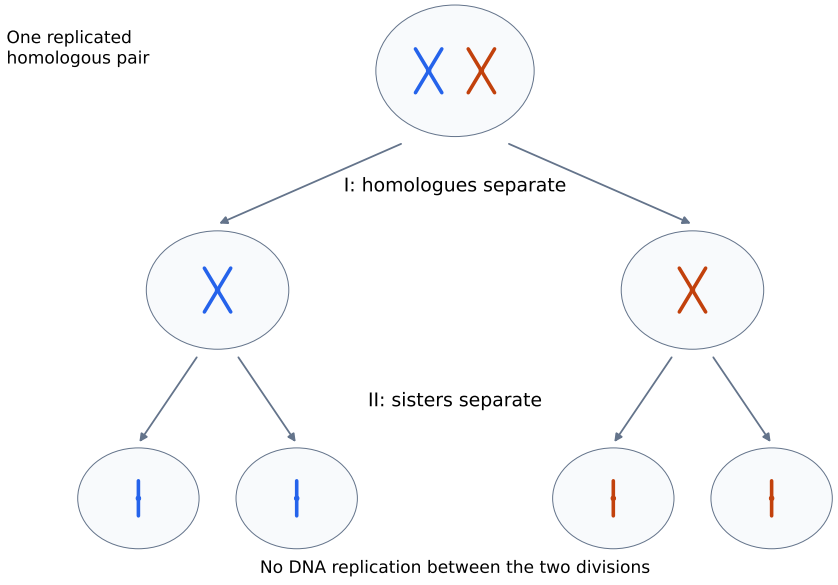
In a practical, use a prepared root-tip slide under a microscope or a supplied micrograph. Locate a region containing dividing cells, then examine several clearly bounded cells at higher magnification. Record a labelled sketch or image for each identified phase, with the chromosome arrangement used as evidence. Keep uncertain cells separate rather than assigning a phase from staining intensity alone.

Meiosis separates homologues first and sisters second

Diploid means two chromosome sets; haploid means one. Meiosis is needed in a sexual life cycle because fertilization joins two haploid contributions. Without reduction somewhere in that cycle, chromosome-set number would double with each generation of fertilization.

After replication, homologous chromosomes pair during meiosis I. Each bivalent contains two homologues and four chromatids. At metaphase I, the bivalents align. Homologues separate in anaphase I while their sister chromatids remain attached. Each product receives one member of each homologous pair, so it is haploid even though each chromosome is still duplicated.

In meiosis II, duplicated chromosomes align and sister chromatids separate, broadly resembling mitotic segregation. No intervening DNA replication is needed. The complete process produces four haploid nuclei from one diploid nucleus. Cytoplasmic division may be unequal, as in oogenesis; four nuclei do not always mean four equal functional gametes.



Colour identifies the two homologues. Meiosis I separates homologues; meiosis II separates sisters. This segregation model omits crossing over so the two events are easy to follow.

For the $2n = 4$ model, replication gives four duplicated chromosomes. Meiosis I gives two products, each with two duplicated chromosomes. Meiosis II gives four products, each with two unduplicated chromosomes. A cell after meiosis I has half as many chromosome sets, not half a set of incomplete genes.

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Crossing over and random orientation generate variation

During prophase I, non-sister chromatids of paired homologues can exchange corresponding DNA regions. Crossing over creates chromatids carrying new combinations of alleles. The exchange is between homologues at corresponding regions; it is not a random transfer of unrelated chromosome pieces. Sister chromatids alone do not account for the usual reshuffling of maternal and paternal alleles.

At metaphase I, each bivalent can orient in either direction relative to the poles. Different bivalents orient independently in the simple model. Thus a product can receive different combinations of maternal and paternal homologues. This is random orientation followed by segregation, not a claim that each gamete must contain exactly half maternal and half paternal chromosomes.

Ignoring crossing over, n independently orienting homologous pairs allow 2^n whole-chromosome combinations across possible meiotic products. For $n = 3$, this is $2^3 = 8$. One meiosis still produces only four nuclei; eight possible types across many divisions does not mean eight products from one division. Crossing over adds further combinations within chromosomes.

Mutation creates new alleles, while these meiotic mechanisms rearrange existing ones. Fertilization adds another source of combination when two gametes join. Distinguish the origin of sequence variation from the redistribution of variants during sexual reproduction.

Non-disjunction can produce an extra chromosome 21

Non-disjunction is failure of chromosomes to separate normally. In meiosis I, both members of a homologous pair may pass to one pole. In meiosis II, sisters may fail to separate. The resulting gametes can contain an extra chromosome or lack one. These errors are changes in chromosome distribution, not automatically a change in one DNA base.

A human gamete containing two copies of chromosome 21 can join a normal gamete containing one. In the usual full-trisomy model, the zygote then has three copies of chromosome 21 and 47 chromosomes in total. Extra chromosome 21 material causes Down syndrome. Other forms involve chromosome rearrangements or mosaicism, so not every case is described completely by the simple 47-chromosome model. NIH explanation of chromosome 21 and non-disjunction.

A simple model of non-disjunction of one pair at meiosis I, followed by normal meiosis II, gives two products with an extra chromosome and two lacking it. If meiosis I is normal but one of its two products has non-disjunction at meiosis II, the four products include two with the usual number, one with an extra and one lacking a chromosome. These are specified segregation models, not probabilities that every human meiosis follows these errors.

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Estimate mitotic index from a defined sample

The **mitotic index** is the fraction of scored cells undergoing mitosis. Count all four mitotic phases in the numerator and all scored cells, including interphase, in the denominator. State whether the result is a fraction or a percentage.

In an invented field, 160 cells are interphase, 20 prophase, 8 metaphase, 7 anaphase and 5 telophase. Total cells = 200; mitotic cells = $20 + 8 + 7 + 5 = 40$. The index is $40/200 = 0.20$, or 20%. A second field with 18 mitotic cells out of 120 gives $18/120 = 0.15$, or 15%. Compare equivalent sampling methods and tissue regions before attributing the difference to a treatment.

Choose fields before seeing which looks most active. Use a clear rule for boundary cells and ambiguous or damaged cells; record exclusions. Include several representative fields and biological replicates. Large nuclei, dark staining or a cell wall alone do not identify mitosis; chromosome arrangement matters.

A snapshot gives a population fraction, not the directly observed duration of one cell's division. If a representative asynchronous population has a steady cycle and comparable survival, a rough phase-time estimate can use its fraction of cells. If 20% are mitotic and the known cycle lasts 15 hours, estimated mitotic duration is $0.20 \times 15 = 3$ hours under those assumptions. Synchronized cells, selective cell loss, non-cycling cells or a drug that arrests mitosis can break the model. A high index can reflect slow exit from mitosis rather than fast production of daughter cells.

AHL: Cell proliferation supports growth and tissue maintenance

Cell proliferation increases cell number through repeated cell cycles. In plant meristems, division supplies new cells for growth; those cells can later expand and differentiate. Early animal embryos also undergo rapid divisions. Initial cleavage can partition existing cytoplasm into more, smaller cells without a matching increase in total embryo mass.

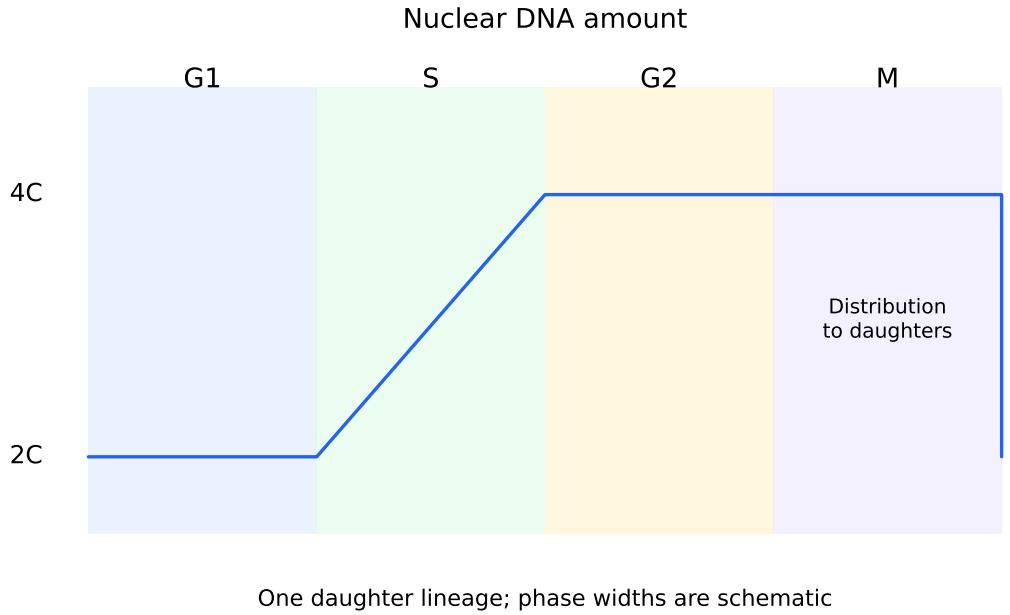
In skin, dividing cell lineages replace cells lost through routine wear. Following a wound, proliferation helps supply cells for repair alongside migration and other tissue processes. Growth of an individual cell and an increase in cell number are distinct: cell enlargement alone is not proliferation, and a larger tissue does not automatically prove a higher instantaneous division rate.

AHL: The cell cycle includes active interphase and regulated division

The cycle proceeds through G1, S and G2, then mitosis and cytokinesis. G1, S and G2 form interphase. Cells grow and synthesize components during interphase; it is not a resting gap in which nothing happens. DNA is replicated during S, while proteins and other components are made as needed across interphase. Mitochondria and

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chloroplasts increase through growth and division of existing organelles.



The line tracks one daughter lineage. DNA amount rises during S and returns to 2C per daughter after division. Phase widths are schematic, not measured durations.

For example, a fictional cycling cell has 6 pg of nuclear DNA in G1. It reaches 12 pg after S and remains at that amount through G2 until the copies are distributed. Each normal daughter receives 6 pg. The whole parent cell at anaphase still contains 12 pg; it does not lose half its DNA just because the chromosomes have begun to move.

Cyclin concentrations rise and fall during the cycle. Passing a checkpoint requires an appropriate cyclin-dependent control system, including a threshold level of a relevant cyclin. A checkpoint can prevent progression when conditions are unsuitable. This is regulated molecular control, not an organism deciding consciously to divide. Different cyclins act at different stages; memorizing specific cyclin names is not needed here.

A high cyclin concentration by itself does not prove every checkpoint condition is satisfied. A cell may pause because of DNA damage or another problem. Similarly, not every cell in a tissue is continuously cycling; some differentiate or leave the proliferating population.

AHL: Loss of growth control can produce different kinds of tumour

Proto-oncogenes normally participate in controlled growth and division. Some mutations convert them to oncogenes with excessive or inappropriate growth-promoting activity. Tumour suppressor genes normally help limit proliferation or support protective controls; loss of their function can remove a restraint. These are different routes to loss of control, not a rule that every mutation turns every gene into an oncogene.

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Accumulated changes can produce a tumour, an abnormal mass of cells. Tumours differ in growth rate and capacity to invade neighbouring tissue or spread. A benign tumour remains localized and does not invade or metastasize in the way a malignant tumour can, though its location or size can still cause harm. Malignant tumours are cancerous and can invade surrounding tissue.

Metastasis is spread of cancer cells to another location, where a secondary tumour can form. The primary tumour is the original site. A secondary tumour derived from the primary retains that cancer's origin; it is not simply an unrelated new cancer of the tissue in which it settles. Fast growth alone does not establish metastasis, and a low measured division fraction does not prove a tumour benign.

Practice

1. Distinguish nuclear division from cytokinesis. Explain why a nucleated eukaryotic cell needs appropriate nuclear distribution before daughter cells separate.
2. Compare animal and plant cytokinesis. Why can equal chromosome distribution accompany unequal cytoplasmic division? Give two examples of unequal division.
3. An invented species has $2n = 6$. Before replication a cell contains 6 pg of DNA. Give the chromosome count, number of DNA molecules and DNA mass after replication but before sister separation.
4. For that species, give the chromosome count in the whole anaphase cell before cytokinesis and in each daughter nucleus after normal mitosis. Explain why the counts differ.
5. A diagram shows duplicated chromosomes individually aligned at the equator; another shows sisters moving apart. Identify the mitotic phases and give the evidence. State how histones, microtubules and motors contribute to division.
6. Track the $2n = 6$ cell through meiosis I and II. Give chromosomes and DNA mass per product after each division, and state when replication occurs.
7. Explain how one diploid nucleus can give four haploid nuclei without a second round of DNA replication. Why does fertilization make reduction necessary in a sexual cycle?
8. Distinguish crossing over from random orientation. With four independently orienting pairs, how many whole-chromosome combinations are possible when crossing over is ignored? Does one meiosis produce that many nuclei?
9. Explain how a gamete with two copies of chromosome 21 can contribute to full trisomy 21. Distinguish this chromosome-distribution error from a base substitution.
10. In the specified one-pair model, compare products of meiosis I non-disjunction followed by normal meiosis II with products when only one meiosis II division has non-disjunction.

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11. Explain why daughter cells need organelles as well as a nucleus. Why is a rise in cell number different from growth of an individual cell? Calculate mitotic index for 24 dividing cells among 160 scored cells.
12. **AHL:** Describe G1–S–G2–M and cyclin-dependent checkpoints. A fictional cell has 8 pg nuclear DNA in G1. Give its mass after S and in each normal daughter. Why is interphase active?
13. **AHL:** Compare proto-oncogene activation and tumour-suppressor loss. Distinguish benign, malignant, primary and secondary tumours; can tumour behaviour be inferred from size alone?
14. **AHL:** An invented sample has 210 interphase, 30 prophase, 12 metaphase, 9 anaphase and 9 telophase cells. Calculate mitotic index as a fraction and percentage. With a 20-hour cycle, estimate mitotic duration under the population model. Explain two sampling precautions and why a drug that arrests metaphase invalidates the claim that a high index proves faster cell production.

Worked answers

1. Nuclear division distributes chromosomes into daughter nuclei; cytokinesis separates cytoplasm into cells. Appropriate distribution helps avoid anucleate products or incomplete chromosome sets. Their timing can overlap, but they are different processes.
2. Animal cells use an actin–myosin contractile ring and cleavage furrow. Plant cells build a cell plate from vesicles, producing separating membranes and wall. Nuclear sets can be distributed appropriately while cytoplasmic amounts differ: human oogenesis produces a large egg lineage and small polar bodies; budding yeast produces a smaller bud.
3. Six chromosomes, each with two sister chromatids: twelve chromatids and twelve DNA molecules, with 12 pg DNA in the whole cell. Replication doubles DNA amount without doubling the attached-chromosome count.
4. Twelve daughter chromosomes in the whole anaphase cell, six moving to each pole. After division, each daughter nucleus has six. Separation changes the counting units before the cell itself separates; it does not double chromosome number in each normal daughter.
5. Individual duplicated chromosomes at the equator support metaphase; separating sisters support anaphase. Histone-associated packaging helps condense DNA; spindle microtubules and motor proteins help position and move chromosomes. Homologous pairs aligned as bivalents would instead suggest metaphase I.
6. After meiosis I, each of two products has three duplicated chromosomes and 6 pg DNA. After meiosis II, each of four products has three unduplicated chromosomes and 3 pg DNA. Replication occurs before meiosis I, not between I and II.

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7. Replication makes two chromatids per chromosome. Homologues separate in meiosis I, reducing two sets to one; sisters separate in II, producing four haploid nuclei. Fertilization restores two sets, so reduction prevents repeated chromosome-set doubling across generations.
8. Crossing over exchanges corresponding DNA between non-sister chromatids of homologues. Independent bivalent orientation redistributes whole homologues. $2^4 = 16$ whole-chromosome combinations are possible across many divisions; one meiosis makes four nuclei, not sixteen.
9. Two chromosome 21 copies from one gamete plus one from the other give full trisomy 21, with 47 chromosomes. Non-disjunction redistributes chromosomes; substitution changes a base. Rearrangement and mosaic forms mean this full-trisomy model does not describe every Down syndrome case.
10. With other steps normal, a meiosis I error gives two products with an extra chromosome and two lacking it. An error in one meiosis II cell gives two normal-number products, one extra and one lacking.
11. Daughters need existing mitochondria and any chloroplasts to maintain organelle functions and growth/division; nuclear DNA alone is insufficient. Proliferation raises cell number, whereas enlargement can occur without division. Index $= 24/160 = 0.15 = 15\%$: a snapshot fraction, not a direct division rate.
12. **AHL:** G1, S and G2 form active interphase: S replicates DNA; biosynthesis and organelle growth/division also support growth. Mitosis and cytokinesis distribute copies. DNA rises from 8 to 16 pg, then each daughter receives 8 pg. Changing cyclin concentrations regulate checkpoints, but other conditions must also be met.
13. **AHL:** Proto-oncogene activation increases growth-promoting activity; tumour-suppressor loss removes restraint. Benign tumours lack malignant invasion/metastasis; malignant tumours can invade. Primary means the original site; secondary means establishment after spread. Size alone establishes none of these behaviours or origins.
14. **AHL:** Total $= 210+30+12+9+9 = 270$; mitotic $= 60$. Index $= 60/270 = 2/9 \approx 0.222$, or 22.2%. Estimated duration $= (2/9) \times 20 = 40/9 \approx 4.44$ hours, assuming a representative asynchronous steady cycling population. Preselect representative fields; apply a consistent boundary/ambiguity rule, record exclusions and repeat suitable samples. Metaphase arrest slows exit and accumulates mitotic cells, so the index can rise while daughter-cell production falls.

Exam tips

Name the boundary and distinguish chromosome count from DNA amount. Identify phases by chromosome arrangement: mitosis separates sisters, meiosis I homologues, meiosis II sisters. Explain variation through non-sister crossing over and independent bivalent orientation.

State the denominator and sampling rules. Phase times need a valid population model.

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Gene expression

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This handout covers all eleven D2.2 objectives: gene expression, transcription control, mRNA degradation, differentiation and epigenetic inheritance. D2.2 is Higher Level only; there is no SL counterpart.

Prerequisites: DNA, transcription and translation, mutations, cell division and the distinction between genotype and phenotype. Numerical datasets and simplified regulatory diagrams are invented teaching models unless a published study is named.

Gene expression connects genetic information with phenotype

A liver cell and a nerve cell can contain essentially the same genome but have different shapes, proteins and functions. The difference need not come from deleting every unused gene. Cells use different parts of their inherited information and regulate how much product they make.

Gene expression is the process through which information in a gene contributes to a functional product and hence to phenotype. For a protein-coding gene, the common sequence is transcription into RNA, translation into a polypeptide, then protein function. An enzyme can alter the rate of a metabolic reaction; a structural protein can affect a cell's shape. Some genes make functional RNAs instead of polypeptides.

Do not treat the arrows as conversion of DNA material into protein. The DNA template is normally retained. Expression can change while the DNA base sequence stays the same, although a mutation in a regulatory or coding region can also affect expression or function.

More transcript does not necessarily mean proportionally more active protein. Translation efficiency, mRNA lifetime, protein degradation, processing and availability of interacting molecules can intervene. Measure the level relevant to the claim: RNA abundance, protein abundance, enzyme activity and a whole-cell phenotype are distinct outcomes.

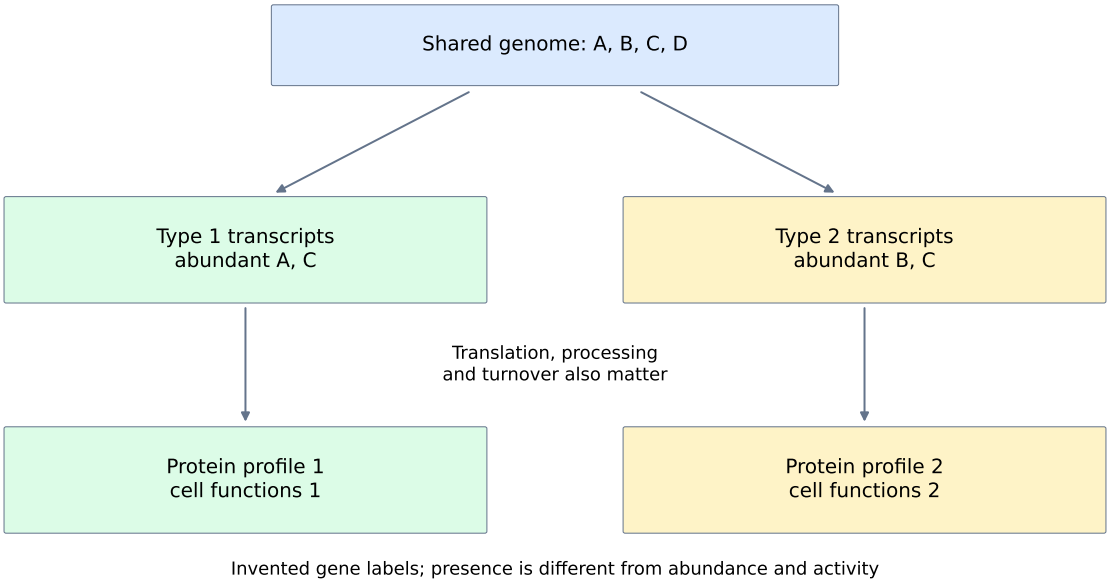
Genome, transcriptome and proteome describe different sets

The genome is the cell's complete genetic information. The transcriptome is the set of RNA transcripts present under specified conditions. The proteome is the set of proteins present. RNA and protein sets and their amounts can change with cell type, developmental stage and environment.

Most differentiated nucleated cells in an individual share essentially the same genome, yet no cell expresses every gene at the same time. A muscle cell requires a different expression pattern from a pancreatic beta cell. These are useful comparisons of spe-

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cialization; mature red blood cells lacking nuclei are not a suitable example of two nucleated cells retaining identical nuclear genomes.



The invented gene labels A–D are placeholders, not real gene names. A gene can remain present when its transcript is absent or below detection.

Suppose two fictional cell types both contain genes A, B, C and D. Type 1 has abundant transcripts A and C; type 2 has abundant B and C. Shared DNA does not require shared transcript abundance. Nor does the presence of transcript C establish that both cells contain equal amounts of active protein C.

Cell differentiation develops patterns of specialization through selective expression. Epigenesis describes the development of these patterns in a multicellular organism. A stable expression pattern can help a daughter cell retain its tissue identity through subsequent divisions, without changing its genotype at the relevant DNA sequence.

DNA-binding proteins regulate transcription

A **transcription factor** is a protein that influences transcription through interactions with particular DNA sequences and other components of the transcription machinery. The sequence matters: binding depends on molecular recognition, not an organism consciously choosing a useful gene.

A promoter is a DNA region associated with initiation of transcription. Suitable transcription factors and RNA polymerase machinery must assemble appropriately for transcription to begin. An enhancer is a regulatory DNA sequence where bound proteins can increase transcription of a target gene. An enhancer may be far from the promoter in the linear DNA sequence; folding can bring the interacting components closer in space.

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Several factors can act together. A cell may express a gene only when the required combination is available, and regulatory proteins can also reduce transcription. A binding event alone does not prove a gene is fully active. Chromatin accessibility, other regulatory partners and the cell's conditions matter.

The promoter and enhancer are DNA, whereas a transcription factor is protein. None needs to be translated into an amino acid before it can act as a regulatory sequence. A promoter mutation can alter factor binding; changing the concentration of a factor can also alter expression without any mutation in the target gene.

A hormone can change the transcriptional context

Cortisol is a steroid hormone. In a responsive cell it can bind an intracellular glucocorticoid receptor. The activated receptor can interact with specific regulatory DNA regions and other proteins to change transcription of target genes. The response depends on which regulatory sites and partners are accessible in that cell. This illustrates hormone-dependent gene regulation; it does not mean cortisol increases transcription of every gene.

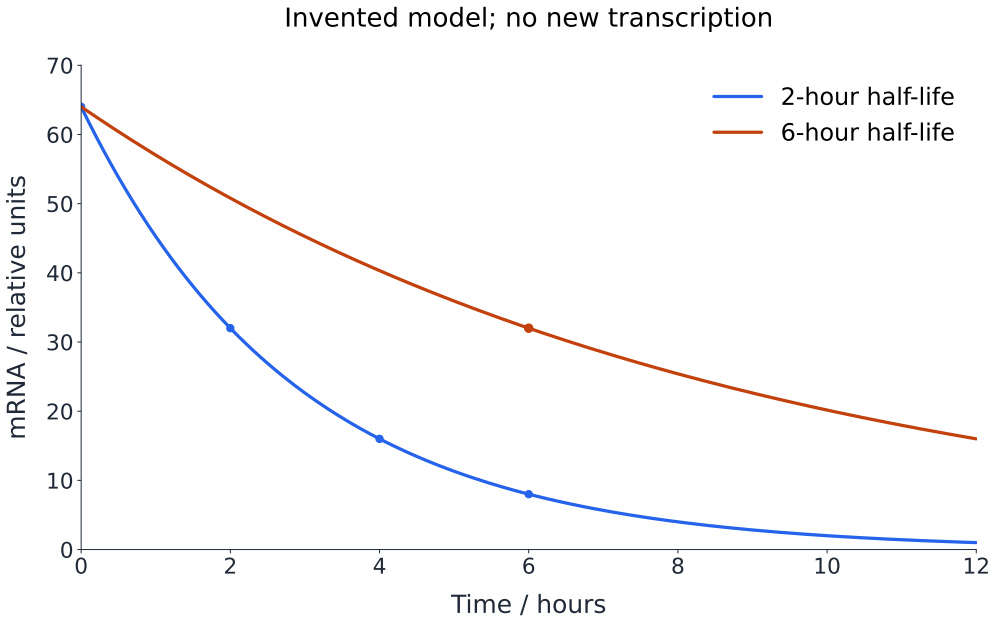
Distinguish this slower change in gene products from a signal that simply changes the activity of an enzyme already present. Both can occur in cell signalling, but they answer different mechanistic questions. For the transcriptional pathway, ask about receptor availability, target-gene RNA and subsequent product or function.

mRNA degradation controls how long a message remains available

An mRNA molecule can be translated repeatedly while it remains intact and available. Nucleases eventually break it down. Human mRNA lifetimes vary greatly, from minutes to days. Shorter persistence can let a cell change its protein-production pattern quickly; a longer-lived message can support translation for longer without being transcribed again each time.

Abundance reflects production as well as removal. A higher measured mRNA amount could mean more transcription, slower degradation or both. Measuring RNA at one time point cannot distinguish these possibilities by itself. A suitable time course after stopping new transcription can help estimate message persistence, although the intervention's other effects must also be considered.

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This model uses constant half-lives of two and six hours. It isolates degradation; real messages and experimental conditions need not follow a single constant half-life.

For a fictional message starting at 64 relative units, a two-hour half-life gives 32 after two hours, 16 after four and 8 after six. With a six-hour half-life, 32 remain after six hours. The difference does not require different starting DNA or different initial RNA abundance.

Protein already made can persist after its mRNA disappears. Conversely, an abundant message can yield little active product if translation is inhibited or the protein is rapidly removed. Match the time scale and measured molecule before explaining a phenotype.

Epigenetic tags change expression without changing the base sequence

An **epigenetic modification** can affect expression without changing the underlying DNA base sequence. DNA methylation and histone modification are examples. A methyl group attached to cytosine does not substitute thymine or another base for that cytosine; the base sequence is preserved.

In the promoter model used here, cytosine methylation represses transcription of the downstream gene. It can help maintain a less active state. Do not generalize this to every methyl group anywhere in a genome: position and molecular context matter.

DNA is packaged around histone proteins in nucleosomes. Histones can also carry methyl groups on particular amino acids. Histone methylation can be associated with repression or activation depending on the residue and modification pattern. The rule "all methylation switches every gene off" is therefore wrong. Specific histone sites and enzyme names are not needed for this unit.

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These tags influence how genetic information is used. They do not erase the distinction between genotype and phenotype: two cells can differ in epigenetic state and gene expression while retaining the same nucleotide sequence. A sequence mutation and an epigenetic change are different explanations requiring different evidence.

Epigenetic inheritance and resetting have different roles

Epigenetic inheritance means that an expression state can pass to daughter cells, or sometimes to offspring, without a change in DNA sequence. Persistence or restoration of DNA methylation and histone states through cell division can help retain a pattern of differentiation.

This does not mean every tag persists indefinitely or every environmentally induced change reaches future generations. Many epigenetic states are reset during germ-cell and early embryonic development. Most tags are removed, while certain parent-of-origin marks can escape particular resetting stages or be maintained through them. Imprints are also erased and established afresh in the developing germ line according to the sex of the individual making the gametes.

Genomic imprinting produces parent-of-origin effects: the activity of an allele can depend on whether it came from the mother or father. Both copies can be present in DNA even when their expression differs. Imprinting does not mean an offspring has only one parent's entire genome.

■ Reciprocal lion–tiger hybrids illustrate why parent of origin matters

A liger has a lion father and tiger mother; a tigon has a tiger father and lion mother. Ligers commonly show greater growth than tigons. These reciprocal crosses involve the same two parent species but reverse which species supplies the maternal and paternal contributions.

The course uses these growth differences to illustrate an imprinting model. Parent-specific expression of growth-regulating genes and differences in retained epigenetic marks can contribute to different growth regulation in reciprocal hybrids. Tags are not all removed and converted into identical expression states at fertilization.

Treat the model with appropriate limits. Body size alone does not identify a particular methylated site or prove that one named gene explains every hybrid. Maternal environment, species-specific regulation and other genetic interactions can also contribute. The key reasoning is that similar species contributions do not guarantee identical expression when parent of origin is reversed.

Environmental exposures can alter methylation patterns

Environmental conditions can change gene expression directly through signalling and can also alter epigenetic states. Air pollution provides an example of changes in DNA methylation. A pollution-related methylation difference is a chemical-tag difference, not automatically a change in the DNA base sequence.

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A 2021 controlled crossover study by Cardenas and colleagues compared diesel exhaust with clean air in 13 volunteers and measured DNA methylation in bronchial epithelial cells. It found methylation differences at particular sites and regions after exposure. This supports an effect on the measured epigenome under those study conditions. It does not establish that every site changes, that every tag persists for life, or that the observed changes are inherited by offspring. Researchers' study summary at the US EPA.

To investigate a proposed regulatory effect, measure the relevant methylation, transcript and functional outcome, with comparable tissue and controls. An association between exposure and a tag is not by itself proof of the entire causal chain from tag to disease. Cell composition, prior exposure, timing and other covariates can affect comparisons.

Monozygotic twins help separate shared DNA from different expression

Monozygotic twins develop from one zygote and share essentially the same inherited DNA sequence. This makes paired comparisons useful when investigating environmental influences on expression. It does not eliminate every possible difference: later mutations, developmental conditions, cell composition and chance processes can also matter.

In a 2005 study, Fraga and colleagues examined 40 monozygotic twin pairs. Fourteen pairs, 35%, differed significantly in the global DNA-methylation and histone-acetylation measures examined. Differences were associated with age; the researchers also compared expression in selected younger and older pairs. These results support epigenetic divergence without requiring different inherited genomes. Original study.

The study did not randomly assign lifelong environments. It therefore cannot prove that one specific lifestyle caused every observed difference. Internal epigenetic drift can also contribute. Nor does 35% mean "35% of all human genes differ" or that the remaining pairs were identical at every possible site.

A stronger design for a particular exposure compares the same tissue, checks cell composition, records exposure and relevant covariates, uses adequate pairs and follows changes over time. A within-pair difference reduces some genetic confounding but is not a complete causal explanation by itself.

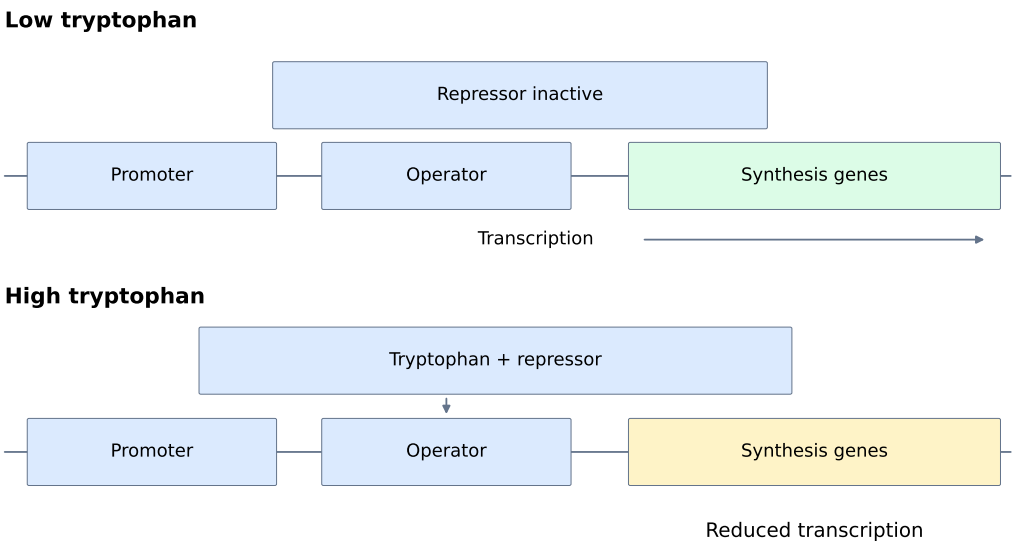
A bacterial end product can regulate the genes that make it

Tryptophan is an amino acid. In the simplified trp-operon repression model, its abundance influences transcription of genes encoding enzymes for tryptophan synthesis.

When tryptophan is scarce, the repressor is inactive in the relevant binding state and does not bind the operator effectively. Transcription of the biosynthetic genes can proceed. When tryptophan is plentiful, it acts as a corepressor: binding changes

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the repressor so it can bind the operator and reduce transcription. The end product therefore helps limit production of enzymes that make more of it.



Repression model only; DNA sequences and protein are different components

The diagram isolates repressor control. It is not a complete map of every regulatory mechanism affecting the real operon.

The operator and promoter are DNA sequences; the repressor is protein; tryptophan is the small molecule that changes its binding behaviour. Tryptophan does not itself become a DNA-bound transcription factor. The response changes transcription of the target biosynthetic genes, not every gene in the bacterial genome.

This is negative feedback in gene expression. It differs from direct inhibition of an enzyme already present, which can change pathway activity faster without first changing transcript supply. Both kinds of control can help regulate metabolism.

Compare expression measurements before calculating fold change

Fold change is a ratio of comparable treatment and control quantities. If normalized transcript abundance is 12 in treatment and 3 in control, fold change is $12/3 = 4$. This is a fourfold level, not a 4% increase. Relative increase is $(12 - 3)/3 \times 100 = 300\%$.

Normalization matters. Suppose target/reference measurements are 60/20 in a control and 120/40 in a treatment. Both normalized ratios equal 3, so normalized fold change is $3/3 = 1$, despite twice as much raw target signal. Equal normalized values do not imply equal absolute cell counts in the original samples.

Report biological replication and variation. A single treatment/control ratio does not establish statistical significance. A transcript increase alone does not prove direct transcription-factor binding or proportional enzyme activity; use appropriate pertur-

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bation and downstream measurements to test those claims.

Practice

1. Explain how two differentiated nucleated cells can retain essentially the same genome but differ in transcriptome, proteome and phenotype.
2. Trace how information in a protein-coding gene can affect a metabolic phenotype. Why does more RNA not guarantee proportionally more active enzyme?
3. Distinguish promoter, enhancer and transcription factor. How can an enhancer distant in linear DNA influence transcription?
4. Explain one hormone-dependent route from cortisol to altered gene expression. Distinguish this from changing the activity of an existing enzyme.
5. An invented mRNA sample starts at 80 relative units and has a ten-minute half-life with no new transcription. Calculate the amount after 30 minutes. Why need protein abundance not fall in the same proportion?
6. Distinguish promoter cytosine methylation from a base substitution. Why is "all histone methylation represses transcription" incorrect?
7. Explain how epigenetic inheritance can preserve a cell's differentiated state. Why does this not prove every acquired tag passes to grandchildren?
8. Identify the parents of a liger and a tigon. Outline the parent-of-origin explanation for their growth differences and a limitation on inferring a specific mechanism from size alone.
9. Describe what the named diesel-exposure study measured. Give one supported conclusion and two claims its findings do not establish.
10. In the twin study, calculate the percentage represented by 14 of 40 pairs. Why does this result not establish that a particular lifestyle caused every difference? Give two useful design controls.
11. Explain the low- and high-tryptophan states of the simplified trp repression model, naming DNA, protein and small-molecule components.
12. Invented normalized transcript abundances are 20 in treatment and 4 in control. Calculate fold change and percentage increase. What additional evidence would help test a claim of greater functional enzyme output?
13. Raw target/reference values are 45/15 in a control and 90/30 in a treatment. Calculate each normalized value and normalized fold change. Explain why the raw target ratio alone would mislead.

Worked answers

1. Cells can retain the same genes while selectively expressing different subsets and amounts. Different transcript sets, translation, processing and degradation yield different protein sets and functions. The genome describes genetic information, the transcriptome RNA present, and the proteome proteins present under stated

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conditions.

2. Transcription makes RNA, translation makes a polypeptide, and a functional enzyme changes reaction activity, potentially affecting phenotype. RNA lifetime, translation efficiency, protein processing/degradation and other required molecules intervene; measure RNA, protein and activity separately.
3. Promoter and enhancer are regulatory DNA; a transcription factor is protein. Promoter-associated machinery supports initiation; enhancer-bound proteins can influence it through spatial interactions as DNA folds. Regulatory partners and accessible chromatin affect the outcome.
4. Cortisol can bind an intracellular glucocorticoid receptor; activated receptor interactions with regulatory DNA and partners alter target transcription. The cell's receptor and regulatory context matter. Direct enzyme activation changes an existing protein's activity without necessarily changing RNA or making new protein.
5. Thirty minutes is three half-lives: $80 \to 40 \to 20 \to 10$ units. A stable existing protein can persist after message degradation; new translation and protein removal have their own time scales.
6. A methyl tag on promoter cytosine leaves the nucleotide sequence intact; substitution changes a base. Promoter methylation represses transcription in this model. Histone methylation can activate or repress depending on the modified site and context.
7. Persistent or restored tags can maintain expression patterns through daughter-cell divisions without sequence change. Germ-cell and early embryo reprogramming removes many tags; inheritance of a particular acquired state across generations requires evidence, not an assumption from mitotic persistence.
8. Liger: lion father, tiger mother. Tigon: tiger father, lion mother. Retained parent-of-origin marks can produce different regulation of growth-related genes in reciprocal crosses. Size alone identifies neither a specific tag nor a complete single-gene cause; maternal and other regulatory/genetic effects may contribute.
9. It compared diesel and clean-air exposure in a controlled crossover design and measured bronchial-cell DNA methylation. Differences at specific sites support an exposure effect on the measured epigenome. They do not establish universal changes at all sites, lifelong persistence, disease causation through those tags or inheritance by offspring.
10. $14/40 \times 100 = 35\%$ of the studied pairs for the measures used. Age and environment were not randomly assigned; internal drift and other differences can contribute. Compare the same tissue/cell composition, record exposure and covariates, and use replicated or longitudinal measurements. Shared inherited DNA reduces some confounding but proves no specific lifestyle mechanism.
11. Low tryptophan: inactive repressor does not effectively bind operator, permitting transcription. High tryptophan: the small molecule acts as corepressor, allowing the repressor protein to bind the DNA operator and reduce transcription of biosynthetic genes. Promoter/operator are DNA; repressor is protein; tryptophan

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is neither.

12. Fold change = $20/4 = 5$; relative increase = $(20-4)/4 \times 100 = 400\%$. Use biological replicates and variation, protein abundance and enzyme-activity measurements with appropriate controls; transcript amount alone does not establish proportional function or significance.
13. Control $45/15 = 3$; treatment $90/30 = 3$; normalized fold change = $3/3 = 1$. Raw target signal doubles, but the reference also doubles. These measurements show unchanged normalized target abundance, not necessarily equal absolute RNA or cell counts in the original samples.

Exam tips

Name the measured molecule and the regulatory stage. Separate DNA sequence, epigenetic state, RNA, protein and function. Distinguish mitotic persistence from inheritance across generations, and association from a tested causal mechanism. For numerical comparisons, normalize first, state the ratio's direction and distinguish fold level from percentage increase.

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Water potential

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

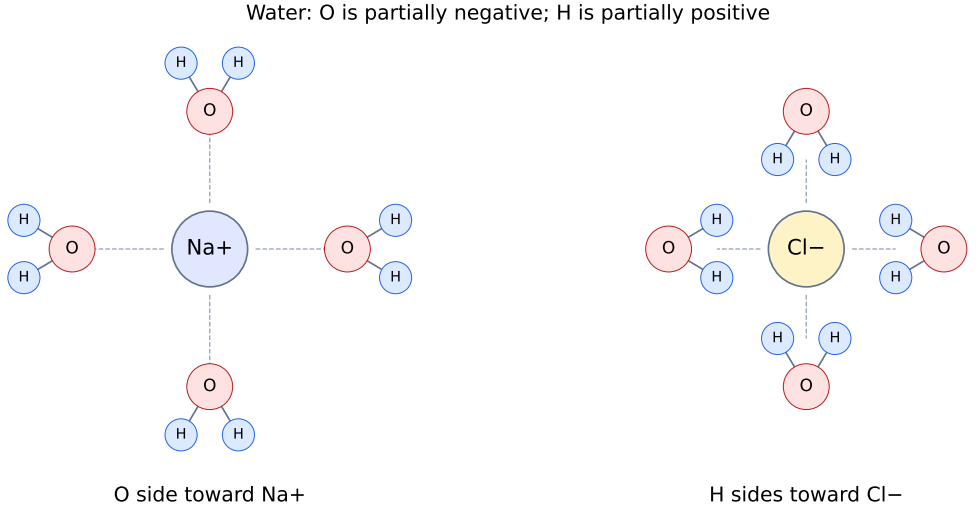
This handout covers D2.3: solvation, osmosis, responses of cells with and without walls, tissue investigations and isotonic applications. It retains the existing qualitative introduction to water potential. Sections marked **AHL** are for Higher Level only.

Prerequisites: water polarity, membranes, diffusion and plant-cell structure. All diagrams, measurements and numerical examples below are invented teaching models, not results from real organisms or examinations.

Solvation connects water polarity with dissolved particles

A potato strip can become firm in one solution and flexible in another. To explain this, begin with interactions between water and dissolved particles, then ask what can cross the cell membrane. The cell wall and any pressure that develops also matter.

Water is polar: oxygen has a partial negative charge and the hydrogen atoms have partial positive charges. In a solution of sodium chloride, water molecules orient their oxygen side toward sodium ions and their hydrogen sides toward chloride ions. Attractions between ions and water help surround and separate the ions. The dissolved salt is not present only as intact solid grains hidden between water molecules.



These schematic hydration arrangements show orientation, not a fixed number or permanent shell of neighbouring water molecules. Dashed attractions are not new covalent bonds.

Polar molecular solutes can interact with water through hydrogen bonding. For ex-

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ample, the hydroxyl groups of glucose can form hydrogen bonds with surrounding water. Glucose dissolving does not require the glucose molecule to split into sodium-like ions. Solvation is the surrounding of solute particles by solvent molecules; water is the solvent in these examples.

Do not describe all solute–water attractions as hydrogen bonds. An ion attracts the appropriately charged region of a polar water molecule; suitable polar groups on molecular solutes can participate in hydrogen bonding. The interactions and particle properties influence how readily substances dissolve.

Osmosis compares solutions across a selectively permeable membrane

Osmosis is the net movement of water through a selectively permeable membrane. For solutions at equal pressure, with solute particles that cannot freely cross that membrane, water moves overall from the less concentrated solution toward the more concentrated solution. Use solute concentration to state this comparison. Individual water molecules move both ways, but unequal rates produce a net transfer.

The membrane is essential. Mixing two solutions without a separating membrane also permits solute diffusion; it is not the same experiment. A selectively permeable membrane can allow water through while restricting particular dissolved particles. Many cells also have water-channel proteins, but a channel does not actively pump water against the relevant gradient.

Tonicity is a comparison relative to a particular cell and membrane. A hypotonic external solution favours net water entry, a hypertonic one favours net water loss, and an isotonic one produces no sustained net change in cell volume under the specified conditions. In a simple classroom model these correspond to lower, higher and equal effective solute concentration outside, respectively.

The qualification about effective solutes matters: equal total solute concentration does not always give equal tonicity if one solute readily enters cells. State the membrane conditions rather than assuming every dissolved substance has the same effect. A solution is not simply “hypertonic” without a comparison.

At dynamic equilibrium, exchange continues but opposing transfers balance. A steady cell volume does not show that all water molecules have stopped. Osmosis itself does not require ATP expenditure to drive water movement, although cells can use energy to regulate solute distributions or remove excess water.

Cells without a wall can swell, burst or shrink

An animal cell lacks a rigid surrounding cell wall. In a sufficiently hypotonic medium it takes up net water and swells; its membrane can eventually rupture. This is lysis. The cell cannot build up the same wall-supported turgor as a plant cell. It is the osmotic gain and limited capacity to expand that matter, not the outside solution physically

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cutting the membrane.

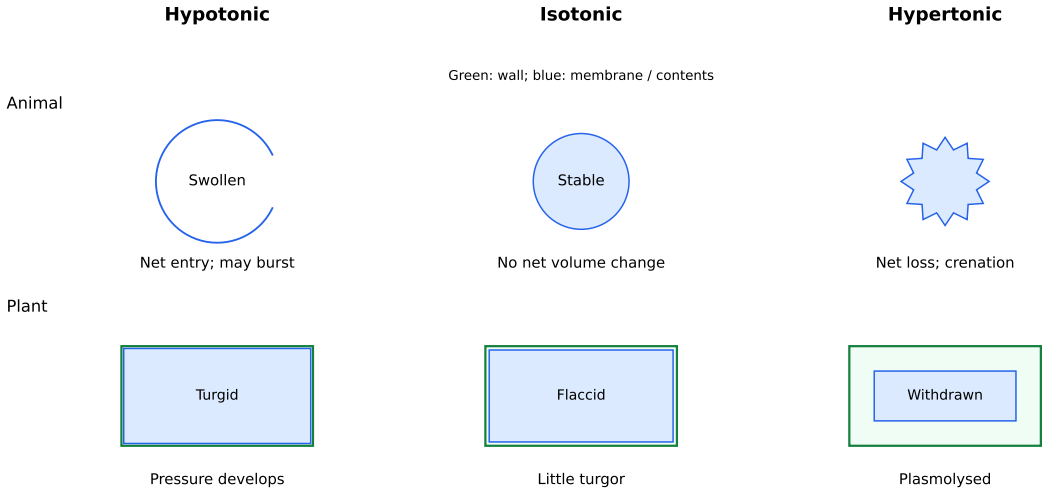
In a hypertonic medium, net water loss reduces cell volume. A red blood cell can develop an irregular, shrunken outline called crenation. This differs from plasmolysis: the latter specifically involves the cell contents and membrane pulling away from a cell wall.

In an isotonic environment, an animal cell can maintain a relatively stable volume while water exchange continues. Multicellular animals regulate tissue fluid so that damaging sustained osmotic swelling or shrinkage is avoided. Isotonic does not mean chemically identical in every respect to the cytoplasm; ions, proteins and other substances can have different distributions.

Freshwater unicellular organisms face a different problem: their surroundings are commonly hypotonic relative to their contents. In organisms such as freshwater Paramecium, contractile vacuoles collect excess water and periodically expel it. Energy-dependent cellular processes support this removal. The water initially entered down an osmotic gradient; the organism does not stop all osmosis simply by living in freshwater.

A plant cell wall changes the outcome of water entry

A plant cell in a hypotonic medium takes up water. Its vacuole and contents expand, pressing the plasma membrane against the wall. The wall resists further expansion and **turgor** develops: pressure of cell contents against the wall. The wall's opposing force limits net entry; it is not a selectively permeable lid that closes every pore.



The plant wall remains outside the membrane. The plasmolysed model shows the membrane and contents withdrawn from the wall; the wall itself has not shrunk to match them.

A turgid cell helps support soft plant tissues. Loss of turgor can make tissue less firm and contribute to wilting. In a simplified isotonic comparison with little wall-supported pressure, a plant cell is flaccid rather than fully turgid. Its wall still exists;

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absence of strong turgor does not mean the wall has dissolved.

In a sufficiently hypertonic medium, water leaves, the vacuole and cell contents shrink, and the membrane can detach from the wall. This is plasmolysis. The wall is relatively permeable to water and many small solutes; the plasma membrane is the main selective boundary for the osmotic comparison.

If a suitable plasmolysed cell is returned to a hypotonic medium, water can re-enter and restore contact and turgor. This is not a guarantee that every damaged cell will recover: exposure duration and membrane injury matter. State the observed structure and the direction of net water movement before naming the condition.

Investigate tissue mass and length with a controlled comparison

Cut plant-tissue pieces from a comparable source, with a consistent diameter and length. Randomly allocate pieces across teacher-approved solution concentrations. Record initial mass or length, expose them for the same time at a controlled temperature, then measure again. Keep solution volume adequate and consistent, and cover containers when evaporation could change concentration.

For mass, blot in a consistent way before weighing so surface solution does not masquerade as water gained by tissue. Avoid squeezing different pieces with different force. Use the same measurement procedure and record instrument resolution. For length, measure comparable axes and avoid treating a curved strip's straight-line distance as its full length.

Use independent tissue pieces as replicates at each concentration. Reweighing the same piece improves a measurement check but does not supply another independent biological sample. Tissue source, age, damage, initial water status and variation among pieces can affect results. A whole tissue is not one perfectly uniform cell compartment.

Percentage change allows a fairer comparison when starting sizes differ:

$$\text{percentage mass change} = \frac{m_{\text{final}} - m_{\text{initial}}}{m_{\text{initial}}} \times 100.$$

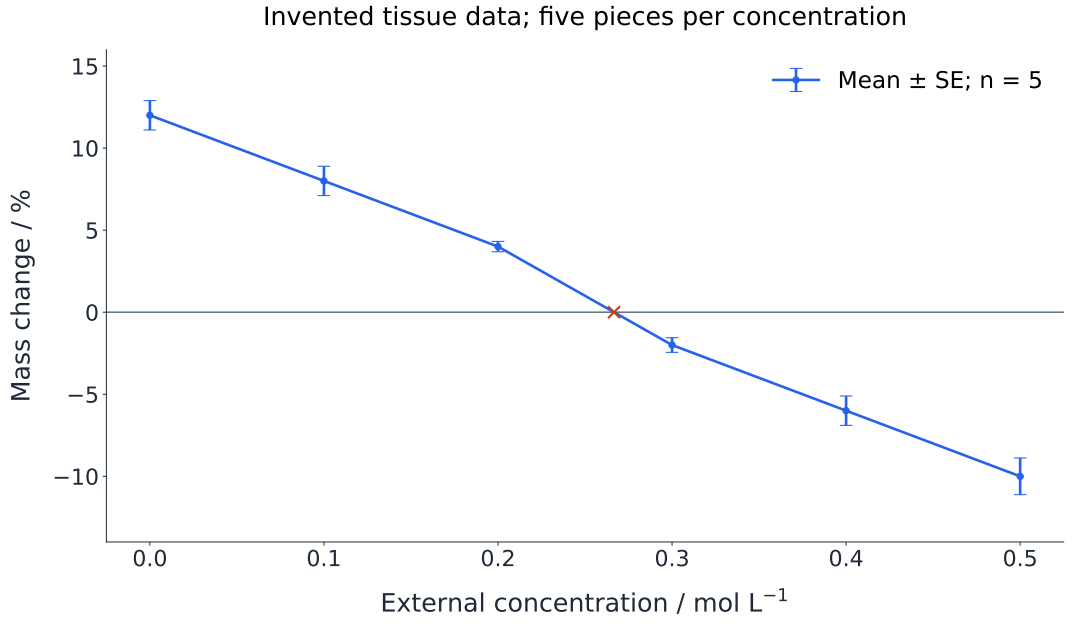
An invented sample changes from 5.00 g to 5.40 g. The increase is 0.40 g and the percentage change is $0.40/5.00 \times 100 = +8\%$. A 4.00 g piece changing to 4.20 g gains 5%. The first has the larger proportional gain. A piece changing from 3.00 g to 3.36 g gains 12%, but the reverse change is $-0.36/3.36 \times 100 \approx -10.7\%$: the denominator changes.

Use the same structure for percentage length change, replacing masses with lengths in consistent units. A strip going from 40.0 mm to 42.0 mm has gained $2.0/40.0 \times 100 = 5.0\%$. A mass and a length percentage need not be equal because volume, shape and tissue structure can change differently.

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Estimate a zero-change point and describe uncertainty

Plot mean percentage change against external solute concentration, with units and clearly identified uncertainty bars. For an invented series at 0.00, 0.10, 0.20, 0.30, 0.40 and 0.50 mol/L, means are +12, +8, +4, −2, −6 and −10%. The zero crossing lies between 0.20 and 0.30 mol/L.



Each concentration has five independent pieces. Bars are invented standard errors of the mean, not the full spread of individual pieces; the line is a visual guide.

If the relationship is approximately linear over that small interval, the crossing estimate is

$$c_0 = 0.20 + \frac{4}{4+2}(0.30 - 0.20) \approx 0.267 \text{ mol/L}.$$

This is interpolation between neighbouring data, not proof of an exact constant for every cell. Sample closer to the crossing with more independent pieces to improve the estimate. A curve that is not locally linear requires a more appropriate fit; do not extrapolate a straight line far outside the measured range.

Standard deviation, SD, describes spread among individual results. Standard error, SE, describes uncertainty in an estimated mean under the sampling model; for independent results it can be calculated as SD divided by the square root of sample size. The course does not require memorizing these statistical formulae. Identify which statistic a graph shows rather than calling all bars simply “error”. More independent pieces can reduce SE without reducing biological variation as measured by SD.

For five invented changes of 3, 4, 5, 4 and 4%, the mean is 4%. The sample SD is about 0.71 percentage points and the SE about 0.32 percentage points. A separate set

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of five independent results with SD 2.0 percentage points has SE about 0.89. Compare spread and uncertainty of the mean separately. Overlapping SE bars alone are not a complete significance test.

A zero net tissue-mass change is consistent with an isotonic condition for that tissue and time interval. It does not show that all molecular exchange stopped, that every cell has the same internal concentration, or that solute and pressure contributions have been measured separately. Leakage of solute, damaged membranes or surface-water errors can also affect the result.

Isotonic solutions protect cells during medical applications

In intravenous fluid treatment, the effective osmotic properties of a fluid matter because large sustained water transfers across cell membranes can harm cells. Appropriately chosen isotonic fluids can help avoid osmotic swelling or shrinkage. Actual fluid selection also depends on composition, purpose and the patient's condition; tonicity alone does not specify a complete clinical treatment. A manufacturer's isotonic sodium chloride label gives one clinical example.

Organs prepared for transplantation are bathed or perfused with controlled preservation solutions. Avoiding damaging osmotic shifts is one requirement. Temperature, oxygen supply where relevant, metabolism and chemical composition also affect preservation; isotonicity alone does not keep an organ alive indefinitely. This example applies the membrane principle rather than supplying a recipe for a clinical fluid.

Water potential connects solute effects and cell pressure

Water potential compares the tendency of water to move relative to a reference. Net water movement is from higher toward lower water potential through a suitable water-permeable pathway. Dissolved solute lowers this tendency, while pressure can oppose the effect of solute in a turgid plant cell.

This explains why a cell containing solutes can stop gaining net water before its internal solute concentration equals the external concentration. Pressure supported by the wall changes the overall balance. A cell at equilibrium can still exchange water molecules in both directions. The following quantitative sections develop this model for HL.

AHL: Water potential is potential energy per unit volume

Water potential is the potential energy of water per unit volume relative to a reference. Use pure water at atmospheric pressure and 20°C as the course reference, assigned zero potential. Values are relative, not measurements of an absolute total energy of all water molecules. Energy per volume has pressure units: $1\ \mathrm{J/m^3} = 1\ \mathrm{Pa}$.

Water-potential values are commonly expressed in kPa; MPa is also useful. Convert before comparing: 1 MPa = 1000 kPa. More negative means lower. Thus −200 kPa is higher than −500 kPa, and net water tends to move from −200 to −500 kPa if

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the pathway allows it. This is movement toward lower potential energy, not an active upward pump.

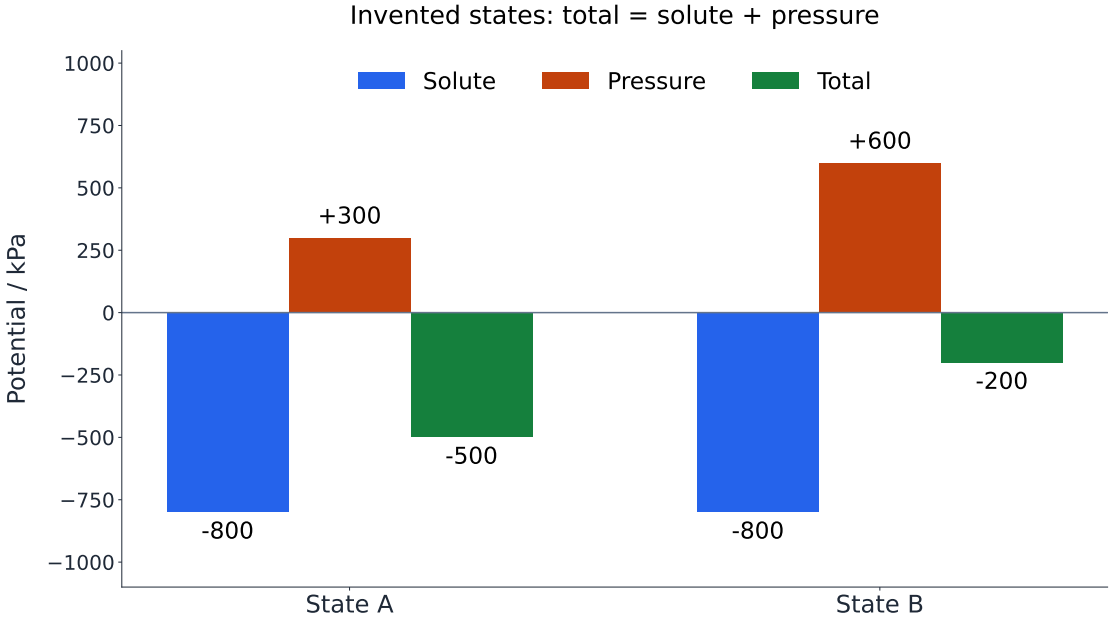
A pressure applied to pure water can raise its water potential above the reference. Dissolving solute lowers its solute component. Water potential is not always negative simply because a biological cell contains solute: the pressure component must also be considered.

AHL: Add solute and pressure contributions before predicting movement

For the simplified cell model used here,

$$\psi_w = \psi_s + \psi_p,$$

where ψ_w is water potential, ψ_s solute potential and ψ_p **pressure potential** . Solute potential is zero for pure water and negative for solutions. Pressure potential in a turgid living plant cell is generally positive relative to the reference. In xylem transporting sap under tension, the pressure component can instead be negative. Tension does not mean a negative amount of water.



Add both signed components. State A has the lower total; the same solute potential can give different water potentials when pressure differs.

An invented cell has $\psi_s = -800$ kPa and $\psi_p = +300$ kPa. Its total is -500 kPa. External solution at -200 kPa has the higher potential, so water initially enters. If solute potential were held at -800 kPa, a pressure of $+600$ kPa would give a total of -200 kPa, matching the outside.

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The final calculation is conditional. In a real cell, entry dilutes solutes as well as raising pressure, so solute potential need not stay exactly −800 kPa. A calculation that fixes it must state that assumption. At equilibrium, equality of total potentials does not require equality of their separate components.

Rearrange the equation when pressure is unknown: $\psi_p = \psi_w - \psi_s$. If a cell has total potential −350 kPa and solute potential −900 kPa, its pressure potential is $-350 - (-900) = +550$ kPa. Subtract the negative value correctly; +550 is not −1250.

AHL: Explain changing plant tissues with both components

In a hypotonic external medium, a typical initially flaccid cell takes up water. Dilution tends to make its solute potential less negative, and wall-supported pressure rises. Both changes raise total water potential until net entry is limited by equality with the outside under the model conditions.

In a hypertonic external medium, net water loss reduces pressure and concentrates internal solutes. Solute potential becomes more negative. With enough loss, turgor falls to zero and the membrane and contents can withdraw from the wall. In a simple incipient-plasmolysis model, pressure potential is approximately zero, so total cell water potential is approximately its solute potential.

Take an invented starting cell with $\psi_s = -700$ kPa and $\psi_p = +400$ kPa: total −300 kPa. Outside at −600 kPa, net water initially leaves. It is wrong to predict entry just because internal solute potential, −700 kPa, is lower than the outside value: that ignores +400 kPa of pressure. As water leaves, both internal components can change.

Do not force the two-term model onto every situation without its assumptions. Height and interactions with surfaces can matter in other water-potential problems. Here compare cells and solutions at comparable height and use the stated solute/pressure model. Tissue mass alone gives no independent measurement of both internal components.

Practice

1. Explain how water molecules orient around sodium and chloride ions. How does glucose solvation differ from salt dissociation?
2. Define osmosis using a membrane and an appropriate solute comparison. What does isotonic mean, and why does it not mean that water molecules stop moving?
3. Predict the initial net water movement and possible outcomes for a red blood cell in hypotonic and hypertonic media. Distinguish crenation from plasmolysis.
4. Explain how a freshwater unicellular organism with contractile vacuoles can persist despite continual osmotic water entry.
5. Describe how a plant cell becomes turgid and how plasmolysis differs. Why is the plasma membrane, rather than the wall alone, the relevant selective boundary?

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6. An invented sample changes from 5.00 to 5.40 g; another changes from 4.00 to 4.20 g. Calculate and compare percentage changes. A third gains mass from 3.00 to 3.36 g and then returns to 3.00 g: compare the forward gain with the reverse decrease.
7. A tissue strip changes from 50.0 to 47.5 mm. Calculate percentage length change. Give four controls for a comparison across external solute concentrations.
8. Invented mean percentage changes are +3% at 0.20 mol/L and −5% at 0.30 mol/L. Estimate the zero crossing by local linear interpolation and give one limitation.
9. Five independent pieces have mean change 4%, SD 2.0 percentage points. State the approximate SE and explain what SD and SE describe. Would reweighing the same piece five times provide five independent pieces?
10. Explain two medical applications of controlled isotonic solutions. Why is isotonicity alone not a complete treatment or preservation protocol?
11. A tissue sample shows zero net mass change. Give one supported inference and three conclusions that cannot follow from that result alone.
12. **AHL:** A cell has solute potential −0.75 MPa and pressure potential +250 kPa. Calculate total water potential in kPa and predict initial movement if the outside is −300 kPa. At the same fixed solute potential, what pressure would balance that outside?
13. **AHL:** Cell A has total potential −200 kPa and solute potential −850 kPa. Find its pressure potential. Cell B has solute potential −900 kPa and pressure +500 kPa. Predict initial net water transfer between A and B through a suitable membrane.
14. **AHL:** A cell has solute potential −700 kPa and pressure +400 kPa; outside is −600 kPa. Explain initial movement and how both components tend to change during water loss. What approximation applies to pressure at incipient plasmolysis?

Worked answers

1. Oxygen faces positive sodium; hydrogen faces negative chloride: ions attract polar water. Glucose's polar groups hydrogen-bond with water without dissociating into ions. Solvation surrounds particles; it does not universally break covalent molecules into ions.
2. Net water transfer through a selectively permeable membrane is toward more concentrated effective solute in the equal-pressure model. Isotonic means no sustained net volume change, with balanced bidirectional exchange. Permeant solutes complicate comparisons using total concentration.
3. Hypotonic: net entry, swelling and possible lysis. Hypertonic: net loss and red-cell crenation. Plasmolysis involves membrane and contents withdrawing from a wall; animal cells lack that wall.
4. Contractile vacuoles collect and expel excess water, supported by energy-dependent processes. Removal balances persistent osmotic entry; water did not initially enter through an ATP pump, nor did osmosis stop.

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5. Net entry expands contents against a resisting wall, developing turgor. Sufficient net loss can withdraw contents from the wall: plasmolysis. The wall passes water and many small particles; the membrane restricts particular solutes.
6. Gains are $0.40/5.00 \times 100 = 8\%$ and $0.20/4.00 \times 100 = 5\%$. The third sample gains 12%, then loses $0.36/3.36 \times 100 \approx 10.7\%$ of its new initial mass. The forward and reverse percentages use different denominators.
7. $(47.5 - 50.0)/50.0 \times 100 = -5.0\%$. Control tissue source/initial condition, dimensions, temperature, duration, solution volume and measuring/blotting. Choose four and explain their effects on comparability. Use adequate independent pieces, not repeated readings of one piece.
8. The fraction across the interval is $3/(3 + 5) = 3/8$; $c_0 = 0.20 + (3/8) \times 0.10 = 0.2375$ mol/L, reasonably reported about 0.24 given these data. Local linearity is assumed; variation, leakage, tissue differences and measurement uncertainty limit the estimate.
9. SE $= 2.0/\sqrt{5} \approx 0.89$ percentage points. SD describes individual-piece spread; SE the estimated mean's uncertainty under the independence model. Repeated weighing of one piece is not biological replication and cannot replace independent pieces.
10. Isotonic intravenous fluids help avoid damaging cell-volume shifts; controlled preservation solutions protect organs against osmotic damage. Composition, physiological purpose and preservation conditions also matter. Isotonicity alone neither specifies treatment nor guarantees indefinite organ survival.
11. Zero net change is consistent with balanced transfer under the conditions. It proves neither stopped molecular motion, identical values in every cell, nor separate solute/pressure measurements. Consider surface water, leakage and damage.
12. **AHL:** -0.75 MPa is -750 kPa. Total $= -750 + 250 = -500$ kPa, lower than outside -300 kPa, so water initially enters. At fixed solute potential, balancing pressure is $-300 - (-750) = +450$ kPa. Real water entry can also dilute solutes.
13. **AHL:** A pressure $= -200 - (-850) = +650$ kPa. B total $= -900 + 500 = -400$ kPa. Net water initially moves A $\rightarrow$ B because -200 is higher than -400 kPa. Compare totals, not the most negative single component.
14. **AHL:** Total $= -700 + 400 = -300$ kPa, higher than outside -600 kPa, so water leaves. Pressure falls; concentrating solutes makes solute potential more negative. Both lower total potential. At incipient plasmolysis, pressure is approximately zero. Components need not stay fixed.

Exam tips

State the membrane, net movement and structural outcome. Distinguish crenation from plasmolysis and equilibrium from stopped exchange. Use initial values for percentage changes; identify SD or SE and uncertainty in a zero crossing.

- **AHL:** Convert units, compare signed totals and allow both components to change.

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Reproduction

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This handout covers D3.1: sexual and asexual reproduction, human reproductive systems and hormonal cycles, fertilization, IVF, and reproduction in flowering plants. Sections marked **AHL** are for Higher Level only.

Prerequisites: meiosis, chromosomes, membranes, hormones and negative feedback. Diagrams are simplified teaching models; numerical datasets are invented unless a published investigation is named. They are not observations from real students or claims about examination papers.

Sexual and asexual reproduction solve different problems

A strawberry runner can establish another plant without fertilization. A flower can instead produce offspring after gametes fuse. Both can increase plant numbers, but they have different consequences for inherited combinations.

Asexual reproduction has no fusion of gametes. Offspring usually have the same inherited genotype as the parent, apart from mutations and other exceptions. Rapid reproduction by an individual already suited to a stable environment can be advantageous. Finding a mate is unnecessary, but similar inherited susceptibility can leave many offspring vulnerable to the same environmental change or disease.

Sexual reproduction involves gamete fusion and produces new combinations of alleles through meiosis and fertilization. Variation can increase the chance that some offspring suit a changed environment. It does not ensure that every offspring is better adapted. Time, energy and successful gamete transfer impose costs; producing a new combination is not automatically an improvement.

Neither process creates exactly the same outcome in every species. Mutation can introduce variation into an asexual lineage. Sexual reproduction between very similar parents may produce relatively little variation. Compare the mechanism and conditions rather than claiming that one method is always superior.

Meiosis and fertilization maintain chromosome sets

A **haploid** cell has one chromosome set in the stated life cycle. A diploid cell has two sets, including pairs of homologous chromosomes. Replication copies DNA before meiosis without changing the number of chromosome sets: each duplicated chromosome has two sister chromatids.

Meiosis I separates homologues and meiosis II separates sisters. Crossing over and independent orientation of homologous pairs generate new allele combinations. **Fertilization** combines the haploid contributions of two gametes, restoring a diploid

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set. Replication alone does not turn a diploid cell into a tetraploid one.

For an invented species with $2n = 12$, a normal gamete contains six chromosomes and the zygote twelve. Ignoring crossing over, six independently orienting pairs allow $2^6 = 64$ whole-chromosome combinations across possible gametes. One meiosis still gives four haploid nuclei, not 64. An invented species with $2n = 20$ has normal gametes with ten chromosomes.

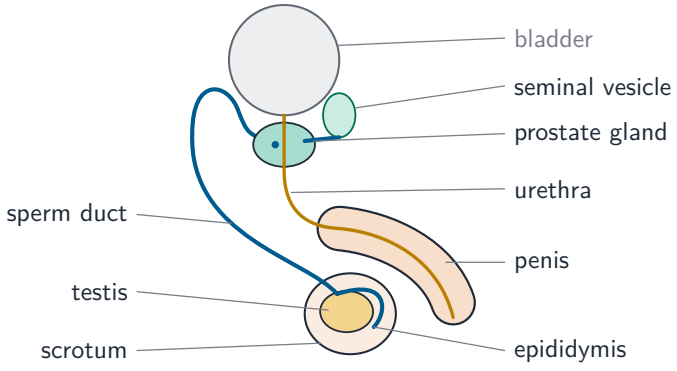
In animals, meiosis directly contributes to gamete formation. Flowering plants include haploid stages that develop after meiosis and produce gametes by mitosis. Do not replace the whole plant life cycle with the claim that each pollen grain is one sperm cell.

Gamete size and movement connect to reproductive strategies

The smaller gamete defines the male reproductive role and the larger gamete the female role. Animal sperm typically travel toward the egg, have relatively little cytoplasm and food reserve, and are produced in large numbers. Eggs are larger and retain resources for early development. In flowering plants, male gametes are carried within the pollen-tube pathway rather than swimming freely through the air.

Producing many small gametes and fewer resource-rich eggs leads to different constraints on reproductive success. Gamete number alone does not determine the number of offspring: transfer, fertilization, development and survival also matter. A flower containing both male and female structures still reproduces sexually if gametes fuse; one organism can supply both reproductive roles.

Human male structures support production and transfer



The bladder provides orientation and stores urine, not sperm. The drawing shows one representative testis and duct; paired structures are present in the typical system.

Testes contain seminiferous tubules where sperm develop and also produce testosterone. The scrotum holds the testes outside the main body cavity, supporting a temperature suitable for sperm production. Sperm mature further and are stored in

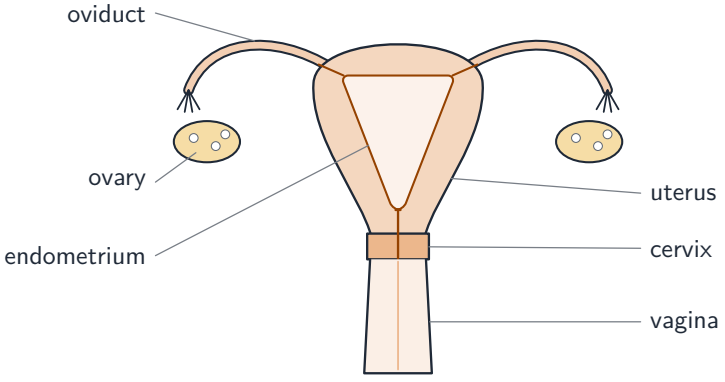
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the epididymis, then pass along the sperm duct, or vas deferens.

Seminal vesicles and the prostate add secretions that contribute to semen and support sperm transfer and function. Semen is not pure sperm. The urethra carries semen through the penis during ejaculation; it also carries urine at other times, rather than being a dedicated sperm-production tube.

Trace the sperm pathway as testis → epididymis → sperm duct → urethra → outside. Glands contribute secretions along the route. For a drawing, place structures and connections before adding leader lines and functions. The bladder, prostate and testis must not be labelled as interchangeable containers.

Human female structures separate fertilization from implantation



An ovary is not a sealed continuation of an oviduct. The endometrium lines the uterine cavity; the cervix is the neck of the uterus above the vagina.

Ovaries contain developing oocytes and produce ovarian hormones. At ovulation an oocyte is released and can enter an oviduct, also called a uterine or fallopian tube. Cilia and muscular activity help move it. Fertilization normally occurs in the oviduct, rather than in the ovary or vagina.

The uterus has a muscular wall and an endometrium that changes during the cycle and supports implantation and pregnancy. The cervix connects the uterus to the vagina through a narrow canal. The vagina receives semen and forms part of the birth canal. Do not draw the urinary outlet as a continuation of the vagina: the urethra is a separate structure.

A released human "egg" is normally a secondary oocyte whose meiosis is not yet complete. The familiar word egg is useful, but it does not mean every meiotic event finished before ovulation.

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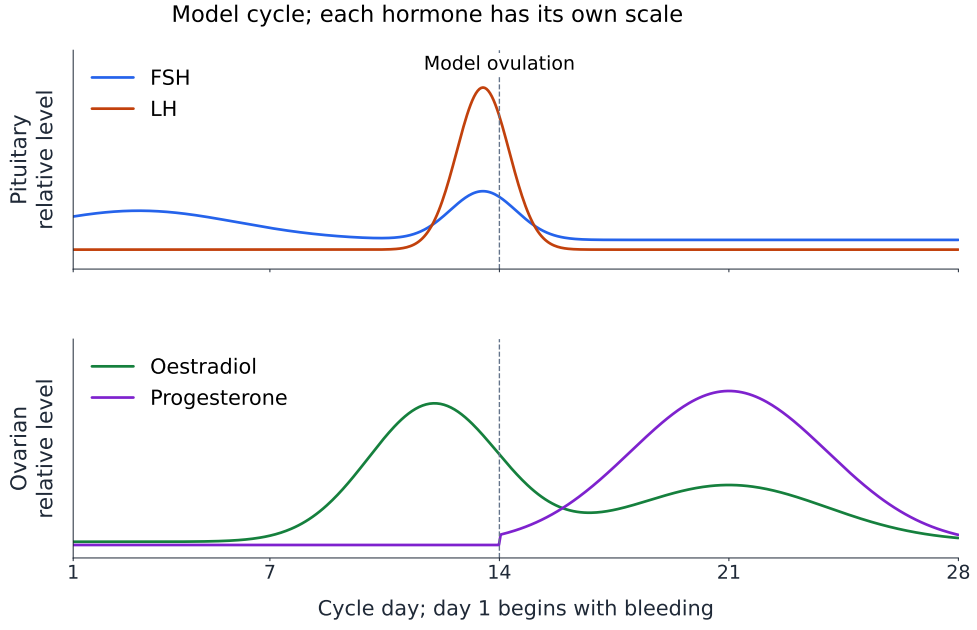
Ovarian and uterine changes are coordinated by hormones

The ovarian cycle concerns follicle development, ovulation and corpus-luteum formation. The uterine cycle concerns changes in the endometrium, including its shedding during menstruation. Together they form the menstrual cycle. Day 1 is the beginning of menstrual bleeding, not the day of fertilization.

FSH from the anterior pituitary promotes follicle development. Developing follicles produce oestradiol, which supports rebuilding of the endometrium. For much of this phase, ovarian hormones exert negative feedback on hypothalamic/pituitary signalling, limiting further stimulation.

Sustained high oestradiol near the end of the follicular phase produces positive feedback in the appropriate physiological context. The resulting LH surge triggers ovulation and development of the corpus luteum from the remaining follicular tissue. A brief rise or low concentration of oestradiol is not the same signal as a sustained high pre-ovulatory level.

The corpus luteum produces progesterone and oestradiol. Progesterone supports a maintained, secretory endometrium, while ovarian hormones exert negative feedback on FSH/LH release. Without a continuing pregnancy signal, the corpus luteum degenerates; progesterone and oestradiol fall, and the endometrium is shed. Falling inhibition also permits recruitment for another cycle.



Each hormone has its own relative scale. Compare its timing, not the absolute concentration of one hormone with another. The 28-day cycle and day-14 ovulation are illustrative, not fixed for everyone.

Read a graph in causal order: follicle growth and oestradiol rise → positive feedback and LH surge → ovulation → corpus luteum and progesterone rise. The progesterone

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maximum is after ovulation, not the trigger for the preceding LH surge. Cycle length and ovulation timing vary; a teaching graph is not a reliable personal fertility prediction.

Human fertilization joins contributions without copying the tail

Sperm reaching the oocyte must interact with its surrounding layers. A sperm plasma membrane fuses with the oocyte membrane, permitting entry of the sperm's contribution, including its nucleus. The paternal tail and mitochondria are normally broken down rather than functioning as a permanent second set of organelles in the embryo.

The maternal and paternal pronuclei bring haploid chromosome complements into the same cell. Their nuclear envelopes break down and the condensed chromosomes participate in the first joint mitotic division, giving two diploid nuclei. "Fusion of gametes" is a useful overall description; it should not force an inaccurate picture of two intact nuclear envelopes simply merging into one permanent envelope before division.

The first divisions distribute the restored diploid complement. Fertilization is distinct from implantation: an embryo must develop and reach the uterus before it can implant. Successful membrane fusion does not alone establish successful pregnancy or later birth.

IVF uses hormones to coordinate several stages

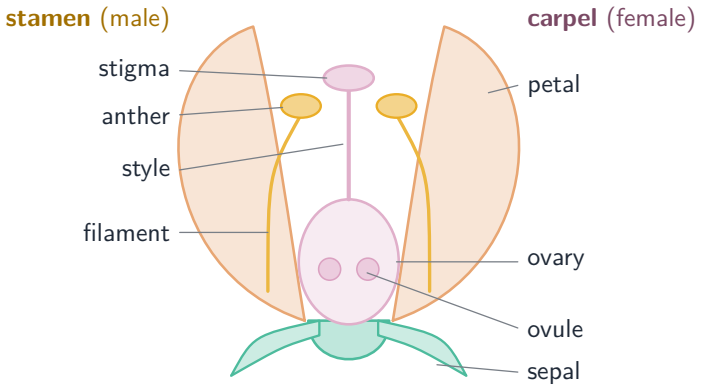
In vitro fertilization means fertilization outside the body under controlled laboratory conditions, followed by transfer of an embryo to the uterus. In a common treatment approach, normal ovarian-cycle signalling is suppressed or controlled, then administered gonadotropins stimulate development of several follicles. This controlled stimulation is often called superovulation.

Monitoring helps coordinate final maturation and oocyte collection. Collected oocytes are exposed to sperm, or a sperm may be introduced by an appropriate laboratory method. Embryos are cultured and a selected embryo can be transferred to the uterus; additional hormonal support may maintain suitable endometrial conditions. The exact regimen varies. Hormones support the physiological stages; they do not guarantee that every collected oocyte fertilizes or every transferred embryo implants. See the NHS outline of IVF stages.

An invented laboratory sequence has 10 collected oocytes, eight fertilized and four embryos reaching a stated later stage. Fertilization is $8/10 = 80\%$ of collected oocytes; later-stage development is $4/8 = 50\%$ of fertilized oocytes or $4/10 = 40\%$ of those collected. These are different denominators, and none is a live-birth rate. State the outcome and population before reporting "success".

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A flower contains structures that make and receive gametes



The stamen comprises anther and filament; the carpel comprises stigma, style and ovary. Ovules are inside the ovary. A whole pollen grain is not a naked male gamete.

Anthers produce pollen grains, supported by filaments. The stigma receives compatible pollen, and the style provides a route toward the ovary. Ovules within the ovary contain the female gametophyte and egg. Petals can attract insects through colour or other signals; nectar and scent can also encourage visits. Sepals protect the developing bud. Avoid assuming that every flower has the same shape or depends on insects.

In the anther, meiosis contributes to haploid spores that develop into pollen grains. The male gametes are produced within the pollen lineage. In an ovule, meiosis and subsequent development produce the embryo sac containing the egg. This connects the plant life cycle with chromosome reduction without pretending that pollen and ovule are equivalent single cells.

Pollination, fertilization and dispersal are different transfers

Pollination transfers pollen from anther to stigma. If compatible pollen germinates, a pollen tube grows through the style toward an ovule and delivers male gametes. One male gamete fuses with the egg to form the diploid zygote that develops into an embryo. In flowering plants a second fertilization produces endosperm, supporting development; the embryo and its food-supporting tissue are different products.

The fertilized ovule develops into a seed, and the ovary commonly develops into a fruit. Seed dispersal transfers seeds away from the parent by mechanisms such as wind, animals or explosive release. It is not pollination, because the transferred structure and life-cycle stage differ. An insect carrying pollen is not transporting an already formed embryo to fertilize a stigma.

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Cross-pollination and self-incompatibility affect variation

Cross-pollination transfers pollen between different plants of the same species. Self-pollination can involve the same flower or another flower on the same plant. Self-fertilization is sexual reproduction: two gametes still fuse, even though one individual supplies them.

Different maturation times for pollen and stigma can reduce self-pollination. Separate male and female flowers, especially on separate plants, can also promote cross-pollination. Animals or wind can move pollen between individuals; a pollinator visiting several flowers on one plant does not necessarily achieve crossing between plants.

Many species have genetically controlled self-incompatibility systems that reject self or otherwise incompatible pollen at an appropriate stage. Pollen may reach a stigma without achieving fertilization. These mechanisms favour crossing and help maintain genetic diversity.

Repeated selfing can increase homozygosity and expose harmful recessive alleles, contributing to inbreeding depression. It need not make every selfed plant unfit: some species regularly self. The outcome depends on genetic background and environment. Outcrossing changes combinations of alleles rather than creating every new allele itself.

Germination mobilizes reserves for embryo growth

After dispersal, a viable seed can remain dormant until suitable conditions and any dormancy requirements are met. Water uptake rehydrates tissues and supports metabolic activity. Oxygen supports aerobic respiration, and a suitable temperature permits enzyme activity. Light requirements vary among species; light is not a universal requirement for every seed to begin germinating.

Stored food is mobilized: enzymes break reserves into soluble products that can be transported and used in respiration and biosynthesis. The embryo grows; the radicle establishes the root and the shoot develops. The seedling initially depends on reserves before photosynthesis can supply enough new organic material.

For an investigation, compare defined conditions using enough viable seeds, independent replicate groups, a consistent germination criterion and a fixed observation period. An invented group with 18 of 24 seeds germinated has a germination percentage of $18/24 \times 100 = 75\%$. A lower value does not identify one cause without controls for viability, dormancy and environment.

AHL: Puberty involves an endocrine control pathway

Increased pulsatile GnRH release from the hypothalamus stimulates the anterior pituitary to release LH and FSH. These act on the gonads, increasing sex-steroid production and supporting reproductive maturation. Testosterone, oestradiol and other relevant signals contribute to secondary sexual characteristics and changes in reproductive function.

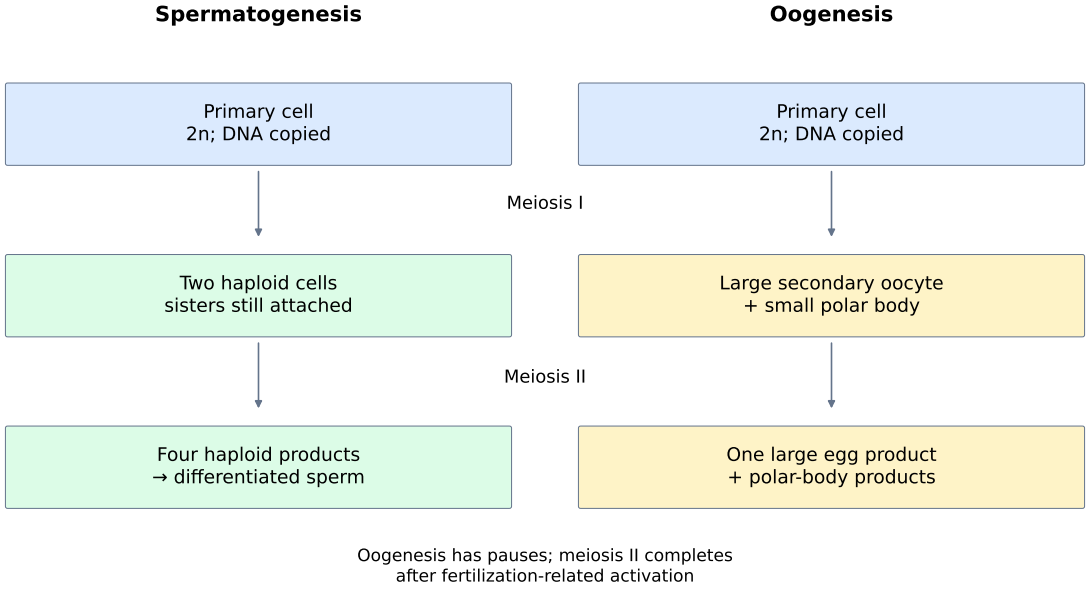
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The hormones are chemical signals acting through responsive cells; they do not physically become gametes. Feedback regulates the axis. Timing and development vary among individuals, and a simplified pathway does not mean every hormone is absent before puberty or that one signal alone explains every developmental change.

AHL: Gametogenesis allocates chromosomes and cytoplasm differently

In spermatogenesis, mitosis maintains precursor populations, cells grow, and meiosis I and II generate haploid products. Differentiation produces streamlined sperm with a flagellum, an acrosome and a midpiece containing mitochondria. Four haploid products can develop from one primary spermatocyte in the simplified normal sequence; mitosis of precursors is not itself the chromosome-halving step.

In oogenesis, precursor mitoses occur before birth. Primary oocytes enter meiosis and pause in prophase I. From puberty, selected oocytes can resume development; unequal division retains most cytoplasm in a large secondary oocyte and gives much less to a polar body. The secondary oocyte normally pauses in metaphase II and completes that division after fertilization-related activation.



Each route begins with one diploid primary cell after DNA replication. Polar-body fate varies; the diagram emphasizes one large functional egg rather than four equal eggs.

Both routes include growth, two meiotic divisions and differentiation, but their timing, numbers and allocation differ. Do not infer that four equal eggs must result because four haploid nuclei are possible. In a model with six primary spermatocytes completing normal development, up to 24 sperm result; six primary oocytes completing the normal

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unequal sequence yield six large functional egg products, not 24.

AHL: Fertilization and early development require controlled transitions

The sperm acrosome reaction releases components that help it penetrate the zona pellucida. After sperm–oocyte interaction, the cortical reaction releases granule contents from the oocyte and modifies the surrounding zona, limiting further sperm penetration and preventing polyspermy. The acrosome belongs to the sperm; cortical granules belong to the oocyte. Do not exchange their roles.

Early mitoses produce more cells while initially partitioning the existing material. A blastocyst develops, with an outer cell population and an inner cell mass. It reaches the uterus and implants in the endometrium. Cell division alone is not implantation: attachment and interaction with the uterine lining are additional steps.

The embryo's developing trophoblast/placental tissues produce human chorionic gonadotropin, hCG, which supports the corpus luteum early in pregnancy. Pregnancy tests use monoclonal antibodies that bind hCG. A test detects a hormone, not a visible embryo or every feature of embryo development. Sampling time and detection threshold affect a negative result; the mechanism does not imply perfect detection immediately after fertilization.

AHL: Placental exchange and progesterone support pregnancy

Placental villi provide a large exchange area between closely apposed maternal and fetal circulations. Maternal and fetal blood normally remain in separate vascular spaces, rather than simply mixing in one shared tube. Oxygen and nutrients pass toward the fetus; carbon dioxide and metabolic wastes pass toward the maternal circulation. Different substances use different transport mechanisms, and the placenta is not an absolute barrier to every harmful agent.

The placenta supports retention of the developing fetus within the uterus by sustaining exchange as it grows. The umbilical vein carries blood from placenta to fetus and the umbilical arteries return it; "vein" and "artery" describe direction relative to the fetal heart, not a universal oxygen-content rule.

Progesterone initially comes mainly from the corpus luteum and later from the placenta. It supports the endometrium and a relatively quiet uterus. Near childbirth, loss of progesterone's inhibitory effect permits greater uterine activity. In humans this includes functional progesterone withdrawal: circulating progesterone need not simply fall before labour. Changes in tissue responsiveness matter. This distinction is supported by human myometrial receptor research.

During labour, cervical stretching promotes oxytocin release from the posterior pituitary, increasing uterine contractions. Stronger contractions cause more stretch, reinforcing oxytocin release: positive feedback. Delivery removes the major stretch-

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ing stimulus. This amplifies a process toward an endpoint; it differs from negative feedback that opposes a deviation to stabilize a variable.

AHL: HRT illustrates why association is not a treatment effect

Earlier observational comparisons associated hormone-replacement therapy with lower coronary-heart-disease incidence. Treatment groups could differ in socioeconomic conditions, health behaviours and access to care. Such confounding can produce an association without the proposed protective hormone effect.

The 2002 Women's Health Initiative randomized trial compared one combined oral estrogen–progestin regimen with placebo in 16,608 postmenopausal women aged 50–79 with an intact uterus. It reported an excess of seven coronary-heart-disease events per 10,000 person-years in that regimen. This challenged the earlier simple cardioprotection claim. Original trial report.

Randomization reduces systematic baseline differences and supports causal inference for the tested comparison. Attrition, adherence, uncertainty and generalizability still matter. The study did not test every formulation, route, age group or purpose of hormone therapy; it does not justify a universal statement about every patient's treatment. Seven extra events per 10,000 person-years is an excess event rate, not "7% of all women" or seven events per individual.

Practice

1. Compare sexual and asexual reproduction in a stable environment and after an environmental change. Give a limitation of each claimed advantage.
2. An invented species has $2n = 8$. State normal gamete and zygote chromosome counts, and the number of whole-chromosome gamete combinations if crossing over is ignored. Distinguish this from the number of products of one meiosis.
3. Explain how differences in gamete size, resources and transfer connect to male and female reproductive roles. Why can a hermaphroditic flower reproduce sexually?
4. Draw the male and female reproductive systems, label the structures taught here, and annotate functions. Trace sperm transport and distinguish the usual fertilization and implantation sites.
5. Explain FSH, LH, oestradiol and progesterone changes in a model menstrual cycle. Identify one positive-feedback interval and one negative-feedback relationship. Explain why ovulation is not fixed on day 14 in everyone.
6. Explain sperm–oocyte membrane fusion and the contributions to the first joint mitosis. Why should fertilization not be confused with implantation?
7. Outline hormonal control in a common IVF approach. Invented results are 12 oocytes collected, nine fertilized and three reaching a stated later embryo stage. Calculate fertilization per collected oocyte and later-stage development using each relevant denominator. Are these live-birth rates?

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8. Draw an insect-pollinated flower and annotate the functions of anther, filament, stigma, style, ovary, ovule, petal and sepal. Explain the route from pollen arrival to an embryo.
9. Distinguish self-pollination, cross-pollination and self-incompatibility. Give two mechanisms promoting crossing and explain how repeated selfing can reduce vigour without claiming that every selfed offspring is unfit.
10. Distinguish pollination from seed dispersal. Explain the need to mobilize reserves during germination and the roles of water, oxygen and suitable temperature.
11. In an invented investigation, 21 of 28 seeds germinate. Calculate the percentage and propose controls and replication for investigating one germination factor. Why would a poor result not identify that factor alone as the cause?
12. **AHL:** Trace GnRH to gonadal steroid effects, then compare spermatogenesis and oogenesis in timing, meiotic divisions, cytoplasm allocation and products. How many sperm and large egg products could eight primary cells of each type yield under the stated normal model?
13. **AHL:** Explain the acrosome and cortical reactions, blastocyst implantation, hCG detection, placental exchange and the changing source of progesterone. Describe the oxytocin feedback loop and explain functional progesterone withdrawal in humans.
14. **AHL:** Explain why an observational HRT–CHD association may be confounded. Describe what the named randomized trial adds and one limit on generalizing it. If its stated excess rate remained constant, how many extra CHD events would correspond to 20,000 person-years? Why is this not a personal risk prediction?

Worked answers

1. Asexual reproduction rapidly propagates a successful genotype without a mate, but shared susceptibility can be harmful after change. Sexual reproduction generates potentially favourable combinations, with transfer costs and no guaranteed improvement. Mutation also varies asexual lineages.
2. Gamete four chromosomes; zygote eight. Four independently orienting pairs give $2^4 = 16$ possible whole-chromosome combinations across gametes. One meiosis produces four haploid nuclei. Replication before meiosis copies chromatids without making the starting cell tetraploid.
3. Small, numerous male gametes carry fewer reserves; larger female gametes retain resources. Transfer and offspring survival constrain success. A hermaphroditic flower supplies both gamete types; fusion remains sexual during self-fertilization.
4. Use the annotated diagrams. Sperm: testis → epididymis → sperm duct → urethra; glands add semen components. Fertilization: oviduct; implantation: uterine endometrium. Ovaries supply oocytes/hormones. Cervix and vagina are separate from the urinary outlet.
5. FSH supports follicles; oestradiol rebuilds endometrium. Sustained high pre-

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ovulatory oestradiol gives positive feedback and an LH surge, triggering ovulation and corpus-luteum formation. Progesterone supports secretory endometrium with negative feedback on gonadotropins. Without pregnancy support, corpus-luteum decline lowers hormones and permits shedding. Timing varies.

6. Membranes fuse; the paternal nuclear contribution enters while tail and mitochondria are normally destroyed. Pronuclear envelopes break down; both complements participate in the first mitosis, giving diploid nuclei. Implantation follows development and uterine arrival.
7. Control/suppress the natural cycle, stimulate several follicles with administered hormones, monitor maturation and collect oocytes; fertilize, culture and transfer an embryo with appropriate endometrial support. $9/12 = 75\%$ fertilized; $3/9 \approx 33.3\%$ of fertilized oocytes or $3/12 = 25\%$ of collected oocytes reach the stated stage. No birth outcome is given.
8. Anther/filament: pollen production/support; stigma/style: reception/tube route; ovary contains ovules with eggs. Petals can attract insects; sepals protect buds. A compatible tube delivers male gametes; egg fertilization produces zygote and embryo. Pollen is neither an embryo nor one naked sperm.
9. Self-pollination stays within one plant; crossing transfers pollen between plants. Different maturation times, separate male/female plants and animal/wind transport promote crossing. Self-incompatibility can block fertilization after pollen arrival. Selfing increases homozygosity, potentially exposing harmful recessives; effects depend on genotype/environment.
10. Pollination moves pollen to stigma; dispersal moves seeds containing embryos. Water supports rehydration/metabolism, oxygen aerobic respiration, and temperature enzyme activity. Mobilized reserves supply respiration and growth before sufficient photosynthesis. Light and dormancy requirements vary.
11. $21/28 \times 100 = 75\%$. Use comparable viable seeds, independent replicate groups, random allocation, controlled other conditions and consistent time/criteria. Viability, dormancy and contamination also affect outcomes; one poor group identifies no unique cause.
12. **AHL:** GnRH stimulates pituitary LH/FSH, which act on gonads and increase sex-steroid production. Both gametogenic routes involve precursor mitosis, growth, two meiotic divisions and differentiation. Spermatogenesis can yield four functional sperm per primary cell; oogenesis pauses before birth and before fertilization, with unequal divisions retaining cytoplasm in one egg. Eight primary cells give up to 32 sperm or eight large egg products in this model.
13. **AHL:** Sperm acrosome activity helps zona penetration; the oocyte cortical reaction modifies the zona to limit polyspermy. A blastocyst implants in endometrium; monoclonal antibodies detect hCG from developing trophoblast/placental tissues. Placental villi support exchange without normal free mixing of the blood supplies. Corpus luteum then placenta provide progesterone. Reduced progesterone effect permits contraction; human functional withdrawal need not require falling circu-

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lating concentration. Stretch → oxytocin → stronger contractions → more stretch is positive feedback, ended by delivery.

14. **AHL:** Socioeconomic and health differences can confound observational comparisons. Randomization supports inference for the tested regimen and population, while adherence, uncertainty and generalizability remain limits. $7 \times (20{,}000/10{,}000) = 14$ excess events under a constant-rate assumption. Person-years are population exposure time; the estimate is neither 7% of women nor a specific individual's probability.

Exam tips

Name the transferred structure, process and location. Separate pollination, fertilization, implantation and dispersal. State hormone sources, targets and feedback direction. Define each rate's outcome and denominator.

- **AHL:** Distinguish sperm acrosome from oocyte cortical granules, chromosome reduction from cytoplasm allocation, and placental exchange from blood mixing. Apply trial findings to their tested comparison and distinguish functional hormone withdrawal from blood concentration.

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Inheritance

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This handout covers D3.2: inheritance through gametes, genetic crosses, genotype and phenotype, dominance patterns, human examples, pedigrees, continuous variation and box plots. Sections marked **AHL** are for Higher Level only.

Prerequisites: chromosome sets, meiosis, fertilization and gene expression. Crosses, families and numerical samples are invented teaching models unless a database or published source is identified. Probabilities predict possible outcomes, not a fixed order of offspring.

Gametes transmit alleles and fertilization restores two sets

Two parents can share a phenotype yet produce an offspring with a different phenotype. The explanation can be inheritance of recessive variants, rather than a new mutation in every such offspring.

An **allele** is a variant of a gene. In a diploid cell, an autosomal gene normally has two copies, one on each member of a homologous chromosome pair. The copies may carry the same or different alleles. A gamete receives one member of each pair and hence one allele at each ordinary autosomal locus. Fertilization combines two haploid contributions and restores the diploid complement.

The **genotype** records the alleles carried at the locus or loci being considered. AA and aa are homozygous; Aa is heterozygous. A capital letter does not mean a physically larger chromosome or a more common allele. A gene specifies a locus and function; its alleles are alternative versions, not two unrelated genes simply because their letters differ.

Genetic crosses require controlled parentage and a phenotype key

In flowering plants, controlled crosses transfer pollen from a chosen parent to a receptive stigma. For a flower capable of selfing, remove its anthers before pollen release where appropriate, protect it from unintended pollen, apply the chosen pollen and label the cross. Bagging and suitable controls help detect contamination. The female gamete is within an ovule in the ovary; applying pollen directly to a leaf does not carry out the intended cross.

The P generation is the parental generation. Its offspring are F1; crossing or selfing appropriate F1 individuals produces F2. These labels describe relationships, not automatically pure-breeding genotypes. Controlled crosses investigate inheritance and develop varieties; selection for one trait can have trade-offs.

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Write four things before a Punnett grid: parental genotypes, the phenotype key, possible gametes, and the inheritance assumptions. Put one parent's gametes along each edge and combine one from each to fill a cell. Each grid cell represents a possible fertilization, not one compulsory individual in a family.

Phenotype depends on genotype and sometimes environment

Phenotype is an observable characteristic, including a measurable biochemical trait. A phenotype may depend mainly on inherited sequence in the stated context, mainly on an environmental event, or on interaction between the two. Ordinary ABO type is useful for a relatively simple inherited trait. A scar from an injury illustrates an acquired effect; height reflects many genes and environmental conditions such as nutrition.

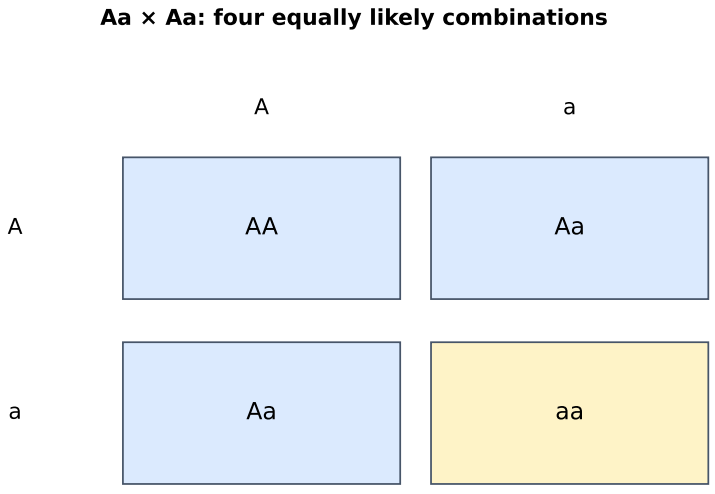
Avoid claiming that every phenotype uniquely identifies genotype. With complete dominance, both AA and Aa can have the same phenotype. Nor does an identical genotype require identical measured values in every environment.

Phenotypic plasticity is the capacity for one genotype to develop different traits in different conditions through changes in expression and development. Sun-exposed and shaded leaves on a plant can differ in structure; changes in skin pigmentation after UV exposure provide another context. Such changes do not require a new inherited DNA sequence. Some are reversible, whereas a developed structure may persist after the environment changes. Plasticity is not proof that an acquired trait will be genetically inherited.

Complete dominance predicts probabilities for a stated cross

In an invented plant, A gives purple flowers and aa gives white flowers under complete dominance. A heterozygote makes A and a gametes in equal proportions in the normal segregation model. Crossing Aa × Aa gives AA, Aa, Aa and aa: genotype probabilities 1/4, 1/2 and 1/4; phenotype probabilities 3/4 purple and 1/4 white.

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The four cells are equally likely under the stated model. They do not prescribe the order or exact composition of a four-offspring family.

A test cross with aa distinguishes possible dominant-phenotype genotypes through offspring evidence. AA × aa predicts all Aa. Aa × aa predicts half Aa and half aa. Observing white offspring supports the heterozygous interpretation under this model; observing only purple offspring in a small sample does not prove AA with certainty.

For Aa × Aa and 20 offspring, the expected number of aa is $20 \times 1/4 = 5$. A real count can differ through sampling. Under independent fertilizations, the probability that two specified offspring are both aa is $(1/4)^2 = 1/16$; it is not $1/2$ because two offspring were mentioned. A previous outcome does not force the next one to complete a ratio.

Dominance describes the heterozygote phenotype. It does not mean stronger, healthier, evolutionarily preferable or common. One functional allele may supply enough enzyme for the dominant phenotype in a particular case; other molecular mechanisms and dominance patterns exist.

PKU connects a recessive genotype with metabolism and environment

Classic phenylketonuria, PKU, is commonly caused by variants affecting the PAH gene. Its product, phenylalanine hydroxylase, normally helps convert phenylalanine to tyrosine. Reduced activity can allow phenylalanine to accumulate and harm development if the condition is untreated. This is a metabolic explanation, not a claim that phenylalanine is always an unwanted substance: it is also an essential amino acid.

The ordinary teaching model is autosomal recessive. Two unaffected carriers can have

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an affected child if each contributes a disease-associated allele. Using A for adequate function and a for the recessive variant, Aa × Aa gives a 1/4 aa probability per pregnancy under the model. Human disease-associated variants are more diverse than a single letter suggests.

Dietary management can change the consequences of the genotype; it does not change an aa genotype into AA. This illustrates genotype–environment interaction. Actual screening and treatment depend on clinical assessment, rather than a classroom Punnett grid. MedlinePlus explanation of PKU genetics.

Multiple alleles and ABO separate population diversity from one genotype

A gene can have many alleles in a population, including variants originating through nucleotide substitutions. A SNP is a single-nucleotide variant at a locus; it need not cause disease. One diploid individual still normally carries only two alleles of an autosomal gene, even if dozens exist in the wider gene pool.

For the ordinary ABO model, use I^A, I^B and i. I^AI^A or I^Ai gives type A; I^BI^B or I^Bi gives B; I^AI^B gives AB; ii gives O. The A and B alleles are codominant in the heterozygote, and each is dominant over i in this model. AB cells express both A and B antigen features, rather than one intermediate antigen called "half A".

An $I^Ai \times I^Bi$ cross gives four equally likely genotypes: I^AI^B, I^Ai, I^Bi and ii. Thus AB, A, B and O each have probability 1/4. An $I^AI^B \times ii$ cross gives half A and half B, with no ordinary-model AB or O offspring. These simplified crosses concern ABO phenotype; they are not a full account of every blood-group system or rare expression exception.

Incomplete dominance differs from codominance

In a model of four-o'clock flowers, Mirabilis jalapa, homozygous red and white parents produce pink heterozygotes. This is incomplete dominance: the heterozygote phenotype is intermediate. Use distinct superscript allele symbols, such as C^R and C^W, rather than implying that one must completely dominate.

Crossing two pink heterozygotes gives 1/4 C^RC^R red, 1/2 C^RC^W pink and 1/4 C^WC^W white. Both genotype and phenotype ratios are 1:2:1. The alleles have not permanently blended into one pink allele: red and white phenotypes can reappear in offspring.

Codominance instead gives distinguishable contributions of both alleles, as in ABO type AB. Dominance categories describe a defined phenotype. Molecular measurements can reveal differences that an outward category hides, so always state which characteristic the cross predicts.

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Sex chromosomes change how an X-linked allele is inherited

In the common human XX/XY model, eggs carry X and sperm carry X or Y. The sperm's sex chromosome therefore contributes to whether the zygote is XX or XY. These chromosomes influence male-typical or female-typical development through gene regulation; actual developmental variations mean the simplified chromosome model does not describe every individual in full.

The X chromosome carries many more genes than the Y. A typical XY individual has one copy of most X-linked loci, so there is no second corresponding allele on Y to mask a recessive effect. Do not write an invented haemophilia allele on Y when the relevant gene is X-linked.

Haemophilia A and B provide X-linked examples involving clotting-factor genes. Write X^H for the model normal allele and X^h for the disease-associated allele. An X^HX^h parent crossed with X^HY gives equal model probabilities of X^HX^H, X^HX^h, X^HY and X^hY. Half of sons are predicted to inherit the affected genotype; one quarter of all offspring are affected sons if X- and Y-bearing fertilizations are equally likely.

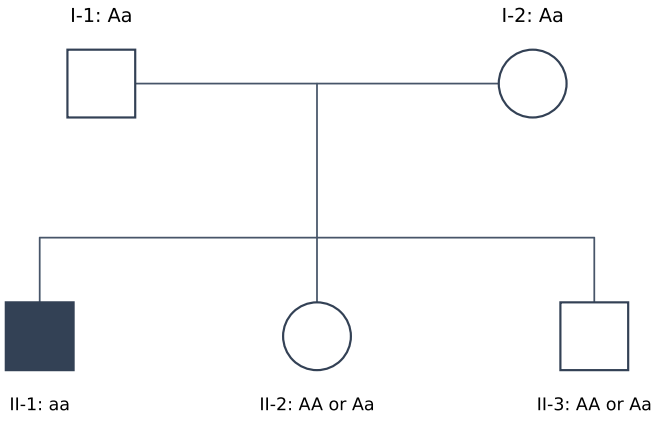
An affected father passes his X to daughters and Y to sons in this model, so he does not transmit his X-linked allele directly to sons. Females can also have haemophilia; heterozygotes can have bleeding symptoms, including through X-inactivation effects. The simple recessive grid must not become "females are always symptomless". MedlinePlus inheritance explanation.

Pedigrees test a model rather than proving it from one person

A pedigree records relationships and a defined trait across generations. In the conventional diagrams here, squares represent males, circles females and filled symbols the studied affected phenotype. State any additional symbol key. A horizontal connection joins parents; vertical descent and a sibling line show their children. Number individuals so an inference refers to a specific person.

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Invented family: test an autosomal recessive model



Square: male; circle: female; filled: affected

This is an explicitly simplified, fully penetrant autosomal model with no new mutation. The family pattern is used to test that model, not to diagnose a real family.

Under a fully penetrant autosomal recessive model, two unaffected parents with an affected aa child must each supply a, so both are Aa. An unaffected sibling can be AA or Aa. Among unaffected offspring of Aa $\times$ Aa, the conditional probability of being Aa is 2/3, because the two Aa possibilities are compared with the three unaffected possibilities, not all four outcomes.

An affected child of unaffected parents is inconsistent with a simple fully penetrant dominant model without new mutation, but real pedigrees can involve new variants, incomplete penetrance or other mechanisms. Small families can fit more than one inheritance pattern. Male-to-male transmission argues against ordinary direct X-linked transmission; absence of an affected son in one small family does not establish X linkage.

Inferring a general model from several observed families is inductive reasoning. Using an assumed model to infer a parent's required genotype is deductive reasoning. Distinguish the provisional model from deductions conditional on it. Close relatives can share the same rare recessive allele through common ancestry, increasing the chance of a homozygous child; that is a genetic reason behind restrictions on close-relative marriage in many societies, not a claim that every such child must be affected.

Continuous variation requires appropriate measurement and summaries

ABO types are discrete categories. Skin pigmentation varies continuously and is influenced by many genes and environmental factors, including UV exposure. Height

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is another continuous measurement. A measurement rounded to whole centimetres is still sampling an underlying continuous variable; discrete recorded numbers do not make the underlying biology an ABO-like category.

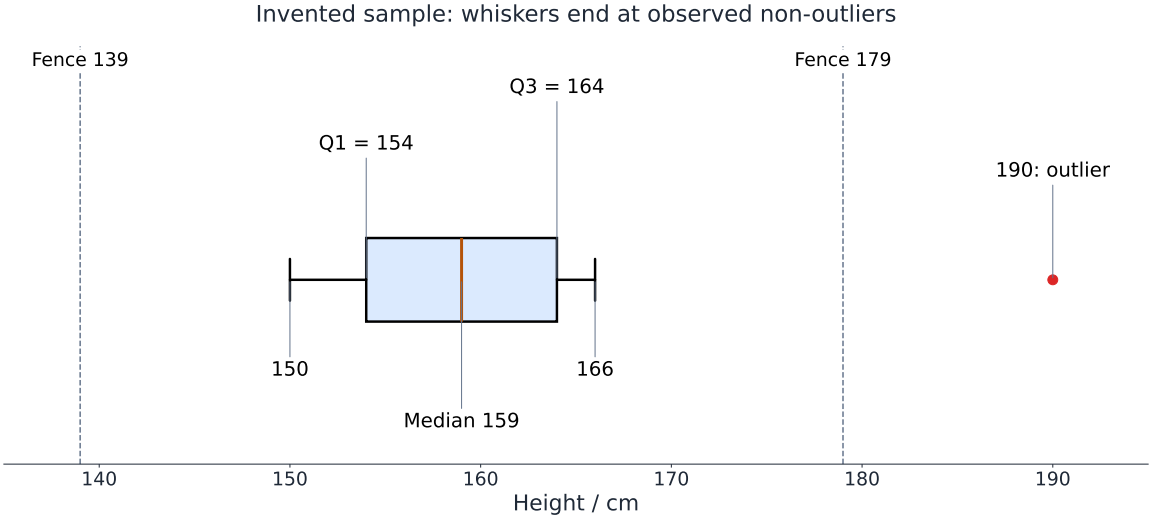
For a numerical sample, the mean uses all values, the median is the middle after sorting, and the mode is the most frequent value, if one exists. Do not assign a numerical mean to arbitrary ABO category codes and treat it as a meaningful biological average.

An invented height sample in cm is 150, 152, 154, 156, 158, 160, 162, 164, 166, 190. The sum is 1612 cm, so mean is 161.2 cm. Median is $(158 + 160)/2 = 159$ cm. There is no mode because no value repeats. The unusually high value pulls the mean upward more than the median; investigate recording errors without deleting a valid observation merely because it is inconvenient.

A box plot distinguishes observed extremes from whisker ends

Using the median-of-halves convention for this ten-value sample, the lower five have median 154 cm and the upper five median 164 cm. Therefore $Q_1 = 154$, median 159 and $Q_3 = 164$ cm; interquartile range, IQR, is 10 cm.

Outlier fences are $Q_1 - 1.5\text{IQR} = 139$ cm and $Q_3 + 1.5\text{IQR} = 179$ cm. The 190 cm observation is above the upper fence and is plotted separately. Whiskers extend to the most extreme observed non-outliers, here 150 and 166 cm, not to the fence values if no observation lies there.



The sample maximum is 190 cm even though the upper whisker is 166 cm. State the quartile convention; different software conventions can give different quartiles for small samples.

Label units and distinguish minimum/maximum observations, quartiles, median and outliers. A point exactly on a fence is not beyond it. A box plot does not show every data value or necessarily reveal multiple clusters, so retain the underlying data where possible.

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AHL: Segregation and assortment explain a dihybrid grid

Segregation places the two alleles of a locus into different normal gametes. For genes on different chromosome pairs, independent orientation at meiosis I makes their combinations independent. An AaBb individual then gives AB, Ab, aB and ab gametes, each with probability 1/4, under the stated equal-viability model.

Crossing AaBb × AaBb produces sixteen equally likely combinations. With complete dominance at each locus, phenotype probabilities are 9/16 A_B_, 3/16 A_bb, 3/16 aaB_ and 1/16 aabb. The underscore means either permitted second allele. The 9:3:3:1 phenotype ratio is not a claim that nine offspring all have genotype AABB.

AaBb × AaBb: independent assortment model

	AB	Ab	aB	ab
AB	AABB	AABb	AaBB	AaBb
Ab	AABb	AAbb	AaBb	Aabb
aB	AaBB	AaBb	aaBB	aaBb
ab	AaBb	Aabb	aaBb	aabb
	A_B_: 9	A_bb: 3	aaB_: 3	aabb: 1

The ratio assumes independent assortment, complete dominance at both loci, random fertilization and no relevant survival or scoring bias.

The double-heterozygote genotype AaBb has probability $(1/2)(1/2) = 1/4$ in this cross. For 160 offspring, the expected phenotype counts are 90, 30, 30 and 10. A test cross AaBb × aabb instead exposes the double heterozygote's gametes and predicts a 1:1:1:1 phenotype ratio under independence.

Mendel's independent-assortment pattern applies to suitable loci, not all genes without exception. Genes far apart on the same chromosome can show recombination frequencies approaching 50% and behave approximately independently in these crosses. Physical presence on the same chromosome and a detectable deviation from independence are related but different claims.

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AHL: Databases connect loci with polypeptide products

A locus is a gene's chromosomal position. Check the organism, approved gene symbol, chromosome, reference assembly and gene product in a curated database. Coordinates from different assemblies must not be subtracted as though they used one common map.

The human ABO locus is on chromosome 9 and encodes glycosyltransferase variants; HBB on chromosome 11 encodes the haemoglobin beta chain. They provide a pair on different chromosomes. APOE and APOC1 are neighbouring loci in the chromosome 19 apolipoprotein cluster and encode apolipoprotein E and C-I, respectively. Use the actual records: ABO, HBB, APOE and APOC1.

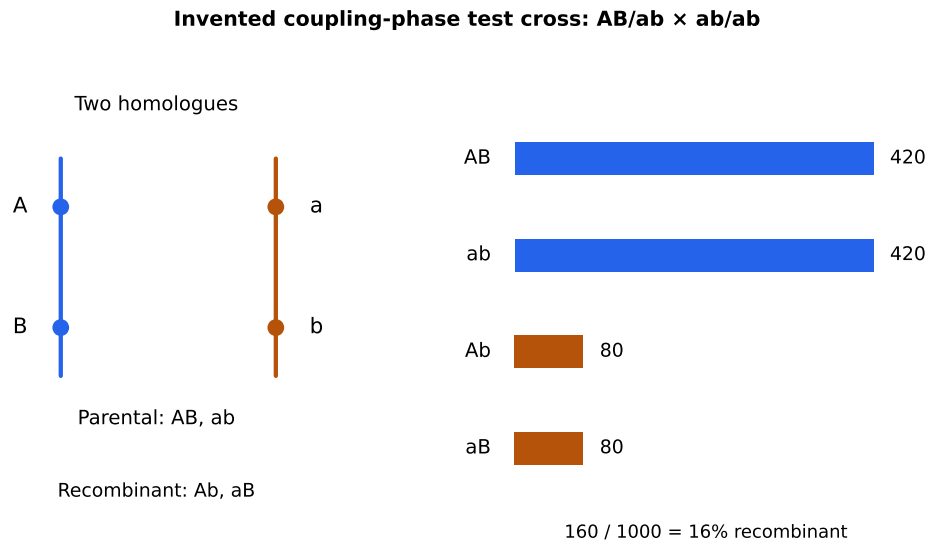
In the checked GRCh38 records, APOE spans 44,905,796–44,909,393 and APOC1 spans 44,914,325–44,919,346 on chromosome 19. These are close physical loci. Physical coordinates alone do not supply a universal recombination probability or show that their products perform identical functions. The crosses below use invented A/B loci, not measured recombination data for these named human genes.

AHL: Linkage requires the phase of the alleles

For two linked loci, show which alleles lie on the same homologue. Coupling phase has A and B on one chromosome, a and b on the other: AB/ab. Draw two vertical chromosome lines with the alleles placed beside them; a slash is a shorthand for that pair, not division of two numbers. Repulsion phase is Ab/aB. Both can have genotype AaBb, but their non-recombinant gametes differ.

Crossing over between the loci can generate recombinant chromatids. From AB/ab, parental combinations are AB and ab; recombinants are Ab and aB. From Ab/aB, those categories reverse. Calling a gamete recombinant requires a known parental arrangement; uppercase letters are not automatically the parental class.

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Use the stated parental phase. The counts assume the test cross reveals gametes without differential survival or phenotype-scoring bias.

An invented AB/ab × ab/ab test cross gives 420 AaBb, 420 aabb, 80 Aabb and 80 aaBb offspring. Total is 1000; recombinants total 160, so observed recombination frequency is $160/1000 \times 100 = 16\%$. The model gamete proportions are AB 42%, ab 42%, Ab 8% and aB 8%.

Do not report 84% recombination by counting the two large parental classes. Recombination frequency is not the proportion of meioses that had any crossing over: a crossover involves particular chromatids, and multiple crossovers can obscure events between widely separated loci. Approximately 50% recombinant classes do not distinguish different chromosomes from sufficiently distant loci on one chromosome by themselves.

AHL: Use chi-squared to compare a cross with a prediction

A goodness-of-fit test compares observed counts O with expected counts E under a stated null hypothesis. For a fully specified four-category prediction with no fitted parameters, degrees of freedom are $4 - 1 = 3$. Use a supplied critical value, here 7.815 at the 5% level.

$$\chi^2 = \sum \frac{(O - E)^2}{E}.$$

For an invented F2 sample of 90, 34, 26 and 10, total 160, the 9:3:3:1 model expects 90, 30, 30 and 10. The contributions are 0, 16/30, 16/30 and 0, so $\chi^2 \approx 1.067$. This is below 7.815: do not reject that null model at the stated level. It does not prove the model uniquely true or establish that every difference was caused by chance.

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For the linked test-cross data 420, 420, 80 and 80, an independence null predicts 250 in each of four categories. Each deviation has magnitude 170; $\chi^2 = 4(170^2/250) = 462.4$. Reject the 1:1:1:1 model at this level. With known phase and the stated assumptions, linkage explains the excess parental combinations; survival or scoring bias would need consideration in a real investigation.

The 5% significance level concerns how a test behaves when its null model is true. It is not a 95% probability that the alternative is true, nor a 5% probability that the null is true. Use independent observations, adequate expected counts, mutually exclusive categories and the same biological outcome definition throughout. Do not calculate this test on percentages while pretending they are the original sample counts.

Practice

1. Define allele, genotype, homozygous and heterozygous. Explain how gametes and fertilization transmit two autosomal alleles to a diploid offspring.
2. Outline a controlled cross between two flowering plants and distinguish P, F1 and F2. Why should unintended self-pollination be controlled?
3. Compare genotype and phenotype using one simple inherited example, one environmental effect and one interaction. Explain phenotypic plasticity without claiming that the DNA sequence must change.
4. For Aa × Aa with complete dominance, give genotype and phenotype probabilities, the expected aa count among 20 offspring, and the probability that two specified offspring are both aa. Contrast Aa × aa.
5. Explain the metabolic basis and recessive inheritance model of PKU. Why can dietary management change consequences without changing the genotype?
6. For $I^Ai \times I^Bi$, give the ordinary-model ABO outcomes. Contrast $I^AI^B \times ii$. Why can a population have three ABO alleles when one person has only two?
7. Two pink Mirabilis heterozygotes are crossed. Give genotype and phenotype ratios and distinguish this from codominance. Do the alleles permanently blend?
8. For $X^HX^h \times X^HY$, give the four possible offspring genotypes. State the probability of an affected son among sons and among all offspring. Explain why an affected father does not directly pass his X-linked allele to a son in the model.
9. Under a fully penetrant autosomal recessive model without new mutation, two unaffected parents have an affected child. Infer their genotypes and the carrier probability for an unaffected sibling. Distinguish the inductive model choice from a deduction conditional on it.
10. For 150, 152, 154, 156, 158, 160, 162, 164, 166 and 190 cm, calculate mean, median, mode, quartiles and IQR using the stated convention. Find outlier fences and distinguish the observed maximum from the upper whisker.
11. Explain why skin pigmentation is a continuous phenotype and why a rounded height measurement is not an ABO-like category. Give a reason to retain the underlying data alongside a box plot.

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12. **AHL:** Derive the gametes and phenotype ratios for AaBb × AaBb and AaBb × aabb under independent assortment. State the expected four phenotype counts for 160 F2 offspring and the probability of genotype AaBb. Give one checked database pair on different chromosomes and one nearby pair on one chromosome, naming their products.
13. **AHL:** For an invented AB/ab test cross, counts are 360 AaBb, 360 aabb, 140 Aabb and 140 aaBb. Identify parental/recombinant classes, calculate recombination frequency and give model gamete proportions. What changes if the parental phase is Ab/aB?
14. **AHL:** An invented F2 has counts 100, 20, 30 and 10 in the four 9:3:3:1 phenotype classes. Calculate expected counts and chi-squared, compare with 7.815 for df = 3, and state a bounded conclusion. Explain why a non-significant result does not prove independence or make the null 95% probable.

Worked answers

1. An allele is a gene variant; genotype records the alleles carried. Homozygotes have matching alleles at the locus and heterozygotes different ones. Normal gametes carry one allele of each ordinary autosomal pair; fertilization combines two haploid contributions.
2. Choose parents, prevent unintended pollen/selfing where appropriate, apply selected pollen to a receptive stigma, protect and label the cross, and use controls. P are parents; their offspring F1; suitable F1 crossings/selfings yield F2. Labels alone do not specify homozygosity.
3. Ordinary ABO illustrates relatively simple inherited variation; an injury scar an acquired environmental effect; height gene–environment interaction. Plasticity changes expression/development of one genotype in different conditions, without requiring an inherited base-sequence change. AA and Aa can share a phenotype under complete dominance.
4. AA:Aa:aa = 1:2:1; dominant:recessive = 3:1. Expected aa = 20/4 = 5; two specified aa offspring = $(1/4)^2 = 1/16$. Aa × aa predicts half Aa, half aa. These are probabilities/expectations, not compulsory family counts.
5. Reduced PAH activity impairs conversion of phenylalanine to tyrosine; accumulation can harm development. In the autosomal recessive model, two carriers have 1/4 affected probability per pregnancy. Management changes exposure/metabolic consequences, not aa into AA; real variants and clinical needs require more than a grid.
6. First cross: AB, A, B and O each 1/4. AB × O: half I^Ai type A, half I^Bi type B. Three population alleles do not require three copies in one diploid genotype. A and B are codominant; both dominate i in the ordinary model.

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7. C^RC^R : C^RC^W : C^WC^W = 1 : 2 : 1 gives red:pink:white = 1:2:1. Pink is intermediate; codominance gives distinguishable contributions of both alleles, such as ABO AB. The alleles segregate without permanent blending.
8. Equal probabilities of X^HX^H, X^HX^h, X^HY and X^hY. Affected probability is 1/2 among sons, 1/4 among all offspring under equal sex-chromosome fertilization. A father gives Y to sons and X to daughters. Females can also be symptomatic; the simplified model does not exclude that.
9. Each unaffected parent supplies a to the aa child: both are Aa. Among unaffected siblings, AA, Aa and Aa give carrier probability 2/3. Inferring a model is induction; genotypes follow deductively under its assumptions.
10. Sum 1612; mean 161.2 cm; median 159 cm; no mode. $Q_1 = 154$, $Q_3 = 164$, IQR 10 cm. Fences 139 and 179 cm; 190 is an outlier. Observed minimum/maximum are 150/190; non-outlier whiskers end at 150/166 cm. The upper fence is not an observed whisker endpoint.
11. Many genes and environment affect pigmentation continuously. Rounding changes recording, not the underlying characteristic. A box plot can hide clusters and individual errors; retain the data.
12. **AHL:** AaBb gives AB, Ab, aB, ab each 1/4 under independence. F2 phenotypes 9:3:3:1 give expected 90, 30, 30, 10; AaBb probability 1/4. Test-cross phenotypes 1:1:1:1. ABO chromosome 9/glycosyltransferase and HBB chromosome 11/beta-globin form a different-chromosome pair. APOE/apolipoprotein E and APOC1/apolipoprotein C-I are close on chromosome 19. Map position alone is not an exact recombination rate.
13. **AHL:** In coupling, AaBb and aabb reveal parental AB/ab gametes. Aabb and aaBb reveal recombinant Ab/aB; $280/1000 \times 100 = 28\%$. Model gametes AB 36%, ab 36%, Ab 14%, aB 14%. In repulsion the identities reverse: these counts give 72% recombinants, strongly conflicting with the model's maximum expected 50% (sample proportions can fluctuate). Check phase, scoring and assumptions.
14. **AHL:** Total 160; expected 90, 30, 30, 10. $\chi^2 = 100/90 + 100/30 + 0 + 0 = 40/9 \approx 4.444$, below 7.815. Do not reject the specified ratio at 5%; other models and sampling uncertainty remain. Significance does not assign a posterior probability to the null or uniquely prove independent assortment.

Exam tips

Write parents, gametes, phenotype key and assumptions before a grid. Distinguish genotype/phenotype and dominance/frequency. State the conditioning group for pedigree and X-linked probabilities. Label plot units, quartile convention, fences and observed whiskers.

- **AHL:** State phase, count recombinants, derive expected counts and use the correct df. Failure to reject does not prove a unique mechanism.

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D.3.3 · Homeostasis

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This handout covers D3.3: internal conditions, negative feedback, blood glucose, diabetes and human thermoregulation. Sections marked **AHL** are for Higher Level only.

Prerequisites: membranes, transport, hormones, cell respiration and blood circulation. All numerical records and organ-flow allocations below are invented teaching data, not patient measurements, diagnostic cut-offs or results from an examination paper.

Homeostasis maintains suitable internal conditions

A person can move from a warm room to a cool street while their cells continue working. The external environment changes more than the conditions immediately surrounding those cells. **Homeostasis** is regulation of the internal environment within suitable limits despite disturbances. Relevant human variables include core temperature, blood pH, blood glucose concentration and blood osmotic concentration.

The internal environment includes blood plasma and tissue fluid around cells; it is not simply the air beside the skin. Enzymes and membranes work within ranges of temperature, pH and solute conditions. Excessive change can alter reaction rates, protein structure, ion gradients or cell volume. Regulation therefore supports coordinated metabolism, rather than keeping a number constant for its own sake.

A set point is a reference around which a variable is regulated. Use a suitable range and allow ordinary fluctuations, measurement error and time delays. A classroom reference of 5.0 mmol/L for blood glucose is a model value, not a universal target for every person and situation. Different measurement sites can also give different temperature readings.

Homeostasis requires continued exchange and energy use. A steady internal concentration can coexist with rapid entry and removal of substances. An unchanged glucose value does not mean that glucose is neither absorbed nor used.

Negative feedback opposes deviations in either direction

A **negative feedback** loop links detection, coordination, signalling and an effector response. A detector responds to the regulated variable; a coordinating process relates that information to a suitable reference; effectors change relevant fluxes. One tissue can perform more than one role. Pancreatic endocrine cells can detect glucose changes and alter hormone secretion without every correction first requiring a separate decision in the brain.

If glucose rises, a response that increases its removal from blood opposes the rise. If it falls, a response that increases supply to blood opposes the fall. As the deviation

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decreases, the corrective drive generally decreases. Negative refers to opposition to the disturbance, not a negative measured concentration or a harmful effect.

Positive feedback reinforces an initial change. For example, cervical stretch and oxytocin can amplify uterine contractions until delivery removes the major stimulus. Such amplification can complete a specific event, but by itself it does not stabilize a variable above and below a reference. Homeostatic control therefore commonly uses negative feedback.

Detection, hormone transport and cellular responses take time. A delayed response can allow a peak or a small overshoot before recovery. Neither a perfectly horizontal line nor instantaneous correction is required for homeostasis. Conversely, every return toward an initial value is not proof of one particular feedback mechanism: the disturbance may simply have ended.

Insulin and glucagon coordinate glucose supply and use

The endocrine pancreas contains islets with beta cells and alpha cells. Rising blood glucose normally stimulates beta-cell secretion of **insulin** . The hormone travels in blood and acts on target cells with suitable receptors. It is a signal, not the enzyme that physically joins glucose molecules into glycogen.

Insulin promotes glucose uptake in skeletal muscle and adipose tissue, including movement of GLUT4 transporters to the surface. It also promotes storage and reduces glucose output by the liver, including promotion of glycogen synthesis and suppression of glucose-producing pathways. These processes lower raised blood glucose. Do not give liver cells the same GLUT4 uptake mechanism as muscle: liver glucose transport and metabolic regulation differ.

Falling glucose normally promotes alpha-cell secretion of **glucagon** . In the liver, glucagon favours glycogen breakdown and glucose production from suitable non-carbohydrate substrates, increasing glucose supply to blood. Glycogen breakdown and glucose synthesis are different pathways. Muscle glycogen mainly supports the muscle's own metabolism; muscle is not simply a second liver exporting its glycogen as free blood glucose.

As glucose returns toward its suitable range, the original stimulus for the increased corrective secretion declines. Basal secretion can remain; "less insulin" does not necessarily mean no insulin. After a meal and during fasting, several hormones, nutrient inputs and activity interact. The two-hormone model explains key opposing actions without claiming that only two substances ever influence glucose.

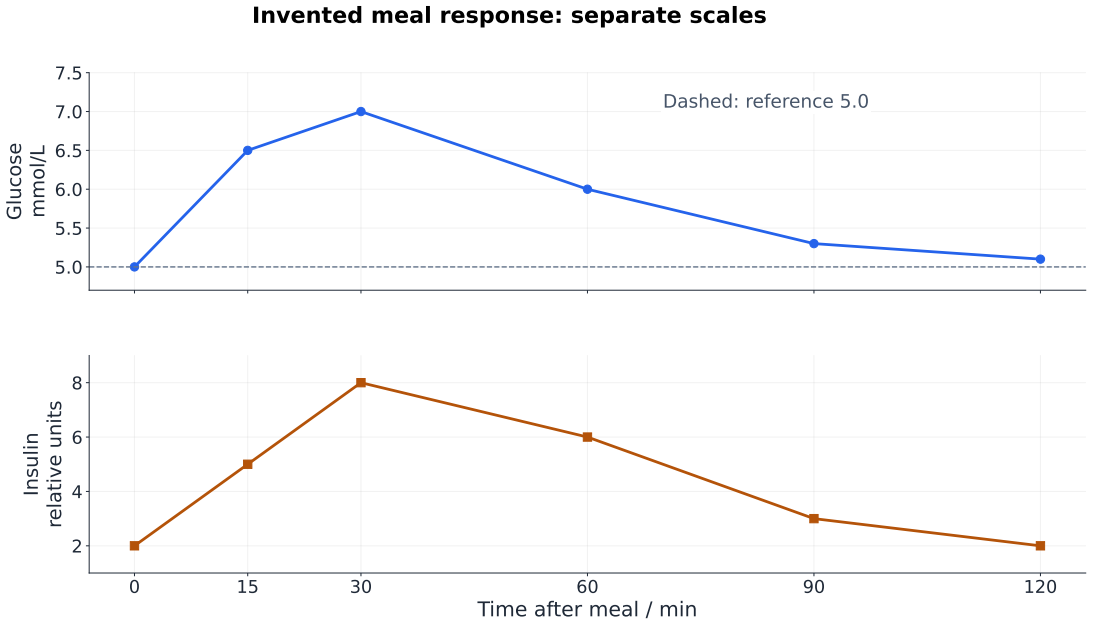
A time course shows sequence and delay rather than unique causation

Consider this invented model record after a meal at time zero. Insulin is given in relative units; those numbers cannot be compared directly with mmol/L as though

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both measured the same substance.

Time / min	Glucose / mmol/L	Insulin / relative unit
0	5.0	2
15	6.5	5
30	7.0	8
60	6.0	6
90	5.3	3
120	5.1	2



Read each vertical scale separately. Insulin rises before the later decline in glucose; the observational sequence is consistent with the model, rather than a unique causal test.

Relative to the model 5.0 mmol/L reference, the peak change is $7.0-5.0 = 2.0$ mmol/L. Percentage deviation uses the reference as denominator: $(2.0/5.0) \times 100 = 40\%$. From the peak to 120 minutes, the fall is $7.0 - 5.1 = 1.9$ mmol/L, or about 27.1% of the peak. These percentages differ because they answer different questions.

The largest observed value occurs at 30 minutes. The true peak might occur between observations, so report sampling interval and measurement uncertainty. A glucose-only trace does not directly measure insulin. Even with both traces, meal absorption, activity and other hormones can also influence the pattern. Manipulating one factor under controlled conditions supplies stronger causal evidence than timing alone.

For supplied or approved observational data, standardize timing, meal description, activity and measurement method. Independent participants provide biological replication; repeated readings from one participant do not become several independent people. Analyse supplied records rather than performing unapproved blood sampling

or provoking abnormal glucose values.

Diabetes involves different failures of the regulatory system

In type 1 diabetes, immune-mediated destruction of pancreatic beta cells usually causes severe insulin deficiency. Inherited susceptibility and environmental influences contribute; eating sugar is not the simple cause. It can begin at any age. Management usually requires replacement insulin, monitoring and planned nutrition/activity. Lifestyle management supports health but does not replace missing insulin. General lifestyle measures do not reliably prevent the autoimmune disease; specific therapies can delay onset in some selected high-risk people. NIDDK overview of type 1 diabetes.

In type 2 diabetes, insulin resistance and inadequate beta-cell compensation cause persistently raised glucose. Insulin may initially be present at high concentrations, while secretion can become insufficient relative to need. Family history, age, inactivity and excess body fat can increase risk; no single characteristic determines an individual's outcome. Appropriate activity, nutrition and weight management can reduce risk or delay onset in susceptible groups. Treatment can include medicines and sometimes insulin; "type 2 never needs insulin" is false. NIDDK overview of type 2 diabetes.

Both types can cause long-term damage when glucose regulation is poor. Similar high-glucose readings do not establish the same underlying mechanism. A classroom graph cannot diagnose a person, and age alone cannot distinguish the types. Explain the physiological difference before linking a management method to the problem it addresses.

Thermoreceptors and the hypothalamus coordinate temperature regulation

Peripheral thermoreceptors in skin detect environmental and skin-temperature changes. Temperature-sensitive regions associated with the hypothalamus also respond to internal conditions. The hypothalamus coordinates neural and endocrine responses with information about temperature, rather than measuring the outside air alone.

Rapid neural pathways can alter sweating, skin-vessel diameter and skeletal-muscle activity. An endocrine pathway can change metabolic heat production over a longer timescale: hypothalamic signals regulate anterior-pituitary TSH secretion, TSH acts on the thyroid gland, and thyroid hormones including thyroxin influence metabolism. Thyroxin is secreted by the thyroid, not by the pituitary. It is not the immediate motor signal for every shiver or drop of sweat.

Effectors include sweat glands, smooth muscle in skin arterioles, skeletal muscle and adipose tissue. Their actions change heat production or exchange. Birds and mammals use physiological and behavioural regulation, but the detailed mechanisms here concern humans. Regulation is not perfect independence from the environment: capacity, water supply, clothing, humidity and activity constrain outcomes.

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Human effectors change heat production, retention and loss

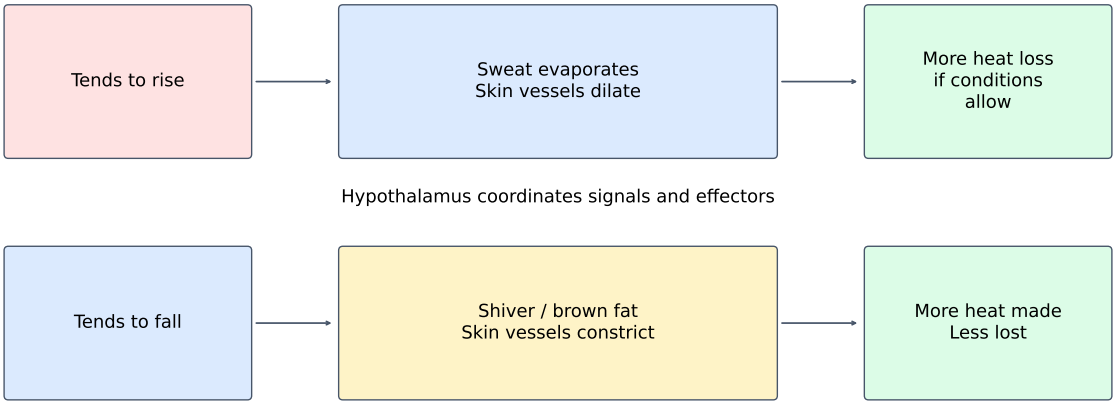
When core temperature tends to rise, vasodilation increases skin blood flow and can increase heat transfer to the surroundings. When it tends to fall, vasoconstriction reduces skin blood flow and conserves heat near the core. The vessels change diameter; capillaries do not move physically nearer to or farther from the skin surface.

Sweat can remove energy when it evaporates from the skin. Water that merely drips away has not provided the same evaporative cooling at that surface. High humidity limits evaporation by reducing the water-vapour gradient. Increased skin blood flow supports heat loss when conditions favour outward transfer; if surroundings are hotter than skin, dry heat exchange can instead bring energy into the body.

Shivering is involuntary repeated skeletal-muscle contraction. Increased metabolism generates heat; it is not purposeful movement to another location. Brown adipose tissue can generate heat through uncoupled respiration. A mitochondrial inner-membrane pathway permits proton return without using ATP synthase, so more of the released energy appears as heat rather than captured ATP. This does not mean respiration has stopped. Brown fat is important in infants and can also be active in adults.

Hair erection uses small muscles attached to follicles. In a furred mammal, raised hairs help trap an insulating air layer. Sparse human body hair makes this a relatively limited insulation mechanism; goosebumps do not equal the thick coat of another mammal.

Human temperature control: oppose the deviation



Name both the effector and how its action changes energy transfer or production. Behaviour can act alongside physiology.

Seeking shade, reducing exertion or changing clothing can limit heat gain; adding insulation, moving to shelter or changing posture can conserve heat. Behaviour and

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physiology are complementary. Negative feedback reduces the corrective response as temperature approaches a suitable range; it does not require all effectors to switch fully on or off together.

Energy balance and measurement qualify temperature conclusions

A positive heat balance means internal thermal energy is increasing. In an invented short interval, heat generation is 160 J/s and heat loss is 120 J/s, giving net retention of $160 - 120 = 40$ J/s. If those rates stay constant for 10 minutes, energy retained is $40 \times 600 = 24\,000$ J, or 24 kJ.

This calculation alone does not give a temperature increase: body mass, effective heat capacity and changes in the exchange rates would also matter. A steady temperature can occur when heat production equals heat loss, even with large flows in both directions.

For a temperature record, state measurement site, device, timing, surrounding conditions and activity. Three repeat values 36.8, 37.0 and 37.2 degrees Celsius have mean 37.0, but repeated readings from one person are not independent participants. A skin reading can change without the same change in core temperature. Avoid treating every recorded difference as a biological effect without considering instrument resolution and method.

AHL: Excretion and osmoregulation are distinct kidney functions

Excretion removes metabolic waste, such as urea formed from amino-acid nitrogen. It differs from egestion of unabsorbed material from the digestive tract. Osmoregulation controls the osmotic concentration of body fluids. Kidneys contribute to both, so changing urine water content does not necessarily change the amount of a waste excreted by the same proportion.

Osmotic concentration counts dissolved particles, with units such as osmol/L. Under an ideal complete-dissociation model, 0.10 mol/L NaCl contributes about 0.20 osmol/L because each formula unit gives two ions. The same molar concentration of a non-dissociating solute gives fewer particles; real solutions can depart from this ideal model. Do not equate osmotic concentration with the concentration of one named ion.

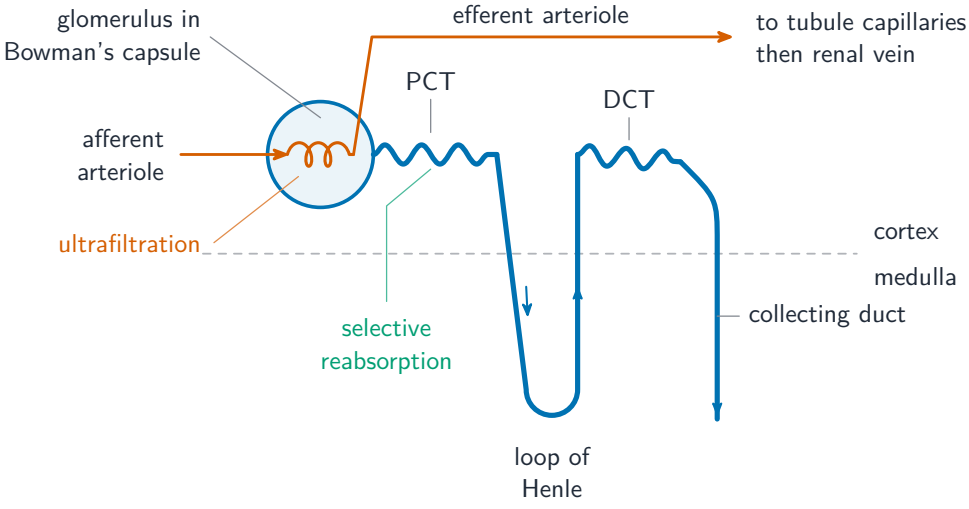
A nephron includes a glomerular filter and a tubule. Blood continues through vessels while filtrate follows a separate tubular route. Urine ultimately enters the renal pelvis, ureter and bladder. The ureter is not the renal vein, and the bladder stores rather than produces urine. NIDDK explanation of kidney function.

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AHL: Ultrafiltration and selective reabsorption act in sequence

Hydrostatic pressure in glomerular capillaries favours passage of water and small solutes across a filtration barrier into Bowman's capsule. Cells and most large plasma proteins are retained in the blood. The resulting filtrate contains useful solutes such as glucose as well as unwanted substances; the filter does not decide which molecule is nutritionally useful. Filtration is pressure-driven, not a pump that actively transports every filtrate component.

The proximal convoluted tubule reabsorbs useful substances into blood. Microvilli provide area; mitochondria support transport requiring energy. Sodium gradients maintained by sodium–potassium pumps help drive cotransport of glucose and other solutes. Water follows appropriate osmotic gradients. These distinct mechanisms must not be collapsed into "every reabsorbed molecule is actively pumped".



Filtration is blood to capsule; reabsorption is tubule to blood. Some substances are also secreted from blood into the tubule. The diagram is a functional model, not a scale section.

In ordinary conditions glucose is almost completely reabsorbed, but transport capacity is finite. A sufficiently large filtered load can leave glucose in urine; its presence is not proof that the kidney actively adds it as a waste. Urea is not simply all removed at the first filter either: tubular handling affects how much is finally excreted.

Ignoring tubular secretion for a specified substance, amount excreted equals amount filtered minus amount reabsorbed. For water in an invented daily model, 180 L filtered and 1.5 L urine imply $180 - 1.5 = 178.5$ L reabsorbed, about 99.17% of filtered water. The large filtered volume is repeated processing, not a claim that the person drinks 180 L each day.

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AHL: The loop supports an osmotic gradient in the medulla

The loop of Henle extends into the medulla. Its ascending limb is relatively impermeable to water; sodium transport, including active transport in its thick segment, helps maintain a high osmotic concentration in the medullary tissue fluid. Water can leave the descending limb more readily. The different permeabilities and opposing flow directions support a medullary gradient.

This gradient makes water reabsorption from a suitably permeable collecting duct possible. Active sodium transport creates or supports conditions for passive water movement; ATP does not directly push water through aquaporins. Do not draw water leaving the ascending limb as freely as salt. Blood flow through the region must also exchange materials without immediately washing away the gradient.

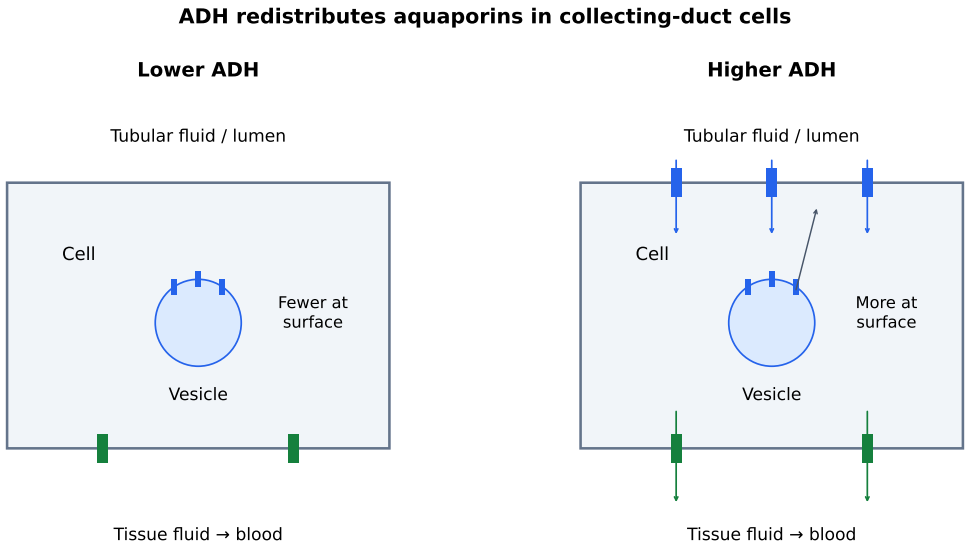
The simplified explanation isolates key roles; urea recycling and other transport processes also contribute in real kidneys. The loop alone does not determine final urine concentration independently of collecting-duct permeability.

AHL: ADH changes aquaporin location in collecting-duct cells

A rise in blood osmotic concentration is detected by osmoreceptors in the hypothalamus. ADH, antidiuretic hormone, is synthesized in hypothalamic neurons and released into blood from the posterior pituitary. Saying "pituitary secretion" should not imply that the posterior pituitary synthesizes the hormone.

ADH acts on collecting-duct cells, promoting insertion of aquaporin-2 channels from intracellular vesicles into the membrane facing the tubular fluid. Water permeability rises. With a suitable medullary gradient, more water is reabsorbed into tissue fluid and returned to blood; urine volume falls and its osmotic concentration generally rises. This response opposes the original increase in blood concentration.

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ADH changes permeability; the osmotic gradient supplies the direction of passive water movement. A second membrane route allows water to leave toward tissue fluid and blood.

When osmotic concentration falls, reduced ADH drive favours removal of these channels from the luminal membrane into vesicles. Less water is reabsorbed in this regulated segment and a larger volume of more dilute urine can result. Other signals, including blood-volume or pressure changes, also affect ADH; this model concerns the osmotic loop.

For an invented constant urea excretion of 0.60 mmol/hour, urine flow 0.120 L/hour gives urea concentration $0.60/0.120 = 5.0$ mmol/L. At 0.030 L/hour, concentration is 20 mmol/L. Fourfold concentration does not mean fourfold urea production: the same amount leaves in less water.

AHL: Organ perfusion changes with activity and total output

Changing arteriole resistance and cardiac output redistributes blood. Vigorous exercise increases skeletal-muscle flow substantially, while gut and kidney flow can decrease. Cerebral flow is regulated and does not simply rise in proportion to total cardiac output. Sleep differs from vigorous exercise, but sleep stage and tissue activity affect the detailed pattern.

These invented allocations illustrate calculations, not fixed normal values for every person or type of exercise. Other organs includes skin and the heart's own circulation; heat-loss demands can influence skin flow.

Region	Sleep / L/min	Wakeful rest / L/min	Vigorous exercise / L/min
Skeletal muscle	0.90	1.00	16.00
Gut	1.10	1.25	0.60
Brain	0.70	0.75	0.75
Kidneys	1.00	1.10	0.40
Other organs	0.80	0.90	2.25
Total	4.50	5.00	20.00

Brain flow remains 0.75 L/min: $0.75/5.00 = 15\%$ at rest versus $0.75/20.00 = 3.75\%$ during exercise. Muscle flow rises sixteenfold, from 20% to 80% of output. A changing total separates absolute flow from percentage.

Kidney flow remains nonzero. Hormones, local flow control and tubular function interact; these allocations alone do not give filtration rate or urinary waste output.

Practice

1. Define homeostasis and identify four human variables it regulates. Explain why a constant concentration need not mean no movement or metabolism.
2. Describe a negative feedback loop for a variable initially above its reference and then below it. Contrast positive feedback and explain why "negative" is not a negative concentration.
3. Trace rising glucose through beta cells, insulin transport and target-cell effects. Distinguish muscle/adipose uptake from liver regulation and explain why insulin is not the glycogen-synthesizing enzyme.
4. Trace falling glucose through alpha cells and glucagon to liver responses. Why is muscle glycogen not simply exported as free glucose by the same route?
5. An invented glucose record rises from 4.8 to 6.0 mmol/L and later falls to 5.0. Calculate the increase, percentage deviation from the initial reference and percentage decrease from the peak.
6. Use the meal-response table to identify the largest observed glucose value and its time. Explain two limits to inferring that insulin uniquely caused the later decline.
7. Compare the physiological basis, risk factors and management principles of type 1 and type 2 diabetes. Correct "only children get type 1", "sugar causes type 1" and "type 2 never needs insulin".
8. Explain the roles of skin thermoreceptors, hypothalamus, anterior pituitary and thyroid in temperature regulation. Distinguish rapid neural responses from the endocrine route.

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9. Explain vasodilation, vasoconstriction, shivering, sweating, brown-fat uncoupling and hair erection. Give one behavioural response and one environmental limit on heat loss.
10. An invented interval has heat generation 180 J/s and loss 130 J/s for eight minutes. Find energy retained. Can this alone give the temperature increase? Explain.
11. Three repeat temperature readings from one person are 36.8, 37.0 and 37.2 degrees Celsius. Calculate the mean and explain why these are not three independent participants. Name two method details needed for a fair comparison.
12. **AHL:** Compare excretion and osmoregulation; trace filtration, reabsorption and the loop's function. With water filtration 150 L/day and urine 1.2 L/day, calculate reabsorbed volume and percentage.
13. **AHL:** Explain osmotic feedback through ADH and aquaporins, naming synthesis/release sites. At urea excretion 0.60 mmol/hour, compare urine concentrations for flows 0.120 and 0.030 L/hour. Why is concentration not production rate?
14. **AHL:** Use the flow table to find brain/muscle fractions at rest/exercise, muscle absolute-flow ratio and absolute kidney-flow change. Why can allocation percentages alone not reveal urine production?

Worked answers

1. Regulation within suitable internal limits: temperature, blood pH, glucose and osmotic concentration. Balanced input and removal keep concentration steady despite continuing fluxes.
2. Above reference, effectors favour a decrease; below, an increase. Corrective drive declines as deviation declines. Negative feedback opposes change; positive feedback amplifies it, as in childbirth contractions. Neither requires a negative measured value.
3. Raised glucose stimulates beta-cell insulin release into blood. Receptor signalling increases muscle/adipose uptake and liver storage while reducing liver output. Liver does not use the same GLUT4 mechanism. Insulin regulates transport/enzymes rather than catalysing glycogen formation.
4. Alpha-cell glucagon release increases liver glycogen breakdown and glucose-producing pathways. Muscle glycogen mainly supports local use rather than direct glucose export. Corrective secretion declines as glucose recovers.
5. Increase $6.0 - 4.8 = 1.2$ mmol/L; $1.2/4.8 \times 100 = 25\%$. Later fall $6.0 - 5.0 = 1.0$ mmol/L; $1.0/6.0 \times 100 \approx 16.7\%$. State the different denominators.
6. Largest observed value: 7.0 mmol/L at 30 minutes; the true maximum may lie between samples. Timing/correlation do not isolate insulin from absorption, activity or other hormones. Glucose alone does not measure insulin.
7. Type 1: usually autoimmune beta-cell loss and insulin deficiency; any age, not simply sugar-caused; replacement insulin. Type 2: resistance plus inadequate compensation; genetic/modifiable risks; activity/nutrition can reduce risk, with

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medicines or insulin used in treatment. Neither age nor one glucose value identifies the mechanism.

8. Skin receptors and hypothalamic mechanisms supply temperature information; hypothalamic neural outputs rapidly alter effectors. Anterior-pituitary TSH stimulates thyroid thyroxin, changing heat generation more slowly. Pituitary does not secrete thyroxin.
9. Skin arteriole dilation raises skin flow; constriction conserves heat. Shivering generates heat; sweat evaporation removes it, limited by humidity. Brown-fat proton return bypassing ATP synthase releases more heat. Raised hairs trap insulating air, with little human benefit. Clothing/shelter/shade alter exchange; hotter surroundings can reverse dry heat transfer.
10. Net retention $= 180 - 130 = 50$ J/s. Eight minutes is 480 s; energy $= 50 \times 480 = 24\,000$ J $= 24$ kJ. Mass, effective heat capacity and changing rates are needed to infer temperature rise.
11. Mean $= (36.8 + 37.0 + 37.2)/3 = 37.0$ degrees Celsius. Repeats share one participant and assess within-person/method variation. Standardize site and device; also timing, activity and surrounding conditions.
12. **AHL:** Excretion removes metabolic waste; osmoregulation controls particle concentration. Glomerular pressure filters plasma into Bowman's capsule while retaining cells/most large proteins. PCT reabsorbs useful solutes/water; ascending-limb salt transport supports the medullary gradient. Reabsorbed water $= 150 - 1.2 = 148.8$ L/day; $148.8/150 \times 100 = 99.2\%$. This is repeated processing, not drinking requirement.
13. **AHL:** Hypothalamic osmoreceptors detect concentration rise. ADH synthesized there is released from posterior pituitary; luminal aquaporin-2 insertion increases permeability, water reabsorption rises and urine volume falls. Low ADH favours retrieval into vesicles. $0.60/0.120 = 5.0$ versus $0.60/0.030 = 20$ mmol/L: fourfold concentration, unchanged excreted amount.
14. **AHL:** Brain: 15%/3.75%, both 0.75 L/min. Muscle: 20%/80%, sixteenfold absolute flow. Renal change $= 0.40 - 1.10 = -0.70$ L/min. Fractions do not measure filtration, reabsorption or waste load; urine output cannot be deduced.

Exam tips

Name variable, detector, signal, target and opposing response. State the percentage reference; distinguish hormone/enzyme, skin/core and heat/temperature.

- **AHL:** Separate blood/filtrate, filtration/reabsorption, gradient/permeability, ADH synthesis/release and absolute flow/output fraction.

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D.4.1 · Natural selection

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This handout covers D4.1: natural selection, sources of variation, competition, abiotic pressures, fitness, heritability, sexual selection and experimental evidence. Sections marked **AHL** are for Higher Level only.

Prerequisites: genes and alleles, meiosis, reproduction and population ecology. Plant comparisons and numerical population models are invented teaching examples. Endler's published guppy results are identified separately; they are not invented measurements or examination questions.

Natural selection connects inherited differences with reproductive success

Imagine a population whose roots vary in depth. During a drought, deeper-rooted plants may obtain more water and leave more viable offspring. If the relevant differences are inherited, later generations can contain more of the contributing alleles. The drought need not instruct each plant to make a useful new allele.

Natural selection is differential survival and reproduction associated with heritable differences in a particular environment. It can change a population over generations. It does not mean that an individual deliberately evolves, that every survivor reproduces, or that a population moves toward a universally perfect form.

Darwin supplied a mechanism for evolutionary change, rather than merely saying that species change. A theory is an explanatory framework supported and tested using evidence, not a guess awaiting promotion to a law. Replacing an influential explanatory framework is a paradigm shift. The selection explanation differed from an account based simply on use, disuse and inheritance of acquired improvements. Modern evolutionary understanding also includes mechanisms other than selection.

Selection operating over immense timescales has contributed to biodiversity through repeated adaptation and divergence. Its local outcome depends on conditions, inherited variation and reproductive success. A change in a measured average alone does not establish which mechanism produced it.

Mutation introduces alleles while sexual reproduction reshuffles them

Variation means differences among individuals. Some variation reflects inherited DNA differences; some reflects environmental effects or interactions. Selection can discriminate between phenotypes, but an evolutionary response requires a heritable contribution to the relevant difference.

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Mutation can introduce new alleles. It can occur before or during exposure to a selection pressure, but useful mutations are not produced because an organism needs them. "Random with respect to need" does not mean all base positions have equal mutation probability or that all mutations have equal effects.

Meiosis produces new allele combinations through crossing over and independent assortment of suitable loci. Random fertilization combines gametes. These processes reshuffle existing alleles; they do not explain the origin of every new allele. In a population with variation, selection affects which combinations contribute to later generations.

A deep-rooted plant could have an inherited tendency, could have grown in a favourable soil patch, or could show plastic growth under local conditions. Measure inheritance and environmental effects rather than assigning every visible difference to a new mutation.

Overproduction and limited resources create competition

Many organisms can produce more offspring than the environment can sustain to reproduction. A plant may produce hundreds of seeds, but only some find suitable space, water, mineral supply and light. Juveniles can compete for food or shelter; reproductive adults can compete for mates. Carrying capacity depends on resource supply and conditions, not a permanently fixed population number.

Intraspecific competition occurs between members of the same species, often with overlapping requirements. Competition need not involve fighting: neighbouring plants can reduce each other's access to light and water. If a heritable feature improves access and eventual reproductive success, its carriers can contribute more alleles to the next generation.

Overproduction alone is insufficient to establish selection. If mortality removes individuals independently of the relevant heritable differences, deaths do not demonstrate a selective advantage for that trait. Conversely, selection can involve differences in mating success or fertility even without conspicuous adult mortality.

Abiotic pressures and biological interactions both affect success

A **selection pressure** is an environmental factor that affects relative survival or reproduction among variants. Low temperature can favour features that maintain function in cold conditions; drought can favour water conservation or access. Extreme weather can affect populations at low as well as high density, so its occurrence need not depend on crowding.

A density-independent disturbance is not necessarily genetically indiscriminate. The same frost can affect genotypes differently if their physiology differs. State both the environmental condition and the inherited feature that changes the outcome.

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Predation, disease and competition are biological pressures. A feature useful against one predator may have a cost elsewhere. Reduced stomatal opening conserves water but can restrict carbon dioxide entry; extensive roots require resources that could otherwise support leaves or reproduction. Adaptation is therefore a context-dependent trade-off, not a label meaning best in every environment.

Fitness measures contribution to later generations

Biological fitness concerns survival value and reproductive contribution of a genotype in a stated environment. It is not simply strength, speed or lifespan. A long-lived individual leaving no descendants may contribute fewer alleles than a shorter-lived individual producing many surviving, fertile offspring.

For an invented comparison, each of two genotypes begins with 20 parents. One group leaves 60 offspring that reach the defined reproductive stage, the other 40. Their mean contributions are 3 and 2 per parent. Taking the first as relative fitness 1, the second has relative fitness $2/3 \approx 0.667$. Use equivalent starting numbers and the same outcome definition.

If one group had twice as many parents, comparing raw offspring totals alone would be misleading. A reproductive-success estimate also depends on time, environment and which offspring stage was counted. Fitness measured in a drought need not remain highest in a wet environment.

Heritable differences must be separated from acquired changes

A scar, a learned skill or an environmentally produced increase in size does not automatically alter the inherited base sequence so offspring receive the same change. An individual can acclimatize without the population genetically evolving. This distinction does not require claiming that every possible inheritance mechanism has been explained by one simple model.

To test the root example, grow offspring from contrasting parents under comparable conditions, using independent families and controlled age, soil and water treatments. A common-garden comparison can reduce environmental differences; growing relatives in only one shared pot would still confound family with pot conditions. Reciprocal environments can reveal genotype–environment interactions.

Evidence is stronger when inherited differences predict both the relevant function and reproductive success, and when population allele measurements agree with the proposed response. Common conditions alone do not remove every parental effect. Nor does failure to observe an increase automatically disprove selection: opposing pressures, gene flow, genetic architecture or limited measurement power can matter.

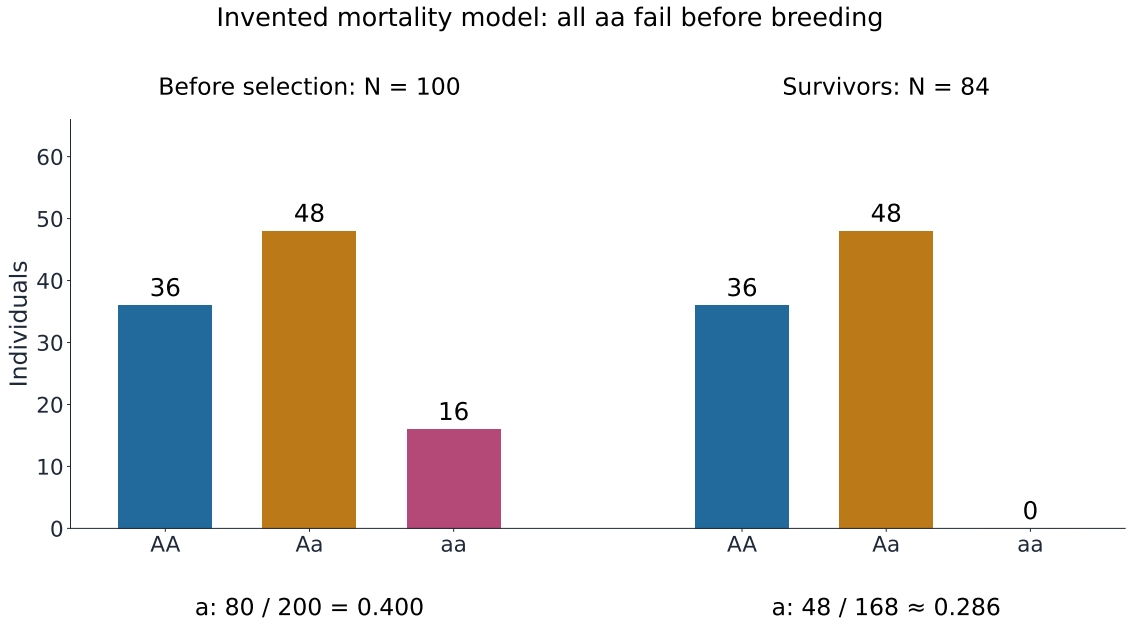
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Allele counts distinguish population change from individual change

An **allele frequency** is the proportion of copies at a defined locus that carry the named allele. For an ordinary diploid autosomal locus, N individuals contribute 2N copies. Count heterozygotes once for each allele, rather than treating each heterozygote as two copies of the same allele.

In an invented 100-plant model, genotypes are AA 36, Aa 48 and aa 16. Copies of a total $48 + 2(16) = 80$ among 200, so frequency is 0.40. Copies of A total $2(36) + 48 = 120$, giving 0.60. Frequencies of the two specified alleles sum to 1.

Suppose all aa plants fail before breeding, while the 36 AA and 48 Aa survive and contribute equally per individual. There are 84 survivors, hence 168 copies. The a frequency among them is $48/168 = 2/7 \approx 0.286$; A frequency is $120/168 = 5/7 \approx 0.714$. No surviving Aa plant changed its own genotype. The composition of the contributing population changed.



After mortality is not automatically a new offspring generation. Transmission carries the survivors' alleles forward; later frequencies depend on the stated breeding and selection conditions.

If those survivors contribute equally to gametes and no further process changes frequencies, a does not have to decline again merely because offspring are born. Continued selection against aa can cause further change, while a can remain hidden in heterozygotes. Root depth in real plants is more complex than this single-locus teaching model.

Another invented record has 90 of 200 copies of a named allele initially and 120 of 200 later: 45% to 60%, an increase of 15 percentage points. Relative increase is

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$15/45 \times 100 \approx 33.3\%$, a different comparison. A rise to 140 of 200 is 70%, ten percentage points above 60%.

A frequency shift alone does not uniquely prove natural selection. Migration, chance sampling of reproduction in a finite population, sampling error and changing sample definitions can also affect observations. Measure reproductive differences and heritability, compare suitable controls, and specify population boundaries and collection times.

Sexual selection can favour traits with survival costs

Sexual selection arises through differences in mating or fertilization success. Competition for access to mates and mate choice can favour physical or behavioural traits even when those traits carry an energetic or predation cost. A benefit in one component of fitness need not be a benefit in every component.

Male birds of paradise provide a context: elaborate plumage and courtship displays can affect attraction of mates, while production, maintenance and display consume resources and can increase exposure. Do not assume that the most conspicuous male necessarily leaves the most offspring under every condition, or that preferences are identical in every population.

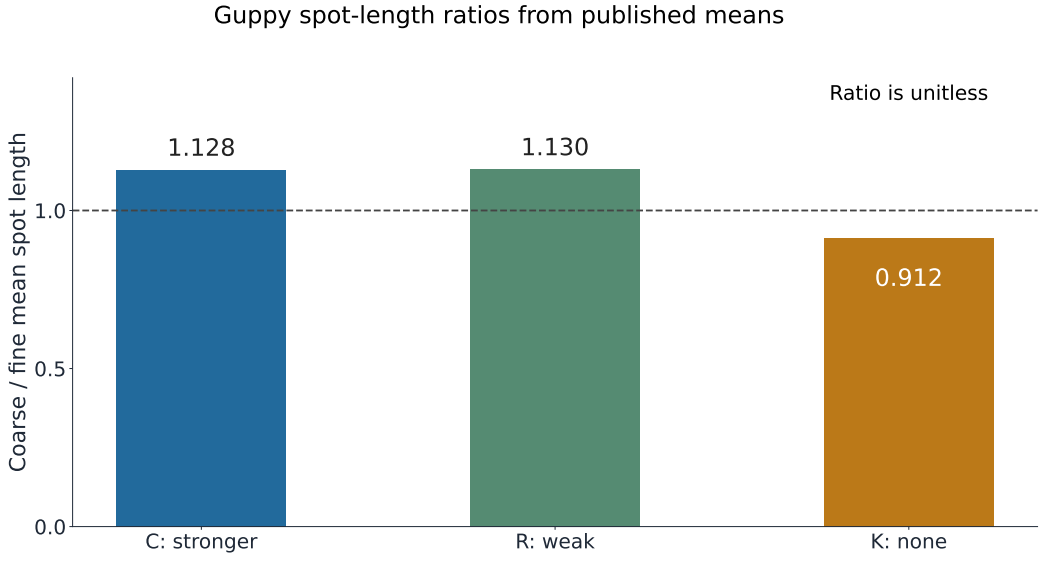
A clear explanation connects inherited variation, a mating-success difference and transmission to offspring. "Females liked it, so males grew new inherited colours" omits the existing variation and differential reproduction. Experiments can compare mate responses while controlling background, light and other visual cues, subject to suitable animal-care requirements.

Endler's guppies show why background and predation must be considered together

Endler's 1980 guppy study varied predation and gravel background. Table 3 reports these mean lengths for all colour spots. C denotes stronger predation, R weaker predation and K no predators. The coarse/fine ratios summarize the published values without converting them into invented measurements.

Treatment	Fine	Coarse	Coarse/fine ratio
C	1.17	1.32	1.128
R	1.54	1.74	1.130
K	1.47	1.34	0.912

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Ratios above one mean larger mean spots on coarse gravel; below one reverses that comparison. No uncertainty interval is supplied here.

Background matching occurred with predation, while the no-predator comparison reversed direction, consistent with a role for sexual selection. Different pattern components responded differently. Read the original paper, table 3 and discussion.

For an experimental comparison, identify the independent variables, measured outcome, treatment allocation and independent replicate units before drawing a conclusion. Many fish in one pond do not supply many independent ponds. A ratio of means is descriptive; it does not by itself establish statistical significance, remove body-size effects or prove one mechanism uniquely. These general evaluation principles apply beyond guppies.

AHL: Gene pools connect inheritance with population evolution

The gene pool comprises the genes and alleles present in a population. A change in its allele frequencies is a genetic description of evolution. Natural selection changes contributions according to heritable differences, while mutation introduces variants, gene flow moves alleles between populations and genetic drift reflects chance changes in finite populations.

Neo-Darwinism integrates natural selection with genetics. Darwin's mechanism did not require knowledge of DNA, but segregation, recombination and inheritance explain how variants are retained and reshuffled rather than permanently blended away. Selection acts through phenotypes; the resulting transmission can change frequencies in the gene pool.

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A pool is not one individual's genotype and does not contain only the most common or beneficial allele. Population boundaries matter: combining distinct groups can hide differences or produce misleading average genotype proportions.

AHL: Human database records show geographical frequency differences

Geographical separation can restrict gene flow, allowing selection, drift and population history to produce different allele frequencies. Human populations still exchange genes; widely separated sample groups must not be described as completely isolated or genetically uniform categories.

For the human ALDH2 variant rs671, the Ensembl record for 1000 Genomes phase 3 gives 33 A copies and 173 G copies in CHB, Han Chinese in Beijing: $33/206 \approx 0.1602$. In CEU, Utah residents with Northern and Western European ancestry, the record lists 198 G copies and no A copies: observed A frequency $0/198 = 0$. These are allele-copy denominators, not 206 and 198 people. The labels are documented by IGSR CHB and IGSR CEU; see the Ensembl rs671 population record.

Record variant identifier, allele orientation, dataset release, sample group, count and denominator before comparing. Do not mix a frequency from one dataset with a denominator from another. These records describe sampled groups, not every person in China, Europe or Utah. Zero observed copies in a finite sample does not prove absence everywhere.

The frequency contrast alone does not identify natural selection as the cause or show its present direction. Distinguishing selection from demographic history requires additional evidence. A database sample supplies a real comparison while preserving uncertainty about mechanism.

AHL: Three selection patterns can affect genetic variation differently

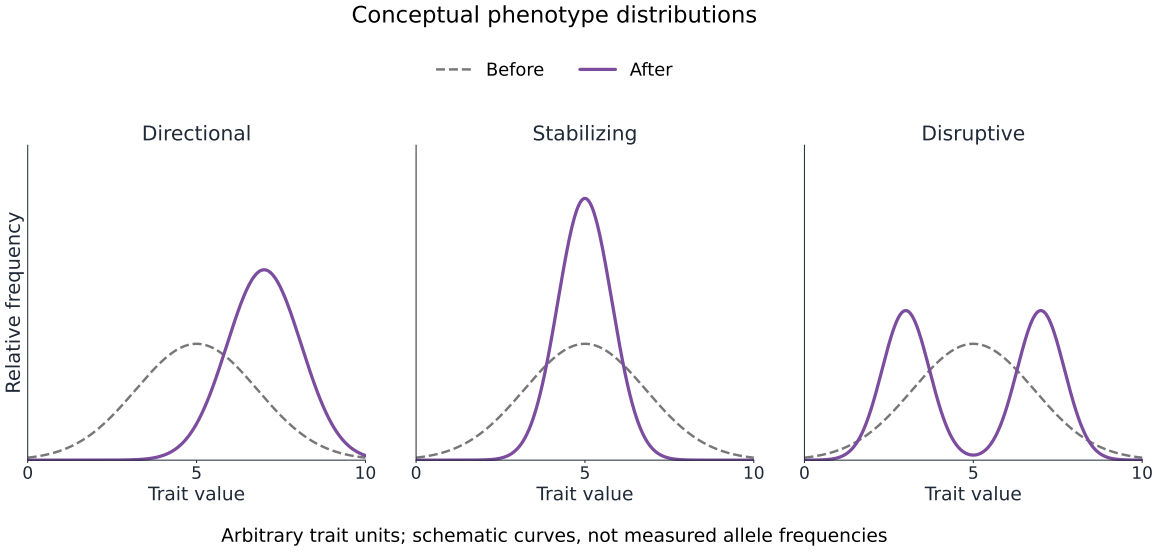
Directional selection favours one end of a trait distribution. If larger roots improve reproductive success in a drought and the difference is heritable, the distribution may shift toward greater depth. It need not continue shifting indefinitely, because costs and environmental conditions can change.

Stabilizing selection favours intermediate phenotypes over extremes. For an invented seed-size model, very small seeds fail to establish while very large seeds reduce the parent's seed number; intermediate size leaves the greatest reproductive contribution. The mean can remain similar while variation narrows. An unchanged trait mean therefore does not show that selection is absent.

Disruptive selection favours both extremes over intermediate phenotypes. In an invented habitat with mainly small soft seeds and large hard seeds, specialized small and large feeding structures might outperform intermediate ones. Two peaks can develop,

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but disruptive selection alone does not prove reproductive isolation or guarantee a new species.



These are conceptual phenotype distributions, not measured population records. Genotyping and breeding evidence are needed to connect a distribution change with particular allele frequencies.

All three patterns can change allele frequencies when phenotypic success has an inherited basis. However, a phenotype-frequency graph is not itself a graph of allele frequencies, and a mean or variance alone does not specify every underlying genotype. Environmental effects can also alter a measured distribution.

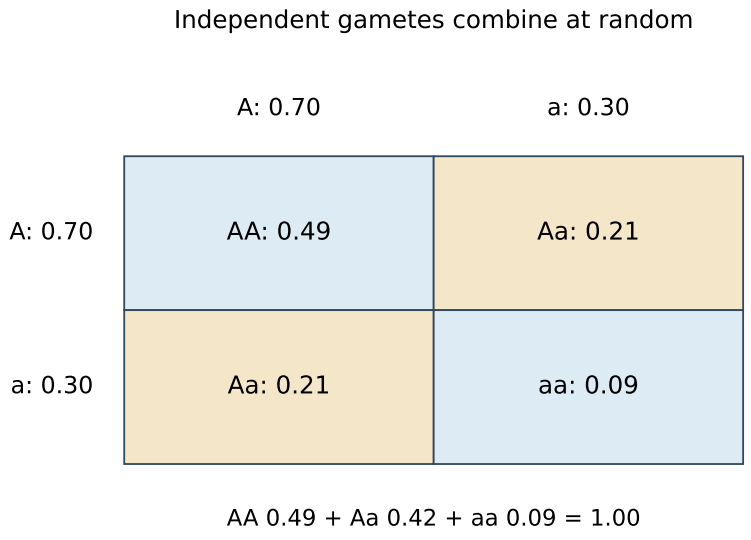
AHL: Hardy–Weinberg predictions begin with random gamete combinations

For a two-allele autosomal locus, let p be frequency of A and q frequency of a. The identity $p + q = 1$ follows from counting all copies of those two alleles; it does not by itself establish equilibrium. If gametes combine randomly, the expected zygote proportions are AA p^2, Aa $pq + qp = 2pq$ and aa q^2.

$$p^2 + 2pq + q^2 = (p + q)^2 = 1.$$

For $p = 0.70$ and $q = 0.30$, predictions are 0.49 AA, 0.42 Aa and 0.09 aa. Among 1000 offspring, expected counts are 490, 420 and 90. These are population expectations under assumptions, not compulsory counts in one small family.

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The two Aa routes each have probability pq. Equal-sized drawing boxes do not mean equal probabilities.

If the recessive phenotype uniquely identifies aa under complete dominance and the equilibrium assumptions are justified, aa frequency 0.09 gives $q = \sqrt{0.09} = 0.30$ and $p = 0.70$. The dominant-phenotype frequency is $p^2 + 2pq = 0.91$, not p. Do not take the square root of the heterozygote frequency and call it q.

When genotype counts are known, direct allele counting works without assuming Hardy–Weinberg proportions: $p = (2N_{AA} + N_{Aa})/(2N)$. For the shared example's survivors, $p = 5/7$ and $q = 2/7$. Random combination then predicts 25/49, 20/49 and 4/49 in the next generation; it preserves those allele frequencies unless another process changes them.

AHL: Equilibrium is a model with testable assumptions

The ideal Hardy–Weinberg model assumes no relevant mutation, migration or selection; random mating at the locus; and a population large enough for negligible drift. Real populations need not satisfy these conditions at every locus.

Non-random mating can change genotype proportions without initially changing allele frequencies. In an invented sample of 100 individuals, 40 AA, 20 Aa and 40 aa give $p = q = 0.50$, but genotype proportions are 0.40, 0.20 and 0.40 rather than the equilibrium predictions 0.25, 0.50 and 0.25. A heterozygote deficit can involve inbreeding or population subdivision; it is not unique evidence of selection.

A departure beyond sampling expectations requires investigating assumptions, scoring and population definition. Agreement is consistent with the model, not proof of every assumption. Separate the calculation, comparison and bounded inference.

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AHL: Artificial selection involves deliberate breeding choices

Artificial selection deliberately chooses breeding parents for desired heritable traits. Crop breeders measure traits, select parents, control crosses and compare descendants under comparable conditions over generations. Selection uses available inherited variation; choosing a parent does not automatically create the desired allele.

A desired trait can bring trade-offs or correlated costs. Related parents can increase homozygosity and expose harmful recessives; reduced diversity can increase shared susceptibility. Record parentage, environment, trait distributions and other performance, not just one striking individual.

Antibiotic treatment unintentionally selects resistant bacteria: natural selection in a human-altered environment. People did not deliberately select breeding parents to obtain the trait. Deliberate reproductive choice differs from unintended environmental selection.

Practice

1. Explain natural selection as a mechanism and why its adoption represented a change in explanatory framework. Distinguish population evolution from an individual's intentional improvement.
2. Compare the roles of mutation, meiosis and fertilization in variation. Correct "a drought produces the useful mutation because the plant needs it".
3. Explain how overproduction and limited light, water or food can create intraspecific competition. Why is high offspring mortality alone insufficient to demonstrate selection for a named trait?
4. Distinguish an abiotic pressure from biological competition. Explain how density-independent weather can still affect genotypes differently, and give one adaptation trade-off.
5. In an invented comparison, 10 parents of genotype P leave 50 offspring reaching a specified reproductive stage; 10 of Q leave 30. Calculate mean contributions and relative fitness of Q if P is 1. Explain why strength or lifespan alone is insufficient.
6. Design a comparison of offspring from deep- and shallow-rooted parents to investigate heritability. Identify environmental controls, independent replicates and a remaining limitation.
7. Allele-copy counts change from 90/200 to 120/200 and later 140/200. Calculate frequencies, percentage-point changes and the relative increase from the first to second observation. Does this alone prove selection?
8. Explain sexual selection using a physical or behavioural bird-of-paradise trait, including a possible survival or resource cost.
9. Interpret the three published coarse/fine ratios in the guppy table. Why can treatment and background change the conclusion?

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10. Explain why many fish from one experimental pond are not many independent ponds, and why a ratio of means alone does not demonstrate significance or remove confounding.
11. For AA 36, Aa 48 and aa 16, calculate A and a frequencies before mortality. If all aa fail and remaining individuals contribute equally, calculate survivor frequencies. Must a decline again in the next generation if no further frequency-changing process acts?
12. **AHL:** Define gene pool and connect inheritance with selection. Compare CHB/CEU rs671 A frequencies and denominators: explain geographical separation and two inference limits. Contrast the three selection patterns, including selection without a changed mean.
13. **AHL:** Under justified Hardy–Weinberg assumptions, 9% of a 1000-person model population has recessive phenotype aa. Calculate p, q, genotype frequencies/counts and dominant-phenotype frequency. Why do known genotypes allow direct allele counting without equilibrium?
14. **AHL:** For 40 AA, 20 Aa and 40 aa, calculate allele frequencies, compare equilibrium predictions and explain the mismatch cautiously. State equilibrium conditions. Distinguish crop breeding from antibiotic-driven resistance and identify a selective-breeding cost.

Worked answers

1. Inherited differences affect reproductive contribution and transmission, changing populations over generations. Selection supplied a new explanatory mechanism, illustrating a paradigm shift. Individual acclimatization need not be inherited evolution.
2. Mutation introduces alleles; meiosis and fertilization reshuffle combinations. Mutations can occur before or during drought, without being directed toward need. Selection changes contributions of available variants.
3. Potential offspring exceed limited resources; same-species requirements overlap. Heritable acquisition differences can affect reproduction. Mortality unrelated to the named trait does not establish its selective advantage.
4. Temperature/drought are abiotic; competition is biological. Frost need not depend on crowding, yet genotypes can differ in response. Water-saving stomatal closure restricts carbon dioxide entry; roots consume resources.
5. P: $50/10 = 5$ offspring per parent; Q: $30/10 = 3$. Relative fitness Q $= 3/5 = 0.60$. Use equivalent groups and outcomes; strength or long life without descendants does not establish contribution.
6. Compare independent families at matched ages; control soil, water and light. Allocate families across replicate pots and, if suitable, reciprocal environments. Persistent differences support inheritance, but parental effects or unmeasured factors can remain.

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7. Frequencies 45%, 60%, 70%; changes +15 then +10 percentage points. First relative increase = $15/45 \times 100 \approx 33.3\%$. Drift, migration and sampling also change records; test inheritance and reproductive differences.
8. Plumage/display can aid mating but cost resources or predator exposure. Heritable variants leaving more descendants can spread. Neither universal preference nor cost-free improvement follows.
9. C/R ratios above one mean larger spots on coarse gravel; K below one reverses this. Background and predator regime interact; pattern components need not respond identically.
10. Pond is the independent treatment unit: fish share its environment. A mean ratio alone supplies no significance test or removal of body-size effects. Replication, allocation, controls and outcome definitions matter.
11. Initially A = $120/200 = 0.60$; a = $80/200 = 0.40$. Survivor N = 84: A = $120/168 = 5/7 \approx 0.714$; a = $48/168 = 2/7 \approx 0.286$. Genotypes did not change. Equal transmission alone need not lower a further; systematic further decline requires a frequency-changing process.
12. **AHL:** A pool contains the population's genes/alleles; genetic inheritance connects differential success with transmission. CHB A = $33/206 \approx 16.02\%$; CEU $0/198 = 0\%$ observed. Separation can restrict gene flow; finite samples and demographic history limit selection inference. Directional favours one end; stabilizing the middle; disruptive both ends. A stable mean can hide reduced variation.
13. **AHL:** $q^2 = 0.09$, q = 0.30, p = 0.70. AA 0.49/490, Aa 0.42/420, aa 0.09/90; dominant phenotype 0.91/910. Direct genotype counts need no equilibrium assumption. Dominant-phenotype frequency is not p.
14. **AHL:** A = $(80 + 20)/200 = 0.50$; a = 0.50. Observed 0.40/0.20/0.40 differs from predicted 0.25/0.50/0.25. Inbreeding or subdivision are possible; selection is not uniquely proven. Conditions: random mating, no relevant selection/migration/mutation, negligible drift in a large population. Crop breeding deliberately chooses parents; antibiotic exposure unintentionally selects bacteria. Breeding can reduce diversity, expose harmful recessives or bring correlated costs.

Exam tips

Link inherited variation, pressure, reproduction and transmission. Count gene copies with the correct denominator. Distinguish percentage points, relative change and evidence of causation.

- **AHL:** Separate alleles, genotypes and phenotypes; justify equilibrium assumptions before square roots. A stable mean or frequency contrast alone does not identify a mechanism.

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D.4.2 · Stability and change

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This handout covers D4.2: ecosystem stability, feedback, mesocosms, keystone species, resource harvesting, agricultural impacts, pollution and rewilding. Sections marked **AHL** are for Higher Level only.

Prerequisites: populations, food webs, energy flow and nutrient cycles. All numerical datasets below are invented teaching examples. Named research and conservation examples are linked separately; none is an examination question or a record of a student investigation.

Ecosystem stability allows continuing change within a persistent system

A forest can keep functioning while individual trees die, seedlings grow and animal numbers fluctuate. Stability means persistence of important ecosystem properties and processes over the timescale considered, not that every organism or population count remains unchanged. A stable concentration can coexist with substantial input and output.

Resistance describes how little a system changes under disturbance; resilience describes its capacity to recover. A forest might resist a mild dry season yet recover slowly after severe fire. State what is being measured: species composition, productivity, canopy cover or nutrient retention need not recover at the same speed.

Fossils provide evidence over much longer periods than modern monitoring. Wing and colleagues' Cerrejón study described a Colombian rainforest flora about 58 million years old, including plant families important in modern Neotropical rainforest. This supports a very long history of rainforest organization. It does not demonstrate that one unchanged community occupied every location continuously, or that all its species were the same as today's.

Energy supply, nutrient recycling and diversity support persistence

Most ecosystems need continuing light input because energy is transferred and eventually dissipated as heat. Nutrients can cycle between organisms and the non-living environment; energy is not recycled in the same way. Decomposers release mineral nutrients that producers can use again, while primary production supplies organic matter to food webs.

Genetic diversity can leave a population with variants that respond differently to drought or disease. Species with partly overlapping functions can provide alterna-

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tives when one declines. Diversity can therefore support resilience, but counting more species does not guarantee resistance to every disturbance. Function, interactions, environmental tolerance and the severity of change all matter.

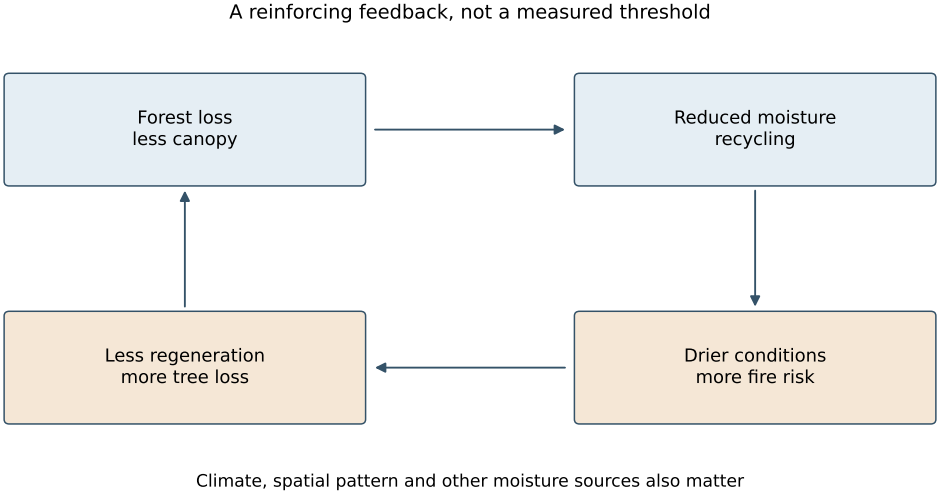
Climatic variables must remain within limits compatible with the community's organisms and processes. Water supply, temperature and disturbance frequency affect survival, reproduction and decomposition. An ecosystem can tolerate ordinary fluctuations yet cross a threshold under a sufficiently large or sustained change.

The **logistic model** describes population growth slowing as density-dependent limits increase. Its **carrying capacity** is the population supportable under specified conditions, not a universal fixed number or a guarantee against overshoot. Population-level logistic growth alone does not establish stability of the entire ecosystem.

Amazon forest loss can weaken the processes that maintain forest

Trees take up soil water and release water vapour through transpiration. Evaporation and transpiration transfer water to the atmosphere and cool surfaces. Atmospheric moisture transport, condensation and rainfall connect forested regions; ocean-derived moisture also matters. Rainfall is not generated exclusively by the trees directly below a cloud.

Removing a large forest area can reduce moisture recycling and alter surface heating and atmospheric circulation. Drier conditions can impair regeneration and favour fire, causing further forest loss. This reinforcing feedback can make a transition difficult to reverse. A tipping point is a threshold beyond which a relatively small additional pressure can produce a large persistent change in system state.



Arrows indicate proposed causal effects, not food-web energy transfer. The diagram gives no numerical tipping threshold.

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The minimum forest area needed to sustain regional processes is uncertain and depends on spatial pattern, climate, drought and fire as well as total area. Avoid turning a proposed threshold into a universally measured cut-off or a prediction that every part of the Amazon changes simultaneously. Flores and colleagues discuss these interacting pressures and uncertainties.

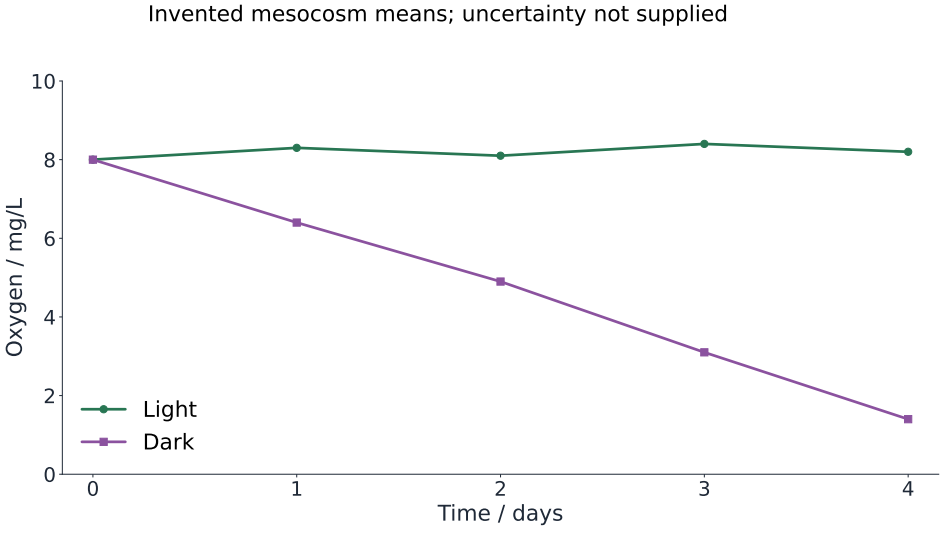
In an invented region, original forest covers 12,000 square kilometres and later covers 9,000. Loss is 3,000 square kilometres; percentage loss is $3000/12000 \times 100 = 25\%$. A further loss to 8,400 is $600/9000 \times 100 \approx 6.67\%$ of the later starting area, but five percentage points of the original area. Always name the baseline.

Mesocosms test a model of a larger ecosystem

A mesocosm is a contained ecosystem model large enough to include interacting organisms and physical conditions. A sealed transparent vessel can exclude matter exchange while allowing light and heat transfer. It is closed to matter, not isolated from energy. Open tanks allow additional inputs and losses; the boundary must match the question.

An aquatic plant–microbial model can connect photosynthetic production, respiration and nutrient recycling. A teacher-approved investigation could compare light availability using replicated vessels with matched water volume, starting community, nutrients, temperature and headspace. Use independent vessels as treatment replicates, random allocation and repeated measurements that do not open the vessel or change the system unnecessarily. Do not seal fish or other animals into a vessel expected to become oxygen-depleted.

The invented records below describe mean dissolved-oxygen concentrations from separate light and dark treatments. They illustrate a prediction; no uncertainty estimate or significance result is supplied.



Stable oxygen does not imply absence of respiration. A falling concentration indicates a net balance, not a direct measurement of every production or consumption rate.

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In light, oxygen production and consumption can approximately balance. In darkness, photosynthetic oxygen production stops while respiration can continue. A decline from 8.0 to 1.4 mg/L over four days is 6.6 mg/L, an average net fall of 1.65 mg/L per day. It does not identify which species consumed each molecule. A sensor drift or unequal starting biomass could also affect interpretation.

Aquatic and microbial models can be easier to maintain than terrestrial sealed systems, but a short successful observation does not prove indefinite stability. Small vessels have artificial boundaries, simplified food webs and limited buffering. Define maintenance, monitoring and welfare criteria before any real investigation; evaluate supplied data when these cannot be met.

Keystone species can have effects disproportionate to their abundance

A keystone species strongly influences community structure relative to its abundance. It need not be the most numerous species or the largest producer. Removing it can alter indirect interactions as well as the organisms it consumes directly.

For example, sea otters eat sea urchins in North Pacific nearshore systems. Reduced grazing pressure can permit kelp to persist, providing habitat for other organisms. Otter loss can therefore affect kelp indirectly through grazers. Other predators, urchin behaviour, storms and temperature also influence outcomes: the same simple cascade is not guaranteed at every coast. See the US Fish and Wildlife Service's account.

To evaluate a keystone hypothesis, compare replicated sites or appropriate before/after and control observations. Measure the proposed intermediate response, such as grazing, rather than only endpoint kelp cover. A correlation between otter abundance and kelp alone does not exclude a common environmental cause.

Renewable harvesting needs replacement, recruitment and precaution

Sustainability means maintaining the capacity of a system or activity to continue over the relevant timescale without undermining that capacity. A **renewable resource** can regenerate; it can still be depleted when removal exceeds replacement. Equal current input and harvest is a simple balance, while a precautionary harvest below uncertain replacement leaves a margin for recovery and error.

For an invented stock balance, with all other gains and losses included in net replacement:

$$\text{next stock} = \text{current stock} + \text{net replacement} - \text{harvest}.$$

A fish biomass of 1,000 tonnes, annual net replacement 100 tonnes and harvest 150 tonnes loses 50 tonnes each year. If those rates remain constant, two years leave 900

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tonnes. With replacement 120 and harvest 80 tonnes, annual gain is 40; three years from 900 leave 1,020 tonnes. These calculations are conditional models: real replacement depends on stock size, age structure, food, temperature and other mortality.

For a terrestrial example, people harvest seeds from Brazil nut trees, *Bertholletia excelsa*, in Amazonian forest. Surviving adults alone do not establish sustainable recruitment: removing too many seeds can reduce the next generation. Peres and colleagues' study of 23 populations associated persistent intensive harvesting with poor juvenile representation. Assess seedlings, juveniles, reproductive adults and harvest intensity over time, with comparable sampling and attention to other habitat differences.

For a marine example, Atlantic cod, *Gadus morhua*, assessment considers spawning biomass, recruitment, age/size structure, landings, discards and mortality. Research surveys help distinguish stock change from changes in fishing effort. See NOAA's stock-management account. A stable catch can conceal falling abundance if effort or technology increases; a falling catch can also reflect a protective quota. Do not treat every cod population as one identical stock.

Quadrat sampling can estimate plant density when positions are selected independently of abundance. For an invented mean of four juveniles per 0.25-square-metre quadrat, density is $4/0.25 = 16$ per square metre; a representative 100-square-metre area gives an estimate of 1,600. Patchiness, counting rules and uncertainty limit extrapolation. Use random coordinates or appropriately stratified sampling, not the most convenient patches.

Agriculture depends on soil, nutrients and wider environmental costs

Crop removal exports nutrients that must be replaced or replenished. Fertilizers can support yield, but poorly timed or excessive applications can increase losses. Nitrogen fixation, organic inputs and mineral fertilizers have different effects and costs; none removes the need for a nutrient budget.

Erosion physically removes soil, reducing rooting volume, organic matter and nutrient stores. Leaching carries dissolved substances downward with water; runoff carries material over the surface. Bare soil, slope, rainfall and cultivation influence these losses. Cover crops, suitable rotations, contour management and maintaining ground cover can reduce risks, depending on the site.

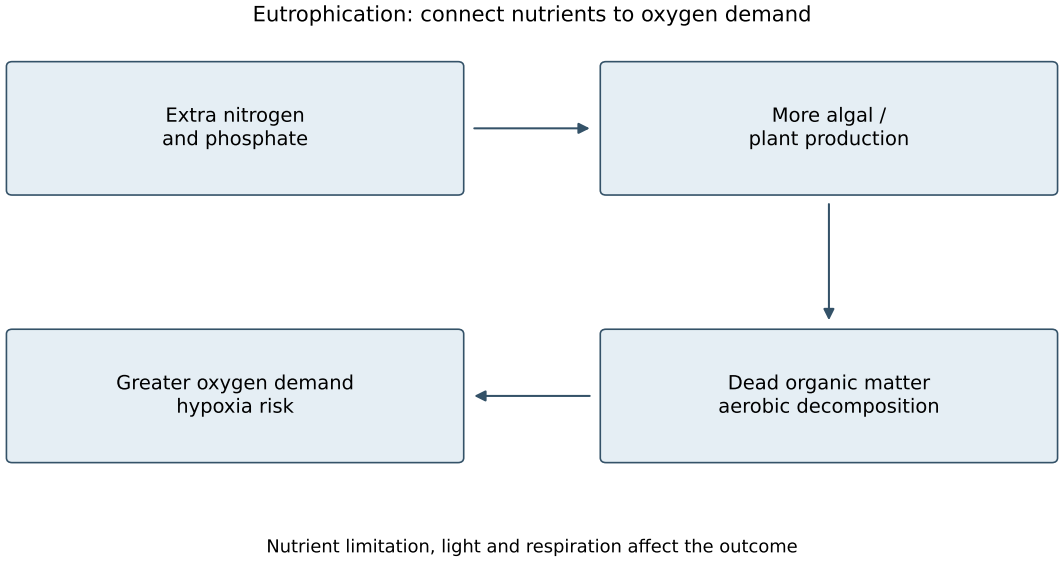
Pesticides can affect non-target organisms and select resistant populations; nutrient and pesticide pollution may affect receiving waters. Fertilizer manufacture, machinery, transport, land-use change and some soil processes contribute to agriculture's carbon footprint. Compare equal output and a stated boundary, rather than one farm's total emissions against another's emissions per kilogram of food. Sustainable assessment includes yield, soil condition, biodiversity, water and long-term input supply.

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Nutrient enrichment can raise oxygen demand in receiving waters

Nitrogen and phosphate fertilizers can reach lakes, rivers and coastal waters through leaching and runoff. Where a supplied nutrient limits growth, enrichment can increase algal or plant production. A dense bloom can shade submerged producers; living organisms respire, and dead organic material supports microbial decomposition.

Aerobic decomposers consume dissolved oxygen. Increased organic supply can therefore raise biochemical oxygen demand, reduce oxygen availability and harm oxygen-sensitive animals. Fish are not usually killed simply because fertilizer "poisons all the oxygen", and photosynthesis during a bloom can temporarily raise daytime oxygen. Day/night changes and decomposition after bloom decline matter.



Enrichment, oxygen demand and dissolved-oxygen concentration are different quantities. Additional environmental factors can alter the final outcome.

A biochemical oxygen demand test measures oxygen consumed under specified incubation conditions. In a simplified undiluted five-day dark test at 20 degrees Celsius, dissolved oxygen falling from 8 to 3 mg/L gives BOD over that interval of 5 mg/L. Another matched sample falling from 8 to 6 gives 2 mg/L. These are concentration differences over the defined test, not automatically rates of 5 and 2 mg/L per day. Formal procedures may require dilution and seed corrections.

Control temperature, starting oxygen and test duration when comparing. Fertilizer reduction, buffer strips and wastewater treatment can reduce nutrient input, but a recovery may lag because stored nutrients and altered food webs remain. Measuring only water colour is insufficient to quantify oxygen stress.

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Persistent pollutants can become concentrated along food chains

Bioaccumulation is build-up within an organism from uptake exceeding elimination. Biomagnification is increasing concentration between trophic levels, often because a consumer eats many contaminated prey while eliminating the pollutant slowly. Compare concentration per equivalent tissue mass, not just total pollutant in differently sized animals.

DDT and its persistent breakdown products can move through food webs. DDE is particularly associated with eggshell thinning and reduced reproductive success in susceptible birds; effects are not identical in every species. For mercury, microbial conversion to methylmercury is important in aquatic systems. Methylmercury can accumulate and biomagnify, damaging nervous systems at sufficient exposure; not every chemical form of mercury behaves identically. See the USGS mercury account.

Invented trophic sample	Pollutant, mg/kg wet tissue
Producers	0.02
Primary consumers	0.10
Small fish	0.50
Predatory fish	2.00

Successive concentration ratios are 5, 5 and 4; overall ratio is $2.00/0.02 = 100$. This is a teaching dataset, not a universal tenfold rule or a real mercury survey. Trophic position, age, tissue type and exposure history must be comparable. The process does not create new pollutant atoms or make energy increase up the food chain.

Large and small plastics harm marine organisms through different pathways

Discarded nets, lines and packaging can entangle animals, restrict movement or cause injury. Ingested plastic can obstruct or damage digestive pathways and interfere with feeding. Effects depend on item shape, size, organism and exposure, not just the total number of items recorded on a beach.

Microplastics are small particles, commonly defined as below 5 mm, including fragments and fibres. They can be ingested by small organisms and enter food webs. Many conventional plastics persist because biological breakdown is slow; fragmentation creates smaller particles rather than automatically mineralizing the polymer. Avoid treating every polymer as chemically identical or claiming that every detected particle causes a demonstrated population decline.

Clear scientific communication can distinguish observed entanglement or ingestion from uncertain long-term effects. Media images can influence perception and action, but a striking image is not an estimate of prevalence. Compare standardized litter surveys, organism observations and interventions such as preventing gear loss and

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reducing avoidable plastic releases.

Rewilding restores processes rather than merely adding attractive species

Rewilding can restore ecological interactions by reintroducing appropriate apex predators or other keystone species, reconnecting habitats and reducing pressures that prevent natural regeneration. Connectivity supports dispersal and recolonization, but design must also consider disease, invasive species, neighbouring land use and human–wildlife conflict. Choosing a species needs evidence about its former role and present habitat.

At Hinewai Reserve on Banks Peninsula, New Zealand, managers support native vegetation regeneration with relatively little interference while controlling selected damaging introduced organisms. Introduced gorse can act as a temporary nurse canopy for native seedlings. This is not a claim that every introduced species should remain everywhere or that restoration requires no management. The reserve's own description explains this context-dependent approach.

Set measurable goals, such as native recruitment, habitat connectivity, water quality or recovery of interactions. Use repeated monitoring and suitable comparisons to distinguish management effects from background change. Restoring a process may take decades, and a visitor's short observation cannot establish complete ecosystem recovery.

AHL: Succession follows changes in conditions and species interactions

Ecological succession is directional change in community composition over time after a new habitat forms or a disturbance changes conditions. Abiotic causes include new exposed substrate, altered water levels or fire effects. Biotic causes include colonization, litter production, shading, competition and modification of nutrient supply. Organisms both respond to and alter their environment.

Primary succession begins on newly available substrate with little or no developed soil, such as a recently exposed glacial surface. Secondary succession follows a disturbance where soil and biological legacies remain, often allowing faster recovery. A forest gap is not a newly created bare-rock surface merely because it has more light.

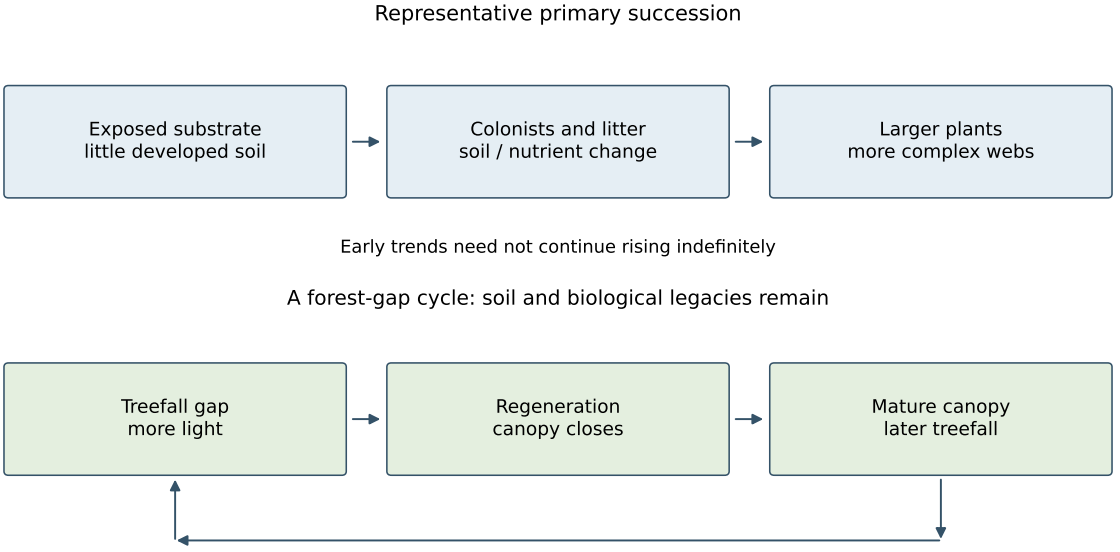
AHL: Primary succession builds soil, production and food-web complexity

A representative glacial-retreat sequence begins with exposed mineral material and colonists tolerant of harsh conditions. Weathering and organic inputs help develop soil. Nitrogen-fixing associations, including alder with *Frankia* bacteria in suitable regions, can increase nitrogen supply. Larger plants can establish as rooting and

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nutrient conditions change, while litter and consumers support more complex food webs and cycling.

Plant size, primary production and diversity often rise during early development, but not every variable increases forever or monotonically. Shade can exclude early colonists; nutrient limitation and soil development alter later productivity. Ecosystem respiration also increases, so greater production does not mean all carbon remains stored.



Conceptual sequences show mechanisms and possible pathways, not a universal timetable or proof that every site converges on the same community.

At Glacier Bay, Alaska, Fastie's 1995 tree-ring reconstruction found multiple pathways related to landscape context and seed arrival. Different-aged sites need comparable starting conditions before they can represent stages of one history; repeated observations strengthen inference.

AHL: Cyclical succession can maintain a mosaic of communities

Treefall in a temperate beech forest opens a canopy gap. Increased light changes regeneration and ground vegetation; growing trees close the canopy and restore shade. Later treefall can repeat this community cycle, which is more than one tree's life history.

Patches at different stages form a mosaic within a persistent forest. Cycles have no fixed duration; severe disturbances can redirect their pathways.

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AHL: Environmental conditions and management affect climax communities

A climax community is a relatively persistent later-successional community under prevailing conditions, not a permanent species list. Climate, disturbances, dispersal and local conditions alter possible endpoints.

Arrested succession prevents an expected later community. Livestock grazing removes tree seedlings and can maintain openness where woodland could establish; not all natural grasslands are destined to become forest. Wetland drainage lowers the water table, altering oxygen, decomposition and establishment; continued drainage can prevent wetland recovery.

Reducing grazing or restoring hydrology can permit change, but seed sources, soils, remaining pressures and time constrain recovery. Name the mechanism maintaining or changing the community.

Practice

1. Explain ecosystem stability, resistance and resilience using a forest example. What does the Cerrejón evidence support, and what does it not prove?
2. Connect energy input, nutrient recycling, genetic diversity and climatic tolerance with persistence. Why is logistic population growth alone insufficient evidence of ecosystem stability?
3. Explain Amazon forest–moisture feedback and threshold uncertainty. For 12,000 → 9,000 → 8,400 square kilometres, calculate successive percentage losses and total loss relative to original.
4. Design a replicated light-treatment mesocosm comparison: controls, boundary and response measure. Interpret the oxygen traces; calculate the dark treatment's average daily net fall and state two limitations.
5. Explain an otter–urchin–kelp keystone effect. Why should an investigation measure the intermediate grazer response and include comparison sites?
6. Define sustainability and renewable resource. With initial stock 1,200 tonnes, annual net replacement 90 and harvest 130 tonnes, calculate annual change and stock after three years. Evaluate the constant-rate assumption.
7. Compare Brazil nut and cod assessments, including recruitment and sampling. Estimate juveniles from six per 0.50-square-metre quadrat across a representative 200-square-metre habitat.
8. Explain erosion, leaching, agrochemical pollution and carbon-footprint boundaries when evaluating agriculture. Give one management action and its mechanism.
9. Explain eutrophication through production, decomposition and oxygen demand. Matched five-day tests fall 9 → 4 and 9 → 7 mg/L: calculate BOD and distinguish interval results from daily rates.

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10. Contrast bioaccumulation/biomagnification using DDT/DDE and methylmercury. Calculate successive and overall table ratios. Compare large/small plastic effects and an evidence limitation.
11. Explain three rewilding mechanisms. Use Hinewai to distinguish minimal interference from no management or removing every introduced plant.
12. **AHL:** Compare primary/secondary succession and abiotic/biotic causes. Explain soil, nitrogen, plant size, production, diversity and food-web changes after glacial retreat. Why may these trends stop or reverse?
13. **AHL:** Explain a forest-gap cycle and persistent patch mosaic. Why are different-aged Glacier Bay sites not automatically one universal sequence?
14. **AHL:** Define climax community and arrested succession. Explain livestock grazing and wetland drainage effects, and limits on recovery after removing pressure.

Worked answers

1. Key properties persist despite turnover. Resistance limits disturbance effects; resilience supports recovery. Cerrejón fossils show rainforest organization and important families about 58 million years ago, not identical communities continuously occupying every site.
2. Energy input replaces dissipation; decomposition recycles nutrients. Genetic variants/overlapping functions support responses within environmental tolerances. One population's density regulation does not establish food-web or biogeochemical stability.
3. Forest loss can reduce moisture recycling, increase dryness/fire and impair regeneration, reinforcing loss. Climate and spatial pattern affect thresholds. Losses: $3000/12000 = 25\%$; $600/9000 \approx 6.67\%$; total $3600/12000 = 30\%$. The second loss is five percentage points of original area.
4. Randomly allocate independent vessels; match water, community, nutrients, headspace and temperature. Sealed matter boundaries still permit energy transfer. Light can balance oxygen production/consumption; dark net fall $= (8.0 - 1.4)/4 = 1.65$ mg/L per day. Separate rates are unknown; sensors/biomass may confound. No uncertainty test is supplied.
5. Otters consume urchins, reducing grazing and indirectly supporting kelp habitat. Grazer measurements test the intermediate mechanism; replicated controls address environmental changes affecting both endpoints.
6. Renewable means able to regenerate; sustainability preserves continuing capacity. Annual change $= 90 - 130 = -40$ tonnes; three years leave $1200 - 3(40) = 1080$ tonnes. Replacement depends on abundance, age and environment; constant rates are assumptions.
7. Brazil nut assessment needs seedling/juvenile replacement, not just surviving adults; cod needs spawning biomass, recruitment and removals, with effort-independent surveys. Density $= 6/0.50 = 12$ per square metre; estimate

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12(200) = 2400. Representativeness, patchiness and counting rules constrain extrapolation.

8. Erosion removes soil; leaching exports dissolved material. Nutrients/pesticides affect non-target systems. Match output and boundaries for carbon comparisons. Ground cover reduces erosion; matching fertilizer to uptake can reduce nutrient loss.
9. Enrichment raises production where nutrients limit growth; shading/dead biomass support decomposition and oxygen demand, risking hypoxia. Matched five-day BOD: $9 - 4 = 5$ and $9 - 7 = 2$ mg/L. These are interval concentration differences, not daily rates; light and timing affect oxygen.
10. Accumulation is within organisms; magnification compares trophic concentrations. DDT/DDE can impair bird reproduction; methylmercury can accumulate with neurotoxic effects. Ratios 5, 5, 4; overall 100. Large items entangle/obstruct; small particles enter organisms/webs. Detection alone does not establish prevalence or population harm.
11. Restore interactions, reconnect habitat and reduce regeneration barriers. Hinewai selectively controls damaging introduced organisms while gorse can temporarily shelter native seedlings. Goals and repeated monitoring test recovery; minimal interference still involves management.
12. **AHL:** Primary starts with little/no developed soil; secondary retains soil/legacies. Substrate/water changes are abiotic; colonization, litter, shade and competition are biotic. Soil/nitrogen development can support larger plants, greater early production/diversity and richer webs/cycling. Later shade, competition and nutrient limits alter trends.
13. **AHL:** Treefall opens a gap; regeneration closes the canopy until another disturbance. Patches differ while forest persists. Glacier Bay sites differ in context/seed arrival; age differences alone do not establish comparable starting conditions or one universal pathway.
14. **AHL:** Climax means relative later persistence under stated conditions. Grazers remove seedlings; drainage alters wetland water, oxygen and establishment. Removing pressure permits change but seed sources, soils, other pressures and time constrain recovery.

Exam tips

Name the boundary, timescale and measured property. Separate energy flow, nutrient cycling, stock, catch and percentage baselines. Trace mechanisms and qualify causal claims.

- **AHL:** Connect organisms with changed conditions. Distinguish a primary sequence, a patch cycle and management that arrests or redirects change.

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D.4.3 · Climate change

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

This handout covers D4.3: human causes of climate change, reinforcing feedbacks, boreal carbon balance, polar habitats, ocean productivity, range shifts, coral reefs and carbon sequestration. Sections marked **AHL** are for Higher Level only.

Prerequisites: energy transfer, carbon cycling, ecological interactions, inheritance and selection. All numerical models and plotted teaching records below are invented. Named research examples are linked separately; they are not invented datasets or examination questions.

Climate evidence concerns patterns rather than one day's weather

Weather is a short-term atmospheric state; climate concerns distributions over longer periods and regions. A cold day in one town cannot refute a global warming trend. Equally, one unusually hot day cannot by itself establish a long-term trend or attribute its whole temperature to one cause.

Compare like-for-like records: location, season, measurement method, reference period and uncertainty matter. A temperature anomaly is a difference from a stated baseline, not the absolute temperature. An invented change from anomaly +0.4 to +1.0 degrees Celsius is a rise of 0.6 degrees, not a 0.6-degree absolute temperature or a meaningful universal percentage warming.

Antarctic ice-core records show associations between atmospheric carbon dioxide and reconstructed temperature over long timescales. A positive correlation does not by itself determine causal direction or exclude feedback and common drivers. Independent physical evidence that greenhouse gases absorb and emit infrared radiation, combined with other observations and modelling, supports a causal warming role. Past natural climate change does not exclude present human influence.

Human activities increase atmospheric carbon dioxide and methane

Burning fossil fuels transfers carbon from long-term geological stores to the active atmosphere–ocean–biosphere system. Deforestation can release stored carbon and reduce future uptake. These changes can raise atmospheric carbon dioxide even while oceans and vegetation absorb some emissions; an absorbing sink need not remove every added molecule.

Methane sources associated with human activity include leaks during fossil-fuel production and transport, anaerobic decomposition in landfills and flooded rice fields,

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and microbial digestion in ruminants. Methane is not released only by industry, and carbon dioxide is not produced only by respiration. Distinguish an emission rate from the resulting atmospheric concentration, which also depends on removal.

Greenhouse gases absorb and emit infrared radiation at relevant wavelengths. Raising their concentrations changes radiative transfer and the energy balance; they do not simply form a solid roof or reflect all outgoing radiation. The natural greenhouse effect makes Earth's surface warmer than it would otherwise be; increasing it changes conditions further. Ozone depletion is a different process.

For an invented surface budget, incoming power 200 units and reflected power 50 give absorbed power $200 - 50 = 150$ and reflected share $50/200 = 25\%$. With incoming 240 and reflected 72, absorption is 168 and reflection 30%. For 180 and 36, absorption is 144 and reflection 20%. These simple budgets isolate reflection; they are not complete planetary temperature models.

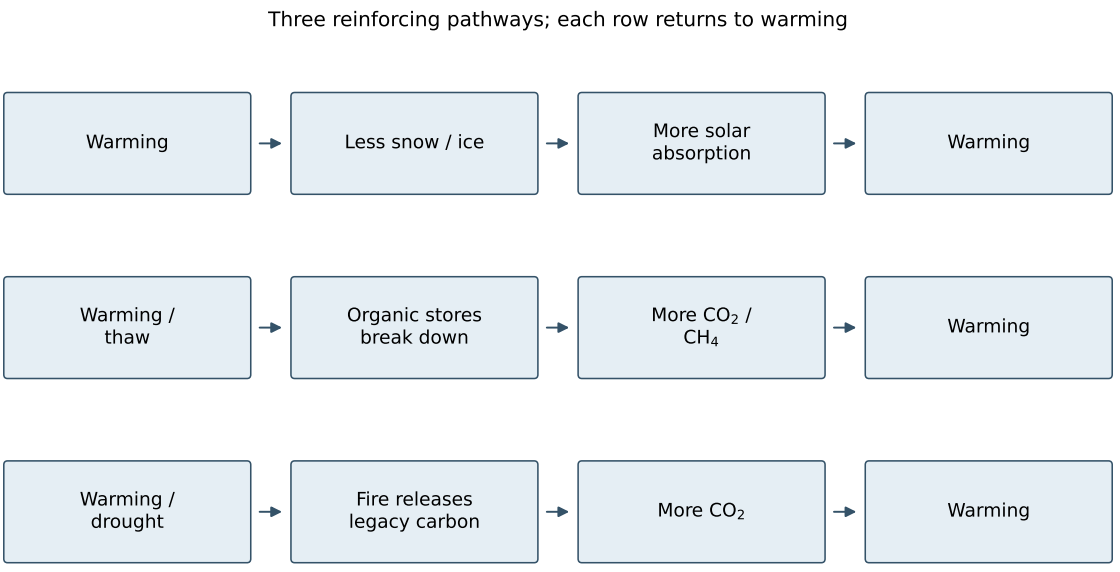
Reinforcing feedbacks can amplify an initial warming

A positive feedback reinforces a change. It is not a claim that the outcome is beneficial or that a variable must become numerically positive. Identify an initial change, an intervening mechanism and an effect that strengthens the initial change.

Loss of reflective snow and ice can expose darker surfaces, increasing absorbed solar energy and promoting further warming and melting. Warming and thaw can expose previously frozen organic material to microbial decomposition. Aerobic pathways release carbon dioxide; waterlogged low-oxygen conditions can support methane production. Ice or frozen soil does not have to contain a pre-made store of every gas later produced.

Peat warming or drying can accelerate decomposition of accumulated organic matter. Drought and fire can release carbon stored in vegetation and soils, while damaged vegetation may take up less carbon. More greenhouse gas can then strengthen warming. Rates depend on moisture, oxygen, substrate and community: decomposition does not rise without limit at every temperature.

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Each row closes back to warming. These are causal pathways, not measured rates or a claim that every location responds identically.

Carbon-rich deep water can release carbon dioxide when circulation brings it toward the surface. Warming can reduce gas solubility; circulation and biological processes also change exchange. This can contribute to feedback, but it does not mean the whole modern ocean is automatically a net carbon source. Local exchange and the global net balance are different quantities.

Boreal forests can switch from accumulating carbon to losing it

Taiga stores carbon in living biomass and organic soils. Warmer conditions, reduced winter snowfall and drought can stress trees, reduce primary production and contribute to forest browning. Fire can burn vegetation and older stored soil carbon, sometimes called legacy carbon. More frequent severe fires can leave less time to rebuild stocks between disturbances.

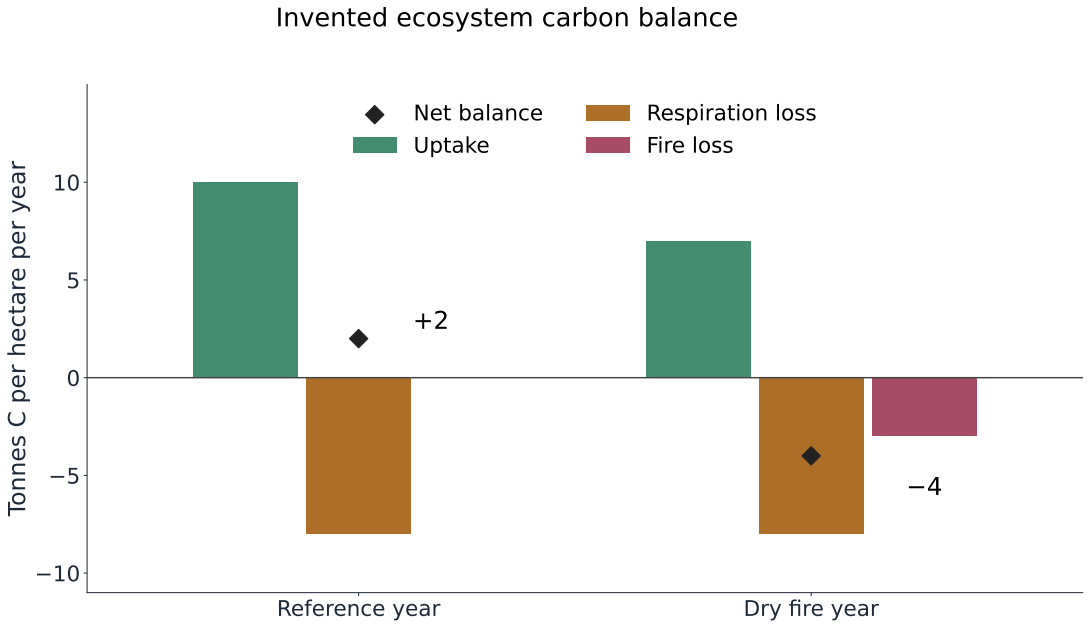
A sink takes up more carbon than it releases over the stated boundary and interval; a source releases more than it takes up. A large standing carbon stock is not automatically a current sink. Crossing a threshold into repeated loss can reinforce warming and make recovery harder, illustrating a possible tipping process.

Walker and colleagues' boreal-soil research used radiocarbon evidence after Canadian fires to investigate combustion of legacy carbon, including vulnerability in dry, younger forests. This supports a mechanism of loss; it does not supply one universal age or temperature at which every boreal forest tips.

For a defined invented hectare, annual uptake is 10 tonnes C and respiratory loss 8, with no fire: net gain is 2 tonnes C. During a dry fire year, uptake 7, respiration 8 and

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combustion loss 3 give $7 - 8 - 3 = -4$ tonnes C: a net source over that year. Even without fire, $7 - 8 = -1$ would remain a loss under those conditions.



Positive net balance means carbon retained by this defined ecosystem. Combustion is counted separately from respiration; other exchanges are excluded by the model.

Report carbon mass consistently: tonnes of C are not tonnes of carbon dioxide. A one-year source observation does not by itself prove permanent collapse; repeated measurements of biomass, soils, production, respiration and disturbance are needed.

Polar ice loss changes breeding platforms and access to food

Landfast sea ice is attached to the coast or another fixed feature; drifting sea ice moves with winds and currents. Both are sea ice, distinct from a land-based glacier. Habitat consequences depend on ice position, season and persistence, not just the annual total area.

Emperor penguins, *Aptenodytes forsteri*, commonly breed on stable Antarctic sea ice. Early breakup can remove the platform before chicks develop waterproof plumage, causing breeding failure. Fretwell and colleagues' satellite study reported evidence of probable failure at four of five colonies studied in the Bellingshausen Sea region during 2022. That regional result is not a claim that every Antarctic colony failed or every chick was directly observed.

Arctic walruses use sea ice as resting platforms near feeding areas over continental shelves. Retreat beyond suitable shallow feeding grounds can increase travel costs or concentrate animals at coastal haulouts. Calves and yearlings can be vulnerable to trampling during disturbance of crowded groups. Walruses feed on benthic organisms, not ice; see the Marine Mammal Commission's account.

Ocean warming and circulation affect nutrient delivery and productivity

Sunlit surface waters can support photosynthesis but become depleted of mineral nutrients. Upwelling and mixing bring nutrient-rich deeper water into the illuminated layer. Temperature, salinity, winds and currents influence this supply; warm water does not manufacture extra nitrate merely by being warm.

A warmer, less dense surface layer can strengthen stratification and reduce vertical mixing with deeper water. Changes in currents or wind-driven upwelling can alter timing and extent of nutrient delivery. Where nutrient supply limits producers, reduced delivery can lower primary production and the energy entering marine food chains. Effects are regional: do not conclude that every current weakens or every coastal upwelling system changes in the same way.

In an invented comparison, production falls from 500 to 400 units per square metre per year: a reduction of $100/500 = 20\%$. At an assumed transfer fraction of 0.10, the next trophic level receives 50 then 40 corresponding units. That is a conditional energy-flow model, not a universal efficiency or a direct prediction of fish catch.

Warming can shift ranges poleward or upslope without guaranteeing escape

A species may establish at a cooler leading edge while losing suitable conditions at a warmer trailing edge. A range shift is a distributional change, not an individual tree walking north or an automatic genetic adaptation. Dispersal, establishment, soil, partners, competition and habitat barriers constrain the response.

Freeman and Class Freeman's New Guinea mountain resurveys documented upslope shifts compared with earlier surveys. Near a summit, moving upward can reduce available habitat; a mountain is not an unlimited staircase. Match survey effort, season and detection when comparing boundaries.

North American trees show varied responses. Sittaro and colleagues' Quebec inventories reported predominantly northward shifts among the studied species, with variation and lag behind warming. Zhu and colleagues' adult–seedling comparison found patterns consistent with contraction for many eastern US species. Juvenile/adult differences suggest recruitment changes but are not identical to tracking every boundary through time. These findings oppose a universal claim that all tree ranges simply move north together.

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Vertical extent is not habitat area. The model illustrates a constraint, not measured New Guinea bird distributions.

In the model, limits 900–1600 m give a 700 m vertical extent; later limits 1200–1800 m give 600 m. The lower boundary rises 300 m, the upper 200 m, and extent contracts by $100/700 \times 100 \approx 14.3\%$. Actual area requires topography and habitat mapping, not simply subtraction of heights.

Coral bleaching and reduced calcification are different threats

Reef-building corals are animals with photosynthetic symbionts that supply much of their organic nutrition. Heat stress can disrupt the association and cause loss of symbionts or their pigments: bleaching. The underlying pale skeleton becomes conspicuous. A bleached coral is stressed but not automatically dead; prolonged or repeated stress can cause mortality and reduce recovery.

Rising carbon dioxide also changes seawater chemistry. Dissolved carbon dioxide increases hydrogen-ion concentration and lowers pH; carbonate availability can decline, making calcium-carbonate skeleton formation more difficult. Acidification means a pH decrease, not necessarily a value below 7. It is not the same mechanism as heat-induced bleaching. See NOAA's ocean-acidification explanation.

Loss of coral cover and structural complexity can remove feeding, shelter and breeding habitats, affecting the wider reef food web. Pollution, disease and destructive disturbance interact with warming and chemistry. Reduced local pressures may improve resilience but cannot by themselves remove global greenhouse-gas forcing.

Carbon sequestration and ecological adaptation address different parts of the problem

Mitigation addresses causes, for example reducing fossil-fuel emissions or increasing durable carbon uptake. **Adaptation** reduces harm from impacts, for example reconnecting suitable habitats or adjusting water management. One project can contribute to both, but an adaptation measure does not automatically reduce atmospheric greenhouse gases.

Afforestation establishes forest on land not recently forested; forest regeneration restores tree cover through recovery. Growing vegetation takes up carbon, but net sequestration depends on soil disturbance, respiration, harvest, fire risk, management and what would otherwise happen. Carbon is not stored permanently merely because a seedling is planted.

Plantations of non-native trees can produce rapid biomass growth under suitable conditions; native regeneration can support habitat and ecological functions. Compare the same timescale, boundary, baseline, biodiversity effects and permanence risks. Species choice, water demand and reflectivity matter; neither approach is automatically best everywhere.

Restoring peat-forming wetlands can raise water tables and slow aerobic decomposition, allowing organic matter to accumulate when production exceeds loss. Peat formation occurs in temperate and boreal zones and can be rapid in suitable tropical systems. Rewetting can also affect methane emissions, so carbon storage and total climate effects need explicit accounting rather than a blanket zero-emission claim.

Policy comparisons require evidence about effectiveness, cost, equity and uncertainty. A cheap intervention that displaces emissions elsewhere may have little net benefit. State which criterion is being judged and whether the estimate includes construction, maintenance, land-use change and later carbon release.

AHL: Phenology studies the timing of biological events

Phenology concerns events such as budburst, flowering, bud set, migration, nesting and seasonal growth. Temperature, accumulated warmth, snowmelt and photoperiod can act as cues. Photoperiod at a fixed latitude and calendar date does not increase simply because the climate warms.

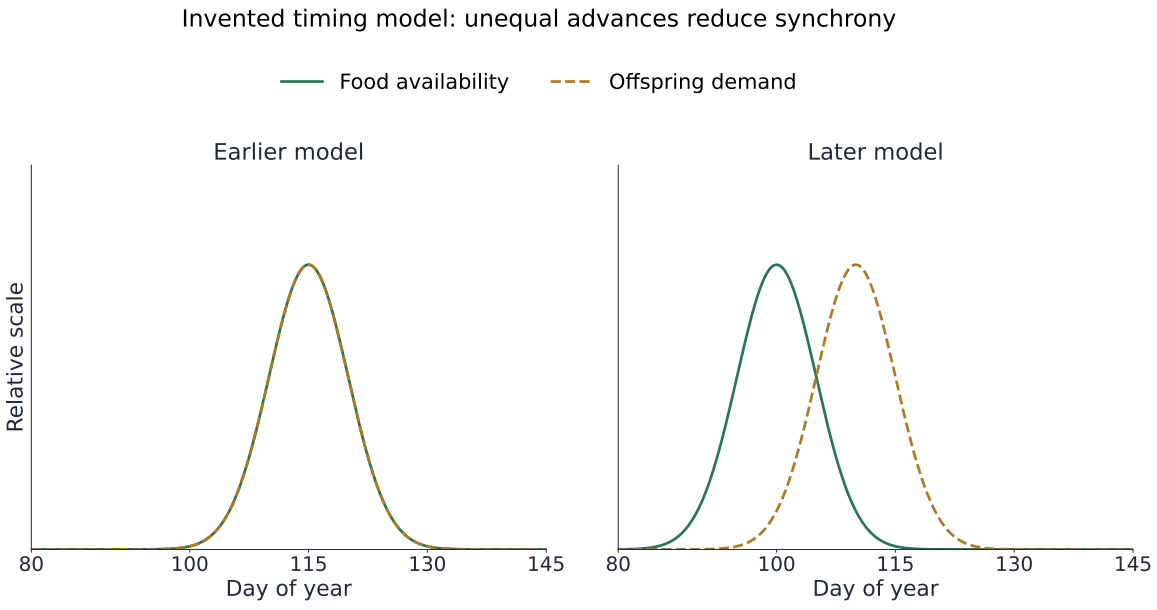
Different species or events can respond to different cues or to different temperature windows. An earlier spring can advance plant growth more than a consumer's arrival or peak offspring demand. Timing change is not automatically evolution: it may be phenotypic plasticity within an individual's inherited response system.

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AHL: Changing synchrony can separate consumers from their food peak

For the Arctic mouse-ear chickweed, *Cerastium arcticum*, earlier warmth and snowmelt can advance spring growth. Migrating reindeer, *Rangifer tarandus*, may not shift arrival equally if their timing depends on different cues, conditions along the route or migration constraints. Reduced overlap with high-quality young plant growth can affect nutrition and reproduction. This case does not require assuming all reindeer eat only one plant or use only day length as a cue.

A second example is the great tit, *Parus major*, and spring caterpillar biomass in north European forest. Visser and colleagues' Hoge Veluwe study found that the estimated caterpillar food peak advanced during 1973–1995 without equivalent advancement of mean laying date. Birds can respond to temperature as well as photoperiod; the relevant cue window, egg production and the delay to chick demand all matter. Do not replace this mechanism with "birds cannot change timing".



The curves have separate relative scales. They illustrate timing overlap, not actual study measurements or equal masses of food and offspring.

In the invented model, food and demand peak on day 115 initially. Later, food peaks on day 100 and demand on day 110: advances of 15 and five days, creating a ten-day difference. Both events advanced; mismatch still increased. Peak dates alone do not determine total usable food: curve width, alternative foods, weather and consumer numbers also matter.

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AHL: Warmer seasons can permit more insect generations

Development of spruce bark beetles such as *Ips typographus* depends on suitable temperatures and season length. A warmer growing season can allow completion of an additional generation where previously only one was possible. This can increase pressure on spruce, especially when drought weakens host defences. Temperature and day length also influence seasonal activity and diapause.

The effect is conditional: excessive heat, unsuitable hosts, predators and other limits can reduce survival. An extra brood from the same parents is not automatically another completed generation. Forest Research's account describes geographical differences in annual generations; no single count applies to every region or year.

In an invented resource-unlimited model, 100 reproductive adults each leave an average of four reproductive offspring: the next generation is 400, and a second completed generation would be 1,600. Real outbreaks need survival, host supply and density effects; multiplying potential reproduction does not prove that many adults actually survive.

AHL: Changed selection can alter inherited variant frequencies

Climate change can alter relative fitness, creating evolutionary change when differences are heritable and affect contribution to later generations. Distributional movement, an individual's acclimatization and altered timing are not automatically evidence of allele-frequency change.

Karell and colleagues' Finnish tawny owl study linked snowy winters with stronger survival selection against the brown colour morph of *Strix aluco*. As winters became milder, that disadvantage diminished and brown-morph frequency increased. Heritable colour variation allowed a population response. The finding does not mean each grey owl turned brown, brown owls are always fitter in warmth, or camouflage alone was proven as the cause; physiological links and other mechanisms require evidence.

In an invented comparison, 40 brown and 60 grey adults experience survival fractions 0.50 and 0.80 in a snowy winter. Survivors are 20 and 48: brown fraction $20/68 \approx 29.4\%$, down from 40%. With survival 0.80 for both, survivors are 32 and 48 and brown fraction remains 40%. These are phenotype proportions among survivors, not direct allele frequencies or a complete multigeneration breeding model.

Practice

1. Distinguish weather, climate, a temperature anomaly and a causal inference. Why does ice-core correlation alone not establish the whole greenhouse mechanism?
2. Explain human carbon dioxide and methane sources, concentration versus emission rate, and infrared absorption/emission. For incoming 240 and reflected 72 power units, calculate absorption and reflected percentage.

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3. Explain three reinforcing feedbacks, including snow/ice, permafrost/peat and fire. Explain how deep-water carbon release can contribute without the whole ocean necessarily becoming a net source.
4. Explain a boreal sink-to-source mechanism and legacy carbon. A model hectare takes up 1,200 kg C annually, loses 900 by respiration and 450 in fire. Calculate net balance and interpret its sign and timescale.
5. Compare emperor penguin and walrus ice-habitat dependence. Give a limitation of the regional penguin study and explain why seasonal timing and location matter.
6. Explain stratification, upwelling and nutrient limitation. Calculate the percentage production fall from 500 to 400 and corresponding next-level supply under assumed 10% transfer; state a limit.
7. Use the New Guinea and North American examples to evaluate a universal northward/upslope escape claim. Calculate changes in model lower/upper limits and vertical extent; why is extent not area?
8. Distinguish heat-induced coral bleaching from acidification and reduced calcification. Explain how coral loss can affect the wider ecosystem without assuming all bleached colonies are dead.
9. Distinguish mitigation and adaptation. Compare afforestation, native regeneration and peatland restoration using boundaries, time, permanence, biodiversity and possible methane effects.
10. For incoming 180 and reflected 36 units, calculate reflection and absorption. Explain why this budget alone cannot predict a unique temperature or a policy's full climate effect.
11. Design a repeated comparison of ecological responses along a climate gradient, controlling sampling effort and timing. Distinguish range movement, plasticity and evidence of evolution; identify a confounder.
12. **AHL:** Define phenology and compare photoperiod/temperature cues using chickweed–reindeer and great tit–caterpillar examples. Calculate food/demand advances and peak separation in the model; why can both advance yet mismatch increase?
13. **AHL:** Explain how climate can increase completed *Ips typographus* generations, with constraints. Calculate one and two model generations from 100 adults and four reproductive offspring per adult; distinguish a sister brood.
14. **AHL:** Explain the tawny owl findings without claiming individual colour change or proven camouflage. Calculate survivor numbers/brown fractions under both model winters and distinguish phenotype frequency from allele frequency.

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Worked answers

1. Weather is short term; climate is a longer distribution. An anomaly uses a baseline. Association can involve feedback or common drivers; radiative physics and independent observations support greenhouse causation.
2. Fossil burning/land change add carbon dioxide; fuel leaks, anaerobic systems and ruminants add methane. Concentration includes removal as well as input. Gases absorb/emit infrared. Absorption $= 240 - 72 = 168$; reflection $= 72/240 = 30\%$.
3. Ice loss increases absorption; thaw/peat change supports microbial gas release; drought/fire releases stored carbon and can reduce uptake. These can reinforce warming. Deep-water ventilation and solubility affect exchange, while the global ocean can still absorb carbon overall.
4. Drought/browning reduce uptake; frequent fire can combust legacy stores. Net $= 1200 - 900 - 450 = -150$ kg C per hectare over the year: a source. A single year does not establish irreversible collapse or a permanent source.
5. Penguins need a stable platform until fledging; early breakup risks chicks. Walrus platforms support resting near benthic feeding; crowding/longer journeys add risks. The 2022 result concerns five studied regional colonies, with probable failure inferred from imagery; it is not a census of all Antarctic chicks.
6. Surface stratification can reduce deep nutrient delivery; changed winds/currents alter upwelling. Production fall $= 100/500 = 20\%$; 10% model supply is 50 then 40. Efficiency and regional response vary; catch cannot be read directly from these values.
7. Responses vary with dispersal, recruitment, barriers and survey design; some ranges lag or contract. Lower limit rises 300 m; upper 200 m. Extent $700 \rightarrow 600$ m contracts $100/700 \times 100 \approx 14.3\%$. Area needs topographic habitat mapping.
8. Heat disrupts symbiosis/pigments; prolonged stress can kill. Carbon dioxide lowers pH and carbonate availability, hindering skeleton formation; pH need not fall below 7. Lost structure and production affect other species. Bleaching can be reversible under suitable conditions.
9. Mitigation addresses drivers; adaptation reduces harm. Compare net additional uptake and avoided losses, baseline, duration, water/soil/biodiversity effects and reversal risk. Plantations are not universally best; native regeneration is not automatically fastest. Rewetting can reduce oxidation while changing methane release.
10. Reflection $= 36/180 = 20\%$; absorption $= 180 - 36 = 144$. Outgoing radiation, heat transfer and other budget terms are omitted; a policy also needs full boundaries and evidence of net effects.
11. Match season, survey effort, methods and habitat; repeat sites and measure temperature plus population responses. A shifted distribution or individual timing response alone is not genetic evolution. Land-use change, detection or food shifts can confound climate attribution.

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12. **AHL:** Phenology concerns event timing; temperature/snowmelt and photoperiod can cue different responses. Chickweed growth and reindeer arrival, or caterpillar peaks and chick demand, need not advance equally. Food advances 15 days; demand five; peak separation grows $0 \rightarrow 10$ days. Width, alternatives and abundance also matter.
13. **AHL:** Suitable warmth/season length can permit another completed generation, but diapause, heat extremes, hosts and mortality constrain outcomes. $100(4) = 400$ then $400(4) = 1600$. Another brood from the original parents is not a new generation completed by their offspring.
14. **AHL:** Heritable colour variants differed in survival under snowy conditions; reduced disadvantage allowed brown frequency to rise. Snowy survivors: $40(0.5) = 20$ brown, $60(0.8) = 48$ grey, brown $20/68 \approx 29.4\%$. Milder model: $32/48$, brown $32/80 = 40\%$. These phenotype counts do not identify allele frequencies or prove one physiological mechanism.

Exam tips

Trace a specified pressure to a biological mechanism and population or ecosystem consequence. State baseline, units, boundary and timescale. Distinguish a model from real study evidence and a plausible mechanism from unique causal proof.

- **AHL:** Keep seasonal timing, completed generations, plasticity and inherited frequency change distinct. A shifted mean or colour proportion needs an appropriate inheritance and sampling model.

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Experimental programme

International Baccalaureate · IB Diploma · Biology · HL

Scope and prerequisites

A tray of seedlings gives different results each day. Is the treatment changing growth, or are starting size, light and measurement differences hiding the answer? An investigation connects a biological explanation to evidence that could challenge it.

This handout develops the experimental programme for both SL and HL: planning, laboratory and field techniques, digital tools, data processing, uncertainty, statistical decisions, conclusions and evaluation. These skills must be practised across the course, not learned only for a final report. The practical skills are shared by both levels.

Prerequisites: units, percentages, biological mechanisms from the relevant topic and spreadsheet basics. All numerical examples and plotted records below are invented teaching data, not published experiments or student submissions. The IB Biology guide gives the programme and assessment requirements; teacher supervision is needed for actual investigations.

A research question defines a biological comparison

Start with a mechanism, then state a measurable question. "Do plants like light?" does not define the organism, treatment or outcome. A sharper question is: how does daily light duration affect change in shoot length of a stated seedling species over seven days, with temperature and watering controlled? Define the measured response rather than replacing growth with an unexplained word such as health.

A hypothesis proposes an explanation; a prediction says what observations would follow under specified conditions. Photosynthesis may supply more material for growth with longer illumination, but other limiting factors and excessive exposure can change the response. A prediction should therefore name the range and expected pattern, not promise that every increase in light always increases growth.

An experiment changes an independent variable and measures a dependent variable. A survey can instead examine two observed variables without assigning either. Correlation between shade and plant density does not alone show that shade caused the difference: soil moisture, disturbance or species identity could also matter. Database investigations and simulations can answer valid questions, but their selection rules, assumptions and data origins still need explanation.

Independent units, controls and pilots protect a comparison

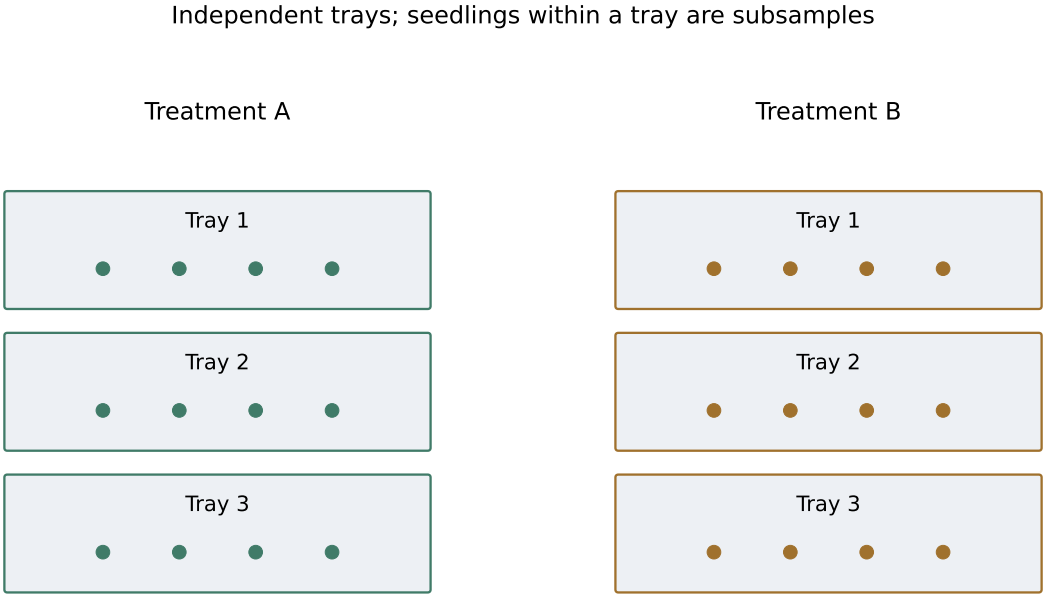
The experimental unit is the independently assigned unit receiving treatment. If light duration is set for a whole tray, five seedlings in that tray are subsamples; they do not create five independent light treatments. Use several independently assigned trays

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per treatment where possible, sample within each, and analyse at the level justified by the design. Measuring one leaf five times checks reading repeatability, not variation among five plants.

Randomly assign eligible units to treatments to reduce systematic allocation differences. If bench position creates a strong light gradient, use blocks of similar positions and randomize treatments within each block. Random allocation helps causal comparisons; random sampling helps represent a target population. Neither automatically repairs a tiny or unrepresentative sample.

A controlled variable is a factor kept comparable, such as seedling age, water volume, soil batch or measurement interval. Explain how it is controlled and why it matters. A control treatment supplies a comparison: for a solvent-delivered substance, a solvent-only control can distinguish the substance from its carrier. A positive control can show that a detection method responds to something known to produce a signal. Not every investigation needs the same type of control.



Three independent trays per treatment; four seedlings per tray are subsamples. The layout illustrates a design, not a required sample size.

Pilot a small version before collecting the main data. Check whether the response changes enough to measure, whether the chosen range is safe and relevant, and whether the apparatus resolves the difference. Choose several treatment levels to reveal shape rather than testing only two extremes. More independent repeats estimate biological variation; more levels describe a response curve. Allocate time to both, using the pilot to justify the balance.

Safety, ethics and a reproducible record belong in the method

Identify a specific hazard, exposure and control. For a low-risk plant investigation, cutting tools, hot water, electricity near liquid, chemical handling and disposal may matter. A vague sentence saying "be careful" does not explain risk reduction. Obtain teacher approval before work; do not culture unknown environmental microbes or expose people or animals to harmful treatments for a class task.

For human observations, consent, privacy, withdrawal and suitable low-risk procedures matter; avoid publishing identifying raw records. Field sampling should respect permission, protected organisms and habitats. Use non-destructive observations where suitable and do not release laboratory organisms or chemicals into the environment.

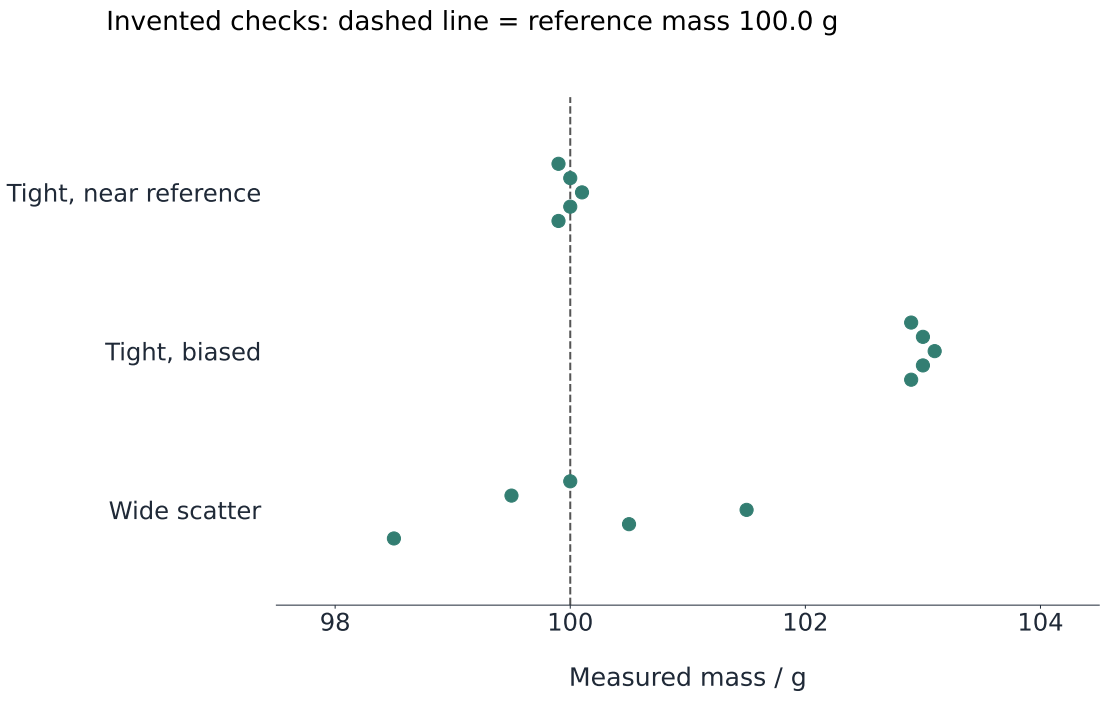
Record dates, source organisms, treatment assignments, apparatus resolution, calibration checks, timings and deviations as work proceeds. Give enough detail for another person to repeat the method. Preserve original raw data separately from calculated values. Record failed readings and their cause; do not erase inconvenient observations or invent replacements. Credit published data, images, code and methods. A shared collection exercise does not make someone else's reasoning or final report your own.

Measurement quality includes accuracy, precision and calibration

Accuracy concerns closeness to a suitable reference or true value; precision concerns closeness of repeated measurements to one another. Validity asks whether the method supports the question and interpretation. A precise sensor can consistently measure the wrong temperature if its calibration is biased. Reliable repetition is useful, but repetition of a confounded design does not establish a valid causal result.

Systematic error is a consistent or predictable measurement bias. A balance zero offset may shift every mass; a scale-factor error may change the slope of its calibration. Random variation creates scatter between readings through measurement processes and biological differences. Averaging independent readings can reduce random uncertainty in a mean; it does not automatically remove calibration bias or all biological heterogeneity.

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All three cases use the same reference. A tight cluster can still lie away from it.

Check a balance with an appropriate reference and tare the container. Read a meniscus at eye level where appropriate; choose glassware and volume for the required precision. Allow a sensor to stabilize, use relevant standards and record units and settings. Calibration applies over a validated range: a single successful check does not guarantee linear behaviour at every value.

Uncertainty expresses a quantified limitation on a result. State its basis: instrument resolution, repeated scatter, calibration information or a stated maximum bound. Half the smallest analogue division can be an appropriate reading estimate in a simple exercise, but it is not a universal uncertainty rule for every instrument. A digital display's last digit alone does not account for all calibration and method errors.

Biological techniques answer different kinds of questions

A light microscope reveals structures within its resolution. Start at low power and use a suitable temporary mount, thin specimen and approved stain when needed. Draw observed features with clear outlines, labels and a scale; distinguish observation from inferred identity. An eyepiece graticule needs calibration against a stage micrometer for the objective used. A scale bar remains useful after an image is resized with the bar; an old printed magnification may not.

For a micrograph, a 20 mm image bar represents 50 micrometres. A cell measuring 32 mm on the same image has actual length $32/20 \times 50 = 80$ micrometres. Image magnification is 20 mm/0.050 mm = 400, a ratio without units. If the image is enlarged, measure the cell and bar on the same copy.

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Chromatography separates components through different interactions with moving solvent and the stationary material. Mark the origin above the solvent surface, avoid dissolving the whole spot into the reservoir, and mark the solvent front promptly. A pigment travelling 36 mm when the front travels 60 mm has $R_f = 36/60 = 0.60$. Both distances start at the origin. Matching a reference under the same conditions supports identification; similar movement alone is not unique chemical proof.

Colorimetry or spectrophotometry measures light transmission/absorbance at selected wavelengths. Use a suitable blank, consistent cuvettes/path length and relevant standards. An invented calibration $A = 0.20c + 0.04$, with concentration c in milligrams per litre and absorbance A dimensionless, gives $c = (0.44 - 0.04)/0.20 = 2.0$ for an unknown of absorbance 0.44. Use interpolation within the standards; turbidity, saturation or a different matrix can invalidate the calibration.

A serial dilution produces a sequence by transferring a measured aliquot into diluent, then mixing before the next transfer. Transferring 1.0 mL into 9.0 mL diluent makes a tenfold dilution with final volume 10.0 mL. Three such steps take a 2.0 milligram-per-millilitre stock to 0.0020 milligrams per millilitre. Use suitable clean equipment and retain the cumulative dilution factor when calculating an original concentration.

Classification uses stated observable characters and suitable identification resources. A dichotomous key gives two contrasting choices at each step; check ambiguous characters rather than forcing a name. Karyograms compare chromosome features; cladograms represent inferred relationships from character or sequence evidence. Molecular models represent selected features and omit others. These techniques support different inferences: a picture or model is not automatically a direct measurement of every feature it depicts.

Field and digital evidence need explicit selection rules

Select quadrat positions by a justified sampling design before seeing which patches contain the target organism. Record area, boundary-counting rules and identification method. Random sampling can estimate an area's mean; a systematic transect can describe change along a measured gradient. Stratify a heterogeneous habitat when needed and weight each stratum appropriately. Four plants in 0.25 square metres give 16 plants per square metre, but one quadrat does not establish the whole site's density.

Sensors provide time series, yet sampling frequency, drift and response delay can change a recorded curve. Image analysis needs a calibrated scale, a stated threshold/segmentation rule and checks against example images; overlapping objects may be counted as one. Databases require matching units, definitions, dates, sampling effort and missing-data rules. A simulated population depends on its model assumptions and parameter choices; changing a parameter tests the model, not automatically the real ecosystem.

Use spreadsheet formulas with correct cell references, clear headings and retained units. Check a few rows independently. A copied formula can systematically divide

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every result by the wrong starting mass. Save processing steps and software settings so a reader can follow the path from raw values to a plotted result.

Raw data, summaries and uncertainty are different objects

Record a raw table with quantity/unit headings, justified precision and relevant qualitative observations. Keep each independent replicate identifiable. Do not average before preserving the original readings. Counts are discrete; length and mass are continuous even when displayed as rounded numbers. A mean represents the sample, not an additional measured organism.

For invented independent rates 4, 5, 5, 6 and 10 millimetres per day, the mean is $30/5 = 6.0$ and median 5. The range is $10 - 4 = 6$. The sum of squared deviations from the mean is $4 + 1 + 1 + 0 + 16 = 22$. The sample standard deviation is $s = \sqrt{22/(5-1)} \approx 2.35$ millimetres per day; the standard error of the mean is $s/\sqrt{5} \approx 1.05$ millimetres per day, under the independence assumptions. SD describes scatter among measurements; SE estimates sampling uncertainty of the mean. Neither is simply the instrument's resolution.

For this small sample, use the stated convention if calculating quartiles: excluding the central observation gives lower half 4, 5 and upper half 6, 10, so $Q_1 = 4.5$, $Q_3 = 8$ and IQR 3.5. Software conventions can differ for short datasets. The value 10 warrants checking, but deleting it because it spoils the pattern is not justified. Investigate a documented recording or apparatus problem; otherwise retain it and discuss influence, or transparently compare justified analyses.

For measured length 50.0 ± 1.0 mm, percentage uncertainty is $1.0/50.0 \times 100 = 2.0\%$. A different result 40 ± 1 cm has percentage uncertainty 2.5%, not 1%. For a difference between readings, adding absolute bounds is a conservative maximum estimate; for a product or quotient, summing fractional bounds is a common first-order maximum approximation. It is not the same as combining independent standard uncertainties in quadrature.

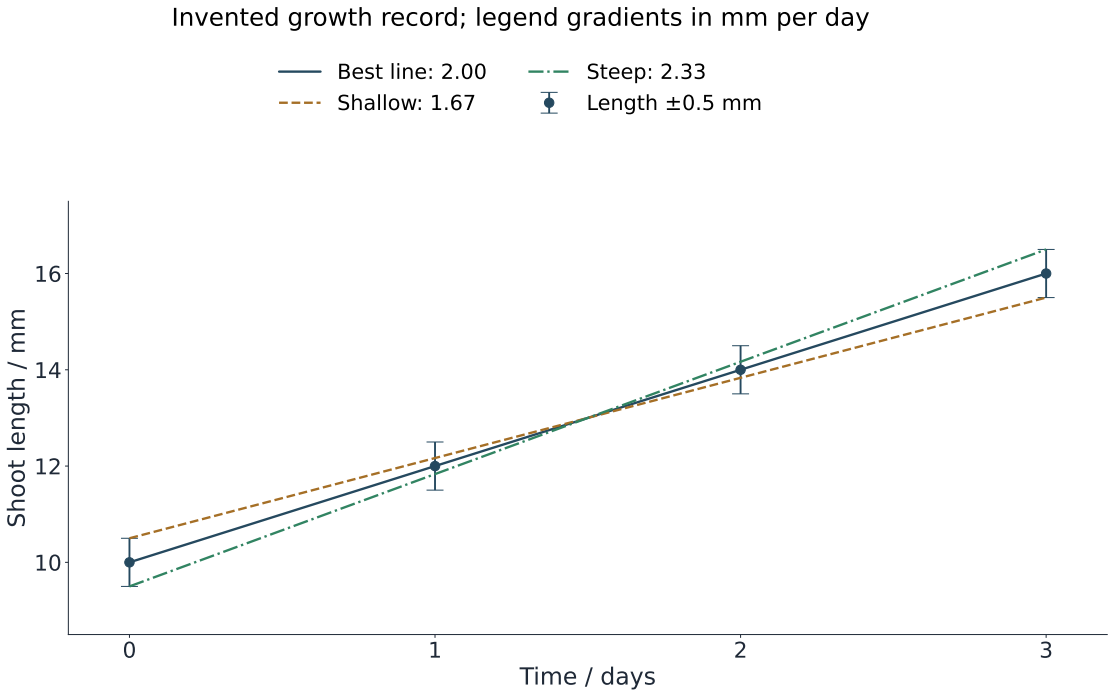
If mass change uses 10.0 ± 0.1 g and 12.0 ± 0.1 g, change is 2.0 ± 0.2 g using summed bounds. A rate from change 6.0 ± 0.4 mm over 3.0 ± 0.1 days is 2.0 mm per day. The summed fractional bound is $0.4/6.0 + 0.1/3.0 = 0.10$, giving an approximate maximum uncertainty of 0.2 mm per day. Correlated errors or a different uncertainty definition require a different treatment; state the convention used.

Graphs reveal rates, intercepts and model limitations

Use a scatter graph for two quantitative variables and a bar chart for separate categories. A histogram shows a continuous distribution in intervals; unequal-width intervals require frequency density when area represents frequency. Box plots compare distributions but do not display each observation. Label axes with quantity and unit, choose useful scales and define all error bars as ranges, SD, SE, confidence intervals or stated bounds.

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An invented linear growth series has time 0, 1, 2, 3 days and length 10, 12, 14, 16 mm. Its gradient is $\Delta L/\Delta t = (16-10)/(3-0) = 2.0$ mm per day. The intercept 10 mm is starting length, not measurement error merely because it is nonzero. A straight relationship is proportional only if it also passes through the origin. On a curved growth trace, a tangent estimates an instantaneous rate while a chord gives an interval average.



Each length has a stated ±0.5 mm bound; time is treated as exact. The two endpoint constructions also pass through the middle bounds in this example.

Using these stated bounds, a shallow plausible line runs from 10.5 to 15.5 mm over three days: gradient $5/3 \approx 1.67$ mm per day. A steep line runs from 9.5 to 16.5 mm: $7/3 \approx 2.33$ mm per day. Their midpoint is 2.00 with half-range about 0.33 mm per day. This is an envelope estimate under the specified assumptions, not a regression confidence interval. For another dataset, check all bars rather than assuming endpoint choices automatically fit.

A best-fit line summarizes a model instead of joining each noisy point. Examine residuals: a curved pattern around a fitted straight line suggests a missing relationship; scatter may change with the predictor. Interpolation stays within observed values; extrapolation needs extra assumptions. A high R^2 describes explained variation under that fitted model and dataset, not the proportion of a response biologically caused by the predictor.

Statistical tests support conditional decisions

Choose a method for the question, data and design. Pearson correlation concerns a linear association; a value near zero can hide a strong curved relationship. A t-test can compare means when its assumptions and independent or paired design match; equal-variance and unequal-variance versions differ. A chi-squared test concerns suitable category counts, with adequate expected counts and appropriate independence. Repeated readings of the same organism cannot be relabelled as independent organisms to increase sample size.

For a preselected significance level 0.05, an invented valid test giving $p = 0.03$ supplies evidence against its null model; $p = 0.12$ does not reach that threshold. A p-value is not the probability that the hypothesis is true or that chance caused the result. A non-significant outcome does not establish zero effect. Report sample size, effect size, variation, assumptions and biological importance; a tiny effect can become statistically detectable in a very large sample.

Ecological estimates also depend on assumptions. A Lincoln estimate with 40 initially marked animals, 50 caught later and 10 marked recaptures gives $40 \times 50/10 = 200$, assuming suitable mixing, retained marks, comparable capture and a sufficiently closed population. For counts 4 and 6 across two species, Simpson's reciprocal form $N(N-1)/\sum n(n-1)$ gives $90/(12+30) \approx 2.14$. State this finite-sample form: the alternative $1/\sum p_i^2$ gives about 1.92 for the same small sample. Do not mix formulas silently or treat an estimate as a census.

Conclusions and evaluation close the chain of reasoning

Answer the actual research question with the direction and size of the observed pattern, its range and uncertainty. Explain how biological knowledge supports an interpretation and which alternatives remain. An experiment with controlled assignment may support a causal claim more strongly than an observational correlation, but only within its method and sampled conditions.

Evaluation links a specific limitation to its likely consequence and a feasible improvement. "Use better equipment" is weaker than explaining that ± 0.5 mm readings make a 0.4 mm change poorly resolved, then choosing a calibrated method of greater resolution. Uncontrolled evaporation could change treatment concentration; cover compatible vessels and check mass loss. If roots within one tray share treatment, add independent trays rather than repeatedly measuring the same roots. Discuss limitations that matter most to the conclusion, not a disconnected list of every possible human mistake.

The individual scientific investigation develops the student's own question, analysis and report under the actual assessment rules. The collaborative sciences project develops joint inquiry and communication across disciplines. A practice handout cannot replace supervised practical work or guarantee an assessment mark. Use feedback to improve reasoning while preserving honest records and independent authorship.

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Practice

1. Improve "Does light help plants?" into a testable question. Give a mechanism-based prediction, dependent variable and two controlled variables.
2. One tray receives each light treatment; four seedlings per tray are read on five days. Identify the unit receiving treatment and explain why this is not 20 independent treatment replicates. Redesign it.
3. A balance gives tightly grouped readings 0.8 g above a reference. Distinguish accuracy, precision and bias. Give an effective improvement and explain why more repeats alone are insufficient.
4. A 25 mm image bar represents 100 micrometres; a cell is 15 mm on that image. Find actual cell length and image magnification. Explain the effect of resizing the image and bar together.
5. A pigment travels 42 mm and solvent front 70 mm from the origin. Find R_f and give one procedural and one identification limit. Three tenfold dilutions start from 5.0 milligrams per millilitre: find the final concentration.
6. An unknown has absorbance 0.64 under calibration $A = 0.20c + 0.04$. Find concentration in milligrams per litre and explain why a blank and validated range matter.
7. For independent rates 2, 3, 3, 4 and 8 mm per day, calculate mean, median, sample SD and SE. Explain why SD and SE answer different questions and why 8 cannot simply be deleted.
8. Find percentage uncertainty in 40 ± 1 cm. Calculate the difference of 15.0 ± 0.2 g and 12.0 ± 0.2 g using summed absolute bounds. Explain why this convention is not automatically a standard uncertainty.
9. A change 8.0 ± 0.4 mm occurs over 4.0 ± 0.2 days. Find rate and approximate maximum uncertainty using summed fractional bounds. State assumptions.
10. For the plotted length series, find best-line rate and intercept, then shallow/steep plausible gradients. Explain why the intercept need not be an error and why the envelope is not a confidence interval.
11. Select an appropriate graph for continuous height against temperature, counts in categories and a height distribution. Explain an error-bar label and one limitation of R^2 as causal evidence.
12. Distinguish correlation, a comparison of means and a category-count test. Interpret $p = 0.03$ and $p = 0.12$ at a preselected 0.05 threshold without assigning probabilities to the truth of a hypothesis.
13. Estimate abundance from initial marks 30, later catch 40 and marked recaptures 8; state two assumptions. For species counts 5 and 5, calculate Simpson's reciprocal index with the stated $N(N-1)$ form.

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14. Design one field or database comparison with explicit selection rules. Identify a safety/ethical issue and write a limitation → consequence → improvement chain. Explain how authentic raw records and independent authorship support the conclusion.

Worked answers

1. Specify species, light durations, interval and change in shoot length. Predict a response within the chosen range using photosynthetic supply while acknowledging other limits. Control starting age/size, water, temperature or soil with stated methods.
2. The tray receives treatment; seedlings are subsamples and days are repeated observations. Assign several independent trays per duration, randomizing within suitable position blocks. Summarize/analyse at the justified experimental-unit level.
3. Precision is high but accuracy is limited by positive bias. Check tare and calibration against suitable references, correcting/replacing the method as justified. Repeats can narrow scatter while retaining the 0.8 g offset.
4. Cell length $15/25 \times 100 = 60$ micrometres. Magnification $25/0.100 = 250$. Resizing changes printed magnification, but the cell-to-bar ratio and inferred actual size remain unchanged when both scale together.
5. $R_f = 42/70 = 0.60$, dimensionless. Keep origin above solvent; mark the front before it disappears. A match is conditional on conditions and is not unique identity proof. Final concentration $5.0/10^3 = 0.0050$ milligrams per millilitre; mix each step.
6. $c = (0.64 - 0.04)/0.20 = 3.0$ milligrams per litre. The blank addresses background; calibration must describe the relevant matrix/range. Unvalidated extrapolation, turbidity or saturation can bias the result.
7. Mean $20/5 = 4$; median 3. Squared deviations sum $4 + 1 + 1 + 0 + 16 = 22$; sample SD $\sqrt{22/4} \approx 2.35$ and SE $\sqrt{22/4}/\sqrt{5} \approx 1.05$ mm per day. SD describes sample scatter, SE uncertainty of the estimated mean under independence. Check 8 and document any justified exclusion; an inconvenient result is not enough.
8. Percentage uncertainty $1/40 \times 100 = 2.5\%$. Difference 3.0 ± 0.4 g under maximum-bound addition. Standard uncertainties describe a different quantity and need an appropriate propagation method, including dependence between measurements.
9. Rate $8/4 = 2.0$ mm per day. Fractional estimate $0.4/8 + 0.2/4 = 0.10$; maximum uncertainty approximately 0.2 mm per day. This first-order bound convention assumes the stated small bounds; it is not a confidence interval or automatic quadrature rule.

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10. Rate 2.0 mm per day; intercept 10 mm is initial length. Shallow $5/3 \approx 1.67$, steep $7/3 \approx 2.33$; midpoint/half-range 2.00 ± 0.33 mm per day. All plotted bounds are met here; time was treated as exact. The result is a geometric envelope, not a probabilistic regression interval.
11. Scatter graph, bar chart and histogram respectively. State whether bars show SD, SE, a range or another interval and give the sample size. A high fitted R^2 can coexist with confounding and does not itself establish causation.
12. Correlation tests association; a suitable t-test compares means; chi-squared can test suitable counts. At 0.05, 0.03 provides evidence against the stated null model while 0.12 does not meet the threshold. Neither proves a hypothesis true or false; assumptions, effect and uncertainty remain relevant.
13. Abundance $30(40)/8 = 150$, conditional on retained marks, mixing, comparable capture and a suitably closed population. Simpson reciprocal $10(9)/[5(4)+5(4)] = 90/40 = 2.25$. These are estimates under stated methods, not exact censuses or a claim of 2.25 observed species.
14. Preselect sites or database inclusion criteria, match definitions/units/timing and record missing data. Explain permission/privacy/habitat or apparatus risks and controls. For example, unequal survey effort biases density comparisons; standardize sampled area and time. Preserve actual records, formulas, exceptions and sources so a reader can trace the inference; inventing a missing value would break that chain.

Exam tips

Name the quantity, unit, experimental unit and uncertainty convention. Separate independent replication from repeated readings and SD from SE. Connect each limitation to the result it could change. Compare an effect's size and biological meaning as well as a test decision. A justified qualified conclusion is stronger than certainty that the method cannot support.

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