Course Companion

AP Biology

Every topic handout in one volume

全部专题讲义合集

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Contents 目录

1. **Chemistry of Life** 2
2. **Cell Structure and Function** 14
3. **Cellular Energetics** 29
4. **Cell Communication and Cell Cycle** 43
5. **Heredity** 54
6. **Gene Expression and Regulation** 65
7. **Natural Selection** 81
8. **Ecology** 98

Structure of Water and Hydrogen Bonding

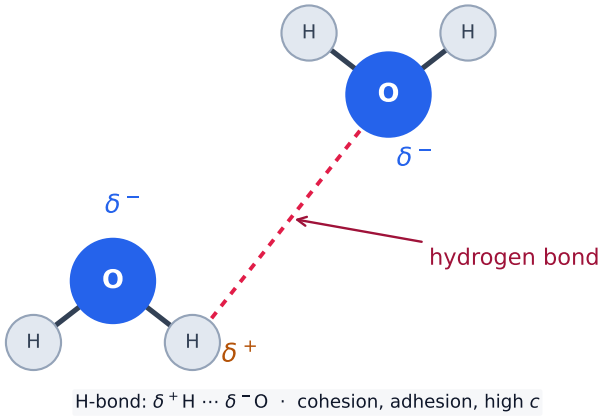
Ice floats, so lakes freeze from the top and fish survive the winter underneath. A single wrong amino acid among hundreds in haemoglobin causes sickle cell disease. Both start at the scale of atoms and bonds, which is where this unit begins.



Ice floats on water because hydrogen bonding makes solid water less dense than liquid water

Water is **polar** 极性: its oxygen pulls electrons more strongly than its hydrogens, giving a slightly negative O and slightly positive H. This lets water molecules form **hydrogen bonds** 氢键 with each other, which explains water's life-supporting properties:

- **Cohesion** 内聚力 and **adhesion** 附着力 (surface tension, capillary action, water rising in plants),
- high **specific heat** 比热容 (resists temperature change, stabilizing organisms),
- high heat of vaporization (evaporative cooling),
- ice floating (less dense solid), and being a great **solvent** for polar and ionic substances.



A hydrogen bond between two polar water molecules

Water as a solvent. When salt (NaCl) dissolves, the slightly negative oxygen ends of water molecules surround each Na^+ ion, and the slightly positive hydrogen ends surround each Cl^- ion. These **hydration shells** pull the ions apart and keep them in solution. Polar molecules such as glucose dissolve because they form hydrogen bonds with water. Nonpolar molecules such as oils cannot, so they clump together away from the water.

> **Worked example (water moving up a plant).** Explain how the properties of water allow it to move up the narrow xylem vessels of a tall tree.

- Water evaporates from the leaves (transpiration), which pulls on the column of water below.
- **Cohesion**: hydrogen bonds between water molecules hold the column together, so the pull is passed all the way down to the roots.
- **Adhesion**: hydrogen bonds between water and the polar walls of the xylem help the water cling and climb, and stop the column from falling back.

An answer earns the marks when it names the property **and** links it to hydrogen bonding: "water is sticky" earns nothing; "hydrogen bonds between water molecules (cohesion) hold the column together" does.

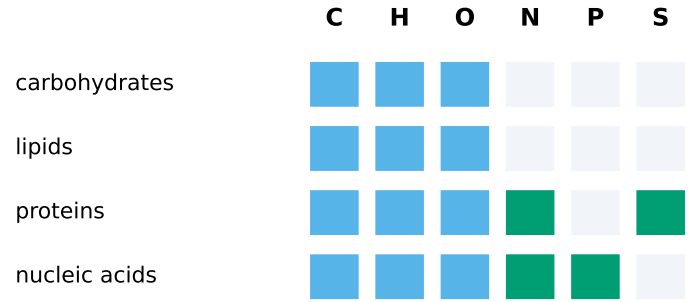
Elements of Life

Living matter is built mostly from a few elements – carbon, hydrogen, oxygen, nitrogen, phosphorus, and sulfur. **Carbon** 碳 is central because it forms four stable covalent bonds, building long chains, branches, and rings – the skeletons of organic biological molecules.

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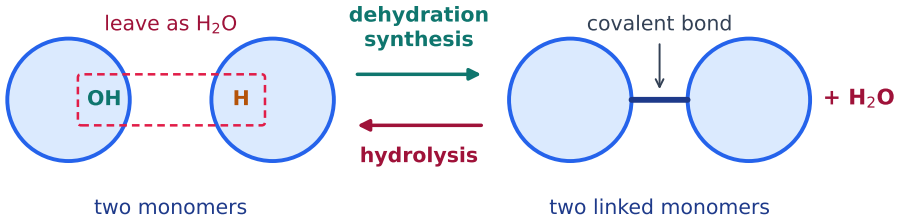
Carbohydrates and fats contain carbon, hydrogen, and oxygen; proteins also contain nitrogen



Which elements each macromolecule contains: proteins contain N and sometimes S, nucleic acids N and P

Introduction to Macromolecules

Many large biological molecules – **macromolecules** 大分子 – are **polymers** 聚合物 built from repeating **monomers** 单体. Lipids are also studied among the major biological molecule classes, but are not generally repeating-unit polymers. Cells join monomers by **dehydration synthesis** 脱水缩合 (removing water to form a bond) and break polymers by **hydrolysis** 水解 (adding water). Four classes: carbohydrates, lipids, nucleic acids, and proteins.



Dehydration synthesis builds polymers and releases water; hydrolysis reverses it

Carbohydrates

Carbohydrates 碳水化合物 are made of sugar monomers (**monosaccharides** 单糖 like glucose). They store energy (starch, glycogen) and provide structure (**cellulose** 纤维素 in plant walls). Many small sugars are polar and dissolve readily in water. Large carbohydrate polymers differ: cellulose, for example, does not dissolve readily in water.

The way the monomers are linked sets the job. Glycogen, the animal energy store, is highly **branched**, with many ends where enzymes can remove glucose quickly when it is needed. Starch in plants is a mix of unbranched and branched chains. Cellulose chains are long and **linear**: they lie side by side, hydrogen-bond to each other, and form strong fibres that make plant cell walls rigid.

Glucose exists in two ring forms, α**-glucose** and β**-glucose**, which differ only in the position of the −OH group on carbon 1. Starch and glycogen are chains of α-glucose: their links make the chain coil into a compact helix, good for storage. Cellulose is made of β-glucose: every other glucose is flipped over, giving straight chains that hydrogen-bond into fibres. Human enzymes that digest starch cannot fit the β links, so we cannot digest cellulose, although it is made of the same glucose monomer. The **monomer** and the **bond** between monomers together decide the polymer's shape and function.

If asked to describe a glucose molecule, say it contains only carbon, hydrogen and oxygen atoms, joined by covalent bonds, with polar O–H groups that hydrogen-bond to water, which is why glucose dissolves.

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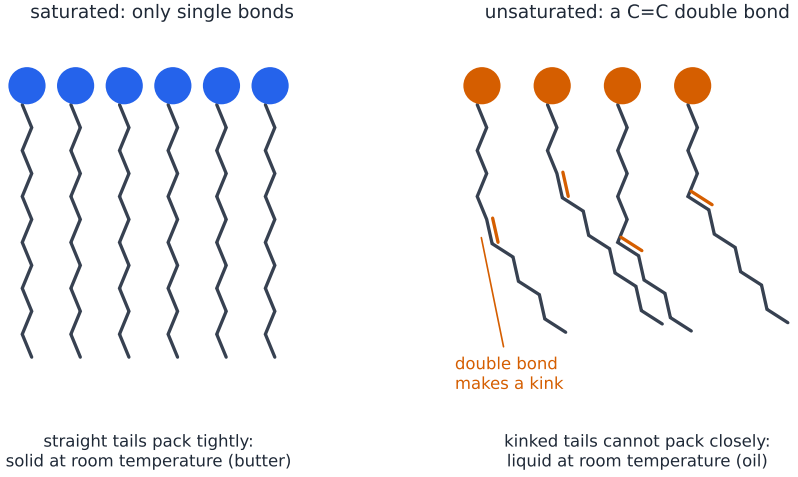


The shapes of storage polysaccharides (starch, glycogen) and structural cellulose

Lipids

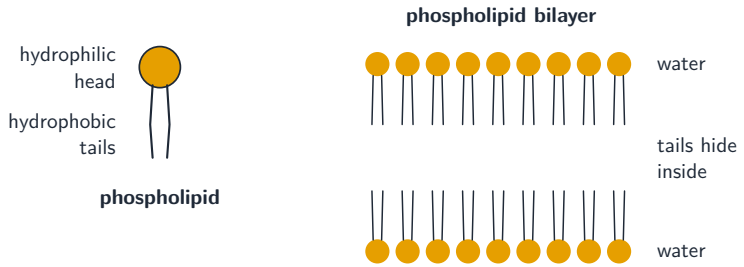
Lipids 脂质 contain large **nonpolar**, water-repelling (**hydrophobic** 疏水) regions. Some also have polar regions, so they are not uniformly nonpolar. They include fats (long-term energy storage), **phospholipids** 磷脂 (which build membranes), and steroids. A phospholipid has a polar "head" and nonpolar "tails," the key to the cell membrane.

Fatty-acid tails come in two kinds. A **saturated** 饱和的 fatty acid has only single bonds between its carbon atoms, so its tail is straight. An **unsaturated** 不饱和的 fatty acid has at least one C=C double bond, which puts a kink in the tail when the double bond is cis. A trans double bond leaves the tail relatively straight. Straight tails pack closely, so saturated fats such as butter are solid at room temperature. Kinked tails cannot pack closely, so unsaturated fats such as olive oil are liquid. For tails of similar length, more cis double bonds generally lower the melting point.

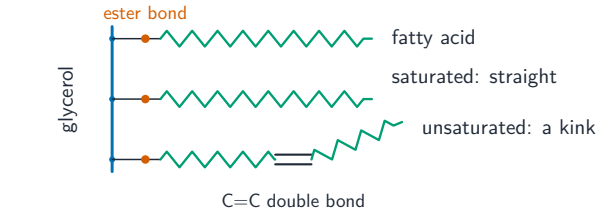


Saturated tails are straight and pack tightly; a cis double bond kinks an unsaturated tail so it cannot pack closely

Lipids do several jobs. Fats store energy and, under the skin of mammals, insulate the body. **Steroids** 类固醇 such as testosterone and oestrogen are hormones that control growth, development and metabolism. **Cholesterol** 胆固醇 sits among the phospholipids of animal cell membranes and keeps them stable.



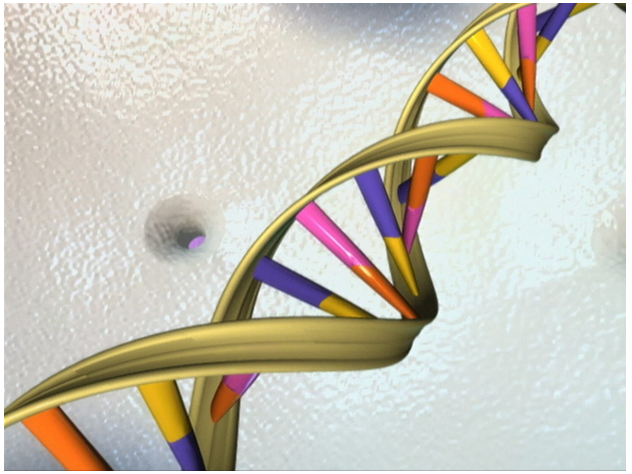
A phospholipid has a polar head and two nonpolar tails, so phospholipids form a bilayer



A triglyceride: glycerol joined to three fatty acids

Nucleic Acids

Nucleic acids 核酸 (DNA and RNA) store and carry genetic information. Their monomers are **nucleotides** 核苷酸, each a sugar, a phosphate, and a nitrogen base. The base sequence encodes instructions; cellular DNA is usually double-stranded, while RNA is usually single-stranded and can fold into paired regions.



Space-filling model of the DNA double helix (NHGRI)

Each nucleotide has three parts: a five-carbon sugar, a phosphate group, and a **nitrogenous base** 含氮碱基 (A, T, C or G in DNA; A, U, C or G in RNA). The sugar of one nucleotide bonds to the phosphate of the next, making a sugar–phosphate backbone with the bases sticking out. A strand has two different ends: the **5′ end** carries a phosphate and the **3′ end** a hydroxyl (−OH). New nucleotides are always added to the 3′ end, so a strand grows in the 5′→3′ direction.

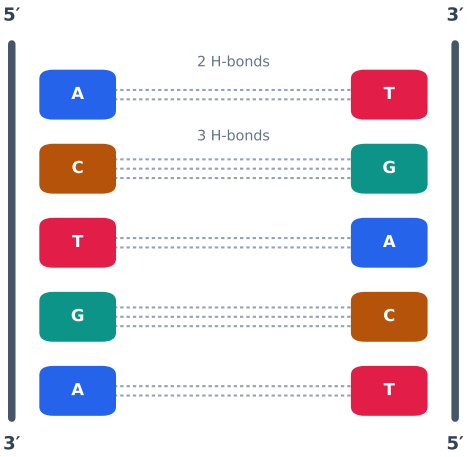
	DNA	RNA
sugar	deoxyribose	ribose
bases	A, T, C, G	A, U, C, G
strands	usually two (double helix)	usually one

The bases are of two kinds. **Purines** 嘌呤 (adenine and guanine) have a double ring; **pyrimidines** 嘧啶 (cytosine, thymine and uracil) have a single ring. Each base pair is one purine with one pyrimidine, so the double helix has the same width all along. A–T pairs are held by **two** hydrogen bonds; C–G pairs by **three**.

> **Worked example (base pairs and stability).** Two DNA samples have the same length and are in the same solution. Sample X is 60% C + G; sample Y is 40% C + G. Which needs a higher temperature to separate its two strands, and why? Sample **X**: it has more C–G pairs, each held by three hydrogen bonds instead of two, so more energy is needed to break the bonds between its strands. The covalent bonds of each sugar–phosphate backbone are not broken when the strands separate.

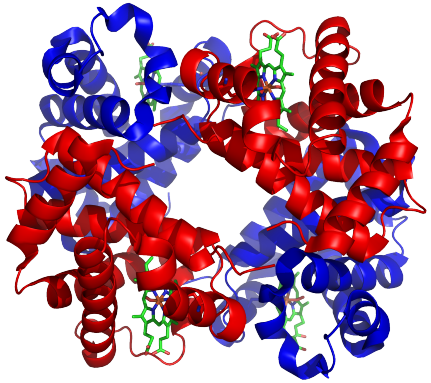
Because every nucleotide carries a negatively charged phosphate, DNA as a whole is **negatively charged**. In an electric field it moves toward the positive electrode, and shorter fragments move faster through a gel. This is how gel electrophoresis and the comet assay separate DNA by size.

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Complementary base pairing holds the two antiparallel strands of DNA together

Proteins



Haemoglobin quaternary structure: four folded polypeptide chains, each holding a haem group

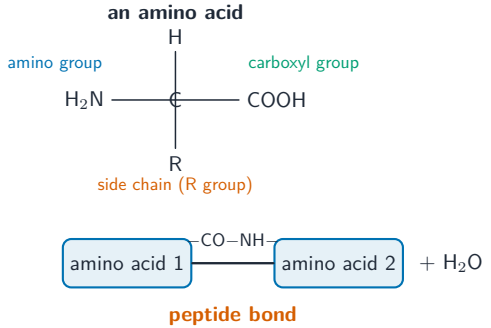
Proteins 蛋白质 are polymers of **amino acids** 氨基酸 joined by **peptide bonds** 肽键. Their amino-acid sequence contributes to their 3-D shape (primary, secondary and tertiary structure; quaternary structure applies when multiple polypeptide chains assemble), and **shape determines function** – as enzymes, transporters, receptors,

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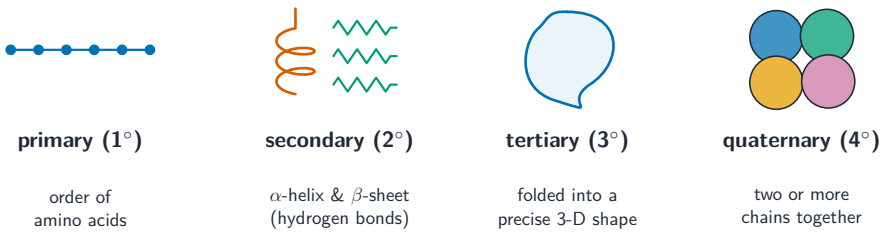
and structural parts. Changing the environment (heat, pH) can **denature** 变性 a protein, unfolding it and stopping its function.

Every amino acid has the same backbone: a central carbon bonded to an amine group ($-NH_2$), a carboxyl group ($-COOH$), a hydrogen, and an **R group** 侧链. Only the R group differs, and it falls into one of three classes: **nonpolar** (hydrophobic), **polar**, or **charged** (acidic or basic). A peptide bond joins the carboxyl group of one amino acid to the amine group of the next, so a chain has an amino (N) end and a carboxyl (C) end. The levels of structure come from different interactions:

- **Primary structure**: the sequence of amino acids.
- **Secondary structure** 二级结构: hydrogen bonds between atoms of the *backbone* fold short stretches into α-helices and β-pleated sheets.
- **Tertiary structure** 三级结构: interactions between *R groups* (hydrogen bonds, hydrophobic clustering, ionic bonds and disulfide bridges) fold the whole chain into its 3-D shape.
- **Quaternary structure**: two or more polypeptide chains held together, as in haemoglobin.



An amino acid, and the peptide bond formed by condensation



The four levels of protein structure

Worked example. Building a polymer from **10 monomers** by dehydration synthesis forms 9 bonds and releases **9 water molecules** – one per bond, so N monomers release $N - 1$ waters. Hydrolysis reverses this exactly: adding those 9 waters breaks the polymer back into 10 monomers. This is why biosynthesis (dehydration) and digestion (hydrolysis) are chemical opposites.

Worked example (Chargaff's rule). In double-stranded DNA, A pairs with T and C pairs with G, so the amount of A equals the amount of T, and C equals G. A sample is 30% adenine. Then thymine is also 30%, which leaves 40% for C and G together: 20% each. The rule fails for single-stranded RNA, which has no partner strand.

Worked example (tracing elements). Which molecules would take up a radioactive label?

- **Sulfur** (^{35}S) is found in some amino acids, so it labels **proteins**, not DNA.
- **Phosphorus** (^{32}P) is in every nucleotide's phosphate group, so it labels **nucleic acids**, and also phospholipids, but not most proteins.
- **Nitrogen** is in amino acids and in nucleotide bases, so it labels both proteins and nucleic acids, but not carbohydrates or fats.

This logic is how Hershey and Chase showed that DNA, not protein, enters a cell to carry the genes.

Worked example (predicting a change in shape). A protein's interior is packed with nonpolar R groups, away from water. A mutation replaces one interior nonpolar amino acid with a charged one. Predict the effect: the charged group is attracted to water and disrupts the hydrophobic core, so the tertiary structure is likely to change. Because shape determines function, the protein may work less well, or not at all.

Common mistakes.

- Calling the O–H bond inside a water molecule a hydrogen bond. That bond is **covalent**; hydrogen bonds are the weaker attractions **between** neighbouring water molecules.
- Saying dehydration synthesis adds water. **Dehydration synthesis** removes a water molecule for each bond; **hydrolysis** adds one to break it.
- Saying humans cannot digest cellulose because it is not made of glucose. It **is** glucose; the β links between the monomers do not fit our digestive enzymes.
- Calling lipids polymers. Fats and phospholipids are not chains of repeating monomers, though they are major biological molecules.
- Saying DNA's two strands run the same way. They are **antiparallel** 反向平行: one runs 5′→3′, the other 3′→5′.

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- Saying a protein's shape comes from its peptide bonds alone. The **R groups** and their interactions (hydrogen bonds, ionic bonds, hydrophobic clustering, disulfide bridges) fold the chain into its tertiary shape.
- Labelling DNA with ^{35}S. DNA contains **no sulfur**; sulfur labels proteins and phosphorus labels nucleic acids.
- Saying unsaturated fats are solid because their double bonds are stronger. Cis double bonds put **kinks** in the tails, so the tails cannot pack closely and the fat stays liquid.
- Mixing up secondary and tertiary structure. Secondary structure comes from hydrogen bonds in the **backbone**; tertiary structure from interactions between **R groups**.
- Saying DNA moves toward the negative electrode in a gel. Its phosphates make it negative, so it moves toward the **positive** electrode.

Practice

Answer each before reading the answers below.

1. Explain, using hydrogen bonds, why sweating cools the body.
2. A DNA sample contains 18% guanine. Find the percentages of the other three bases.
3. How many water molecules are released when 25 amino acids join into one polypeptide?
4. A cell is grown with radioactive ^{32}P. Which two of the four major biological molecule classes contain phosphate groups that can become radioactive?
5. A student says: "Boiling an egg breaks the peptide bonds in its proteins." Correct the student.

Answers

1. Evaporating water must break many hydrogen bonds, which takes a large amount of energy (a high heat of vaporization). That energy is taken from the skin as the sweat evaporates, cooling it.
2. C = 18%, so G and C together are 36%, leaving 64% for A and T: A = 32% and T = 32%.
3. $25 - 1 = 24$ water molecules, one per peptide bond.
4. Nucleic acids (DNA and RNA) and lipids containing phosphate, such as phospholipids. Other phosphate-containing molecules, such as ATP, can also be labelled.
5. Heat denatures the protein: it breaks the weak bonds (such as hydrogen bonds) that hold the folded shape. The peptide bonds, and so the primary structure, stay intact.

Exam tips

- Trace every property of water back to its **polarity and hydrogen bonding** (cohesion, high specific heat, solvent power).
- Know the four macromolecule classes and their monomers; polymers are built by **dehydration synthesis** and broken by **hydrolysis**.
- For proteins, **structure (shape) determines function** – heat or a pH change **denatures** them and stops them working.
- In DNA the bases pair A–T and C–G on **antiparallel** strands; RNA is single-stranded and uses U.
- Link each molecule to its role (carbohydrates: energy/structure; lipids: membranes/storage; nucleic acids: information).

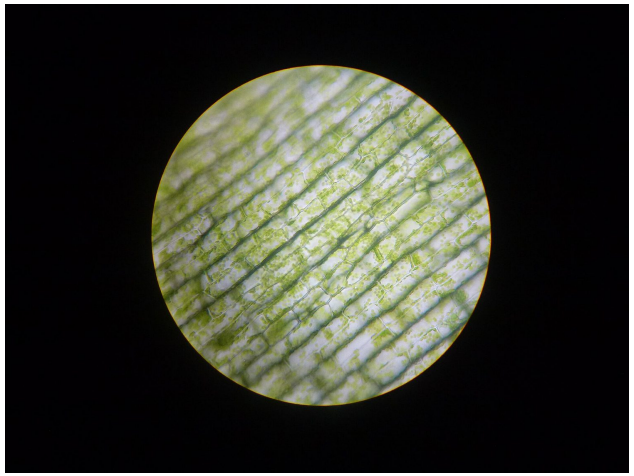
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Cell Structure and Function

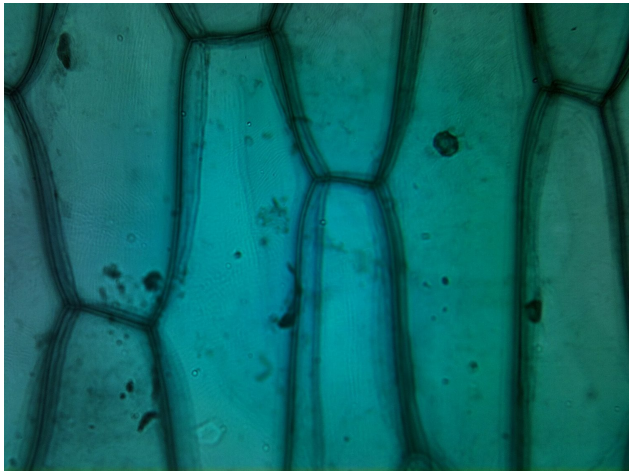
AP Biology

Organelles and the Endomembrane System

In cystic fibrosis, one amino acid is missing from a membrane protein called CFTR, which should carry chloride ions out of lung cells. In the most common mutation, the protein is synthesised but folds incorrectly, and the cell's quality checks reject it inside the endoplasmic reticulum, so it never reaches the cell surface. Without it, the mucus in the lungs becomes thick and sticky. To understand the disease, and many exam questions built on it, you need to know where proteins are made, how they travel through the cell, and how the membrane controls what crosses it.

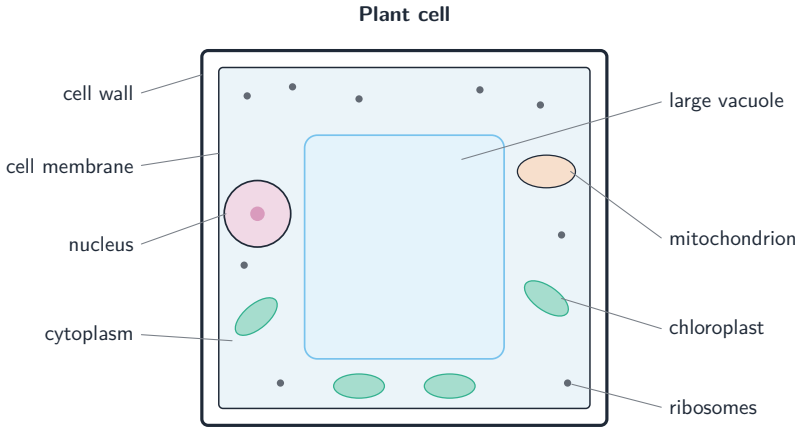


Elodea leaf cells packed with green chloroplasts (light micrograph)

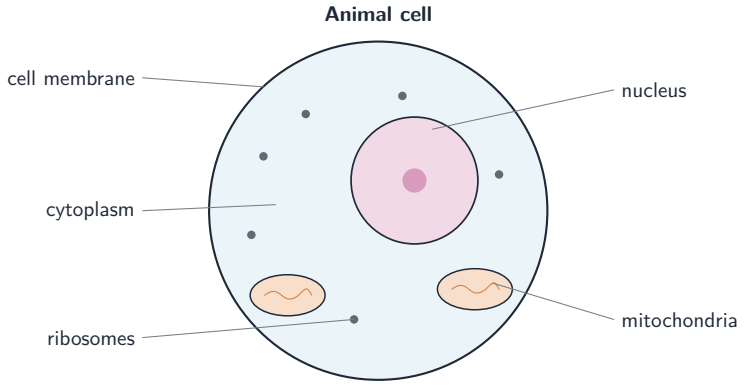


Onion epidermis cells under a light microscope – cell walls and nuclei clearly visible

A eukaryotic cell divides its work among **organelles** 细胞器. The **endomembrane system** 内膜系统 is a connected set that makes, modifies, and ships molecules: the **nucleus** 细胞核 (holds DNA), rough and smooth **endoplasmic reticulum** 内质网 (protein and lipid synthesis), the **Golgi** 高尔基体 (modifies and packages), **lysosomes** 溶酶体 (digestion), and vesicles that carry material between them. **Mitochondria** 线粒体 (respiration) and **chloroplasts** 叶绿体 (photosynthesis) have their own membranes.



A plant cell also has a cell wall, a large vacuole, and chloroplasts



A generalised animal cell and its organelles

What each organelle actually does

The exam asks you to name a structure **and** say what it does, so learn them as pairs.

Ribosomes 核糖体 are made of **ribosomal RNA** 核糖体核糖核酸 (rRNA) and protein. They have no membrane, and they are found in **every** form of life – bacteria, archaea and eukaryotes alike. That shared presence is evidence of common ancestry: a structure this central was already present in the last common ancestor of all living things. A ribosome reads **messenger RNA** 信使核糖核酸 (mRNA) and builds the protein it codes for. Free ribosomes make proteins for use inside the cell; ribosomes bound to the rough endoplasmic reticulum make proteins for export or for the membrane.

The endoplasmic reticulum (ER) is a network of membranes that runs through the cytoplasm, helps the cell keep its shape and carries materials around inside it. **Rough ER** 粗面内质网 is covered with ribosomes: the proteins they make are threaded into the ER, the first step for proteins that will go to the membrane, to a lysosome, or out of the cell. **Smooth ER** 滑面内质网 has no ribosomes; it makes lipids and breaks down (detoxifies) harmful substances.

The Golgi complex 高尔基复合体 is a series of flattened, membrane-bound sacs. A protein arriving from the endoplasmic reticulum is passed through them, and three things happen to it:

- it arrives folded from the ER and is chemically modified – most often by **glycosylation** 糖基化, the attachment of short sugar chains;
- it is **sorted and targeted**, so the modified sugar or phosphate tag decides which compartment the protein is sent to;
- it is packaged into a vesicle for transport.

So the Golgi is not storage: it is the cell's finishing and dispatch department, and its

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modifications *determine* what a protein does and where it ends up.

Lysosomes are membrane-enclosed sacs of **hydrolytic enzymes** 水解酶 that digest material – worn-out organelles, engulfed bacteria, and food particles taken in by the cell. Their enzymes work best at a low pH, which the lysosome maintains inside itself; keeping them enclosed protects the rest of the cell. (A lysosome is a good example of compartmentalization: an enzyme that would damage the cytoplasm is safe inside its own membrane.) Lysosomes also take part in **apoptosis** 细胞凋亡, programmed cell death, in which a cell is dismantled deliberately, for example to sculpt fingers from a webbed embryonic hand.

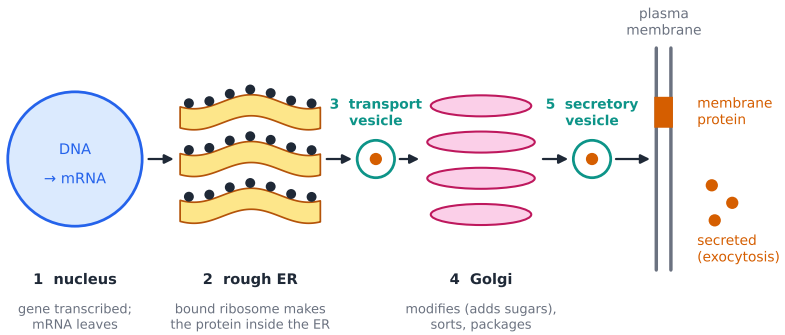
Vacuoles 液泡 are membrane-bound sacs with many different roles: storing water, ions, nutrients or waste, and in a plant cell the large central vacuole also presses outwards on the cell wall to keep the cell firm. Animal cells have many small vacuoles instead of one large one.

A **cell wall** 细胞壁 surrounds the membrane of plant and fungal cells and most bacterial and archaeal cells, but not animal cells. It gives the cell shape and strength, stops it bursting when it takes in water, and acts as a barrier to some substances.

Mitochondria and **chloroplasts** each have a **double membrane** and their own circular DNA and ribosomes – the evidence behind endosymbiotic theory below. The outer membrane of a mitochondrion is smooth; the inner membrane is folded into **cristae** 嵴, which give it a much larger surface area for the reactions of aerobic respiration. The two membranes also make separate compartments for different stages of respiration. Chloroplasts, found in plants and algae, are where photosynthesis happens.

■ Following a protein through the cell

A protein that ends up in the plasma membrane, or is secreted, follows one route. Free-response questions often ask you to draw or describe it.

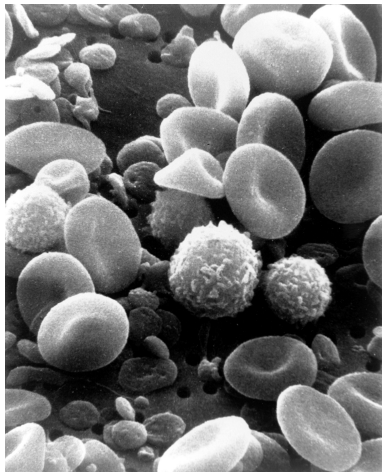


mRNA leaves the nucleus; a membrane or secreted protein is then made at the rough ER and travels through the Golgi to the plasma membrane

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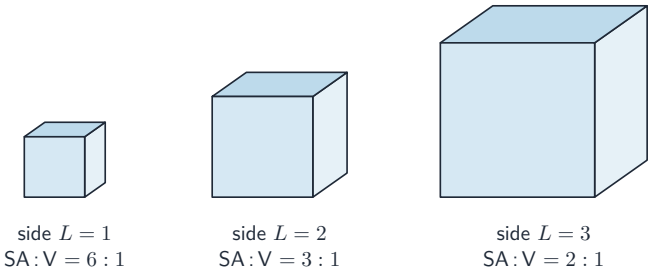
> **Worked example (where is the faulty protein?).** CFTR is a membrane protein, so it is made by ribosomes **on the rough ER**, not by free ribosomes. From the ER it goes in a vesicle to the Golgi, then in another vesicle to the plasma membrane. In the most common cystic fibrosis mutation the protein folds wrongly, and the ER keeps misfolded proteins back, so the mutant protein is found **in the ER**, not in the membrane. If instead a mutation changed only the protein's ATP-binding site, the protein could still fold and reach the membrane, but it would not work there.

Why Cells Stay Small



Scanning electron micrograph of red blood cells – no nucleus, high surface area for gas exchange

Cells stay small because of the **surface-area-to-volume ratio** 表面积与体积比. As a cell grows, its volume rises faster than its surface area, so its membrane cannot exchange materials fast enough for the interior. Staying small (or being flat or folded) keeps enough surface to serve the volume.



a bigger cube (or cell) has a **smaller** surface area : volume ratio

The surface-area-to-volume ratio falls as a cell gets bigger

Worked example. A cube-shaped cell 2 μm on a side has surface area $6 \times 2^2 = 24$ μm^2 and volume $2^3 = 8$ μm^3, so its surface-area-to-volume ratio is $\frac{24}{8} = 3$ μm^{-1}. Double the side to 4 μm: surface area $= 6 \times 4^2 = 96$, volume $= 4^3 = 64$, ratio $= \frac{96}{64} = 1.5$ μm^{-1}. Doubling the size **halved** the ratio – the larger cell has far less membrane per unit of interior, which is why cells stay small.

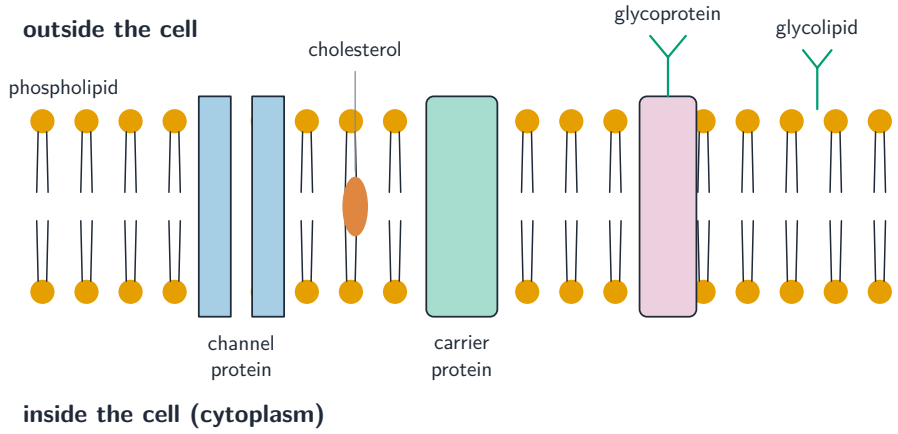
Worked example (a sphere). A spherical cell of radius 3 μm has $SA = 4\pi r^2 = 113$ μm^2 and $V = \frac{4}{3}\pi r^3 = 113$ μm^3, a ratio of 1.0 μm^{-1}. For any sphere the ratio is $3/r$, so it falls as the radius grows. Cells that need a lot of exchange increase their surface without growing: **microvilli** 微绒毛 on gut cells, root hairs on plant roots, and the cristae inside mitochondria are all folds or projections that add membrane.

The same rule works for whole organisms. A mouse has far more surface per kilogram than an elephant, so it loses heat much faster and must burn food faster to stay warm. In general, the smaller the animal, the higher its metabolic rate *per unit of body mass*.

The Fluid Mosaic Model of the Membrane

The cell membrane is a **phospholipid bilayer** 磷脂双分子层 – polar heads facing the water, nonpolar tails inside – studded with proteins. The **fluid mosaic model** 流动镶嵌模型 pictures it as a fluid sheet in which lipids and proteins drift. **Cholesterol** and the degree of unsaturation tune its fluidity.

Membrane proteins follow the same rule as phospholipids. The part of a protein that crosses the bilayer has mainly **nonpolar** (hydrophobic) R groups, which touch the fatty-acid tails. The parts that stick out into the cytoplasm or the outside fluid have **polar and charged** R groups, which touch water. Short sugar chains attached to some proteins (glycoproteins) and lipids (glycolipids) face the outside and let cells recognise each other.



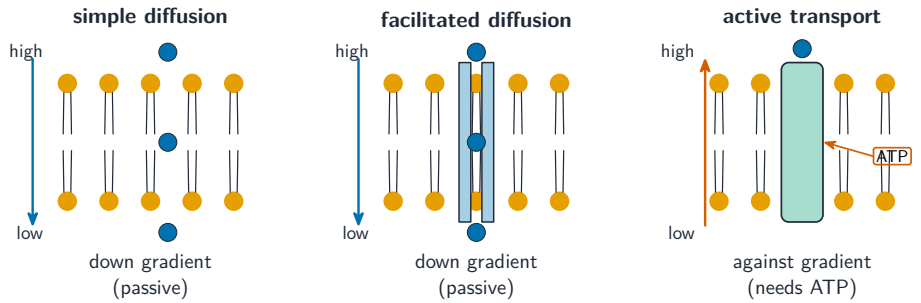
The fluid mosaic model of the cell membrane

What Can Cross the Membrane

The membrane is **selectively permeable** 选择透过性. Small nonpolar molecules (O_2, CO_2) cross freely. Water, small but polar, crosses the bilayer only in small amounts and moves mostly through channel proteins called **aquaporins** 水通道蛋白. Large or charged/polar particles (ions, glucose) cannot pass the nonpolar core without help. This selectivity lets the cell control its internal environment.

The nonpolar core is the reason. Steroid hormones such as oestrogen are nonpolar, so they dissolve in the core and cross the membrane by simple diffusion, then bind receptors *inside* the cell. Ions such as Na^+, K^+ and Cl^- are charged, so the hydrophobic tails stop them; they need **channel proteins** 通道蛋白 or carriers even to move down their electrochemical gradients (which depend on both concentration and charge). Because ions carry charge, moving them across a membrane makes one side more positive than the other: the membrane becomes polarized.

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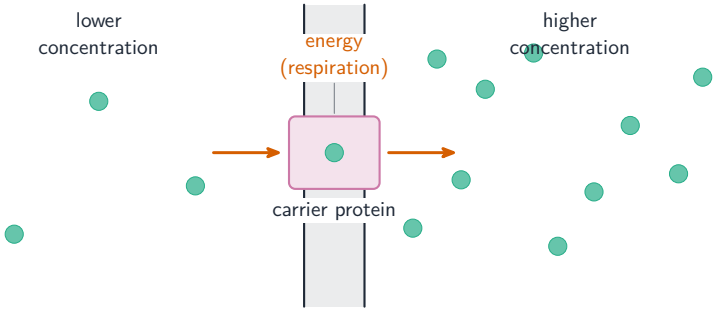


Three ways substances cross the membrane

Passive and Active Transport

- **Passive transport** 被动运输 moves substances **down** their concentration gradient (high → low) without cellular energy input – **diffusion** 扩散.
- **Active transport** 主动运输 moves substances **against** the gradient (low → high), requiring energy, directly from ATP or indirectly from another gradient. Protein pumps like the sodium–potassium pump use ATP directly.

The **sodium-potassium pump** 钠钾泵 uses one ATP to move 3 Na^+ out of the cell and 2 K^+ in, both against their gradients. Because more positive charge leaves than enters, it helps keep the inside of the cell negative: the membrane potential that nerve and muscle cells depend on. Active transport always needs a membrane protein and a source of energy; when a cell runs out of ATP, the gradients slowly run down.

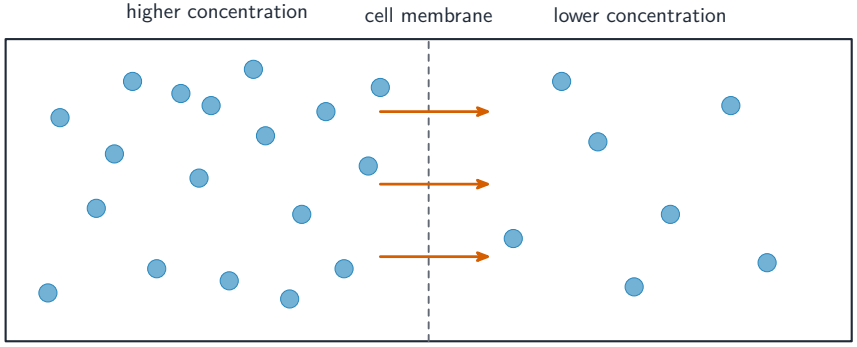


Active transport moves particles against the gradient, using ATP and a carrier protein

Facilitated Diffusion

Facilitated diffusion 易化扩散 is passive transport through a membrane protein – a **channel** or **carrier** 载体 – for particles that cannot cross the lipid alone. It still goes down the gradient and needs no energy, but its rate can saturate when all the proteins are busy.

Reading a rate graph. Free-response questions often plot the rate of uptake against the concentration outside the cell. For simple diffusion through the bilayer, the rate keeps rising in proportion to the concentration. For facilitated diffusion, the rate rises at first and then levels off: every channel or carrier is already in use, so more solute cannot make it faster. A level section is therefore evidence that a membrane protein is involved, and a cell with more of those proteins levels off at a higher rate.



Diffusion: particles spread from higher to lower concentration, down the gradient

Tonicity, Water Potential, and Osmoregulation

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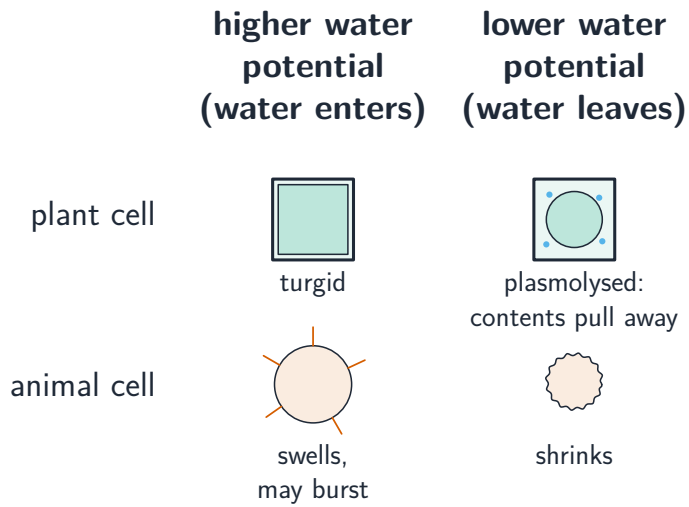


Red onion cells in hypertonic solution: the membrane pulls away from the wall (plasmolysis)

Osmosis 渗透 is the diffusion of water across a membrane. **Tonicity** 张力 compares concentrations of solutes that cannot cross the membrane: in a **hypotonic** 低渗 solution a cell gains water (may burst); in a **hypertonic** 高渗 one it loses water (shrinks); in an **isotonic** 等渗 one there is no net change. **Water potential** 水势 predicts the direction of water movement (water moves to lower water potential), and is the sum of a pressure term and a solute term: $\Psi = \Psi_p + \Psi_s$, where the solute potential $\Psi_s = -iCRT$. **Osmoregulation** 渗透调节 is how organisms control this balance.

Freshwater protists live in water that is hypotonic to their cytoplasm, so water flows in all the time. A **contractile vacuole** 收缩泡 collects it and pumps it out, using energy. Put the protist in a medium with even less solute and water enters faster, so the vacuole must fill and empty more often. In a plant cell, water entering the large central vacuole pushes the membrane against the cell wall, giving turgor pressure; the wall stops the cell from bursting.

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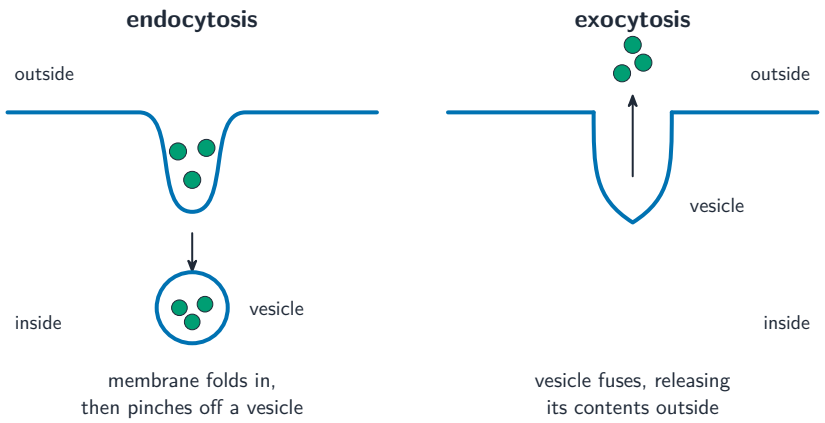


How plant and animal cells respond to solutions of different water potential

> **Worked example.** For a 0.1 M sucrose solution ($i = 1$) in an open beaker (so pressure potential $\Psi_p = 0$) at 25 °C ($T = 298$ K, $R = 0.0831$ L · bar/mol · K): $\Psi_s = -iCRT = -(1)(0.1)(0.0831)(298) \approx -2.48$ bar, so $\Psi \approx -2.48$ bar. A plant cell whose interior is $\Psi = -1.0$ bar sits in this solution: since the solution is **more negative**, water moves *out* of the cell into the solution, and the cell loses turgor.

Mechanisms of Membrane Transport

Large materials move in bulk by **vesicles**: **endocytosis** 内吞 brings material **in** (the membrane engulfs it), and **exocytosis** 外排 sends material **out** (a vesicle fuses with the membrane). Both require energy and let cells import and secrete large molecules.



Endocytosis brings material in; exocytosis releases it out

Compartmentalization Inside the Cell

Membranes create separate **compartments** 区室 so incompatible reactions can run at once and conditions (pH, ion levels) can be tuned locally. This organization boosts efficiency – the internal membranes also add surface area for reactions.

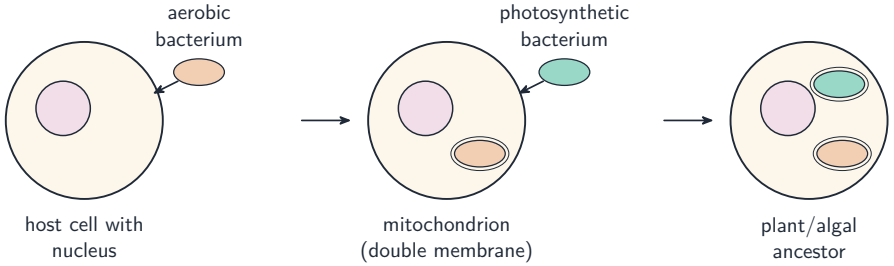
Examples to use as evidence:

- A **lysosome** keeps its digestive enzymes at about pH 5, while the cytosol is about pH 7.2. The enzymes work best at the lower pH, so they are less active in the cytosol; substantial leakage can still damage or kill the cell.
- The inner membrane of a mitochondrion is folded into **cristae**, which gives a much larger area for the proteins of the electron transport chain. It also separates two compartments, so a proton gradient can build up between them.
- In a chloroplast, the **thylakoid** membranes hold the light-reaction proteins, and protons build up inside each thylakoid.

The Origins of Cell Compartmentalization

The **endosymbiotic theory** 内共生学说 explains mitochondria and chloroplasts: they arose when a larger cell engulfed free-living prokaryotes that then lived inside it. The evidence – their own circular DNA, their own ribosomes, and double membranes – supports this shared evolutionary origin.

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evidence: own circular DNA, 70S-type ribosomes, division within the cell, double envelope, sequence relationships to bacteria

Engulfed prokaryotes became mitochondria and chloroplasts.

Prokaryotes 原核生物 (bacteria and archaea) usually have no membrane-bound organelles, but their cells still have specialised regions: the DNA sits in an area called the nucleoid, and ribosomes are spread through the cytoplasm. In bacteria, the enzymes of the Krebs cycle work in the cytosol; in eukaryotes they work inside mitochondria. Eukaryotic cells keep many internal membranes that divide them into specialised regions.

Exam skill (experiments). Many unit 2 free-response questions describe a real experiment. The **independent variable** is what the researchers changed (for example, the ouabain concentration); the **dependent variable** is what they measured (the pump's activity). A **control** is a group treated the same way except for the factor being tested, such as normal kidney cells beside diseased ones, or cells with no inhibitor. When asked to "justify a control", say what it rules out: it shows that any difference is caused by the factor tested, not by something else in the method.

Common mistakes.

- Using "hypertonic" without saying compared with what. Tonicity always compares **two** solutions: the solution outside is hypertonic **to the cell** when it has a higher concentration of solutes that cannot cross the membrane than the cell's cytoplasm.
- Moving water the wrong way. With equal pressure potential, water moves **towards** the hypertonic side, which has the **lower** water potential. When pressures differ, compare total water potentials.
- Dropping the minus sign in $\Psi_s = -iCRT$. Adding solute always makes the solute potential **more negative**.
- Forgetting i for an ionic solute. NaCl gives two particles, so $i = 2$ and Ψ_s is about twice that of sucrose at the same molarity.
- Saying water crosses the membrane only through aquaporins. A little crosses the bilayer directly; aquaporins make it much faster.

- Calling facilitated diffusion active because it uses a protein. Any movement **down** the gradient is passive, with or without a channel or carrier.
- Saying membrane proteins are made by free ribosomes. Proteins for the membrane or for export are made by ribosomes **on the rough ER**.
- Saying the Golgi makes proteins. Ribosomes make them; the Golgi **modifies, sorts and packages** them.
- Saying ions cross the membrane because they are small. They are blocked by the nonpolar core because they are **charged**, however small.
- Saying the sodium-potassium pump moves equal numbers of ions. It moves **3 Na^+ out and 2 K^+ in** per ATP.

Practice

Answer each before reading the answers below.

1. Calculate the surface-area-to-volume ratio of a cube-shaped cell of side 1 µm and of side 3 µm. Which exchanges materials more efficiently, and why?
2. Calculate the solute potential of a 0.10 M sucrose solution at 295 K ($\Psi_s = -iCRT$, $R = 0.0831$ L · bar/(mol · K)). A potato cell with $\Psi = -3.0$ bars is placed in it. Which way does water move?
3. Predict what happens to a red blood cell placed in a hypertonic solution, and explain.
4. Which of these cross the phospholipid bilayer without a protein: O_2, glucose, Na^+, a steroid hormone? Explain.
5. The sodium-potassium pump moves 3 Na^+ out and 2 K^+ in per ATP. Explain why this is active transport.
6. In an experiment, dialysis bags containing 0, 0.2, 0.4 and 0.6 M sucrose are placed in distilled water and weighed after 30 minutes. Identify the independent variable, the dependent variable and an appropriate control.
7. Give two pieces of evidence that mitochondria were once free-living prokaryotes.

Answers

1. 1 µm: $6 \div 1 = 6$ μm^{-1}; 3 µm: $54 \div 27 = 2$ μm^{-1}. The smaller cell has more membrane per unit volume, so it exchanges materials faster relative to its needs.
2. $\Psi_s = -(1)(0.10)(0.0831)(295) = -2.45$ bars. Water moves from higher water potential (-2.45) to lower (-3.0), so into the potato, which gains mass.
3. It shrinks (crenates): water potential is lower outside, so water leaves the cell by osmosis.
4. O_2 and the steroid hormone, because they are small or nonpolar and dissolve in the hydrophobic core. Glucose (large, polar) and Na^+ (charged) need channels or carriers.

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5. It moves both ions against their concentration gradients and uses energy from ATP hydrolysis.
6. Independent: sucrose concentration in the bag. Dependent: percent change in mass. Control: a bag containing distilled water (0 M).
7. Any two: they have their own circular DNA; they have a double membrane; their ribosomes resemble bacterial ribosomes; they divide by a process like binary fission.

Exam tips

- Use the two AP-formula skills: **surface-area-to-volume ratio** (why cells stay small) and **water potential** $\Psi = \Psi_p + \Psi_s$ ($\Psi_s = -iCRT$).
- Water moves toward the **lower (more negative) water potential**; predict swelling or shrinking from tonicity (hypo/hyper/isotonic).
- **Passive transport and osmosis need no energy** (down the gradient); **active transport** needs energy, directly from ATP or indirectly from another gradient (against the gradient).
- Distinguish diffusion, facilitated diffusion (a channel/carrier protein), and active transport.
- Explain membrane selectivity from the **phospholipid bilayer** – small non-polar molecules cross, large/charged ones need help.

Cellular Energetics

AP Biology

Enzymes as Biological Catalysts

Cyanide kills in minutes, even while oxygen is still reaching the blood: the cells cannot use it. Severe poisoning also disrupts breathing. Cyanide binds to one protein at the very end of the electron transport chain in their mitochondria. Oxygen can no longer accept electrons, the chain stops, and the cells run out of ATP. One blocked step in a long pathway is enough. This unit is about those pathways: the enzymes that run each step, and how cells capture energy from light and release it from food.

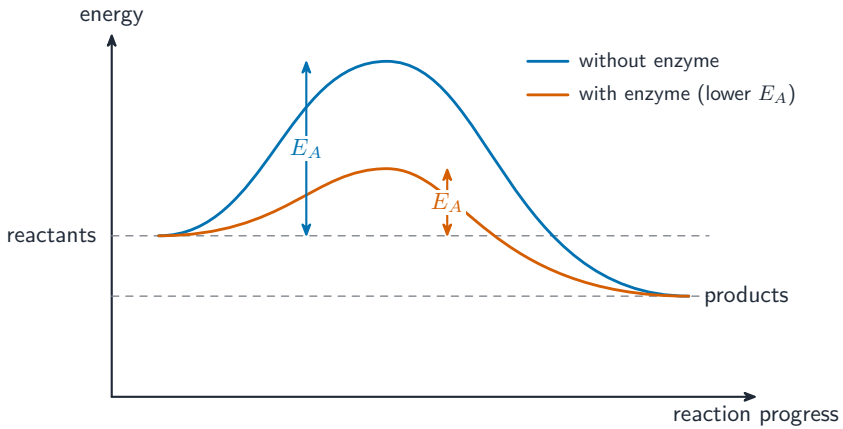


Biological washing powder uses enzymes that work at moderate temperature and pH

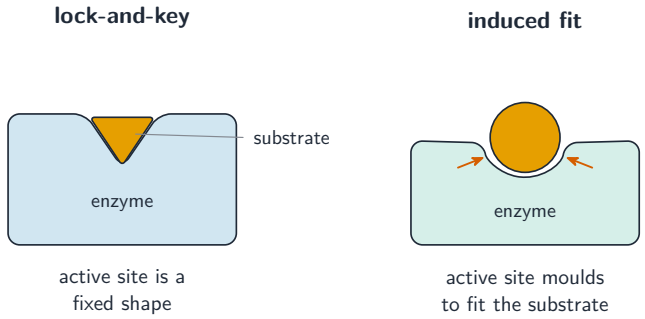
An **enzyme** 酶 is a biological **catalyst** 催化剂, usually a protein (some RNA molecules also catalyse reactions), that speeds a reaction by lowering its **activation energy** 活化能, without being used up. Each enzyme has an **active site** 活性位点 that binds a specific **substrate** 底物 (the "lock and key" or induced fit), so enzymes are highly specific. They do not change whether a reaction is favorable – only how fast it goes.

Fit means both **shape and charge**. The active site's shape matches the substrate, and the R groups lining it carry charges or polar groups that attract opposite charges on the substrate. A substrate with the right shape but the wrong charge pattern will not bind well. When the substrate binds, it forms an **enzyme-substrate complex**; the enzyme then releases the products and can be used again.

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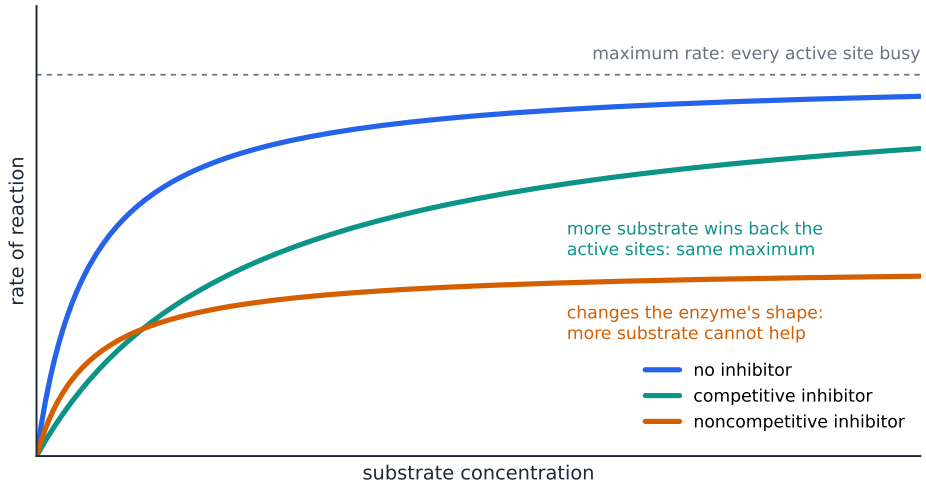
An enzyme lowers the activation energy of a reaction



The lock-and-key and induced-fit models of enzyme action

Environmental Impacts on Enzyme Function

Enzyme activity depends on conditions. Each enzyme has an **optimal** temperature and pH. Excessive heat or extreme pH can make a protein enzyme **denature** 变性 (lose shape) and stop working; smaller pH changes can reduce activity by changing active-site charges without unfolding the protein. **Substrate concentration** raises the rate until the enzyme saturates. **Inhibitors** 抑制剂 slow enzymes – **competitive** ones block the active site, **noncompetitive** ones bind elsewhere and change the shape.



Rate against substrate concentration: a competitive inhibitor can be outcompeted by more substrate; a noncompetitive inhibitor lowers the maximum rate

At low substrate concentration, adding substrate speeds the reaction because more collisions fill more active sites. At high concentration every active site is busy, so the rate levels off. The graph shows how to tell the two kinds of inhibitor apart: adding lots of substrate overcomes a competitive inhibitor, but not a noncompetitive one. Product concentration matters too: if products build up, the reaction slows, and in many pathways the final product itself switches off an early enzyme (feedback inhibition). Denaturation is sometimes **reversible**: if the temperature or pH returns to normal before the protein has been badly damaged, it can fold back and work again.

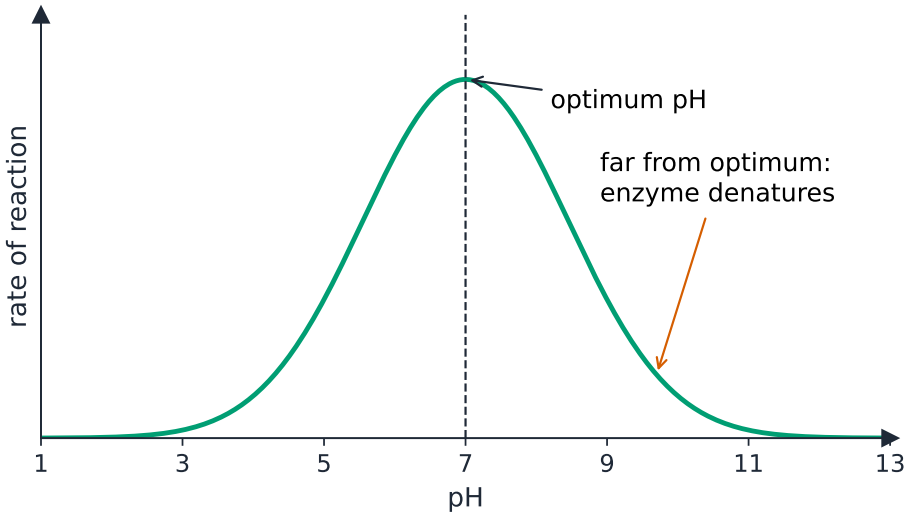
Worked example (a blocked pathway). A cell makes amino acid B in three steps: substrate $\xrightarrow{\text{enzyme 1}}$ X $\xrightarrow{\text{enzyme 2}}$ Y $\xrightarrow{\text{enzyme 3}}$ B. The product of enzyme 2 is Y. If the pH changes so that enzyme 3 denatures, it can no longer bind Y: B is not made, and Y builds up. The pH change breaks the hydrogen and ionic bonds that hold enzyme 3's shape, so its active site no longer fits Y in shape and charge.

Worked example (an enzyme experiment). Students add the same volume of potato extract, which contains **catalase**, to hydrogen peroxide at five temperatures, and collect the oxygen for 2 minutes.

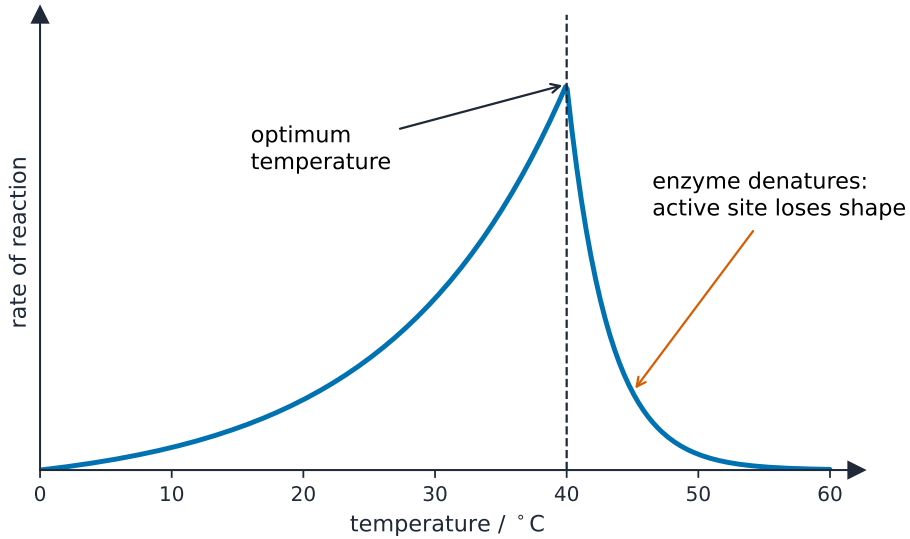
Temperature (°C)	**10**	**20**	**30**	**40**	**50**
O_2 collected in 2 min (mL)	6	12	20	14	2

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- **Variables.** The **independent variable** 自变量 is temperature; the **dependent variable** 因变量 is the volume of oxygen (or the rate). **Controlled variables** 控制变量 include the volume and concentration of peroxide, the amount of extract, and the pH.
- **Rate.** At 30 °C the rate is $20 \div 2 = 10$ mL O_2 per minute; at 10 °C it is 3 mL per minute.
- **Claim and reasoning.** The rate rises from 10 to 30 °C because molecules move faster and collide with active sites more often. Above about 30 °C it falls sharply because the enzyme's hydrogen and ionic bonds break, the active site changes shape, and fewer substrate molecules can bind (denaturation).
- **Control.** A tube with boiled extract is a **negative control**: if it gives no oxygen, the oxygen in the other tubes came from the enzyme, not from peroxide breaking down by itself.
- **Limit.** With readings only every 10 °C, the optimum could be anywhere between 20 and 40 °C. Repeating each temperature, and adding readings at 25 and 35 °C, would make the estimate more reliable.



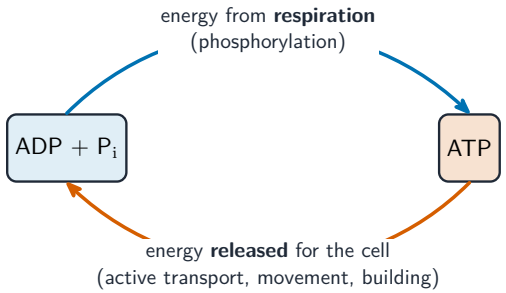
Each enzyme has an optimum pH



Rate rises to an optimum temperature, then falls as the enzyme denatures

Cellular Energy and ATP

ATP (adenosine triphosphate) is the cell's energy currency. **Hydrolysing** its last phosphate (ATP + H_2O → ADP + P_i) **releases** free energy to power cellular work, and making ATP from ADP + P_i takes energy in. The energy comes from the products being far more stable than ATP, not from "breaking a bond": breaking any bond takes energy. Cells constantly recycle ATP, coupling energy-releasing reactions to energy-requiring ones.



The ATP-ADP cycle stores and releases energy

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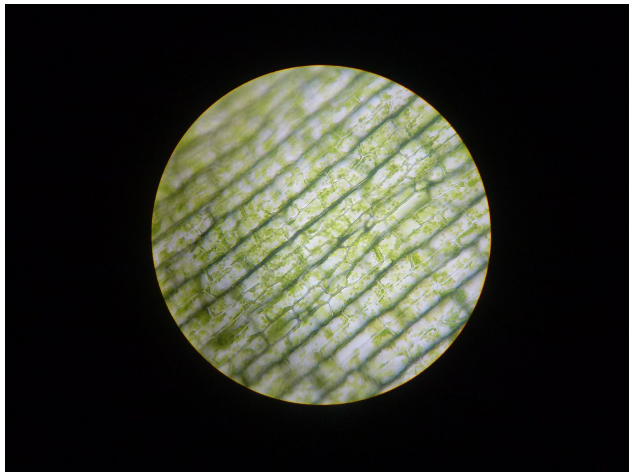
Life and the laws of thermodynamics

A living cell is highly ordered, and that order is often mistaken for a violation of physics. It is not.

- **The first law** 热力学第一定律 says energy is conserved: it changes form but is never created or destroyed. A cell does not make energy – it *transforms* the chemical energy of glucose, or the light energy of the Sun, into the chemical energy of ATP.
- **The second law** 热力学第二定律 says that in any energy transfer, some energy is lost as heat and the **entropy** 熵 of the universe increases. A cell builds order inside itself, but only by releasing more disorder to its surroundings, mostly as heat.

Living systems need a **constant input of free energy** 自由能 to maintain organised processes over time. That energy can come from current intake or stored resources. An organism does not necessarily die when external intake stops, but cannot maintain these processes indefinitely without replenishing its energy supply.

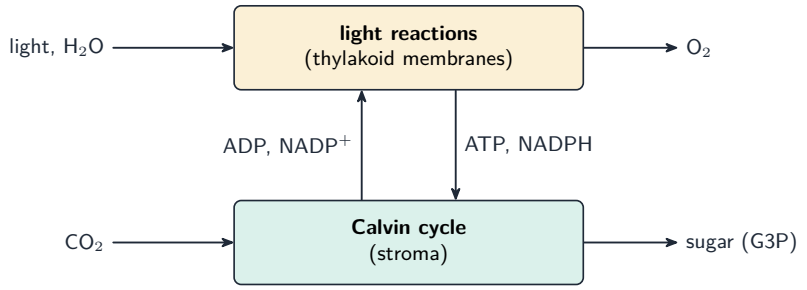
Photosynthesis



Chloroplasts fill plant mesophyll cells – the organelles of photosynthesis

Photosynthesis 光合作用 captures light energy to build sugar from CO_2 and water, releasing O_2. It has two stages:

- The **light reactions** (in the thylakoid membranes) use light to make ATP and NADPH and split water, releasing oxygen.
- The **Calvin cycle** 卡尔文循环 (in the stroma) uses that ATP and NADPH to fix CO_2 into sugar.



The light reactions make ATP and NADPH; the Calvin cycle spends them to fix CO_2 into sugar

So light energy becomes chemical energy stored in glucose.

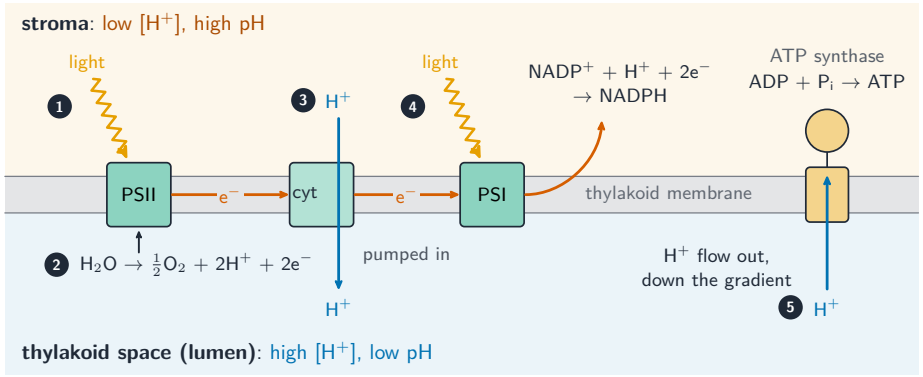
> **Worked example (leaf disks).** Leaf disks with the air removed sink in a sodium bicarbonate solution, which supplies CO_2. Under light they photosynthesise, oxygen collects in the leaf spaces, and the disks float. A lamp placed closer makes the disks float sooner: more light means a faster rate of the light reactions and more O_2. **Predict:** in plain water with no added bicarbonate, the disks generally float more slowly or may stay down. Plain water still contains some dissolved CO_2, but its lower availability limits the Calvin cycle; less $NADP^+$ and ADP are regenerated, so the light reactions slow too. The time for half the disks to float is a convenient measure, and a shorter time means a faster rate.

The light reactions, step by step

The light reactions happen in the **thylakoid** 类囊体 membrane. Thylakoids are flattened sacs stacked into piles called grana, which pack a large membrane area into each chloroplast; the fluid around them is the stroma. Two groups of pigments and proteins, **photosystem** 光系统 II (PSII) and photosystem I (PSI), absorb light. Between them is a chain of electron carriers. Follow the numbers in the figure:

1. Light energy excites electrons in the chlorophyll of PSII to a higher energy level, and they leave PSII.
2. PSII replaces them by splitting water in the thylakoid space: $H_2O \rightarrow \frac{1}{2}O_2 + 2H^+ + 2e^-$. **This is where the oxygen comes from**: it comes from water, not from CO_2.
3. The electrons pass along the chain. Their energy is used to pump H^+ from the stroma into the thylakoid space, so a **proton gradient** 质子梯度 builds up.
4. Light excites the electrons again at PSI. They then reduce $NADP^+$ to NADPH in the stroma.
5. H^+ flow back into the stroma, down their gradient, through **ATP synthase** ATP 合酶, which uses that flow to make ATP from ADP and P_i.

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The light reactions: electrons flow from water to NADPH, and the H^+ gradient they build drives ATP synthase

Cyclic electron flow. Electrons from PSI can also be passed back to the carriers between the two photosystems instead of to $NADP^+$. They pump more H^+ on the way, so the cell makes ATP but no NADPH and splits no water. The Calvin cycle uses ATP and NADPH in a ratio of 3 : 2, more ATP than the linear pathway alone gives, and cyclic flow tops up the ATP. When NADPH builds up and $NADP^+$ runs short, electrons from PSI have nowhere else to go, so more of them take the cyclic route.

Making ATP using the energy of a proton gradient is called **chemiosmosis** 化学渗透. The same mechanism makes most of the ATP in mitochondria (see below). Only the location differs.

The Calvin cycle in numbers

The Calvin cycle runs in the **stroma** 基质. It needs no light directly, but it stops in the dark because it runs out of ATP and NADPH. It has three phases:

- **Carbon fixation** 碳固定: the enzyme **rubisco** 核酮糖二磷酸羧化酶 joins CO_2 to a 5-carbon sugar, RuBP. The 6-carbon product splits at once into two 3-carbon molecules.
- **Reduction** 还原: ATP and NADPH turn these into the 3-carbon sugar G3P.
- **Regeneration** 再生: most of the G3P is rearranged, using more ATP, to make RuBP again.

Three turns fix 3 CO_2 and give one spare G3P. Making one glucose (6 carbons) takes 6 turns, which use 18 ATP and 12 NADPH.

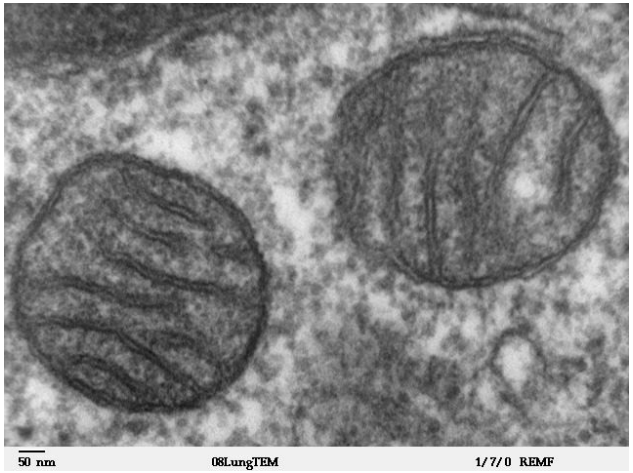
Where the oxygen in the air came from

Photosynthesis did not only feed the first organisms; it changed the planet. Scientific evidence supports the claim that photosynthesis by **prokaryotes** 原核生物 – specifically **cyanobacteria** 蓝细菌 – produced the oxygen that built Earth's **oxygenated atmosphere** 含氧大气.

The evidence is physical: **banded iron formations** 条带状铁建造 in rocks about 2.4 billion years old record iron dissolved in the ocean being oxidised and precipitating out, which needs free oxygen. Before that event, called the Great Oxidation Event, the atmosphere held almost none.

Two consequences matter for the rest of the course. Free oxygen made **aerobic respiration** possible, which releases far more ATP per glucose than anaerobic pathways do. And it was lethal to much of the anaerobic life already present, so it changed which organisms could survive – an early example of organisms changing their own environment.

Cellular Respiration

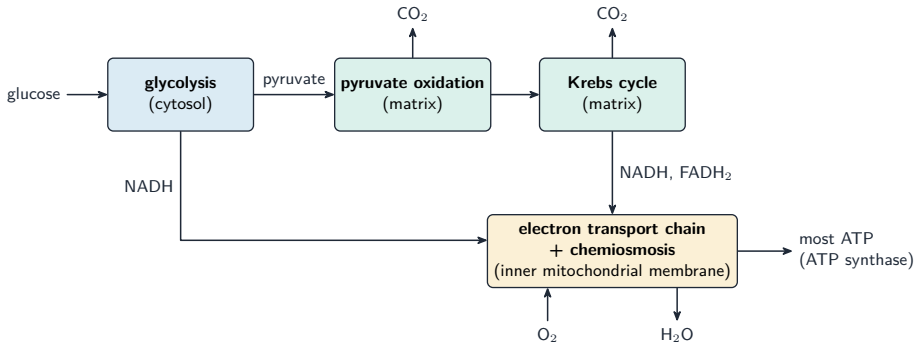


TEM of a mitochondrion: folded inner membrane (cristae) multiplies surface for respiration

Cellular respiration 细胞呼吸 releases the energy in glucose to make ATP, mostly using oxygen. Its stages:

- **Glycolysis** 糖酵解 (in the cytoplasm) splits glucose, making a little ATP.
- The **Krebs cycle** 克雷布斯循环 (mitochondrial matrix) releases CO_2 and loads electron carriers.

- The **electron transport chain** 电子传递链 (inner membrane) uses those electrons to pump protons and make most of the ATP, with oxygen as the final electron acceptor.



The stages of aerobic respiration and where in the cell each happens

Without oxygen, cells use **fermentation** 发酵 to keep glycolysis running, making far less ATP. Photosynthesis and respiration are complementary – the products of one are the reactants of the other.

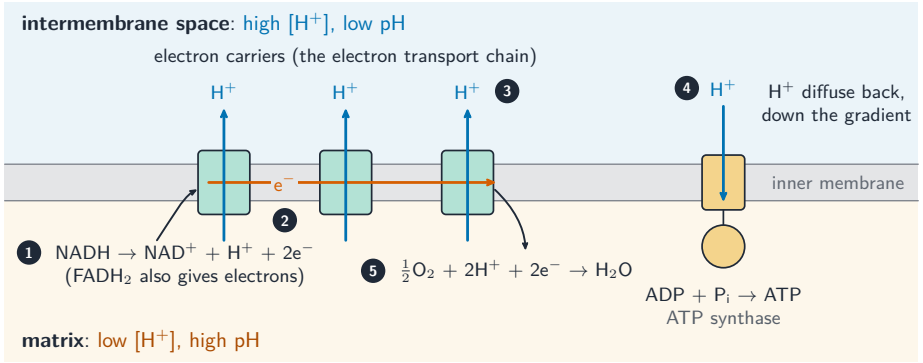
Following the electrons

Glucose is oxidised in steps. At each step, electrons (with H^+) are loaded onto the carriers NAD^+ and FAD, which become NADH and $FADH_2$.

- **Glycolysis**: glucose (6C) → 2 **pyruvate** 丙酮酸 (3C), giving a net 2 ATP and 2 NADH.
- **Pyruvate oxidation** 丙酮酸氧化: in the matrix, each pyruvate loses one CO_2 and becomes acetyl-CoA (2C), giving 1 NADH.
- **Krebs cycle**: each acetyl-CoA is oxidised to 2 CO_2. Per glucose (two turns) this gives 2 ATP, 6 NADH and 2 $FADH_2$.

So the six carbons of glucose all leave as CO_2 before the electron transport chain is reached. The chain then gets the electrons, not the carbon.

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Chemiosmosis in mitochondria: the chain pumps H^+ out of the matrix, and their flow back through ATP synthase makes ATP

Compare the two membranes:

	Chloroplast	Mitochondrion
electrons come from	water	NADH and $FADH_2$
energy that moves them	light	the electrons' own energy, released step by step
H^+ pumped into	thylakoid space	intermembrane space
final electron acceptor 最终电子受体	$NADP^+$	O_2 (forms water)
ATP synthase faces	the stroma	the matrix

Why fermentation is needed. Glycolysis needs NAD^+. Without oxygen, the chain stops, NADH is not turned back into NAD^+, and glycolysis would stop as well. Fermentation passes the electrons from NADH to pyruvate (or to a molecule made from it). In animals this makes lactate; in yeast it makes ethanol and CO_2. This **regenerates NAD^+** so that glycolysis can keep making its 2 ATP. Fermentation itself makes no extra ATP.

What happens if the gradient leaks. A substance that lets H^+ cross the membrane without passing through ATP synthase is an **uncoupler** 解偶联剂. The chain keeps running and oxygen is still used, but little ATP is made: the energy is released as heat. Brown fat in newborn babies and hibernating mammals does this on purpose, using a channel protein, to keep the body warm.

Common mistakes.

- "Photosynthesis releases the oxygen from CO_2." The O_2 comes from **water**, split at PSII.

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- "The electron transport chain makes ATP." The chain pumps H^+; it is **ATP synthase** that makes the ATP, powered by the H^+ flowing back.
- Mixing up the compartments. In the chloroplast, H^+ build up **inside the thylakoid**. In the mitochondrion, they build up in the **intermembrane space**, not in the matrix.
- "Fermentation makes ATP." Only its glycolysis step does; the extra step only regenerates NAD^+.
- "The Calvin cycle is the dark reaction, so it happens at night." It runs in the light, when ATP and NADPH are being made.
- "Oxygen is used in the Krebs cycle." Oxygen is used only at the **end of the chain**. The Krebs cycle stops without it because NAD^+ and FAD are not regenerated.
- "Blocking ATP synthase makes the proton gradient disappear." The opposite: with no way back, H^+ pile up and the gradient gets **steeper**.
- "A competitive inhibitor lowers the maximum rate." Enough substrate outcompetes it, so the maximum rate is unchanged; a **noncompetitive** inhibitor lowers it.
- Describing an active site by shape alone. It fits its substrate in **shape and charge**.

> **Worked example.** Aerobic respiration of one glucose nets about **2 ATP** from glycolysis, **2 ATP** from the Krebs cycle, and about **28 ATP** from oxidative phosphorylation, for ≈ **32 ATP** total. With no oxygen only glycolysis runs, so fermentation nets just **2 ATP** per glucose – roughly 16 times less energy, which is why aerobic pathways dominate in oxygen-rich cells.

Fermentation comes in two common forms. In muscle cells short of oxygen, and in the bacteria that cause tooth decay, pyruvate takes the electrons from NADH and becomes lactate (the ion of **lactic acid** 乳酸). Lactate formation itself consumes a proton; it is not the cause of muscle acidification. In yeast, pyruvate is turned into ethanol and CO_2, which makes bread rise.

Exam skill (predict the effect of a block). Free-response questions often name one step that is blocked and ask what changes. Work along the pathway in both directions.

- **ATP synthase blocked** (for example by oligomycin): H^+ cannot flow back, so the proton gradient becomes *steeper*, ATP production falls, and the chain slows because pumping against an ever-steeper gradient gets harder; oxygen use falls.
- **The end of the chain blocked** (cyanide, or an enzyme deficiency): electrons cannot reach oxygen, NADH is not turned back into NAD^+, and the Krebs cycle stops. Glycolysis can continue only through fermentation, so **lactate production rises** and ATP falls to 2 per glucose.
- **Pyruvate cannot enter the mitochondrion** or cannot be oxidised: glycolysis still makes its NADH, but the Krebs cycle gets no acetyl-CoA, so Krebs-cycle NADH and $FADH_2$ fall, and the cell turns to fermentation.

- **An uncoupler** lets H^+ leak back without ATP synthase: the chain speeds up and oxygen use rises, but the energy is released as heat. Brown fat in newborn babies and hibernating mammals does this on purpose to keep warm.

Exam skill (hypotheses and graphs). The **null hypothesis** 零假设 says the factor being tested has *no* effect, for example "temperature has no effect on the rate of ATP synthesis". Use a **line graph** when the independent variable is continuous, such as time or temperature, and a **bar graph** when it is in separate categories, such as treatment groups. Label both axes with units, and add error bars when the data give a spread or confidence interval.

Practice

Answer each before reading the answers below.

1. A competitive inhibitor and a noncompetitive inhibitor each slow an enzyme. Which effect can be overcome by adding more substrate? Explain.
2. Explain why the rate of an enzyme-catalysed reaction falls sharply above the optimum temperature.
3. A plant releases 12 mL of O_2 in 4 minutes. Calculate the rate, and state a null hypothesis for an experiment testing light intensity on this rate.
4. State where each takes place: the light-dependent reactions, the Calvin cycle, the electron transport chain of respiration.
5. Cyanide blocks the last protein of the electron transport chain. Predict and explain the effect on ATP production.
6. Explain why fermentation lets glycolysis continue without oxygen.

Answers

1. Competitive: it competes for the active site, so a high substrate concentration outcompetes it. A noncompetitive inhibitor binds elsewhere and changes the enzyme's shape, so more substrate does not help.
2. High temperature breaks the hydrogen bonds and other interactions that hold the enzyme's shape. The active site changes shape (denaturation), so substrate no longer fits.
3. $12 \div 4 = 3$ mL/min. Null hypothesis: light intensity has no effect on the rate of oxygen production.
4. Thylakoid membranes; the stroma of the chloroplast; the inner mitochondrial membrane.
5. Electrons can no longer pass to oxygen, so the chain stops and protons are no longer pumped. The proton gradient disappears, so ATP synthase makes far less ATP (only glycolysis continues).

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6. Fermentation oxidizes NADH back to NAD^+, which glycolysis needs as an electron acceptor.

Exam tips

- An **enzyme lowers the activation energy** and is not used up; its **active site** is specific to one substrate (lock and key).
- Rate rises with temperature only up to the **optimum** – beyond it the enzyme **denatures** and the rate falls (unlike an ordinary reaction).
- Know the ATP↔ADP cycle: **hydrolysis** of ATP to ADP + P_i releases energy to power the cell (never write "breaking the bond releases energy").
- Write the overall equations: photosynthesis **stores** energy (builds glucose); respiration **releases** it (breaks glucose down) – they are opposites.
- Match each stage to its location and whether it needs oxygen.

Cell Communication and Cell Cycle

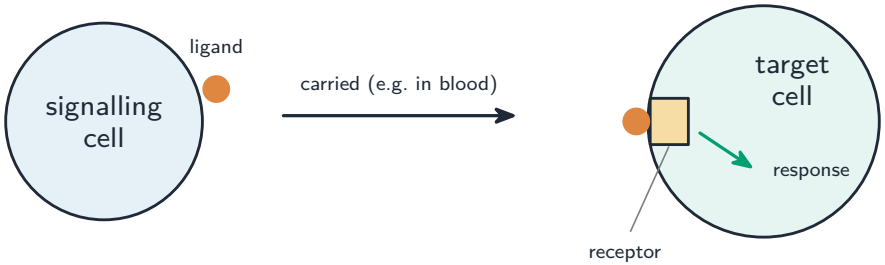
AP Biology

Cell Communication

A single molecule of **adrenaline** 肾上腺素 arriving at a liver cell can release millions of **glucose** 葡萄糖 molecules within seconds. A single faulty switch in the same kind of pathway can make a cell divide without stopping. This unit follows a signal from the outside of a cell to its response, and shows where the control can fail.

Cells coordinate by sending and receiving chemical **signals** 信号. In chemical signaling, a **ligand** 配体 binds a **receptor** 受体 on a **target cell** 靶细胞. The ligand may be released or held on a cell surface. Signals travel over different ranges: **direct contact** (**cell junctions** 细胞连接), **local** signaling (nearby cells, like **neurotransmitters** 神经递质), and **long-distance** signaling (**hormones** 激素 carried through the blood in animals; plants use other transport routes).

Each range has classic examples. In **direct contact**, an immune cell that has engulfed a pathogen shows pieces of it on its surface, and a helper T cell must touch it to read them. **Local** signals include neurotransmitters, morphogens that tell embryo cells where they are, and **quorum sensing**, in which bacteria release a chemical and switch on new genes (such as those for glowing or for forming a biofilm) only when enough of them are nearby. **Long-distance** signals include insulin, human growth hormone, thyroid hormones, testosterone and oestrogen.



A signal molecule (ligand) binds a matching receptor on the target cell

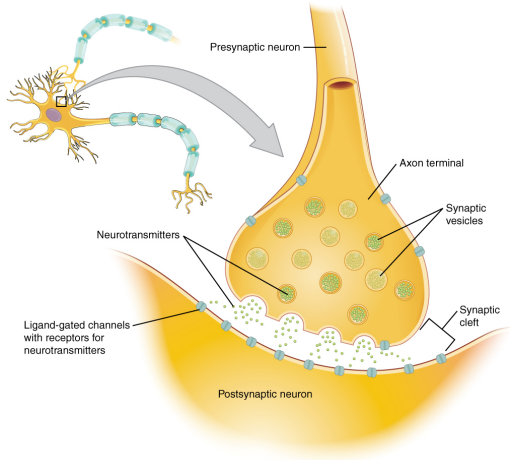
Introduction to Signal Transduction

Signal transduction 信号转导 converts an outside signal into a cellular response in three stages: **reception** 接收 (ligand binds receptor), **transduction** 转导 (a relay of molecules inside the cell), and **response** 响应 (a change in the cell's activity, such as switching on a gene). Receptors are specific, so a cell only responds to signals it can receive.

Where the receptor sits depends on the ligand. Large or polar ligands, such as peptide hormones and neurotransmitters, cannot cross the membrane, so they bind receptors **on the cell surface**. Small nonpolar ligands, such as steroid hormones (oestrogen, testosterone), cross the membrane and bind receptors **in the cytoplasm or nucleus**; the receptor–hormone complex often acts directly on genes. A receptor has a ligand-binding part that fits only its ligand; when the ligand binds, the receptor's part *inside* the cell changes shape, and that change starts transduction.

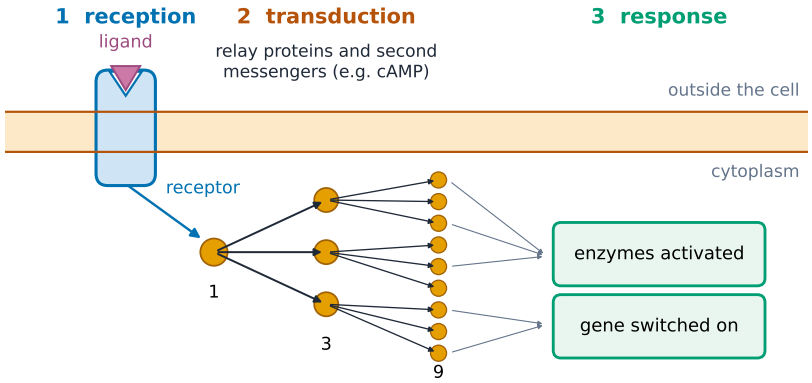
Two kinds of surface receptor appear often on the exam:

- **G protein-coupled receptors** (GPCRs). The ligand changes the receptor's shape, so it activates a G protein inside the membrane. An inactive G protein holds GDP; the active receptor makes it swap GDP for GTP, and the GTP-bound G protein switches on an enzyme, such as the one that makes cyclic AMP. The G protein then breaks GTP down to GDP and turns itself off.
- **Ligand-gated ion channels**. The ligand opens (or closes) a channel. Acetylcholine binding to receptors on a muscle cell opens channels that let Na^+ in, which starts the muscle contracting.



A chemical synapse: signal transduction passes a message across a gap with a ligand and receptor

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The three stages of cell signalling: reception at the membrane, transduction by a relay cascade that amplifies the signal at each step, and the response

Signal Transduction Pathways

In the transduction stage, the signal passes through a **pathway** 通路 – often a **cascade** 级联 of proteins that activate one another, frequently **amplifying** 放大 the signal so a few ligands trigger a large response. **Second messengers** 第二信使 (like **cyclic AMP** 环磷酸腺苷 or calcium ions) spread the signal quickly through the cell. A change in one step can alter the whole outcome.

Many cascades work by **phosphorylation**. Enzymes called kinases add a phosphate group, taken from ATP, to the next protein in the relay. The phosphate is charged, so it changes the protein's shape, which switches it on (or, for some proteins, off). Each kinase activates many copies of the next one, so the signal is amplified. Enzymes called phosphatases remove the phosphates, which switches the pathway off again when the signal stops.

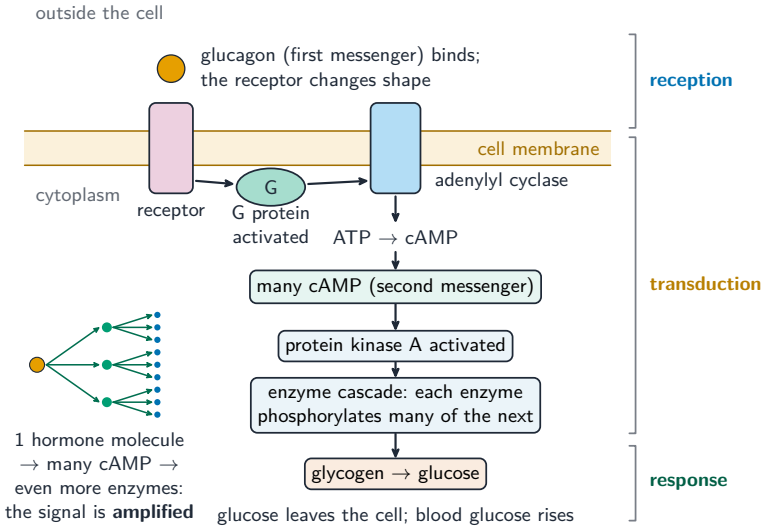
The response can be a change in which genes are expressed, a change in an enzyme's activity (adrenaline switches on the breakdown of glycogen to glucose in liver cells), cell division, or even **apoptosis** 细胞凋亡, programmed cell death.

Worked example (toxins and drugs). Chemicals that act on any step can switch a pathway on or off.

- *Cholera toxin* changes the G protein in gut cells so that it cannot break down its GTP. The G protein stays on, the cell makes cyclic AMP nonstop, and chloride ions and water pour out of the cells into the gut: severe diarrhoea.

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- A mutation that stops GTP from replacing GDP does the opposite: the G protein can never be switched on, so the ligand binds but there is **no response**.
- *Acetylcholinesterase* normally breaks down acetylcholine in the gap between nerve and muscle. Inhibit it, and acetylcholine stays bound to the receptors, the channels keep opening, and the muscle keeps being stimulated, leading to spasms.



A signalling cascade amplifies the message inside the cell

Worked example (amplification: epinephrine and glycogen). Epinephrine (adrenaline) binds a G protein-coupled receptor on a liver cell. Suppose one activated receptor switches on 10 G proteins, each G protein activates one adenylyl cyclase that makes 100 cAMP molecules, and the cascade of kinases after that multiplies the signal 1000 times more. How many active enzymes of the final step (glycogen phosphorylase) result from one epinephrine molecule? $10 \times 100 \times 1000 = 10^6$: one hormone molecule leads to about a million active enzymes, each of which breaks off many glucose units from glycogen. This is why tiny concentrations of a hormone give a large, fast response. Because epinephrine never enters the cell, the response depends entirely on the receptor and the relay; a drug that blocks the receptor, or an inhibitor of adenylyl cyclase, stops glycogen breakdown even when the hormone is present.

Worked example (yeast mating pheromones). Haploid yeast cells come in two mating types, **a** and α. Each type releases a **pheromone** 信息素 that binds a receptor on cells of the **other** type. The receptor activates a G protein and a kinase cascade, which switches on **transcription factors**; these turn on the mating genes, so the cells stop dividing, grow towards each other and fuse into a diploid cell. Predict the effect of a mutation that removes the α-pheromone receptor from type **a** cells. The **a** cells can no longer detect α-pheromone, so no transduction happens, the mating genes stay off, and the cells cannot mate, although they still make their own a-pheromone and divide normally. This example shows signaling between **single-celled** organisms, and a response that is a change in **gene expression**.

Morphogens. In an embryo, a **morphogen** 形态发生素 is released from one region and diffuses away, so its concentration falls with distance. Cells read the local concentration: a high concentration switches on one set of genes, a lower one another set. In the fruit fly, the protein Bicoid, high at the head end of the egg, determines which end develops as the head; an embryo with no Bicoid develops without a head and thorax.

Reading error bars

Experimental results are often shown as means with **error bars**, usually ±2 standard errors of the mean ($\pm 2\,\mathrm{SE}_{\bar{x}}$), which gives roughly a 95% confidence interval.

Worked example. Liver cells were treated three ways, and the cAMP made was measured (mean ±2 SE, arbitrary units): no hormone 5 ± 2; epinephrine 48 ± 6; epinephrine plus a receptor-blocking drug 9 ± 4.

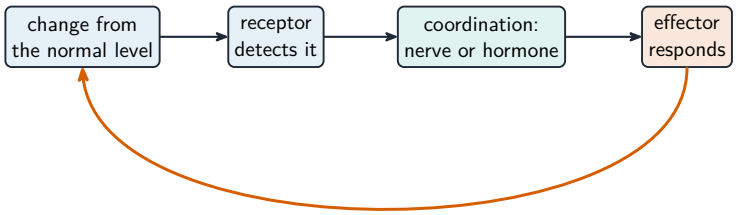
- Epinephrine vs no hormone: the error bars (3–7 and 42–54) do **not** overlap, so the increase is very likely a real (statistically significant) difference.
- No hormone vs epinephrine plus drug: the bars (3–7 and 5–13) **overlap**, so overlap alone cannot establish whether the difference is significant. The mean with the drug is much nearer the untreated mean, consistent with strong inhibition.
- A claim to support: epinephrine increases cAMP production through its receptor, because blocking the receptor greatly reduces the increase.

Overlapping 95% confidence intervals can still accompany a statistically significant difference; use an appropriate statistical test to decide. Bars that do not overlap suggest a real difference. Error bars do not tell you the cause; that comes from the controls in the design.

Feedback

Feedback 反馈 keeps systems balanced:

- **Negative feedback** 负反馈 counteracts a change to restore a **set point** 设定点 (like a **thermostat** 恒温器) – it maintains **homeostasis** 稳态.
- **Positive feedback** 正反馈 amplifies a change to push a process to completion (like childbirth **contractions** 宫缩 or **blood clotting** 血液凝固).



Negative feedback detects and corrects a change

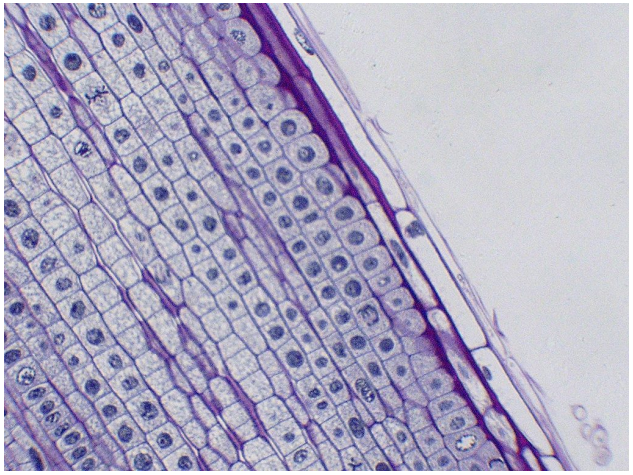
Worked example (blood sugar). After a meal, blood glucose rises. The pancreas releases **insulin** 胰岛素, which makes body cells take up glucose and the liver store it as glycogen, so blood glucose falls back to its set point. Between meals, glucose falls; the pancreas releases glucagon, which makes the liver break glycogen down and release glucose. Each hormone *reverses* the change that triggered it, so both are negative feedback. Positive feedback examples include milk release while a baby feeds (suckling causes more milk release, which encourages more suckling) and fruit ripening: ripening fruit releases the gas ethylene, which makes nearby fruit ripen and release more ethylene.

Cell Cycle

The **cell cycle** 细胞周期 is the life of a cell from one division to the next: **interphase** 间期 (G1 growth, S **DNA replication** DNA 复制, G2 preparation) followed by M phase, including **mitosis** 有丝分裂 and **cytokinesis** 胞质分裂, which normally produce **daughter cells** 子细胞 with the same **chromosome** 染色体 number and very similar genetic information. Interphase takes most of the time; DNA is copied only in S phase.

- **G1**: the cell grows and is metabolically active, copying its organelles.
- **S**: each chromosome (spread out as chromatin) is copied, giving two **sister chromatids** joined at a centromere. In animal cells the centrosome also duplicates during S phase.
- **G2**: the cell makes proteins and replenishes ATP stores in preparation for division.
- **G0**: many cells, such as most nerve cells, leave the cycle and stop dividing. Some can return to the cycle when the right signal arrives.

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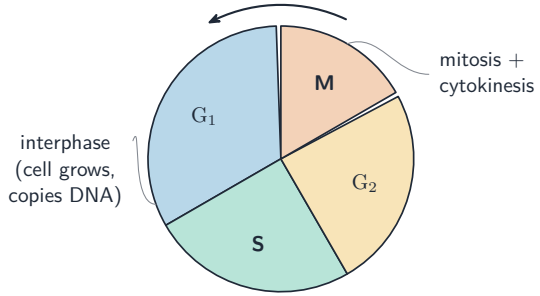


Onion root-tip cells at different stages of mitosis (light micrograph)

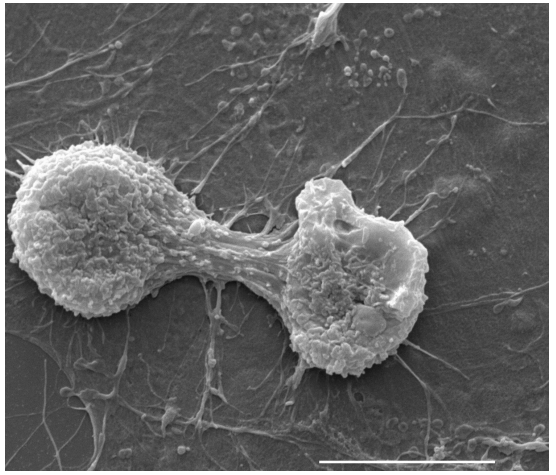
Mitosis passes a complete, identical genome to two daughter cells. It is used for growth, repair of tissues, and asexual reproduction, in four steps:

1. **Prophase** 前期: the chromatids condense into thick, visible chromosomes, the spindle starts to form, and the centrosomes move to opposite poles.
2. **Metaphase**: spindle fibres line the chromosomes up across the middle of the cell.
3. **Anaphase** 后期: the sister chromatids separate and are pulled to opposite poles.
4. **Telophase** 末期: the spindle breaks down and a new nuclear envelope forms around each set of chromosomes.

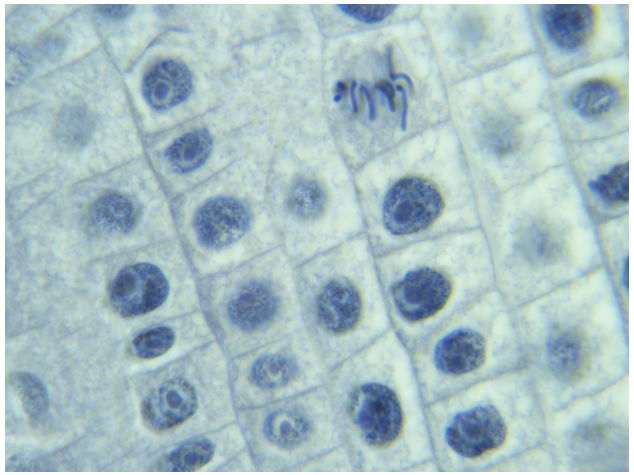
Cytokinesis then divides the cytoplasm: a cleavage furrow pinches an animal cell in two, while a plant cell, held in by its wall, builds a new cell plate across the middle. Chromosomes are visible under a microscope only during division because only then are they tightly condensed; in interphase the DNA is spread out as chromatin.



The cell cycle: interphase, then mitosis and cytokinesis



A fibroblast dividing: cytokinesis splits one cell into two as mitosis ends



Onion root tip at metaphase: chromosomes line up before sister chromatids separate

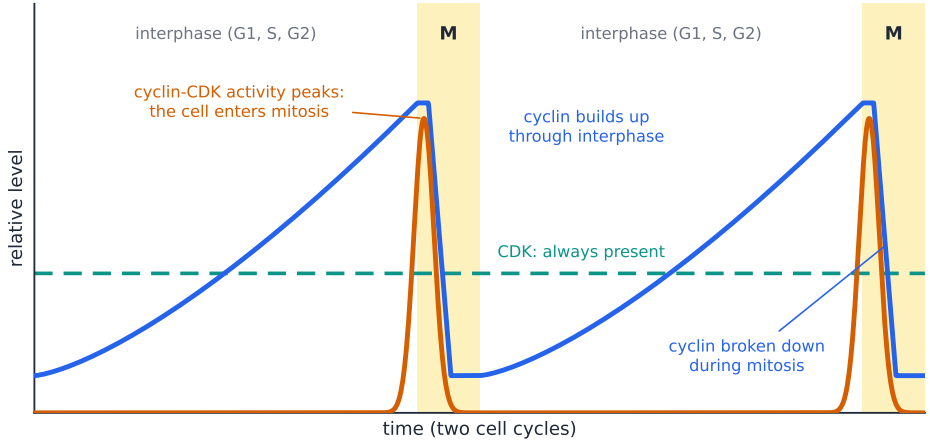
Regulation of Cell Cycle

The cycle is controlled at **checkpoints** 检查点 that verify conditions before proceeding (Is the DNA intact? Are chromosomes attached?). Internal signals (**cyclins** 周期蛋白 and their **kinases** 激酶) and external signals drive the cycle forward. When this control fails – for example, a **mutation** 突变 that ignores a checkpoint – cells divide uncontrollably, which underlies **cancer** 癌症.

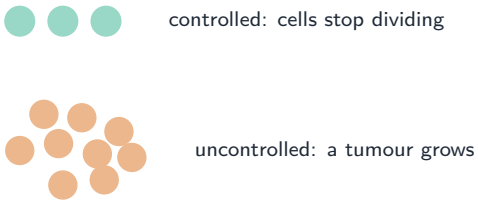
The main checkpoints are at the end of G1 (is the cell big enough, and is the DNA undamaged?), at the end of G2 (has all the DNA been copied correctly?), and in

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metaphase (is every chromosome attached to the spindle?). The driving force is a pair of proteins: a **cyclin-dependent kinase** (CDK) is always present, but it is active only when bound to a **cyclin**. Cyclin builds up through interphase; when enough cyclin–CDK complex has formed, it phosphorylates target proteins that push the cell into mitosis. Then the cyclin is destroyed and the cycle resets. A cell with damaged DNA that cannot be repaired is normally sent into apoptosis instead of dividing.



Cyclin builds up through interphase; cyclin-CDK activity peaks to start mitosis, then the cyclin is broken down



Uncontrolled division from failed checkpoints forms a tumour

Worked example. Signal amplification in a cascade: one hormone activates one receptor, which switches on about 100 relay proteins, and each of those makes about 1,000 second-messenger molecules – so a single signal produces roughly $100 \times 1000 = 10^5$ product molecules. This is why a hormone concentration as low as 10^{-9} M can trigger a large cellular response.

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Worked example (predict the effect of a mutation). In a pathway, a ligand activates a G protein, which switches on an enzyme that makes a second messenger. A mutation leaves the G protein permanently **active**, even with no ligand. Predict the result: the enzyme keeps making second messenger and the response continues **without** the signal. The cell can no longer switch the response off. In growth-signal pathways, this kind of always-on mutation is a step toward cancer.

Worked example (timing the cell cycle). In an onion root tip, 200 cells are counted: 160 in interphase, 20 in prophase, 8 in metaphase, 6 in anaphase and 6 in telophase. The cycle takes about 24 hours.

- The **mitotic index** 有丝分裂指数 is the fraction of cells in mitosis: $\dfrac{20+8+6+6}{200} = 20\%$.
- For this rough phase-duration estimate, assume an asynchronously dividing sample and use phase fractions as approximate time fractions; growth and sampling biases can affect the estimate. Interphase: $\dfrac{160}{200} \times 24 = 19.2$ hours. **Metaphase** 中期: $\dfrac{8}{200} \times 24 \approx 1.0$ hour.

DNA content through the cycle. A cell starts G1 with an amount x of DNA. It doubles to $2x$ during S phase and stays at $2x$ through G2 and mitosis. At cytokinesis each daughter cell returns to x. A graph of DNA per cell against time rises gradually during S, then drops at division.

Practice

Answer each before reading the answers below.

1. Name the three stages of cell signalling, in order.
2. A drug blocks the receptor for a hormone. Predict the effect on the target cell's response, and explain.
3. In a root-tip count, 25 of 250 cells are in mitosis and the cycle lasts 20 hours. Find the mitotic index and the time spent in mitosis.
4. Classify each as positive or negative feedback: (a) body temperature falls and shivering begins; (b) contractions during childbirth release oxytocin, which strengthens the contractions.
5. A student says: "A cell in G2 has the same amount of DNA as a cell in G1." Correct the student.

Answers

1. Reception, transduction, response.
2. The response is reduced or absent. The hormone cannot bind, so the signal never starts the transduction pathway, even though the hormone is present.

3. Mitotic index $= \dfrac{25}{250} = 10\%$; time in mitosis $\approx 0.10 \times 20 = 2$ hours.
4. (a) Negative feedback: shivering raises the temperature back toward the set point.
   (b) Positive feedback: the change is amplified until the baby is born.
5. DNA is copied in S phase, between G1 and G2, so a G2 cell has **twice** the DNA of a G1 cell. The amount halves again only when the cell divides.

Common mistakes.

- Saying all receptors are on the cell surface. Receptors for small nonpolar ligands, such as steroid hormones, are **inside** the cell.
- Saying a phosphate group always switches a protein on. Phosphorylation changes the shape; for some proteins that means **off**.
- Calling the insulin response positive feedback because insulin "increases" glucose uptake. It **reverses** the rise in blood glucose, so it is negative feedback.
- Saying overlapping error bars prove there is no difference. Overlapping confidence intervals alone **cannot decide statistical significance**; an appropriate test is needed.
- Saying chromatids separate in metaphase. They line up in metaphase and separate in **anaphase**.
- Saying CDKs are made and destroyed each cycle. **Cyclin** levels rise and fall; the CDK stays, and is active only when cyclin binds.

Exam tips

- In signal transduction name the three stages: **reception** → **transduction** → **response**.
- **Negative** feedback reverses a change to keep conditions steady (homeostasis); **positive** feedback amplifies a change to completion (childbirth, clotting).
- Order the **cell cycle**: interphase (grow, copy DNA in S phase) then mitosis → two **identical** daughter cells.
- The DNA is copied **once**, in S phase, so each daughter gets a full copy.
- Failed **checkpoints** allow uncontrolled division → cancer.

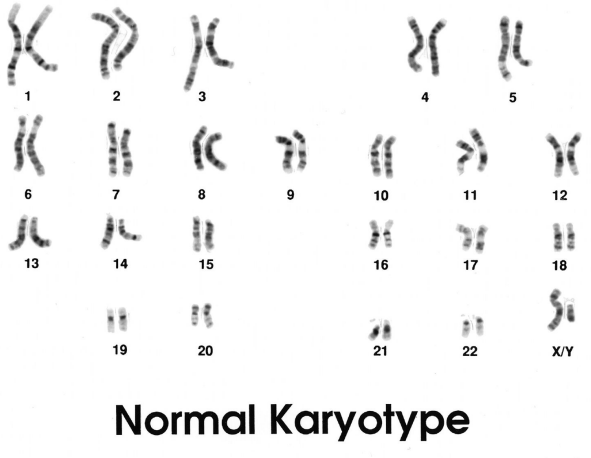
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Heredity

AP Biology

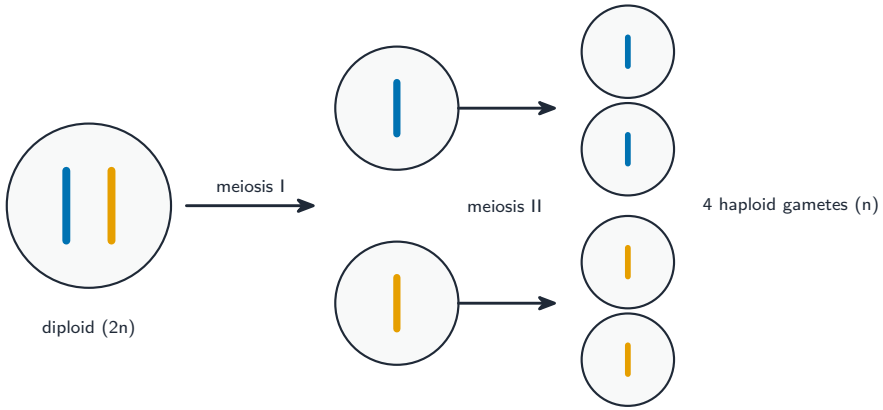
Meiosis

Two brown-eyed parents can have a blue-eyed child. Colour blindness is far more common in boys than girls. Two genes on the same chromosome are inherited together, except when they are not. Each of these follows from what chromosomes do in meiosis, and each can be turned into a probability you can calculate.



A human karyotype: 22 pairs of autosomes plus the sex chromosomes

In animals, **Meiosis** 减数分裂 produces haploid cells used in forming **gametes** 配子 (eggs and sperm), with **half** the diploid chromosome number, so fertilization restores the full set. Not all four meiotic products necessarily become functional gametes. One diploid cell divides **twice** to give four haploid cells. Meiosis I separates **homologous chromosomes** 同源染色体 (reducing the number); meiosis II separates sister chromatids (like mitosis).



Meiosis halves the chromosome number in two divisions

What happens in each division:

- **Prophase I**: homologous chromosomes pair up side by side (synapsis), and non-sister chromatids can cross over at points called chiasmata. The spindle forms and the nuclear envelope breaks down.
- **Metaphase I**: homologous *pairs* line up across the middle of the cell.
- **Anaphase I**: the homologues separate; sister chromatids stay joined.
- **Telophase I and cytokinesis**: two haploid cells form, each chromosome still made of two chromatids.
- **Meiosis II** is like mitosis: chromosomes line up singly (metaphase II), the centromeres split and sister chromatids are pulled apart (anaphase II), and four haploid cells result.

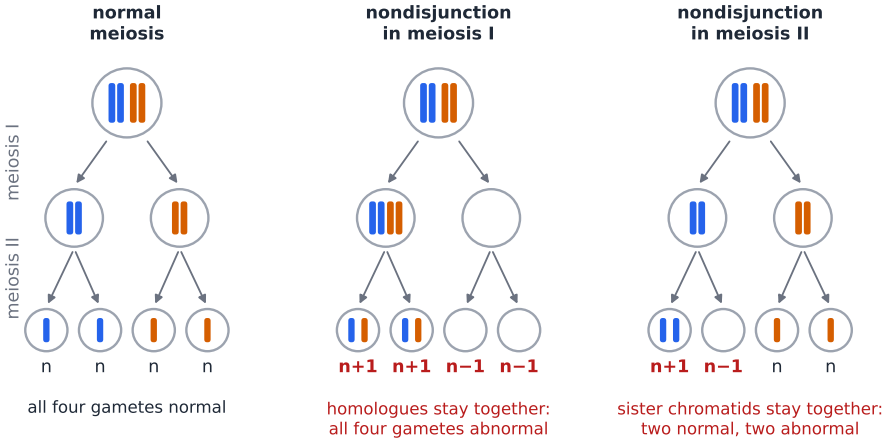
The spindle grips each chromosome at its **kinetochore**, a protein structure at the centromere. In **metaphase I**, both kinetochores of one homologue attach to microtubules from the **same** pole, and the other homologue's to the opposite pole, so whole homologues move apart in anaphase I. In **metaphase II**, the kinetochore of each **chromatid** attaches to a microtubule from a **different** pole, so the sister chromatids are pulled apart in anaphase II.

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	Mitosis	Meiosis
divisions	one	two
cells produced	two	four
chromosome number	same as parent (diploid stays diploid)	halved (**diploid** 二倍体 → **haploid** 单倍体)
daughter cells	genetically identical	genetically different
homologues pair up?	no	yes, in prophase I
used for	growth, repair, asexual reproduction	making gametes

Both use a spindle to move chromosomes, and both copy the DNA only once, in the S phase before division. Both depend on chromatids or homologues separating correctly.

Nondisjunction 不分离 is a failure of separation. If a pair of homologues fails to separate in meiosis I, *all four* gametes are abnormal: two have an extra chromosome (n+1) and two are missing one (n−1). If sister chromatids fail to separate in meiosis II, only the two gametes from that cell are affected. A zygote formed from an abnormal gamete has the wrong number of chromosomes (**aneuploidy** 非整倍性); trisomy 21, the cause of Down syndrome, is the best-known example.



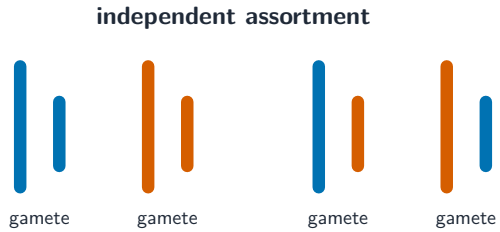
Nondisjunction in meiosis I makes all four gametes abnormal; in meiosis II, only two

A **triploid** organism (three sets of chromosomes) is usually sterile: in meiosis I the three copies of each chromosome cannot pair off evenly, so almost every gamete gets an unbalanced set. Seedless bananas and watermelons are triploid.

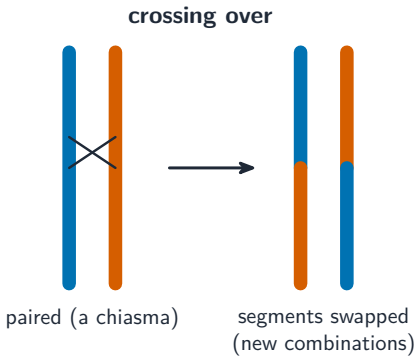
Meiosis and Genetic Diversity

Meiosis and fertilization generate variation in three ways, so offspring differ from parents and each other:

- **Crossing over** 交叉互换: non-sister chromatids of homologous chromosomes exchange corresponding segments during prophase I.
- **Independent assortment** 自由组合: each homologous pair lines up and separates randomly.
- **Random fertilization**: any sperm can meet any egg.



Independent assortment produces many gamete combinations



Crossing over swaps segments between homologous chromosomes

Together these create enormous variation – the raw material for evolution.

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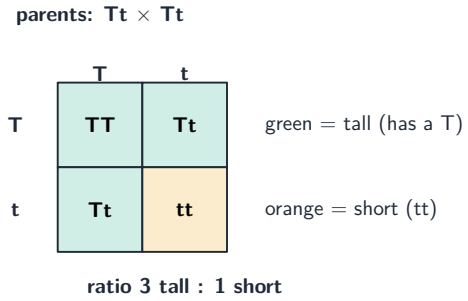
> **Worked example (counting combinations).** A cell with n homologous pairs can line them up in 2^n ways, so independent assortment alone gives 2^n kinds of gamete. A human has $n = 23$, so $2^{23} \approx 8.4$ million kinds of egg or sperm, before crossing over adds any more. Random fertilization then pairs any one of these with any of the other parent's: 8.4 million $\times$ 8.4 million $\approx 7 \times 10^{13}$ combinations for one couple.

Mendelian Genetics



Natural variation in snail shell banding – heritable differences selection can act on

A gene's alternative versions are **alleles** 等位基因. An organism's **genotype** 基因型 (its alleles), together with environmental influences, contributes to its **phenotype** 表型 (its traits). Under complete dominance, a **dominant** 显性 allele masks a **recessive** 隐性 one in a heterozygote. In the diploid Mendelian model, the two alleles **segregate** into different gametes, and unlinked genes assort **independently**. A **Punnett square** 庞纳特方格 predicts offspring probabilities; an Aa $\times$ Aa cross gives a 3:1 phenotype ratio under complete dominance. **Homozygous** 纯合 means two identical alleles; **heterozygous** 杂合 means two different.



A monohybrid Punnett square giving a 3:1 ratio

The two probability rules.

- **Product rule** (for independent events that must **both** happen): $P(A \text{ and } B) = P(A) \times P(B)$.
- **Sum rule** (for mutually exclusive events, **either** of which will do): $P(A \text{ or } B) = P(A) + P(B)$.

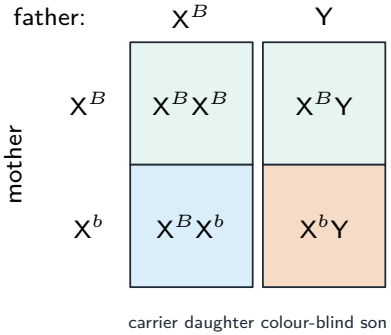
> **Worked example (sum rule).** In $Aa \times Aa$, what is the probability that an offspring is heterozygous? It can get A from the mother and a from the father ($\frac{1}{2} \times \frac{1}{2} = \frac{1}{4}$), **or** a from the mother and A from the father (another $\frac{1}{4}$). These are mutually exclusive, so add: $\frac{1}{4} + \frac{1}{4} = \frac{1}{2}$.

> **Worked example.** For a **dihybrid** cross of two independent genes, $AaBb \times AaBb$, use the multiplication rule instead of a 16-box square. Each gene alone gives $\frac{3}{4}$ dominant, so the chance an offspring shows **both** dominant traits is $\frac{3}{4} \times \frac{3}{4} = \frac{9}{16}$, and the chance of the fully recessive $aabb$ is $\frac{1}{4} \times \frac{1}{4} = \frac{1}{16}$. Multiplying two independent 3:1 ratios is what produces the classic 9:3:3:1 pattern.

Non-Mendelian Genetics

Many traits do not follow simple dominance:

- **Incomplete dominance** 不完全显性: heterozygotes are a blend (red × white → pink).
- **Codominance** 共显性: both alleles show fully (AB blood type).
- **Multiple alleles**, **polygenic** 多基因 traits (many genes, like height), **pleiotropy** (one gene, many effects), and **sex-linked** 伴性 genes (on sex chromosomes, often the X chromosome) all give more complex ratios.



Sex linkage gives different results for sons and daughters

Three more patterns come up often:

- **Non-nuclear inheritance.** Mitochondria and chloroplasts have their own DNA and are shared out randomly when cells divide. In animals, mitochondria normally come from the egg, not the sperm. A mother can pass a mitochondrial DNA mutation to sons and daughters, but an affected father normally does not pass it on. If the mother carries a mixture of normal and mutant mitochondrial DNA, the amount inherited and the severity of disease can differ among children. In many plants, chloroplasts come from the ovule, so a leaf-colour trait carried in chloroplast DNA, such as white-striped leaves, follows the mother plant; some species inherit chloroplasts from the father or both parents.
- **Other sex-determination systems.** Birds use Z and W chromosomes: ZZ is male and ZW female, so in birds it is females who show Z-linked recessive traits more often. In bees, males develop from unfertilized eggs and are haploid. In many reptiles, the temperature at which the eggs are kept decides the sex.
- **Lethal alleles.** If one genotype dies before birth, the expected ratio changes. Crossing two heterozygotes for a lethal recessive allele gives $1:2:1$, but the homozygous recessive quarter never appear, so the survivors are in a $1:2$ ratio, a sign that an allele is lethal in double dose.

> **Worked example (chi-square test).** To check whether real data fit a predicted ratio, use $\chi^2 = \sum \dfrac{(o-e)^2}{e}$. A monohybrid cross predicts 3:1, so of 80 offspring you expect 60 dominant and 20 recessive, but you observe 55 and 25. Then $\chi^2 = \dfrac{(55-60)^2}{60} + \dfrac{(25-20)^2}{20} = \dfrac{25}{60} + \dfrac{25}{20} = 0.42 + 1.25 = 1.67$. With 1 degree of freedom the critical value at $p = 0.05$ is 3.84; since $1.67 < 3.84$, we **fail to reject** the null hypothesis – the deviation is within chance.

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Worked example (a sex-linked cross). Red–green colour blindness is caused by a recessive allele on the X chromosome. A carrier mother (X^BX^b) has children with a father of normal vision (X^BY).

- Daughters get the father's X^B, so **none** is colour-blind; half are carriers (X^BX^b).
- Sons get the father's Y and either maternal X, so **half** the sons are colour-blind (X^bY).

Boys need only one recessive allele to show the trait, because the Y carries no copy of the gene. That is why X-linked recessive conditions are far more common in males.

Linked genes and recombination

Genes on the **same** chromosome are **linked** 连锁. Nearby genes tend to be inherited together; distant genes can behave as if unlinked because multiple crossovers can hide exchanges. In a testcross, **recombination frequency** 重组频率 measures recombinant offspring. It estimates short gene separations but approaches a maximum expected value of 50%; it does not keep increasing with map distance:

$$\text{recombination frequency} = \frac{\text{number of recombinant offspring}}{\text{total offspring}} \times 100\%.$$

Worked example. A test cross of a dihybrid ($AaBb \times aabb$) gives 445 $AaBb$, 435 $aabb$, 62 $Aabb$ and 58 $aaBb$ offspring. The two large classes match the parents' combinations; the two small ones are **recombinants**. Recombination frequency $= \frac{62+58}{1000} = 12\%$, so the genes are about 12 map units apart. Unlinked genes would give four roughly equal classes, near 25% each.

Reading a pedigree

A **pedigree** 系谱图 shows a trait across generations. For a simple Mendelian trait, assuming complete penetrance (everyone with the disease genotype shows it) and no new mutation, two useful tests are:

- **Two unaffected parents with an affected child**: the trait is **recessive**. Each parent must carry a hidden allele.
- **An affected mother with an unaffected son**: the trait cannot be X-linked recessive, since every son receives his only X from his mother.

Environmental Effects on Phenotype

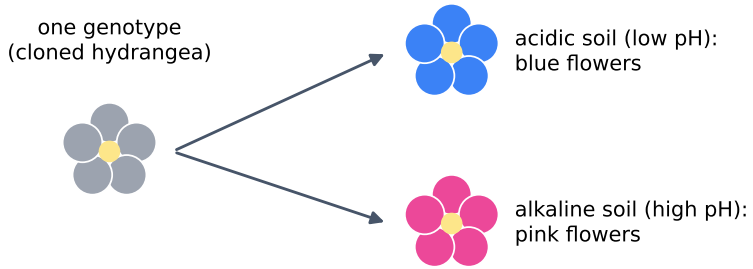
Phenotype is not set by genes alone – the **environment** also matters. Temperature, nutrition, and other factors can change how genes are expressed (a Himalayan rabbit's dark fur where it is cold, a plant's height with more sunlight). So identical genotypes can give different phenotypes in different conditions.

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The ability of one genotype to produce different phenotypes in different environments is called **phenotypic plasticity** 表型可塑性. Examples you may be asked about:

- **Height and weight** in humans depend on alleles at many genes, but also on nutrition and health in childhood.
- **Hydrangea flower colour**: the same plant has blue flowers in acidic soil (where aluminium ions are available to the pigment) and pink flowers in alkaline soil.
- **Seasonal fur colour** in arctic animals such as the arctic fox and snowshoe hare: shorter day length in autumn triggers changes in gene expression, so the new fur grows **white** for winter camouflage, and brown again in spring.
- **Sex determination by temperature** in many reptiles: in some turtles, eggs incubated at a warmer temperature develop as females and cooler eggs as males, because temperature changes the expression of genes for the enzymes that make sex hormones.
- **Mating type in yeast**: a haploid yeast cell switches on the genes for mating (and grows towards a partner) only when it detects the **pheromone** made by a cell of the **opposite** mating type nearby.
- **The Himalayan rabbit and Siamese cat**: an enzyme for dark pigment is denatured at normal body temperature, so only the cooler ears, nose, feet and tail are dark.

Worked example. Two genetically identical cuttings of a hydrangea are planted, one in soil of pH 5 and one in soil of pH 8. Their flowers are blue and pink. Explain. The plants have the same genotype, so the difference in phenotype must be caused by the **environment**: the soil pH changes the availability of aluminium ions, which bind to the flower pigment and change its colour. A question like this tests whether you can separate a genetic cause from an environmental one; the evidence is that the genotype is controlled (identical clones) while one factor of the environment is changed.



The same genotype gives different phenotypes in different environments: hydrangea flower colour depends on soil pH

Common mistakes.

- Thinking earlier children change the odds for the next one. Each fertilization is independent: if two carriers already have three unaffected children, the chance the fourth is affected is still $\frac{1}{4}$.
- Adding probabilities that should be multiplied. Use the **product rule** for "this **and** that" (independent events) and the **sum rule** for "this **or** that" (different ways to get the same result).
- Saying a son inherits an X-linked allele from his father. A son gets his only X from his **mother**; a father passes his X to every daughter and his Y to every son.
- Treating a recombination frequency of about 50% as tight linkage. Values near 50% mean the genes assort independently (far apart or on different chromosomes); small values mean they are close together.
- Writing that a chi-square result "proves" the hypothesis. If χ^2 is below the critical value you **fail to reject** the null hypothesis; the data are simply consistent with it.
- Using the wrong degrees of freedom. df = number of phenotype classes $-$ 1 (for example 3 for a 9:3:3:1 test).
- Saying sister chromatids separate in anaphase I. In anaphase I the **homologues** separate; sister chromatids separate in anaphase II.
- Saying nondisjunction in meiosis II makes all four gametes abnormal. Only the two gametes from the cell where it happened are affected.
- Predicting a mitochondrial trait from the father. Mitochondria come from the **egg**, so these traits follow the mother.
- Saying the environment changes an organism's genes. In these examples of phenotypic plasticity, the environment changes how the genes are **expressed** without changing the DNA sequence. Environmental mutagens can change DNA in other contexts.

Practice

Answer each before reading the answers below.

1. Give the expected genotype ratio from $Aa \times aa$.
2. In $AaBbCc \times AaBbCc$, with independent genes, what is the probability of an *aabbcc* offspring?
3. A man with haemophilia (X-linked recessive, X^hY) has children with a woman with two normal alleles (X^HX^H). Predict the sons and the daughters.
4. A cross expected to give a $1:1$ ratio produces 88 and 72 of 160 offspring. Find χ^2 and decide at $p = 0.05$ (critical value 3.84 for $df = 1$).
5. A student says: "Genes that are far apart on the same chromosome are inherited together most strongly." Correct the student.

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Answers

1. 1 Aa : 1 aa.
2. $\left(\frac{1}{4}\right)^3 = \frac{1}{64}$.
3. No son is affected: sons receive the father's Y. Every daughter is a carrier, $X^H X^h$: she receives the father's only X, which carries h.
4. Expected 80 and 80: $\chi^2 = \dfrac{8^2}{80} + \dfrac{8^2}{80} = 1.6$. Since $1.6 < 3.84$, the difference is not significant; the data are consistent with 1 : 1.
5. The reverse is true. Crossing over between distant genes is frequent, so they recombine often and can appear almost independent (recombination frequency near 50%). Genes close together rarely recombine and are the ones inherited together most strongly.

Exam tips

- Contrast mitosis (2 identical, full chromosome number) with **meiosis** (4 genetically varied haploid products, half the number; not all necessarily become functional gametes).
- Explain variation from **crossing over, independent assortment, and random fertilisation**.
- Use the multiplication rule for dihybrid crosses (each gene's 3:1 multiplied), and a **chi-square** test ($\chi^2 = \sum \frac{(o-e)^2}{e}$) to judge observed vs expected ratios.
- Keep **genotype** (the alleles) separate from **phenotype** (what you see) – AA and Aa can look the same.
- Recognise non-Mendelian patterns: incomplete dominance, codominance, and sex linkage.

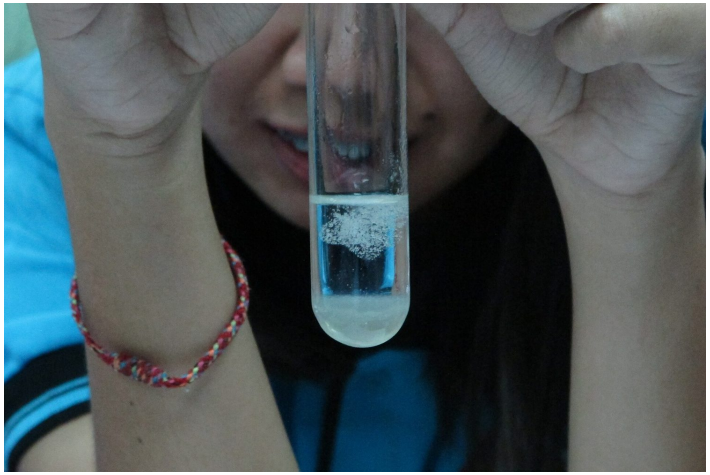
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Gene Expression and Regulation

AP Biology

DNA and RNA Structure

Your liver cells and your nerve cells carry exactly the same DNA, yet one makes bile and the other carries signals. This unit is about how a cell reads its DNA, and how it chooses **which** parts to read.



Spooling precipitated DNA from a cell extract – DNA is a real, visible molecule

DNA carries genetic information as a **double helix** 双螺旋 of two strands. Its **nucleotides** 核苷酸 pair by rule – A with T, G with C (**complementary base pairing** 互补配对) – so one strand specifies the other. The strands run **antiparallel** 反平行. **RNA** is single-stranded, uses **uracil** (U) instead of thymine, and has ribose sugar.

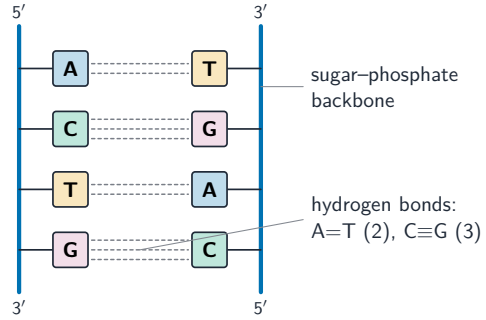
The bases come in two shapes. **Purines** 嘌呤 (adenine and guanine) have two rings; **pyrimidines** 嘧啶 (cytosine, thymine and uracil) have one ring. Every base pair is one purine with one pyrimidine, so the helix has the same width all along its length. This pairing rule is the same in every living thing.

> **Worked example (base percentages).** A sample of double-stranded DNA is 30% adenine. Find the other three bases.

1. A pairs with T, so T = 30%.
2. That leaves $100 - 60 = 40\%$ for G and C together.
3. G pairs with C, so G = C = 20%.

This only works for **double-stranded** DNA. In single-stranded RNA, A need not equal U.

RNA does three jobs, and its shape suits each one: **mRNA** carries the code from the nucleus to a ribosome, **tRNA** carries an amino acid and matches it to a codon, and **ribosomal RNA** 核糖体 RNA (rRNA) is a main building block of the ribosome itself.



the two strands run in opposite directions (antiparallel)

DNA: two antiparallel strands held by complementary base pairs

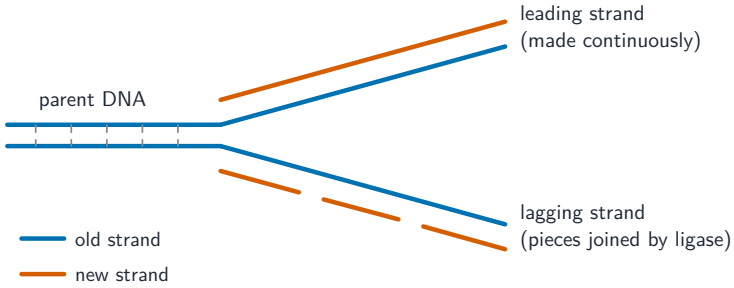
How the DNA is packaged differs between the domains, and the CED asks for both.

- **Prokaryotic** organisms typically have a **single circular chromosome** 环状染色体 in the cytoplasm, often with extra small circles called **plasmids** 质粒. Plasmids copy themselves and often carry useful genes, such as antibiotic resistance. Some eukaryotes, such as yeast, have plasmids too.
- **Eukaryotic** organisms typically have **multiple linear chromosomes** 线性染色体, and each is condensed by wrapping around proteins called **histones** 组蛋白. That packing is what fits two metres of DNA into a nucleus a few micrometres across – and because a tightly wound region is hard for the transcription machinery to reach, the packing is itself a level of gene regulation.

DNA Replication

Before a cell divides, DNA is copied **semiconservatively** 半保留复制: the helix unwinds and each old strand templates a new one, so each daughter helix has one old and one new strand. **DNA polymerase** 聚合酶 adds nucleotides following base-pairing rules, building the new strand and proofreading as it goes.

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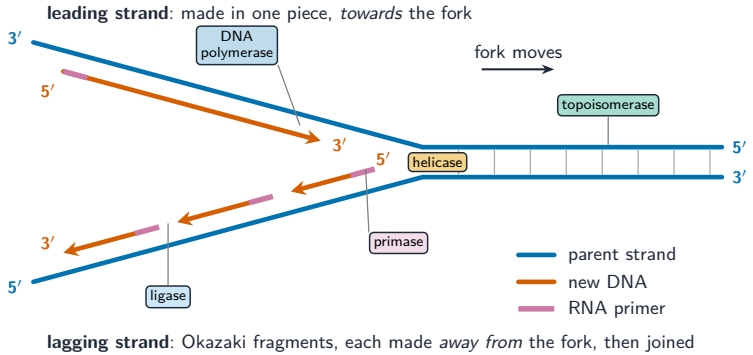


each new molecule = one old strand + one new strand (semi-conservative)

Semi-conservative DNA replication at a replication fork

The rule that explains the fork. The two strands of DNA are **antiparallel**: one runs 5′ → 3′, the other 3′ → 5′. DNA polymerase can add nucleotides only to the **3′ end** of a growing strand, so every new strand grows 5′ → 3′. Each enzyme at the fork has one job:

- **helicase** 解旋酶 unwinds the double helix and separates the strands at the fork.
- **topoisomerase** 拓扑异构酶 works ahead of the fork, relieving the twisting that unwinding causes.
- **primase** 引物酶 lays down a short **RNA primer** RNA 引物, because DNA polymerase cannot start a new strand on its own; it can only extend one.
- **DNA polymerase** adds nucleotides to the 3′ end. RNA primers are later removed, and DNA polymerase fills the gaps with DNA.
- **ligase** 连接酶 joins the separate pieces of DNA into one strand.

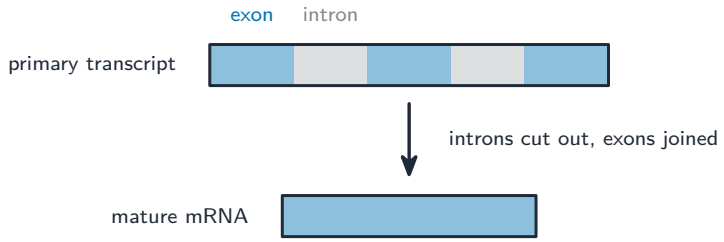


Because new DNA can only grow 5′ → 3′, one strand is made continuously and the other in short pieces

The **leading strand** 前导链 grows towards the fork, in the same direction as the fork moves, so it is made in one piece. The **lagging strand** 后随链 must grow away from the fork, so it is made in short **Okazaki fragments** 冈崎片段. Each needs its own primer, and ligase joins them.

Transcription and RNA Processing

Transcription 转录 copies a gene's DNA into **messenger RNA** 信使核糖核酸 (mRNA). **RNA polymerase** RNA 聚合酶 reads the **template strand** of DNA $3' \to 5'$ and builds a complementary RNA $5' \to 3'$, the same direction rule as DNA polymerase. The other DNA strand, the **coding strand**, has the same sequence as the mRNA, except with T for U. RNA polymerase needs no primer. In eukaryotes the mRNA is then **processed**: a cap and tail are added, and **introns** 内含子 are removed by splicing, leaving the **exons** 外显子 in the mature transcript. Some retained exon regions are untranslated, so exon does not mean only protein-coding sequence.



Introns are removed and exons joined to make mature mRNA

The **5′ cap** added during processing is a modified guanine nucleotide – a **7-methylguanosine cap** – and its job is recognition: it **helps the ribosome recognise and bind the mRNA**, as well as protecting the transcript from degradation as it leaves the nucleus. The **poly-A tail** 多聚腺苷酸尾, a long run of adenine nucleotides at the 3′ end, makes the mRNA more stable. An mRNA that lasts longer can be translated more times, so it makes more protein.

One gene, many proteins. A cell can keep different sets of exons from the same primary transcript. This **alternative splicing** 可变剪接 gives different mature mRNAs, and so different proteins, from one gene. It is one reason humans make far more kinds of protein than they have genes.

> **Worked example (spliced length).** A primary transcript is 15 kb long, but the mature mRNA is 7 kb.

1. Ignoring the small length added by the cap and tail, about 8 kb of introns were removed by splicing. (Cap and tail are added, so they cannot explain a loss.)

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2. If the full gene, introns included, is put into a bacterium, the mRNA stays about 15 kb long. Bacteria generally cannot splice out these eukaryotic introns, so they stay in.
3. The protein is then usually wrong: the intron sequences are translated too, often adding stop codons. This is why genetic engineers put a copy **without** introns into bacteria, made from the mature mRNA.

Translation

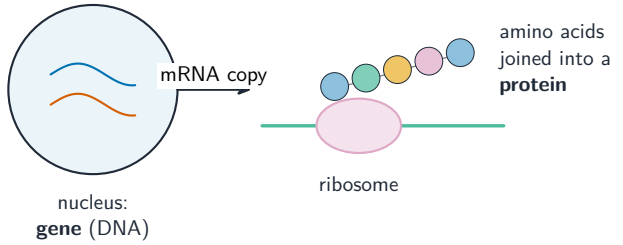
Translation 翻译 builds a protein from the mRNA at the **ribosome** 核糖体. The mRNA is read in three-base **codons** 密码子, each specifying an amino acid or a stop signal (the genetic code). **Transfer RNA** 转运核糖核酸 brings the matching amino acid, and the ribosome links them into a polypeptide until a stop codon ends it. This is the "central dogma": DNA → RNA → protein.

Where it happens. Ribosomes are free in the cytoplasm of all cells, and also on the surface of the rough ER in eukaryotes. A prokaryote has no nucleus, so ribosomes start translating an mRNA **while it is still being transcribed**. In eukaryotes, transcription (in the nucleus) and translation (in the cytoplasm) are kept apart.

One code for all life. Nearly all living things use the same genetic code: the same codon gives the same amino acid in bacteria, plants and humans. This shared code is evidence that all life has a **common ancestor**. It is also why a human gene put into a bacterium can make a human protein, such as insulin.

Several codons, one amino acid. There are 64 codons but only 20 amino acids, so most amino acids have more than one codon. The tRNAs for some of these codons are rarer than others. A ribosome waits longer at a codon whose tRNA is rare, so that codon is translated more slowly.

The exception: retroviruses. In a **retrovirus** 逆转录病毒 such as HIV, the genes are stored as RNA. The enzyme **reverse transcriptase** 逆转录酶 copies the viral RNA into DNA, which is then put into the host's chromosome. Information flows RNA → DNA, the reverse of the usual direction.

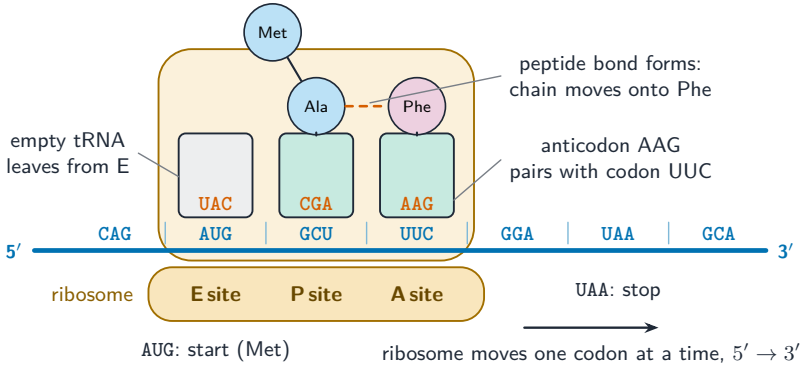


mRNA is read by a ribosome to build a protein from amino acids

Inside the ribosome. The ribosome has three sites for tRNA: **A** (where a new tRNA

arrives), **P** (holding the tRNA with the growing chain) and **E** (exit).

1. **Initiation**: the small subunit binds the mRNA and finds the **start codon** 起始密码子 AUG. A tRNA carrying methionine pairs with it, and the large subunit joins.
2. **Elongation**: a tRNA whose **anticodon** 反密码子 is complementary to the next codon enters the A site. A **peptide bond** 肽键 joins the chain in the P site to the new amino acid. The ribosome then moves one codon towards the 3′ end, and the empty tRNA leaves from the E site.
3. **Termination**: a **stop codon** 终止密码子 (UAA, UAG or UGA) reaches the A site. No tRNA matches it, so a release factor frees the finished polypeptide.



Each codon is matched by a tRNA anticodon; the chain passes from the P-site tRNA to the amino acid in the A site

Worked example (DNA to protein). The template strand of a gene reads 3′-TAC CGA AAG ATT-5′.

1. The mRNA is complementary to the template strand, with U instead of T: 5′-AUG GCU UUC UAA-3′.
2. Read the codons from the start codon, using the genetic code table: AUG = Met, GCU = Ala, UUC = Phe, UAA = stop.
3. The polypeptide is Met–Ala–Phe. The stop codon adds no amino acid.

The tRNA anticodons are 3′-UAC-5′, 3′-CGA-5′ and 3′-AAG-5′. They have the same sequence as the template DNA, but with U for T.

Worked example (protein length). Protein 1 is coded by 234 nucleotides and protein 2 by 495. Assume all of them code for amino acids.

1. Each amino acid needs one codon of 3 nucleotides.
2. Protein 1: $234 \div 3 = 78$ amino acids. Protein 2: $495 \div 3 = 165$ amino acids.
3. Protein 2 has $165 - 78 = \mathbf{87}$ more amino acids.

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Common mistakes.

- Saying DNA polymerase "reads 5′ → 3′". It **builds** the new strand 5′ → 3′, by reading the template 3′ → 5′.
- Saying the lagging strand is made "more slowly" or "backwards". It is made in the same 5′ → 3′ direction, but in pieces, because it runs away from the fork.
- Using T in mRNA, or reading the genetic code table with **anticodons**. The table is written in mRNA **codons**.
- Giving the stop codon an amino acid.
- Saying a ribosome makes the amino acids. It **joins** amino acids brought to it by tRNA.

Regulation of Gene Expression

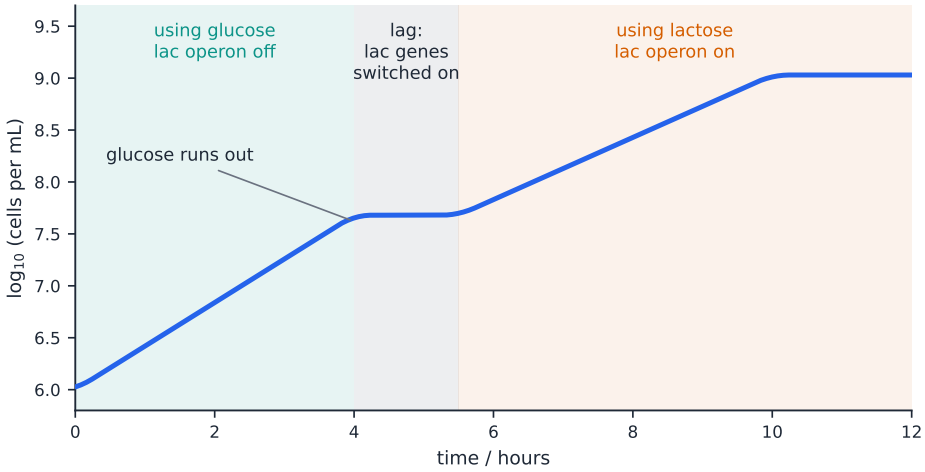
Cells control **which** genes are expressed and **when**. In prokaryotes, **operons** 操纵子 switch groups of genes on or off. In eukaryotes, regulation happens at many levels – which genes are transcribed (transcription factors, promoters, enhancers), RNA processing, and after translation. This lets a cell respond to its environment without changing its DNA.

Always on, or on when needed. Some genes are expressed all the time, in every cell, because every cell needs their products: they are **constitutive** 组成型 genes. A gene for a glycolysis enzyme such as GAPDH is one. Others are **inducible** 诱导型: they are switched on only when a signal is present. Which happens is controlled by **regulatory sequences**, stretches of DNA near the gene that regulatory proteins bind to.

Two kinds of operon. An operon is a group of genes under one **promoter** 启动子, so they are switched on and off together.

- The **lac operon** is **inducible**. Its genes break down lactose. A **repressor** 阻遏蛋白 protein sits on the operator and blocks RNA polymerase. Allolactose, made from lactose, binds the repressor and changes its shape so it leaves the operator, so the genes are transcribed only when lactose is present.
- The **trp operon** is **repressible** 阻遏型. Its genes make the amino acid tryptophan. The genes are normally on. When tryptophan is plentiful, it binds the repressor and activates it, so the repressor blocks transcription. The cell stops making what it already has.

When glucose is there too. Bacteria prefer glucose. While glucose is present, the lac operon stays mostly off even with lactose around. Given both sugars, the bacteria grow on glucose first, pause while the lac genes are switched on, then grow on lactose.



Given glucose and lactose, the bacteria use glucose first; the lag is the time taken to switch the lac operon on

Worked example (reading the graph). Which sugar do the bacteria prefer, and why is there a flat section?

1. The first rise happens while glucose is present, so glucose is the preferred sugar.
2. The flat section starts when glucose runs out. The cells have not yet made the enzymes for lactose, so they cannot grow.
3. Allolactose, made from lactose, inactivates the repressor; with glucose depleted, the lac genes are strongly transcribed and translated, and growth starts again on lactose.

Changes on top of the DNA. Epigenetic 表观遗传的 changes alter gene expression without changing the base sequence, and they can be reversed.

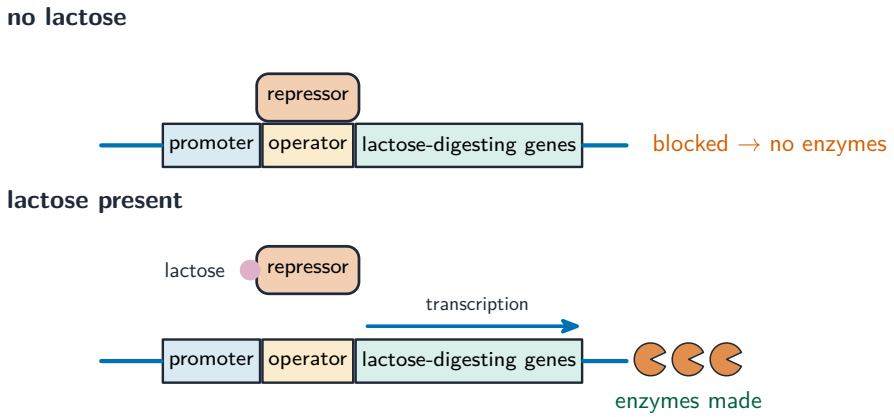
- **DNA methylation** DNA 甲基化, adding methyl groups to DNA, usually **switches genes off**.
- **Histone acetylation** 组蛋白乙酰化 loosens the DNA around the histones, so the transcription machinery can reach the genes and they are **switched on**. Removing the acetyl groups packs the DNA tightly again.

Many control points. In a eukaryote, a gene's output can be changed at every step: how tightly its DNA is packed, whether it is transcribed, how its mRNA is spliced, how long the mRNA lasts, how often it is translated, and whether the protein is modified afterwards. For example, adding a phosphate group can change a protein's shape and switch it on or off. So a tissue can contain plenty of one mRNA but little of its protein, if translation of that mRNA is blocked.

Genes switched together. Most eukaryotic genes are transcribed individually

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rather than in operons (some eukaryotes do have operons), but groups of genes can still be controlled together. Genes far apart in the genome can share the same regulatory sequence, so one transcription factor switches all of them on at once. A heat shock, for instance, switches on a whole set of heat-shock protein genes together.



The lac operon switches genes on only when lactose is present

Gene Expression and Cell Specialization

Every cell in a body has the **same** DNA, yet cells differ because they express **different** genes – **differential gene expression** 差异表达. This is how one fertilized egg produces many specialized cell types (muscle, nerve, skin); signals during development turn specific genes on and off.

How the switch works. RNA polymerase cannot start transcription on its own. Proteins called **transcription factors** 转录因子 bind to the promoter, and to **enhancers** 增强子 that can be far upstream or downstream of the gene. Together they recruit RNA polymerase. Each cell type makes its own mix of transcription factors, so each turns on its own genes. **Negative regulators**, such as repressors, bind the DNA and block transcription.

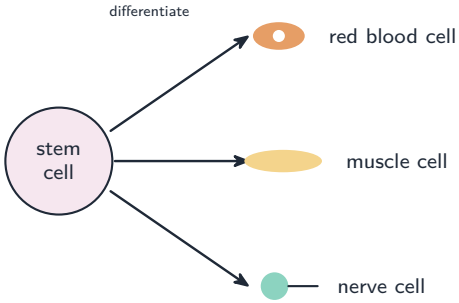
During development one transcription factor often switches on the gene for the next, so genes are expressed in a fixed order, a cascade. A cell is a muscle cell because the factors in it switch on muscle proteins, such as actin and myosin, and not the proteins of a nerve cell.

Small RNAs. Some short RNA molecules regulate genes after transcription. A **microRNA** 微小核糖核酸 or siRNA is loaded into a protein complex (one protein in it is called AGO2). The small RNA base-pairs with a matching mRNA, and the complex then cuts the mRNA or blocks its translation. Less protein is made, though

the gene is still transcribed.

> **Worked example (small-RNA data).** In normal cells the mean amount of gene H mRNA is 40 units. In cells with no working AGO2, it is 100 units.

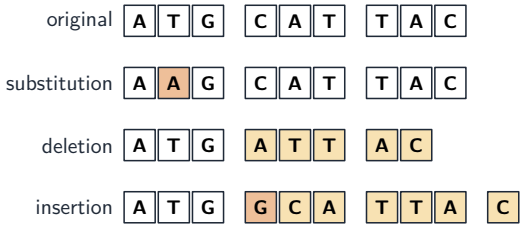
1. Percent increase $= (100 - 40) \div 40 \times 100 = \mathbf{150\%}$.
2. Without AGO2, the mRNA is no longer cut, so it builds up. This supports the claim that small RNAs normally lower gene H expression.
3. If gene G mRNA hardly changes when AGO2 is lost, small RNAs play a smaller part in regulating gene G.



A stem cell differentiates into specialised cell types

Mutations

A **mutation** 突变 is a change in the DNA sequence. **Point mutations** change one base (silent, missense, or nonsense); **insertions/deletions** can cause a **frameshift** 移码 that garbles everything downstream. Mutations in gametes are heritable; they may be harmful, neutral, or beneficial – and mutations supply the variation on which natural selection acts.



Substitution, deletion, and insertion mutations

Where mutations come from. Mutations arise when DNA polymerase makes a copying error that is not repaired, or from outside causes: radiation such as UV light and X-rays, and reactive chemicals (**mutagens** 诱变剂). They happen at random. They are not made **because** the organism needs them.

- A **silent** mutation changes a codon to another codon for the same amino acid, so the protein is unchanged.
- A **missense** 错义 mutation changes one amino acid. Its effect depends on where: in an enzyme's active site it can destroy the enzyme, elsewhere it may change little.
- A **nonsense** 无义 mutation creates an early stop codon, so the protein is cut short.
- A **frameshift** after an insertion or deletion shifts the reading frame of the codons after it, and usually makes a useless protein. An insertion or deletion of **three** bases keeps the reading frame.

Whether a mutation helps or harms depends on the **environment**. A mutation in the MC1R gene gives dark fur: harmful on pale sand, but it hides pocket mice living on dark lava rock.

> **Worked example (codon chart).** A mutation changes amino acid 229 from threonine to alanine. Threonine's codons are ACU, ACC, ACA and ACG; alanine's are GCU, GCC, GCA and GCG.

1. The codons differ only in the **first** base: A → G.
2. So a single-base substitution (a missense point mutation) is the likely cause, for example ACU → GCU.
3. In the template DNA the matching change is T → C.

Whole-chromosome errors change the phenotype too. Mutation is not only a change of base: **errors in mitosis or meiosis can result in changes in phenotype**. If chromosomes fail to separate – **nondisjunction** 不分离 – a gamete receives an extra copy or none, and the resulting zygote has an abnormal chromosome number (**aneuploidy** 非整倍性). Trisomy 21 is the familiar example. Because a whole chromosome carries hundreds of genes, the phenotypic effect is far broader than a point mutation's. Parts of a chromosome can also be lost, copied twice, turned around or moved to another chromosome; these changes in **structure** also cause genetic disorders.

Bacteria swap genes sideways. Prokaryotes do not have meiosis, but they gain new genes by **horizontal gene transfer** 水平基因转移, from cells that are not their parents.

- **Transformation** 转化: a cell takes up DNA from its surroundings, for example from a dead cell.
- **Transduction** 转导: a virus carries DNA from one bacterium to another.

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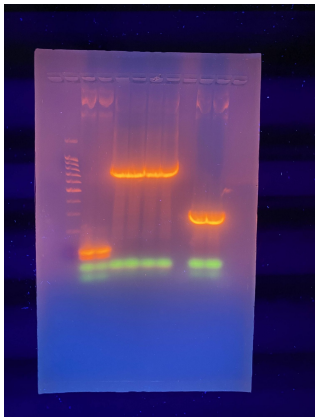
- **Conjugation** 接合: one cell passes DNA, often a plasmid, to another through direct contact. **Transposition** is a different process: a **transposon** 转座子 ("jumping gene") moves from one place in the DNA to another. It does not by itself transfer DNA between cells.

This is how a gene for antibiotic resistance spreads quickly through a population of bacteria, even between species. Viruses add variation too: if two related viruses infect the same cell, their genetic material can mix. In flu, whole RNA genome segments can be exchanged by reassortment, producing new combinations.

Biotechnology



A thermal cycler repeatedly heats and cools samples to amplify DNA (PCR)



Agarose gel under UV light: DNA fragments separate by size as bright bands

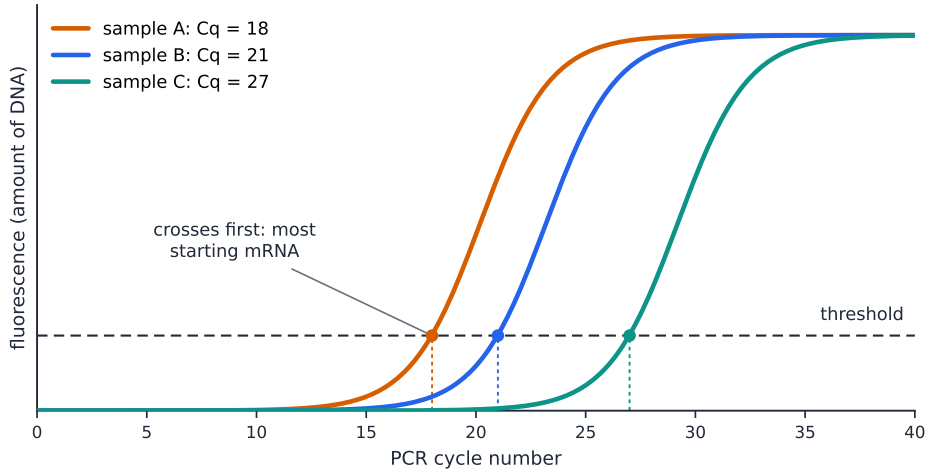
Biotechnology 生物技术 tools manipulate genetic material: **PCR** 聚合酶链反应 copies DNA, **gel electrophoresis** 凝胶电泳 separates DNA fragments by size, **restriction enzymes** and cloning move genes between organisms, and **CRISPR** edits sequences. These techniques enable genetic testing, engineered organisms, and medical treatments.

PCR in three steps, repeated about 30 times.

1. **Denature** at about 95 °C: the two DNA strands separate.
2. **Anneal** at about 55 °C: short DNA primers bind to each end of the target region.
3. **Extend** at about 72 °C: a heat-stable DNA polymerase builds new strands from the primers.

With ideal amplification, each cycle doubles the target, so after n cycles one copy has become 2^n copies.

Measuring gene expression: qPCR. To measure how much of one mRNA a tissue makes, scientists copy its mRNA into DNA and run PCR while a dye makes the DNA glow. The **Cq** is the cycle at which the glow crosses a threshold. A sample with more starting mRNA reaches the threshold sooner, so a **lower Cq means more mRNA**.



The earlier a curve crosses the threshold, the more starting mRNA the sample had

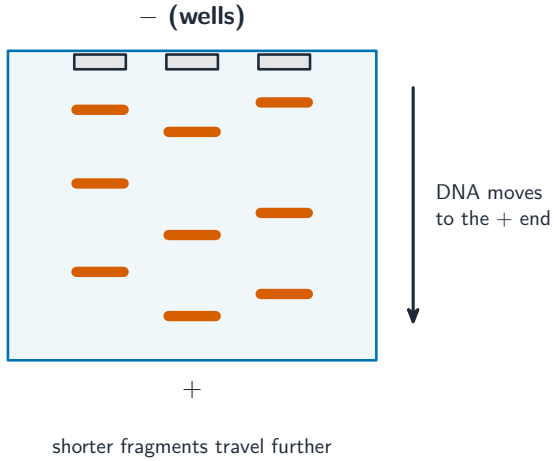
> **Worked example (comparing Cq).** Sample A has Cq = 18 and sample B has Cq = 21. How much more starting mRNA is in A? Assume equal sample input and reverse-transcription efficiency, and ideal doubling in each PCR cycle.

1. A reaches the threshold $21 - 18 = 3$ cycles earlier.
2. Each cycle doubles the DNA, so A started with $2^3 = 8$ times as much.
3. Sample C (Cq = 27) has the least: $2^9 = 512$ times less than A.

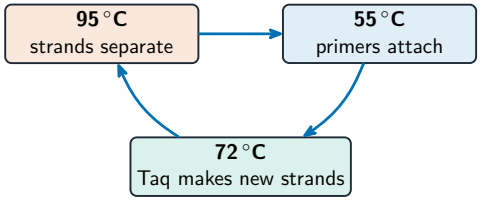
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Other tools.

- **Bacterial transformation**: a gene is put into a plasmid, and bacteria take up the plasmid. The plasmid also carries a gene for antibiotic resistance, so only bacteria that took it up grow on a plate containing the antibiotic. The bacteria then copy the gene each time they divide (gene **cloning** 克隆) and can make its protein.
- **DNA sequencing** DNA 测序 reads the order of bases. Comparing sequences shows how closely species are related, and identifies a species from a small sample. Environmental DNA (eDNA) from water can show a fish lives in a lake without anyone catching it, because primers that match only that species' DNA give a PCR product only if its DNA is there.
- A **genetically modified organism** 转基因生物 has DNA altered using genetic engineering. A transgenic organism carries introduced DNA from another species, but an organism edited without adding foreign DNA can also be genetically modified.



Gel electrophoresis separates DNA fragments by length



The polymerase chain reaction doubles the DNA each cycle

> **Worked example.** A template DNA strand `3'-TAC GGA TTC-5'` is transcribed into mRNA `5'-AUG CCU AAG-3'`. A ribosome reads three codons: AUG = Met (start), CCU = Pro, AAG = Lys – coding for **Met–Pro–Lys**. Changing the third base of a codon often gives the same amino acid, because the genetic code is **degenerate**, which softens the effect of many mutations.

Reading DNA identifies individuals. Because the number of repeats at certain non-coding loci varies from person to person, **analysis of DNA can be used for forensic identification**: amplify those regions by PCR, separate the fragments by gel electrophoresis, and compare the pattern of band positions with a reference sample. A match is a statement about probability rather than certainty, which is why several loci are tested rather than one.

More common mistakes.

- Saying a **higher** Cq means more mRNA. It means **less**: the sample needed more cycles to reach the threshold.
- Saying gene expression is measured by the amount of DNA. Every cell has the same DNA; expression is the amount of **mRNA** or **protein**.
- Assuming more mRNA always means more protein. Small RNAs, mRNA stability and translation control all come between them.
- Saying mutations happen "to help the organism adapt". They are random; selection then acts on them.
- Mixing up inducible and repressible operons. Lactose switches the lac genes **on**; tryptophan switches the trp genes **off**.

Practice

Answer each before reading the answers below.

1. A DNA template strand reads 3′-TAC GGA TTT-5′. Give the mRNA and the amino-acid sequence (AUG = Met, CCU = Pro, AAA = Lys).
2. Explain why one new DNA strand is made continuously and the other in Okazaki fragments.

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3. A substitution changes the codon CAG (Gln) to UAG (stop). Name the type of mutation and predict its effect on the protein.
4. Describe the state of the *lac* operon when lactose is absent and when it is present.
5. Name three ways eukaryotic pre-mRNA is processed, and explain how one gene can code for more than one protein.
6. In gel electrophoresis, which fragments travel farthest, and why does DNA move toward the positive electrode?
7. A liver cell and a nerve cell have the same DNA but different proteins. Explain.

Answers

1. mRNA 5′-AUG CCU AAA-3′: Met-Pro-Lys.
2. DNA polymerase adds nucleotides only to the 3′ end. One strand (leading) runs in the direction the fork opens; the other (lagging) is made in short pieces in the opposite direction, later joined by ligase.
3. A nonsense mutation: translation stops early, giving a shortened protein that is usually non-functional.
4. Absent: the repressor binds the operator and blocks RNA polymerase, so the genes are not transcribed. Present: allolactose binds the repressor, which changes shape and leaves the operator, so the genes are transcribed.
5. A 5′ cap, a poly-A tail, and splicing to remove introns. Alternative splicing joins different exons, giving different mRNAs and proteins.
6. The smallest fragments move fastest through the gel. DNA is negatively charged (phosphate groups), so it moves toward the positive electrode.
7. Different genes are expressed: different transcription factors switch on different sets of genes in each cell type.

Exam tips

- Know the **central dogma** DNA → RNA → protein: transcription makes mRNA, translation reads it in three-base **codons** at the ribosome.
- Remember RNA is single-stranded and uses **U** instead of T; replication is **semi-conservative**.
- Every cell has the **same DNA** – cells differ because they express **different genes** (differential gene expression).
- A **mutation** is a change in the DNA sequence and may be harmful, neutral, or beneficial (the raw material for selection).
- Link biotech tools to their jobs: PCR copies DNA; gel electrophoresis separates fragments by size.
- In data questions, name the **direction** of an effect and back it with numbers from the graph or table, such as a percent change or a Cq difference.

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Natural Selection

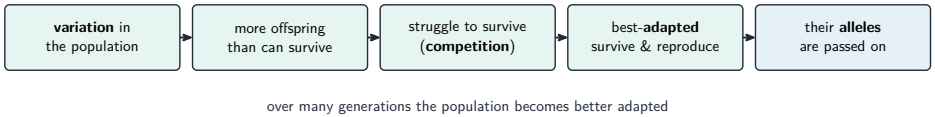
AP Biology

Introduction to Natural Selection

In 1977 a drought hit the Galápagos island of Daphne Major. Small, soft seeds ran out, and finches with deeper beaks were better able to crack the large, hard seeds that were left. Most finches died. The next generation had beaks about 4% deeper. Scientists watched evolution happen in a single year.

Evolution 进化 is a change in the heritable traits of a population over generations. **Natural selection** 自然选择 is its main driver: individuals vary, some variations are heritable, more offspring are produced than survive, and those with traits better suited to the environment survive and reproduce more. Over time, helpful traits become more common.

Darwin's key point was **competition** 竞争: there are never enough resources for every individual, so some survive and reproduce better than others. This is **differential survival and reproduction**. And because the biotic and abiotic environment changes from year to year, which variation is favoured can change too. After a wet year in which soft seeds were plentiful, small-beaked finches did better again.



Natural selection: the best-adapted survive, reproduce, and pass on their alleles

Natural Selection

Selection acts on **variation** 变异. **Fitness** 适合度 means reproductive success, not strength. Selection comes in modes: **directional** (favors one extreme), **stabilizing** (favors the average), and **disruptive** (favors both extremes). The environment does the "selecting," so a trait that helps in one setting may not in another. Well-suited traits are **adaptations** 适应.



Peppered moth dark form (carbonaria) – industrial melanism is a classic natural-selection case study

The same allele can help or harm. The sickle-cell allele causes sickle-cell disease in people with two copies. But people with one copy are protected against malaria, so in places where malaria is common the allele stays frequent. Likewise, the alleles that made insects resistant to DDT spread only where DDT was sprayed.

Variation in molecules matters too. A cell's set of molecules can vary, and that variation can affect fitness. A plant with several forms of an enzyme, each working best at a different temperature, can survive in a wider range of climates. A population of mussels in salty water has more of one form of an enzyme that helps cells balance salt.

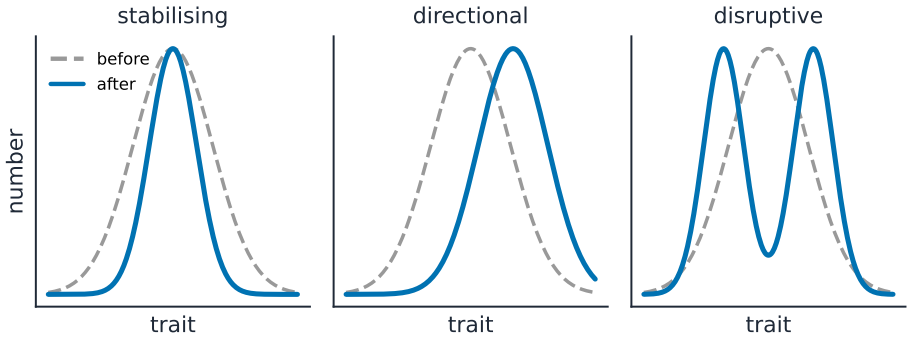
Choosing a mate. Sexual selection 性选择 acts on traits that help an individual win a mate, even when they make it easier for predators to see. Male guppies with more coloured spots attract more females. Where there are no predators, the number of spots rises over the generations; where a predator hunts by sight, spots become fewer, because brightly spotted males are eaten first.

> **Worked example (selection from data).** On an island, the mean toe-pad area of anole lizards rose after strong hurricanes.

1. **Variation**: the lizards' toe pads differed in size before the storm, and the difference is partly heritable.
2. **Selection**: lizards with larger toe pads could cling to branches in high winds, so more of them survived.
3. **Inheritance**: the survivors bred, so the next generation had larger toe pads on average.

This is **directional** selection. Islands hit by more storms experience stronger selection for larger pads.

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Stabilising, directional, and disruptive selection

Artificial Selection

In **artificial selection** 人工选择, humans – not nature – choose which individuals breed, selecting for desired traits (crops, livestock, dogs). It works the same way as natural selection and, being fast and visible, is strong evidence that selection can reshape populations.

From one species of wild mustard, farmers have bred cabbage (selected for leaves), broccoli (flower buds), kohlrabi (stem) and kale. Artificial selection usually **lowers** genetic diversity, because only a few chosen individuals breed. A crop field of near-identical plants can be wiped out by one new disease.



Selective breeding makes a wanted feature more common over generations

Population Genetics

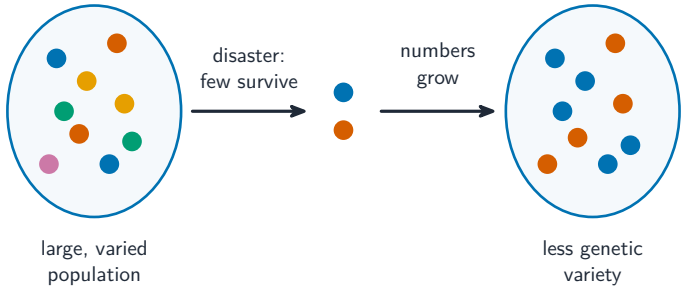
Population genetics 群体遗传学 studies the pool of alleles in a population. Evolution is a change in **allele frequencies** 等位基因频率 over time. Besides natural selection, allele frequencies change through **mutation** (new alleles), **gene flow** 基因流 (migration), and **genetic drift** 遗传漂变 (random change, strongest in small populations – the bottleneck and founder effects).

- In the **bottleneck effect** 瓶颈效应, a disaster cuts a population to a few survivors **for at least one generation**. The survivors' alleles are a random sample, not the best-adapted ones, and many alleles are lost.
- In the **founder effect** 奠基者效应, a few individuals start a new population, for example on an island. The new population has only the founders' alleles, so its allele frequencies can be very different from the original population's.

Selection is not random; drift is. Drift changes allele frequencies by chance, whatever the alleles do. It can even make a harmful allele common in a small population. Drift can make a small population differ from other populations of its species. **Gene flow** works the other way: moving alleles between populations tends to keep them alike and can slow their divergence into separate species.

> **Worked example (genetic rescue).** A small, isolated population of snakes declined: few young survived, and many had defects. After males from a large population nearby were released into it, survival rose.

1. The small population had lost genetic variation by drift and inbreeding, so many offspring were homozygous for harmful recessive alleles.
2. The new males brought new alleles (gene flow).
3. Offspring were then more often heterozygous, so the harmful recessive alleles were masked, and more young survived.



A population bottleneck reduces genetic variety

Hardy-Weinberg Equilibrium

The **Hardy–Weinberg** 哈迪-温伯格 model gives the allele and genotype frequencies expected when a population is **not** evolving. With allele frequencies p and q ($p+q=1$):

$$p^2 + 2pq + q^2 = 1,$$

where p^2 and q^2 are the homozygotes and $2pq$ the heterozygotes. It holds only under five conditions:

1. a large population (so no genetic drift),
2. no migration (no gene flow),
3. no new mutations,
4. random mating,
5. no natural selection.

Real populations never meet all five. That is why the model is useful: it is a **null hypothesis** 零假设, the "nothing is happening" prediction. A statistically supported departure shows that at least one model assumption is not met. It does not by itself prove that allele frequencies are changing: non-random mating can alter genotype frequencies without changing allele frequencies.

> **Worked example.** In a population in Hardy–Weinberg equilibrium, 16% of individuals show the recessive phenotype, so $q^2 = 0.16$ and $q = \sqrt{0.16} = 0.4$. Then $p = 1 - q = 0.6$. The predicted **carrier** frequency (heterozygotes) is $2pq = 2(0.6)(0.4) = 0.48$, i.e. 48%, and the homozygous dominants are $p^2 = 0.6^2 = 0.36$, i.e. 36%. As a check, $0.36 + 0.48 + 0.16 = 1$. This is the standard route: recessive phenotype $\to q^2 \to q \to p \to$ everything else.

> **Worked example (testing for equilibrium).** In a sample of 200 plants, 90 are AA, 40 are Aa and 70 are aa.

1. Count alleles: there are 400 in total. A alleles: $2(90)+40 = 220$, so $p = 220/400 = 0.55$ and $q = 0.45$.
2. Expected counts if in equilibrium: $AA = 0.55^2 \times 200 = 60.5$, $Aa = 2(0.55)(0.45) \times 200 = 99$, $aa = 0.45^2 \times 200 = 40.5$. Expected counts may be fractional and sum to 200.
3. There are far fewer heterozygotes (40) than expected (99), so the population is **not** in equilibrium. Non-random mating, such as self-pollination, is one likely cause. A chi-square test (unit 5) would show whether the gap is too big to be chance.

When the genotypes are given as counts, always calculate p by counting alleles. Do not take the square root of a homozygote frequency unless you are told the population is in equilibrium.

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$$p + q = 1$$
$$p^2 + 2pq + q^2 = 1$$

p	frequency of allele *A*
q	frequency of allele *a*
p^2	frequency of genotype *AA*
$2pq$	frequency of genotype *Aa*
q^2	frequency of genotype *aa*

H–W equilibrium: no selection, mutation, migration, drift, or non-random mating

Hardy–Weinberg: allele frequencies p and q give genotype frequencies p^2, $2pq$, q^2

Evidence of Evolution



Archaeopteryx fossil: feathers plus reptilian features – evidence of common ancestry

Multiple independent lines support evolution: the **fossil record** 化石记录, **homologous structures** 同源结构 (shared anatomy from common ancestry), **vestigial structures** 痕迹器官, shared **embryology**, and molecular evidence – the near-universal genetic code and matching DNA/protein sequences. The evidence comes from many sciences: geography, geology, physics, biochemistry and mathematics.

Dating fossils. There are three main ways:

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- **Rock layers.** Lower layers of sedimentary rock are older, so a fossil's layer gives its age relative to other fossils.
- **Radioactive decay.** Isotopes decay at a fixed rate. Carbon-14 has a half-life of about 5730 years, so it dates remains up to about 50 000 years old; isotopes that decay more slowly date older rocks.
- **Geographical data**, such as where continents were when the rock formed.

> **Worked example (carbon dating).** A bone has 25% of the carbon-14 that living tissue has.

1. 100% → 50% is one half-life; 50% → 25% is a second.
2. Age ≈ 2 × 5730 ≈ **11 500 years**.

Comparing molecules. The more alike two species' DNA or protein sequences are, the more recently they shared an ancestor. Humans and chimpanzees differ in about 1% of their DNA; humans and mice differ much more. Morphological homologies, including vestigial structures such as the tiny hip bones of whales, point the same way.

Common Ancestry

All life shares a **common ancestor** 共同祖先, shown by universal features: the same DNA/RNA machinery, the same genetic code, ribosomes, and core metabolic pathways in all organisms. The more features and sequences two species share, the more recently they diverged.

What all eukaryotes share. Comparative evidence indicates that the common ancestor of living eukaryotes had these typical features:

- **membrane-bound organelles**, such as the nucleus and mitochondria;
- **linear chromosomes**;
- **genes that contain introns**.

Linear chromosomes and introns are not exclusive to eukaryotes: some prokaryotes have them too.

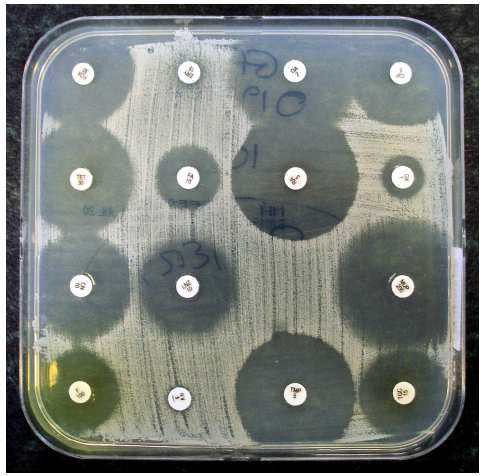
> **Worked example (glycolysis).** Use these facts to argue that glycolysis first appeared in the common ancestor of all living things: (a) nearly all organisms use it; (b) it happens in the cytoplasm; (c) it does not need oxygen.

1. (a) A pathway found in almost all living things was most likely inherited from one ancestor, not invented many times.
2. (b) It needs no membrane-bound organelle, so it could run in the earliest cells, which had none.
3. (c) The early atmosphere had almost no oxygen, so the first cells needed a way of making ATP without it.



Archaeopteryx: transitional fossils link major groups and support common ancestry

Continuing Evolution



Antibiotic sensitivity plate: clear zones show which antibiotics kill the bacteria

Evolution is ongoing and observable: antibiotic-resistant bacteria, pesticide-resistant insects, and rapid changes in fast-breeding species. Because environments keep changing, selection keeps acting – evolution has no endpoint.

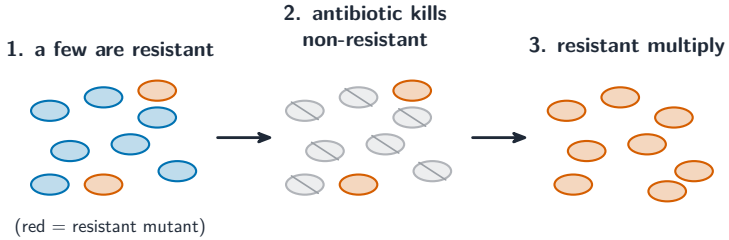
The CED gives four kinds of evidence that evolution is still going on:

1. **Genomes change over time**; we can measure new mutations and changed allele frequencies from one generation to the next.

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2. The **fossil record** shows continuous change.
3. **Resistance evolves** to antibiotics, pesticides, herbicides and cancer chemotherapy drugs.
4. **Pathogens evolve** and cause new diseases, such as new strains of flu each year.

Why chemotherapy can fail. A tumour contains many genetically different cells. A drug kills most of them, but any cell that happens to resist it survives and divides. The tumour grows back, now made of resistant cells. This is natural selection inside one body.



How antibiotic resistance spreads through a population by natural selection

Phylogeny

A **phylogenetic tree** 系统发育树 (cladogram) diagrams evolutionary relationships, with branch points marking common ancestors and shared derived traits grouping related species. Trees are hypotheses, revised as new (especially molecular) data arrive.

Tree or cladogram? A **phylogenetic tree** shows evolutionary relationships. Its branch lengths may represent genetic change or time, depending on the scale; a time-calibrated tree uses fossils or a **molecular clock** 分子钟. A **cladogram** 分支图 shows only the order of branching, with no time scale.

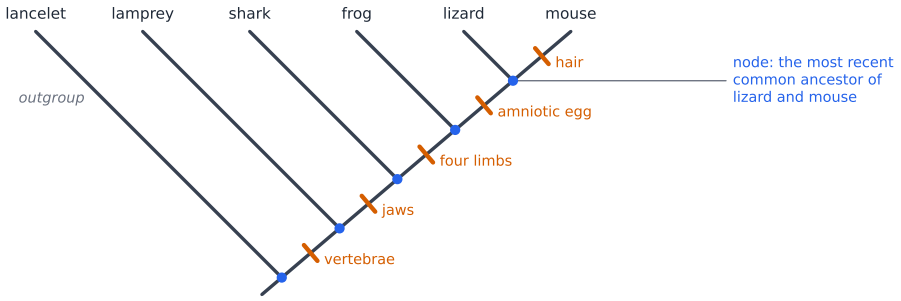
Reading one.

- Each **node** is the **most recent common ancestor** of everything above it.
- Two groups are most closely related if they share the most recent node. Do not read relatedness from how close the tips are.
- The **outgroup** 外类群 is a lineage known from independent evidence to lie outside the group being studied. Comparing it with the group helps root the tree and identify ancestral characters.

Building one from a table. Use **shared derived characters** 共有衍征: new characters found in some groups and their descendants, but not in the outgroup.

	vertebrae	jaws	four limbs	amniotic egg	hair
lancelet	–	–	–	–	–
lamprey	✓	–	–	–	–
shark	✓	✓	–	–	–
frog	✓	✓	✓	–	–
lizard	✓	✓	✓	✓	–
mouse	✓	✓	✓	✓	✓

1. The lancelet has none of the characters, so it is the outgroup and branches off first.
2. For this nested table, group animals by their shared derived characters: hair groups the mouse within amniotes, and four limbs group the amniotes with the frog. Counting characters alone is not a general rule for building trees.
3. Mark each character **once**, on the branch where it first appeared. Every group above the mark has it.



Each character is marked once, where it first appeared; every lineage above the mark has it

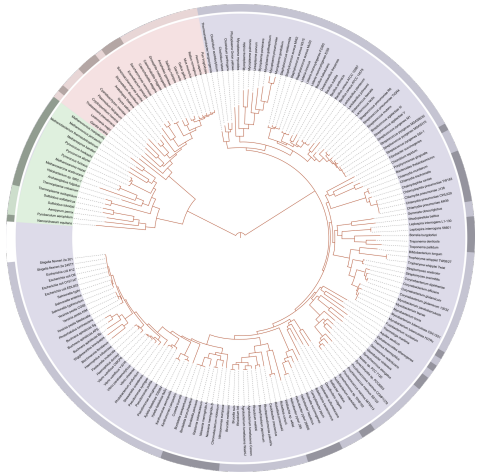
Molecules beat looks. Molecular data, such as DNA or amino-acid sequences, usually give more accurate trees than morphology. Unrelated species can look alike because they live in similar ways (convergent evolution), but they rarely share long stretches of identical DNA by chance. For a simple example with comparable rates of change, the two species with the **fewest differences** are likely to be closest relatives. Rates can differ among lineages, so the most different sequence does not by itself identify the outgroup.

> **Worked example (molecular clock).** Humans and chimpanzees differ at 12 positions in a gene, and fossils show they split about 6 million years ago. Humans and gorillas differ at 18 positions.

1. Rate = 12 differences ÷ 6 million years = 2 differences per million years.
2. Humans and gorillas: 18 ÷ 2 = **9 million years** ago.

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3. This assumes the gene changes at a steady rate in every lineage.



The tree of life: phylogeny groups organisms by shared ancestry from a common origin

Speciation

Speciation 物种形成 is the origin of new species, usually when populations become **reproductively isolated** 生殖隔离 and diverge. **Allopatric** speciation follows a geographic split; **sympatric** speciation happens without one. Once populations can no longer interbreed, they are separate species.

The **biological species concept** 生物学物种概念 defines a species as a group whose members can interbreed and produce **fertile, viable offspring**, but cannot do so with other groups. It does not work for organisms that reproduce asexually, or for fossils.

Barriers that keep species apart. Prezygotic 合子前隔离 barriers stop a zygote forming at all:

- **habitat**: they live in different places, such as in water and on land;
- **temporal**: they breed at different times of day or year;
- **behavioural**: different courtship songs or displays;
- **mechanical**: their reproductive parts do not fit;
- **gametic**: sperm and egg cannot fuse.

Postzygotic 合子后隔离 barriers act after a hybrid zygote has formed: the hybrid dies young, or it is **sterile** (a mule, from a horse and a donkey), or its own offspring are weak.

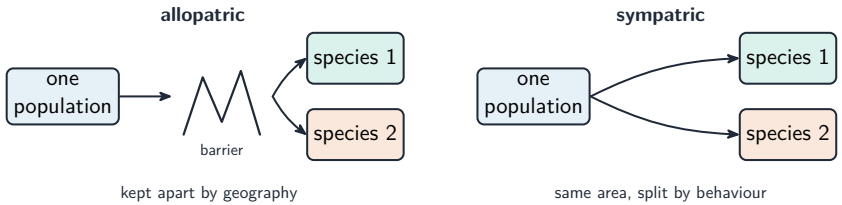
How fast? In **gradualism** 渐变论, species change slowly and steadily over hundreds of thousands or millions of years. In **punctuated equilibrium** 间断平衡, long periods with little change are broken by short bursts of rapid change. Many fossil records show

the second pattern.

Spreading out and converging.

- **Divergent evolution** 趋异进化: related populations adapt to different habitats and become more and more different.
- **Adaptive radiation** 适应辐射: one ancestor gives rise to many species quickly, when new habitats open up. The Hawaiian *Drosophila* flies and Darwin's finches arose this way.
- **Convergent evolution** 趋同进化: unrelated species face similar selective pressures and evolve similar traits, such as the streamlined bodies of sharks and dolphins. These are **analogous** traits, not evidence of common ancestry.

Speciation without a barrier. The apple maggot fly *Rhagoletis* once laid its eggs only on hawthorn. Some flies began to use apple trees, which fruit at a different time. Flies mate on the fruit where they hatched, so the two groups now breed apart and are splitting into two species while living side by side. This is sympatric speciation. In plants, a mistake in meiosis can make a polyploid offspring that cannot breed with its parents: a new species in one generation.



Allopatric and sympatric speciation

Variations in Populations

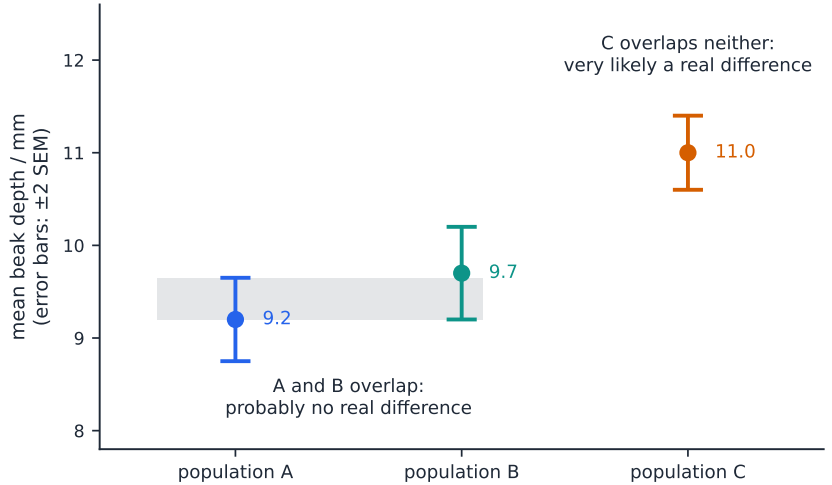
Genetic **diversity** 多样性 helps a population survive change – if conditions shift, some variants may already be suited to them. Low diversity (as in an endangered species) leaves a population vulnerable. Variation arises from mutation and the reshuffling of meiosis and sexual reproduction.

- A genetically diverse population is more **resilient**: when a new disease, drought or predator arrives, some individuals are likely to have alleles that let them survive.
- A population with little diversity can be wiped out by one change. In the 1840s Irish farmers grew almost entirely one variety of potato, so potato blight destroyed nearly the whole crop. California condors and black-footed ferrets were each once reduced to fewer than 30 animals, and lost much of their variation.

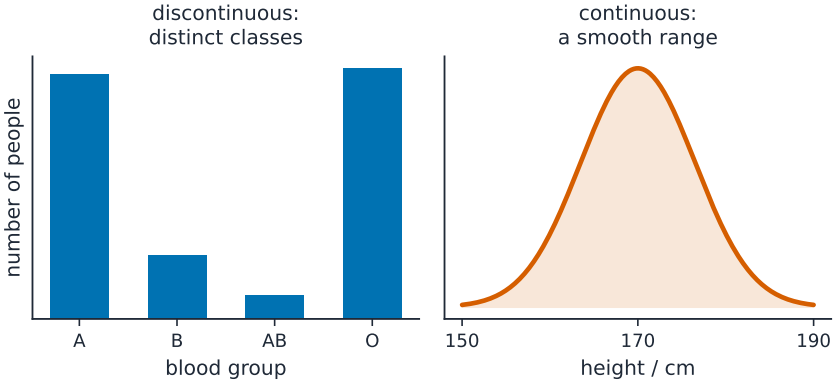
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- An allele that helps in one environment can harm in another. Some resistance alleles carry a fitness cost: bacteria with them may grow more slowly without the antibiotic, so the allele may become rarer when treatment stops.

Is a difference real? FRQs often give sample means with error bars of ±2 standard errors of the mean (SEM), about a 95% confidence interval. If the bars of two groups **do not overlap**, the difference between the means is very likely real (statistically significant). If they overlap, an appropriate statistical test is needed: overlapping confidence intervals can still accompany a significant difference.



Overlapping ±2 SEM bars alone cannot decide significance; non-overlapping bars suggest a real difference



Discontinuous and continuous variation

Origins of Life on Earth

Evidence suggests early Earth's conditions could form simple organic molecules (the Miller–Urey type experiments), which assembled into polymers, then self-replicating **RNA** (the "RNA world"), and eventually membrane-bound cells. The first cells were prokaryotes; eukaryotes arose later through **endosymbiosis** 内共生.

The timeline from geology.

- Earth formed about **4.6 billion years ago**.
- Early Earth cooled while impacts continued. Zircon evidence suggests liquid water, and potentially habitable conditions, existed by about **4.3–4.4 billion years ago**; a sharp habitability boundary at 3.9 billion years is not established.
- Some of the oldest widely accepted fossil evidence of life, from layered microbial mats like stromatolites, is about **3.5 billion years** old.

The timing and process of the origin of life remain uncertain; the geological record constrains them but does not give an exact date.

The RNA world. This hypothesis says RNA was the first genetic material. RNA can store information **and** act as a catalyst (an RNA enzyme, a **ribozyme**). The CED gives its three assumptions:

1. Genetic information was passed on by **copying RNA**.
2. That copying needed **base pairing**.
3. Proteins coded by genes were **not** the catalysts yet.

Even today, the catalytic core of every ribosome is RNA, which supports this idea.



Stromatolites: layered microbial mats are among the oldest evidence of life on Earth



A hydrothermal vent: hot chemical-rich water may have powered early metabolism at life's origin

Common mistakes.

- Saying organisms evolve a trait **because they need it**. Variation arises at random by mutation; selection only changes how common existing variants are.
- Saying the antibiotic or pesticide **causes** the resistance mutation. Resistant variants were already present; the chemical kills the others, so the resistant ones leave more offspring.
- Reading a cladogram's tip order as relatedness. Relatedness comes from the **most recent shared branch point**; two tips next to each other are not always closest relatives, and branches can rotate about a node without changing the tree.
- Saying humans evolved from chimpanzees. Humans and chimpanzees share a **common ancestor**; neither species is the ancestor of the other.
- Mixing up homologous and analogous structures. **Homologous** structures share an ancestral origin (human arm, whale flipper); **analogous structures** 同功结构 do a similar job but evolved separately (bird and insect wings), so only homologous structures show common ancestry.
- Using the dominant-phenotype frequency as p^2. Dominant phenotype $= p^2 + 2pq$; always start from the recessive phenotype, q^2.
- Saying genetic drift is natural selection. Drift is **random**; it does not favour better-adapted alleles.
- Saying an individual evolves or "adapts" during its life. Populations evolve; individuals survive or do not.
- Saying overlapping 95% confidence intervals rule out a statistically significant difference. Overlap alone cannot decide; use an appropriate test.

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- Saying a cladogram shows how long ago groups split. Only a phylogenetic tree with a time scale does.
- Choosing the outgroup by its name. Choose an outgroup known to lie outside the group studied; neither the fewest characters nor the most sequence differences alone guarantees this.
- Saying a population in Hardy–Weinberg equilibrium has equal allele frequencies. Equilibrium means the frequencies are **not changing** between generations, whatever their values.

Practice

Answer each before reading the answers below.

1. In a population in Hardy-Weinberg equilibrium, 16% show the recessive phenotype. Find q, p and the frequency of heterozygotes.
2. List the five conditions for Hardy-Weinberg equilibrium.
3. A cross is expected to give a $3:1$ ratio. The offspring are 84 purple and 36 white. Calculate χ^2 and decide whether the data fit (critical value 3.84, 1 degree of freedom).
4. Explain how a population of bacteria becomes resistant to an antibiotic. Why is it wrong to say an individual bacterium evolves resistance?
5. Distinguish homologous structures from analogous structures, with an example of each.
6. DNA differences: species A and B differ at 2 sites, A and C at 8, B and C at 8. Assume comparable rates of change and independent evidence that C is the outgroup. Which two are most closely related, and how would C appear on a rooted tree?
7. Explain how a population bottleneck can change allele frequencies.

Answers

1. $q^2 = 0.16$, so $q = 0.4$ and $p = 0.6$. Heterozygotes: $2pq = 0.48$.
2. No mutation, random mating, no natural selection, a very large population (no genetic drift), and no migration (gene flow).
3. Expected 90 and 30. $\chi^2 = \dfrac{(84-90)^2}{90} + \dfrac{(36-30)^2}{30} = 0.4 + 1.2 = 1.6$. $1.6 < 3.84$, so we fail to reject the null hypothesis: the data fit a $3:1$ ratio.
4. Variation already exists; a few bacteria carry resistance alleles. The antibiotic kills the rest, and the survivors reproduce, so the frequency of resistance rises over generations. Populations evolve; individuals do not change their genes in response.
5. Homologous structures share an ancestral origin but may differ in function (human arm, bat wing). Analogous structures have similar functions but evolved separately (bird wing, insect wing).

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6. A and B. C branches off first (an outgroup), and A and B share a more recent common ancestor.
7. A sudden fall in population size leaves a random sample of survivors, so some alleles are lost or become more common by chance, and genetic variation falls.

Exam tips

- State natural selection cleanly: **variation → differential survival and reproduction → helpful traits become common**.
- **Fitness = reproductive success**, not strength or health.
- Evolution is a change in **allele frequencies** in a **population** – individuals do not evolve.
- Use **Hardy–Weinberg** ($p^2 + 2pq + q^2 = 1$): start from the recessive phenotype $q^2 \to q \to p$.
- Cite independent **evidence** (fossils, homologous structures, DNA) and explain speciation via reproductive isolation.

Ecology

AP Biology

Responses to the Environment

In 1995 wolves were returned to Yellowstone National Park after 70 years away. Wolf predation helped reduce elk numbers and browsing pressure, contributing to willow recovery in some places. Water availability, other predators and beavers also shaped the response; attributing all vegetation and river changes to wolves alone is too simple. This unit is about how such links work.

Organisms sense and respond to their surroundings in ways that aid survival and reproduction. **Behaviors** may be **innate** (inherited, like reflexes and instincts) or **learned**. Responses such as migration, hibernation, and phototropism, and signals between organisms, are shaped by natural selection because they improve fitness.



Sunflowers face the light: organisms detect and respond to environmental stimuli

■ The behaviours the CED lists by name

These are the illustrative behaviours an exam question builds its scenario from, so learn what each one *is* and what it *buys the organism*:

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Behaviour	What it is	Why it aids fitness
taxis 趋性	movement **directed** towards or away from a stimulus	reaches food, light or moisture, or escapes a hazard
kinesis 动性	a change in movement speed or turning rate, without steering towards or away from the stimulus	keeps the animal in favourable conditions by moving less there and more elsewhere
nocturnal and diurnal activity 夜行性与昼行性	being active at night, or by day	avoids predators or heat, and matches activity to when food is available
territorial marking 领域标记	scent, sound or visual marks that claim an area	secures food, nesting sites and mates without repeated fighting
coloration 体色	pigmentation in animals and flowering plants	warning, camouflage, mate attraction – and in flowers, attracting pollinators
foraging 觅食 by bees and others	searching for and collecting food	efficient foraging returns more energy than it costs, which is what selection acts on

△ **Taxis and kinesis are the pair most often confused.** Taxis has a *direction*: the woodlouse walks towards the damp. Kinesis changes movement speed or turning rate without directional steering: the woodlouse moves quickly in dry air and slows down in damp air, so it ends up in the damp without ever steering there.

Life-history strategy 生活史策略 is the pattern of when an organism grows, reproduces and dies, and it too is shaped by selection: a **biennial** plant stores resources in its first year and flowers in its second; **reproductive diapause** pauses reproduction until conditions improve, so offspring arrive when they can survive. Both trade the timing of reproduction against the chance that it succeeds.

Plants respond too. Photoperiodism 光周期现象 is a response to day length: many plants flower only when nights reach a certain length, so they flower in the right season every year. **Phototropism** is growth towards light. When a caterpillar chews a leaf, many plants respond to the **herbivory** 植食作用 by making bitter or toxic chemicals, and some release chemicals into the air that attract the caterpillar's predators.

Sending signals. Organisms communicate with **visual**, **sound**, **touch**, **electrical** and **chemical** signals. Signals show dominance, mark territory, lead others to food

and attract mates. A bird's song both holds its territory and attracts a mate; a honeybee's dance tells other bees where flowers are. A signal changes another organism's behaviour, so it changes who survives and reproduces.

- The **fight-or-flight response** prepares an animal's body to fight or run when it senses danger.
- **Predator warnings**, such as a meerkat's alarm call, make the whole group hide.

Working together. Cooperative behaviour usually increases both the individual's fitness and the group's survival. Wolves hunting in a pack can kill prey too large for one wolf; fish in a school and birds in a flock are harder for a predator to pick off; bees in a colony share the work. An animal that helps its relatives at a cost to itself still passes on copies of its own alleles, because relatives share many of them. This is **kin selection** 亲缘选择. Worker bees do not breed at all, but they help their mother, the queen, raise their sisters.

Innate or learned, both are selected. A behaviour, inherited or learned, spreads if it increases survival and reproduction. Parent birds feeding their chicks, courtship displays and efficient foraging are all examples.

Energy Flow Through Ecosystems

Energy enters most **ecosystems** 生态系统 as sunlight, is captured by **producers** 生产者 (photosynthesizers), and passes to **consumers** 消费者 along a **food chain** 食物链. Each level is a **trophic level** 营养级. Only about **10%** of energy transfers up each level (much of the rest is released as heat, or remains in uneaten material and wastes), so food chains are short. Producers have the most energy available, but need not be the most numerous organisms. Energy **flows** through and is lost, while matter (carbon, nitrogen) **cycles**.

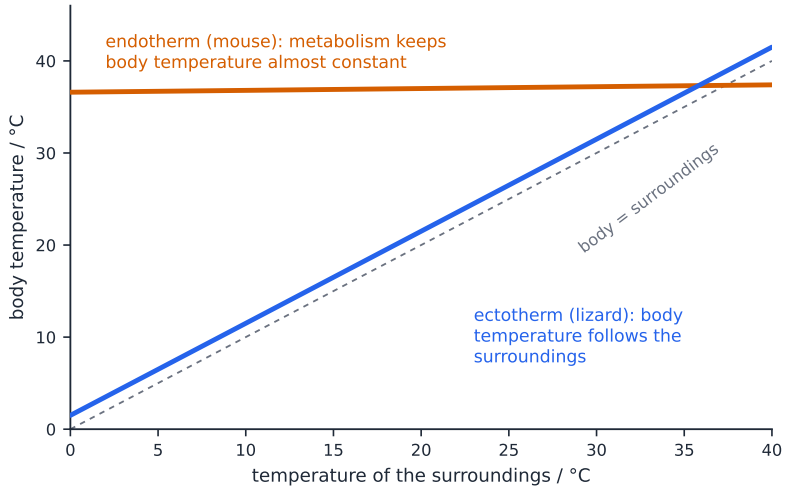
Ecological levels. A **population** is one species in one area; a **community** is all the populations there; an **ecosystem** is the community plus its non-living surroundings; a **biome** 生物群系 is a large region with a typical climate and vegetation, such as a desert or a tropical rainforest.

Making and using food. Autotrophs 自养生物 capture energy from the environment.

- **Photosynthetic** organisms capture light energy. Together they set the ecosystem's **primary productivity**, the rate at which producers store energy.
- **Chemosynthetic** 化能合成 organisms capture energy from small inorganic molecules such as hydrogen sulfide, without sunlight; many use oxygen as an electron acceptor. Bacteria at deep-sea vents feed whole communities this way, with no sunlight at all.

Heterotrophs 异养生物 get their energy by eating organic matter: carbohydrates, lipids and proteins. They include herbivores, carnivores, omnivores, scavengers and **decomposers** 分解者. Decomposers break down dead matter and wastes, returning nutrients to the soil.

Warm or cold. Endotherms 恒温动物, such as birds and mammals, use the heat from their own metabolism to keep their body temperature almost constant. **Ectotherms** 变温动物, such as lizards and insects, gain most of their heat from the surroundings; they warm up by basking in the sun and cool down in the shade.



An endotherm holds its temperature steady; an ectotherm's follows its surroundings

Keeping warm is expensive. A mouse uses many times as much food energy as a lizard of the same mass, but it can stay active on a cold night. Smaller endotherms lose heat faster, because they have more surface area for each gram of body.

Energy budgets. If an organism takes in more energy than it uses, the extra is **stored** and used for **growth** and **reproduction**. If it takes in less, it loses mass, has fewer offspring and eventually dies. Some organisms change how they reproduce with the energy available. Aphids and water fleas reproduce asexually, quickly, when food is plentiful, and switch to sexual reproduction when conditions get worse. Many animals and plants breed only in the season when food is easiest to find.

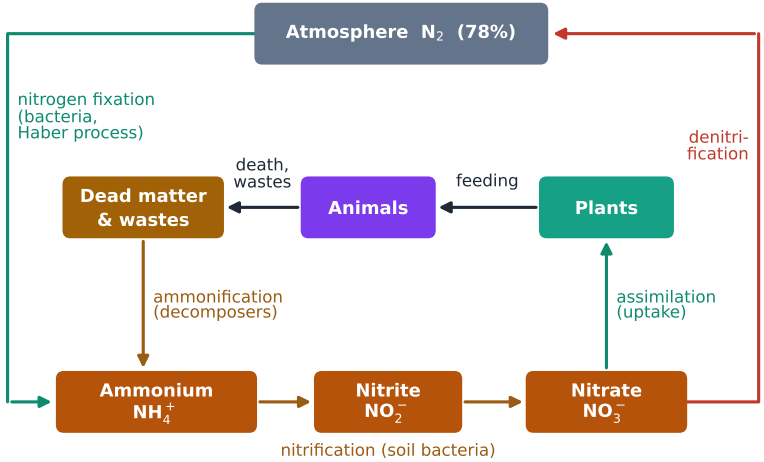
Changing the energy supply. Fewer producers, or less sunlight reaching them, means less energy for every level above. For example, after a volcanic eruption fills the air with dust, plants grow less. Fewer herbivores can then be supported, and fewer carnivores still. A top level, such as the quaternary consumers, may disappear entirely.

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How matter cycles

Every **biogeochemical cycle** 生物地球化学循环 moves atoms between **reservoirs** 储库 (non-living stores such as the air, the ocean and rocks, and living stores in organisms). Atoms are never used up, only moved: each cycle shows the **conservation of matter**. The cycles are linked; for example, the water cycle carries nitrates and phosphates through the soil.

- **Water.** Reservoirs: oceans, surface water, the air, living organisms. Processes: evaporation, condensation, precipitation and **transpiration** 蒸腾作用 (water lost from leaves).
- **Carbon.** Photosynthesis takes CO_2 from the air into carbohydrates; respiration and decomposition return it. Burning fossil fuels releases carbon that was locked away for millions of years, so CO_2 in the air rises.
- **Nitrogen.** The air, 78% N_2, is the largest reservoir, but most organisms cannot use N_2. Soil microorganisms carry out each step, shown below.
- **Phosphorus.** Weathering of rocks releases phosphate (PO_4^{3-}) into soil and water. Producers absorb it and build it into DNA, ATP and phospholipids; consumers get it by eating; decomposers return it. Unlike the other three cycles, phosphorus has **no gas stage**, so it moves slowly and often limits growth.

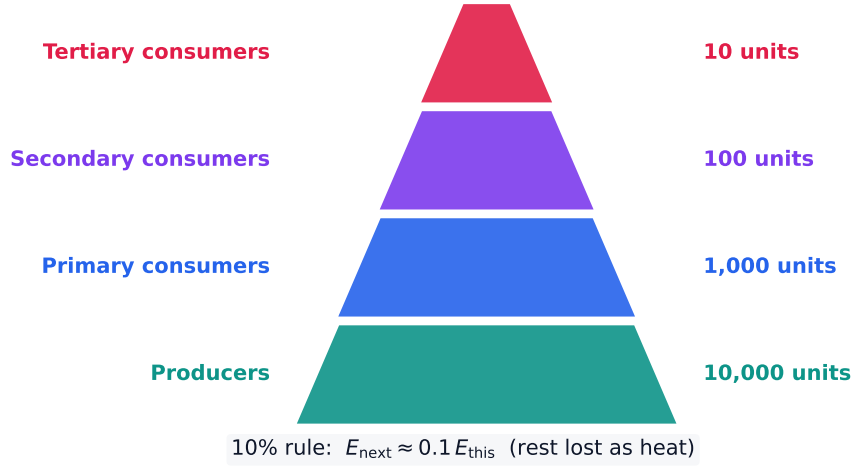


Bacteria carry out nearly every step of the nitrogen cycle

1. **Nitrogen fixation** 固氮作用: bacteria (some in the root nodules of legumes) turn N_2 into ammonia.
2. **Nitrification** 硝化作用: other soil bacteria turn ammonium into nitrite, then nitrate.

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3. **Assimilation**: plants take up nitrate or ammonium and use it to make amino acids and nucleotides.
4. **Ammonification** 氨化作用: decomposers turn the nitrogen in dead matter and wastes back into ammonium.
5. **Denitrification** 反硝化作用: bacteria in waterlogged, low-oxygen soil turn nitrate back into N_2.



Only about 10% of the energy passes to the next trophic level

> **Worked example.** Suppose producers capture 10,000 kcal. Applying the 10% rule, primary consumers receive about 1,000 kcal, secondary consumers 100 kcal, and tertiary consumers only 10 kcal. Only about 10% reaching each next level, with the rest released as heat or remaining in uneaten material and wastes, is why food chains rarely exceed four or five levels – there is too little energy left to support another.

Population Ecology

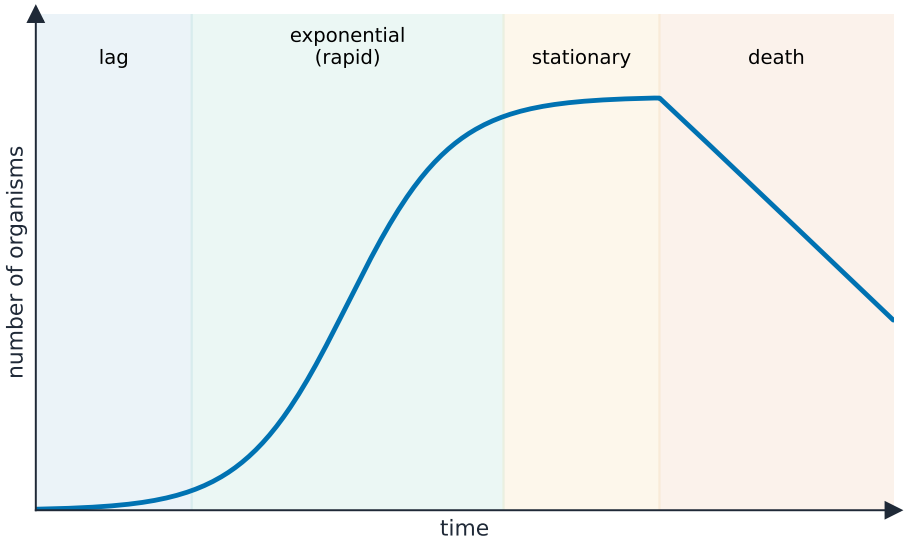
A **population** 种群 is the individuals of one species in an area. Its growth depends on birth, death, immigration, and emigration. Ignoring migration, the change in size is simply births minus deaths:

$$\frac{dN}{dt} = B - D$$

With no limits, each individual adds offspring at the maximum rate $r_{\max}$, and the population grows **exponentially**:

$$\frac{dN}{dt} = r_{\max} N$$

Worked example (exponential growth). A population of 500 bacteria has $r_{\max} = 0.2$ per hour. Its growth rate now is $0.2 \times 500 = 100$ cells per hour. When it reaches 5000 cells, it grows ten times as fast, 1000 cells per hour: the bigger it gets, the faster it grows, which gives the J-shape. **Exponential growth** 指数增长 (J-shaped) happens with unlimited resources; **logistic growth** 逻辑斯蒂增长 (S-shaped) levels off at the **carrying capacity** 环境容纳量 K – the maximum the environment can sustain – following $\frac{dN}{dt} = r_{\max} N \frac{K - N}{K}$.



A population in a closed culture: lag, exponential growth, a stationary phase where the curve levels off, then decline

Worked example. A population has $r_{\max} = 0.5\ \text{yr}^{-1}$, carrying capacity $K = 1000$, and current size $N = 400$. Then $\frac{dN}{dt} = 0.5 \times 400 \times \frac{1000 - 400}{1000} = 0.5 \times 400 \times 0.6 = 120$ individuals per year. The $\frac{K-N}{K}$ term is why growth is fastest near $N = K/2$ and slows toward zero as N approaches K.

Worked example (reading K from a graph). A yeast culture grows quickly, then levels off at about 8×10^6 cells per mL from day 5 onwards.

1. The carrying capacity is the level where the curve flattens: $K \approx 8 \times 10^6$ cells/mL.
2. Growth stops because something runs short or builds up: the sugar is used up, or toxic wastes such as ethanol accumulate.
3. Adding fresh medium would raise K; the population would grow again until a new limit was reached.

Effect of Density on Populations

Some factors depend on crowding, others do not:

- **Density-dependent** 密度制约 factors intensify as a population grows – competition, predation, disease.
- **Density-independent** 非密度制约 factors act regardless of density – weather, natural disasters.

These factors regulate population size around the carrying capacity.

Carrying capacity is the number of a species that the ecosystem's resources can support **long term**. As a population nears it, density-dependent factors bite harder: each individual gets less food, disease spreads more easily between crowded animals, and predators find prey more easily. Births fall and deaths rise until they balance, giving the S-shaped logistic curve. A population that **overshoots** K damages its resources, then crashes below K.



A dense deer herd: density-dependent factors such as disease and food competition intensify as numbers rise

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Community Ecology

A **community** 群落 is all the interacting populations in an area. Key interactions: **competition** 竞争 (for shared resources), **predation** 捕食, **symbiosis** 共生 – **mutualism** (both benefit), **commensalism** (one benefits, other unaffected), and **parasitism** (one benefits, other harmed). These relationships shape which species coexist.



Clownfish among sea-anemone tentacles – a textbook mutualism

Describing a community. A community is described by **which species** it has and by its **species diversity**: how many species there are, and how evenly the individuals are shared between them. **Simpson's diversity index** 辛普森多样性指数 combines both:

$$\text{diversity index} = 1 - \sum \left(\frac{n}{N}\right)^2$$

where n is the number of one species and N the total of all species. It runs from 0 (one species only) towards 1 (many species, evenly spread).

> **Worked example (Simpson's index).** Community A has 40 oaks, 30 maples and 30 birches ($N = 100$). Community B has 80 oaks, 10 maples and 10 birches.

1. A: $1 - (0.4^2 + 0.3^2 + 0.3^2) = 1 - (0.16 + 0.09 + 0.09) = 0.66$.
2. B: $1 - (0.8^2 + 0.1^2 + 0.1^2) = 1 - (0.64 + 0.01 + 0.01) = 0.34$.
3. Both have three species, but A is more **even**, so it is more diverse.

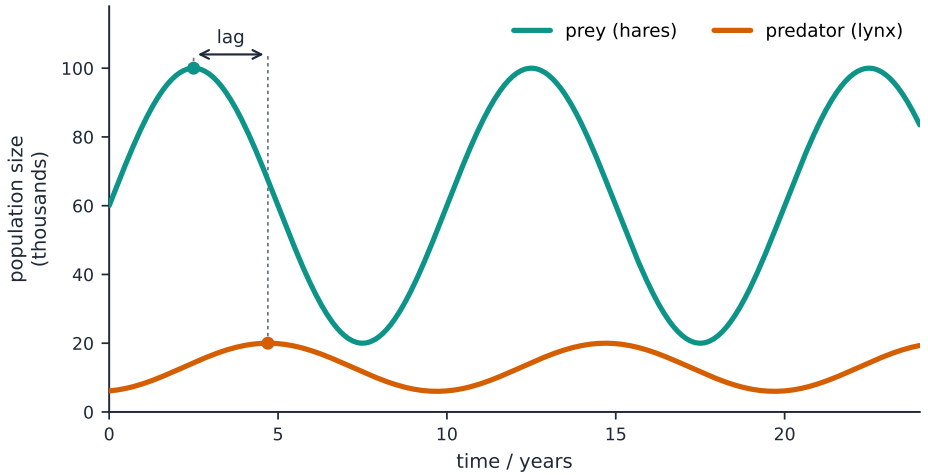
Effects as + and −. Each interaction can be written by its effect on each species:

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Interaction	Effect on species 1 / species 2	Example
competition	− / −	lions and hyenas for the same prey
predation, herbivory	+ / −	lynx eating hares
mutualism 互利共生	+ / +	bees and flowers; nitrogen-fixing bacteria in root nodules
commensalism 偏利共生	+ / 0	barnacles on a whale
parasitism 寄生	+ / −	tapeworms; a cuckoo laying its eggs in a warbler's nest

A relationship can change with conditions. Great spotted cuckoos parasitise carrion crow nests. In a study in northern Spain, their chicks released a foul-smelling secretion that deterred predators. When predation pressure was high, this could benefit the host brood, bringing the relationship closer to mutualism; the benefit is not found in every population.

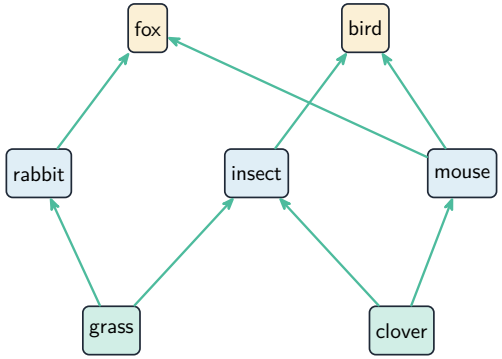
Predators and prey cycle. When prey are plentiful, more predators survive and breed, so the predator population rises, a little later. More predators eat more prey, so the prey fall; then, short of food, the predators fall too, and the prey recover.



The predator population peaks a little after the prey population, because predators need time to breed

Other ways populations shape a community.

- **Niche partitioning** 生态位分化: similar species reduce competition by using a resource differently. Several warbler species feed on insects in the same tree, but each in a different part of it.
- **Competitive exclusion**: if two species need exactly the same limited resource, the better competitor eventually removes the other.
- **Trophic cascade** 营养级联: a change at one trophic level ripples down the chain. Yellowstone's wolves helped reduce elk browsing, contributing to willow recovery in some places alongside other ecological factors.



A food web links several food chains together

Biodiversity



Coral reef outcrop: high biodiversity in a productive marine ecosystem

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Biodiversity 生物多样性 is the variety of life – genes, species, and ecosystems. Higher diversity generally makes a community more **resilient** 有韧性, better able to withstand and recover from disturbance. A **keystone species** 关键种 has an outsized effect, so losing it can collapse the community.

Why diversity gives resilience. An ecosystem with few parts, and little variation between them, is **less resilient** to change. This is true for natural ecosystems and for artificial ones such as farms.

- With many species at each trophic level, a predator that loses one prey species can switch to another. With only one, it starves.
- A forest of many tree species loses only some trees to a new disease. A plantation of one tree species can lose them all.
- After a disturbance, some species in a diverse community are likely to survive and fill the gaps.

What holds diversity up. Keystone species, producers, and essential abiotic factors (such as water and nutrients) and biotic factors maintain an ecosystem's diversity. A **keystone species** has an effect far larger than its numbers. When the sea star *Pisaster* was removed from rocky shores, mussels took over the rocks and the number of species fell from 15 to 8. Sea otters keep sea urchins in check; without otters, the urchins eat whole kelp forests.

> **Worked example (removing a species).** In community A, the top predator eats five prey species. In community B, it eats only species PP1. Explain why removing PP1 affects B more.

1. In A, the predator can switch to its other four prey, so its numbers change little.
2. In B, the predator loses its only food source, so its population crashes.
3. Its other effects on community B, such as controlling herbivores, are lost too, so the change spreads further.



Biodiversity at three levels: genetic, species, and habitat

Disruptions in Ecosystems



Deforested landscape – habitat loss is a major disruption to ecosystems

Ecosystems change from natural and human causes – climate shifts, invasive species, habitat loss, and pollution. A disturbance can trigger **ecological succession** 生态演替 (the community rebuilds over time). Because species are interconnected, a change to one – especially a keystone or a trophic level – can ripple through the whole ecosystem.

Variation meets the environment. An **adaptation** is a genetic variation that is favoured by selection because it gives an advantage in a **particular** environment. Mutations are **not directed** by the environment; the variation is already there before the change, and the change only selects among it. In **heterozygote advantage** 杂合子优势, the heterozygote is fitter than either homozygote. With malaria present, people with one sickle-cell allele survive best, so both alleles stay in the population.

Invasive species. An **invasive species** 入侵物种 is one brought into a new area, by accident or on purpose, that spreads and harms the ecosystem. It succeeds because its new home lacks its natural **predators**, **parasites** and **competitors**, or because it outcompetes native species for resources.

- **Kudzu**, a vine from Asia planted in the USA, grows over and shades out whole forests.
- **Zebra mussels**, carried to the Great Lakes in ships' ballast water, filter out the plankton that native species eat and block water pipes.

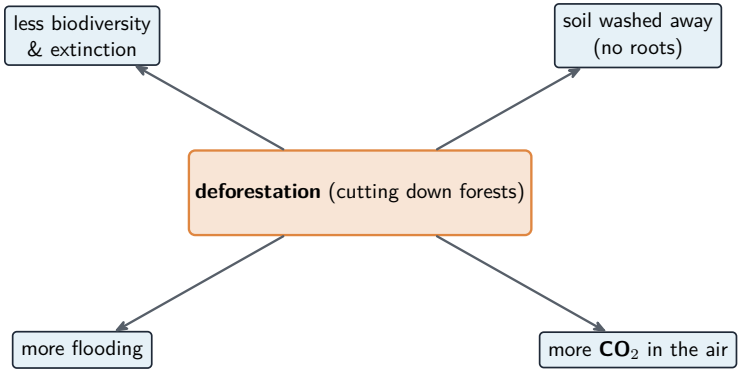
The same species may not be invasive somewhere else, where the community already has predators or competitors that hold it in check.

Human activities speed up change.

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- **Biomagnification** 生物放大: a toxin that is not broken down or excreted, such as DDT or mercury, becomes more concentrated at each trophic level. Top predators get the highest dose; DDT made the eggshells of eagles and ospreys so thin that they broke.
- **Eutrophication** 富营养化: fertiliser runs off into lakes. Algae grow fast, then die; decomposers break down the dead algae and use up the oxygen in the water, and fish die.
- **Introduced diseases**: Dutch elm disease, a fungus spread by beetles, killed most elm trees in Europe and North America; potato blight devastated Ireland's potato crop, which relied heavily on a few susceptible varieties.
- Logging, urbanisation and **monocropping** (growing one crop over large areas) destroy habitats and lower diversity.

Geological and weather events. Ecosystems also change for reasons humans do not control. **El Niño** warms the eastern Pacific every few years, which moves rainfall and cuts fish numbers off Peru. **Continental drift** separated and joined land masses: when the Isthmus of Panama formed about 3 million years ago, North and South American animals mixed, and many South American species died out in competition with the newcomers. A **meteor impact** 66 million years ago ended the age of the dinosaurs. Global climate change is moving the ranges of many species towards the poles and up mountains. Biogeography, the study of where species live, records these changes.



Deforestation lowers biodiversity and causes erosion, flooding, and higher CO2

Common mistakes.

- Saying energy is recycled in an ecosystem. **Matter** cycles; energy flows one way and leaves as heat.
- Saying the 90% "lost" energy is destroyed. It is still energy, mostly released as **heat** in respiration, or left in parts that are not eaten or digested.

- Placing the fastest logistic growth at the carrying capacity. Growth is fastest near $N = K/2$; at $N = K$ the term $\frac{K-N}{K}$ is zero, so $\frac{dN}{dt} = 0$.
- Calling a flood or a frost density-dependent because it kills many individuals. A factor is density-dependent only if its effect **per individual** grows as the population becomes more crowded.
- Mixing up taxis and kinesis. **Taxis** is directed movement towards or away from a stimulus; **kinesis** changes movement speed or turning rate without steering towards or away from the stimulus.
- Calling a keystone species "the most common species". A keystone species has an effect far **larger than its numbers** suggest; a sea star or a wolf can be rare and still hold the community together.
- Saying an organism "decides" to adapt, or that the environment causes the right mutation. Selection acts on variation that already exists.
- Mixing up nitrification and nitrogen fixation. **Fixation** turns N_2 gas into ammonia; **nitrification** turns ammonium into nitrate.
- Saying a higher Simpson's index means more individuals. It means more species, more evenly spread.
- Drawing food-web arrows from predator to prey. Arrows show the **direction energy flows**: from the organism eaten to the one that eats it.
- Calling every symbiosis mutualism. Symbiosis includes **mutualism** (+/+), **commensalism** (+/0) and **parasitism** (+/−).

Practice

Answer each before reading the answers below.

1. Producers in a food chain store 10 000 kcal. Estimate the energy available to tertiary consumers, and explain the loss.
2. A population has $r_{\max} = 0.1$ per year, $N = 500$ and $K = 1000$. Use $\dfrac{dN}{dt} = r_{\max} N \dfrac{K-N}{K}$ to find the growth rate.
3. Classify each as density-dependent or density-independent: a drought; disease; competition for nesting sites.
4. A community has three species with 5, 5 and 10 individuals. Calculate Simpson's diversity index, $1 - \sum (n/N)^2$.
5. Sea otters eat sea urchins, which eat kelp. Predict what happens to the kelp forest if otters are removed, and name the otter's role.
6. Explain why an invasive species often spreads rapidly in a new ecosystem.

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Answers

1. About 10 kcal (10% passes at each of three transfers). Most energy is lost as heat from respiration, and some is not eaten or not digested.
2. $0.1 \times 500 \times \dfrac{500}{1000} = 25$ individuals per year.
3. Drought: density-independent. Disease and competition: density-dependent.
4. $N = 20$: $\sum = 0.0625 + 0.0625 + 0.25 = 0.375$, so the index is 0.625.
5. Urchins increase and overgraze the kelp, so the kelp forest declines along with the species that depend on it. The otter is a keystone species.
6. It often has no natural predators, parasites or competitors there, so its population is not controlled and it out-competes native species.

Exam tips

- Apply the **10% rule**: about 90% of energy is lost at each trophic level, so food chains are short.
- Distinguish **exponential** (J) from **logistic** (S) growth; the latter levels off at the **carrying capacity** ($\frac{dN}{dt} = r_{\max} N \frac{K-N}{K}$).
- Separate **density-dependent** (competition, disease, predation) from **density-independent** (weather) limiting factors.
- Name community interactions (competition, predation, symbiosis) and the effect of a **keystone species**.
- Explain how disturbing one species – especially a keystone or a whole trophic level – ripples through the ecosystem.
- For a **null hypothesis**, state that the treatment has **no effect**: "Caffeine in nectar has no effect on how often bees return to a flower."
- For data on a carrying capacity, read K where the curve levels off, and name the limit: food, space, or the build-up of wastes.

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