

1 • Molecules, Diet, Transport and Health

Pearson Edexcel • International A-Level • Biology

From molecules to a working circulation

A red blood cell leaving your lung carries oxygen on haemoglobin, rides an artery, squeezes through a capillary narrower than itself, releases the oxygen to a respiring cell, and returns through a vein. Every structure in that journey is built from the molecules this unit starts with, and every exam question about it asks you to connect structure to function.

WBI11 Molecules, Diet, Transport and Health is the first IAS unit: a written paper of 1 hour 30 minutes, 80 marks, all compulsory. It assesses Topic 1 (Molecules, Transport and Health) and Topic 2 (Membranes, Proteins, DNA and Gene Expression). Expect data questions on health studies, calculations with standard form, and extended answers that name structures and explain mechanisms.

Water, carbohydrates and food tests

Water is the transport medium because it is a **polar solvent**. In a water molecule, oxygen pulls the shared electrons more strongly than hydrogen: each O–H bond has a slight negative end at O and a slight positive end at H. This is the **dipole** nature of water. Opposite charges between neighbouring molecules form **hydrogen bonds**, and polar or charged particles (glucose, sodium ions, amino acids) dissolve because water clusters around them.

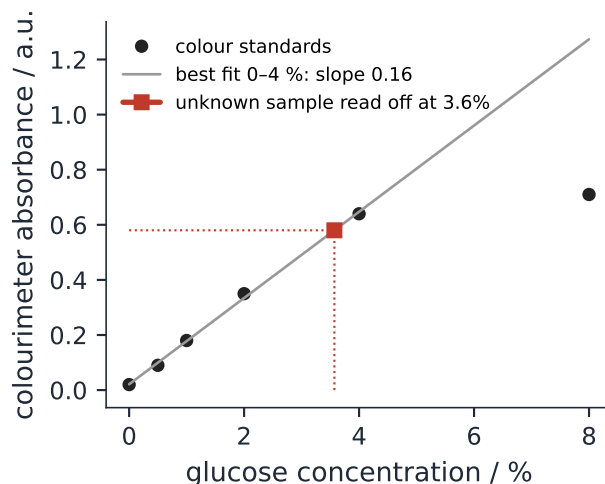
Carbohydrates are joined and split by two reactions:

- **Condensation** removes a water molecule as a **glycosidic bond** forms between two sugars.
- **Hydrolysis** adds water back and breaks that bond.

Two glucose molecules condense to maltose; glucose plus fructose give sucrose; glucose plus galactose give lactose. Long chains make polysaccharides: **amylose** (1,4-linked, coils into a helix, compact), **amylopectin** and **glycogen** (both 1,4-chains with 1,6-branches; glycogen is the animal store in liver and muscle). Branching means many chain ends, so glucose can be released quickly; the helix means a lot of energy fits in a small space.

■ Core Practical 1: estimating sugar and starch concentration

Reducing sugars reduce blue Benedict's reagent on heating, giving a brick-red precipitate. To estimate an unknown concentration, compare the colour against standards of known concentration, or read the solution in a colourimeter.



Calibration of Benedict's reagent: absorbance of colour standards against glucose concentration, with an unknown read off the straight part of the line.

Worked check. Standards of 0, 0.5, 1, 2 and 4 % glucose give absorbances 0.02, 0.09, 0.18, 0.35, 0.64. An unknown gives 0.58. On the straight section, each 1 % adds about 0.16 absorbance, so the unknown is near $0.58/0.16 \approx 3.6\%$. Reading beyond the standards (extrapolating past 4 %) is less reliable than measuring within them – a point the examiners like you to make. Iodine turns blue-black with starch; intensity of the blue-black gives the same kind of estimate for starch.

Lipids: triglycerides and ester bonds

A **triglyceride** is one glycerol plus three fatty acids, joined by three condensation reactions that form **ester bonds**. **Saturated** fatty acids have no C=C bonds: straight chains pack closely, so animal fats are solid at room temperature. **Unsaturated** acids have one or more double bonds that kink the chain, packing is looser, and oils are liquid. Lipids store more energy per gram than carbohydrates because they are more reduced.

Why animals need a pump: the heart and vessels

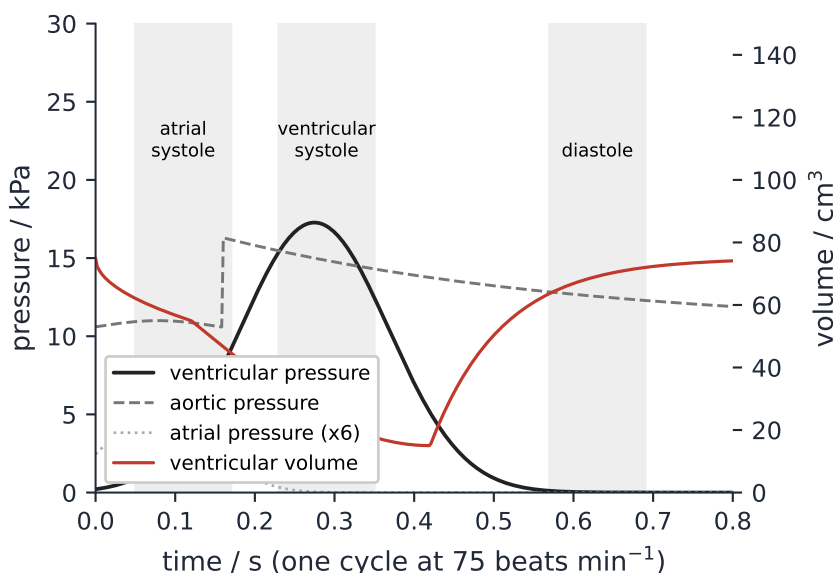
Diffusion supplies cells only a few tenths of a millimetre thick. A big organism needs **mass transport**: a pump and pipes that carry every cell's supply and waste. The three vessel types match three jobs:

Vessel	Wall	Why
Artery	thick muscle and elastic fibres, small lumen	carries blood at high, pulsing pressure; elastin smooths the pulse
Capillary	one cell thick endothelium	short diffusion path, huge total surface area; tissue fluid leaks out at the start
Vein	thin wall, wide lumen, valves	low pressure return; valves and the surrounding muscles stop backflow

The mammalian heart is two pumps in one organ. The right side sends blood to the lungs; the left side, with the thicker ventricle wall, sends blood to the body. Atria receive; ventricles deliver. The coronary arteries feed the heart muscle itself – blockage there is a heart attack.

■ The cardiac cycle

One beat has three phases. **Atrial systole** squeezes the last blood into the ventricles. **Ventricular systole** raises ventricular pressure above arterial pressure, the semilunar valves open, and blood is driven out. **Diastole** relaxes the muscle, pressure falls, the semilunar valves slam shut (the second heart sound), and the chambers refill from the veins.

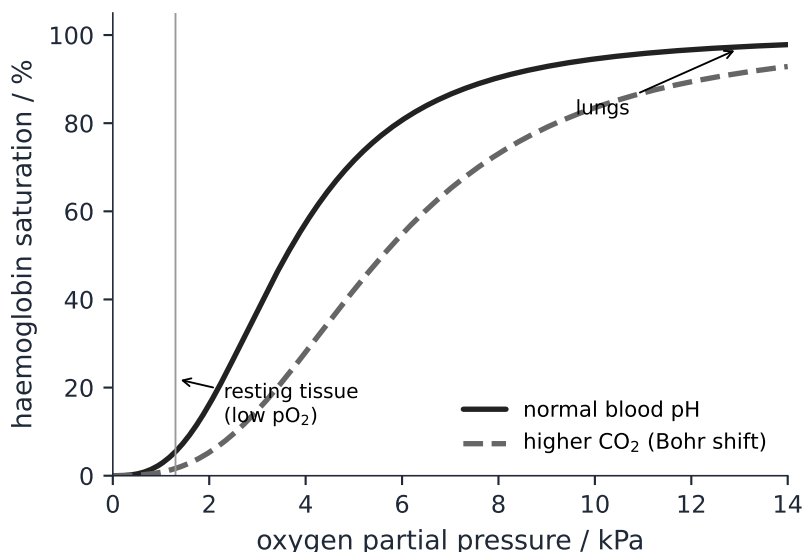


One cardiac cycle: ventricular and aortic pressure with ventricular volume. Valves change state whenever the two pressure curves cross.

Read the figure the way an examiner wants: valve state is decided by pressure difference, never by an instruction. Atrio-ventricular valves open when atrial pressure exceeds ventricular pressure; semilunar valves open when ventricular pressure exceeds aortic pressure.

Haemoglobin and the oxygen journey

Haemoglobin is a protein of four polypeptide chains, each folded around a **haem** group carrying one Fe^{2+} ion that binds one O_2 molecule – four in total. Binding the first oxygen changes the shape of the whole molecule and makes the next bindings easier: **cooperative binding** gives the oxygen dissociation curve its shallow-then-steep S shape.



Oxygen dissociation curves: the sigmoid curve for normal blood and the rightward Bohr shift when carbon dioxide rises.

In the lungs (high pO_2) the curve flattens near 100 % saturation. In resting tissue near 1–2 kPa the curve is steep, so a small fall in pO_2 unloads a large amount of oxygen – exactly where it is needed. When tissue respire hard it releases more carbon dioxide; hydrogen ions attach to haemoglobin and shift the whole curve to the right (the **Bohr effect**), forcing even more oxygen to unload in the busiest tissue.

Worked check. At pO_2 2 kPa the normal curve reads about 30 % saturation and the shifted curve about 15 % – haemoglobin holds only about a third as much oxygen at that partial pressure, so most of its four sites are released to the tissue sooner.

Cardiovascular disease: a chain of events

Atherosclerosis begins with damage to the **endothelium** lining an artery. The inflammatory response grows, low-density lipoproteins invade, smooth muscle and connective tissue build a **plaque**, the artery narrows and hardens, and a clot can finally block it or break loose.

Clotting itself is a cascade. Damage releases **thromboplastin**, which (with calcium ions) converts the soluble plasma protein **prothrombin** into the enzyme **thrombin**; thrombin converts soluble **fibrinogen**

into the insoluble mesh of **fibrin** that traps platelets and cells.

Blood pressure is reported as systolic over diastolic. Hypertension, smoking, high blood LDL-cholesterol, diet, inactivity, age, gender and genetics are **risk factors**. Several risk factors can act together; the size of the combined risk depends on the factors and the population.

■ Reading health-risk data

Questions 8 and 9 of every paper live here. Four habits cover almost every mark:

- **Correlation is not causation.** Two variables moving together may share a hidden cause, or the link may be chance.
- Judge the study: How large was the sample? How was it selected? What was controlled? Was it blind? Are there **confounders** such as age or smoking?
- Do the arithmetic carefully: percentage change, ratios in standard form, per-capita rates.
- Ask who or what is missing: non-responders, unpublished studies, extrapolated lines.

Worked check. A study finds gum disease correlates with endothelial damage. Before claiming one causes the other you would want a mechanism, a dose–response pattern, and confounders (diet, smoking, age) excluded – and the question “were the groups matched for blood pressure?” is exactly a confounder question.

Diet, cholesterol and treating CVD

Energy balance decides body mass. Overweight and obesity are screened by **BMI** (mass in kg divided by height in metres squared; 18.5–24.9 is the healthy band) and **waist-to-hip ratio** (WHR). Dietary saturated fat raises blood LDL-cholesterol; unsaturated fat and soluble fibre lower it.

Worked check. A woman of mass 74 kg and height 1.66 m: $BMI = 74/1.66^2 = 74/2.76 = 26.8$ – just into the overweight band. Waist 84 cm and hips 98 cm give $WHR = 84/98 = 0.86$.

Treatments all carry risks alongside benefits:

- **Antihypertensives** (beta blockers, ACE inhibitors, diuretics) lower blood pressure.
- **Statins** (for example simvastatin) block a liver enzyme in cholesterol synthesis, so cells take up LDL from blood.
- **Anticoagulants** (warfarin) and **antiplatelet** drugs (aspirin) reduce clotting, so bleeding risk rises.

A question asking you to “discuss” wants both sides plus a judgement anchored in the data given.

Membranes and transport

Every exchange surface is a membrane doing controlled business. The **fluid mosaic model** is the accepted picture: a phospholipid bilayer (fluid because the tails can slide), studded with intrinsic proteins that span the bilayer (channels, carriers), extrinsic proteins, cholesterol that controls fluidity, and glycoproteins and glycolipids for recognition.

Four transport routes:

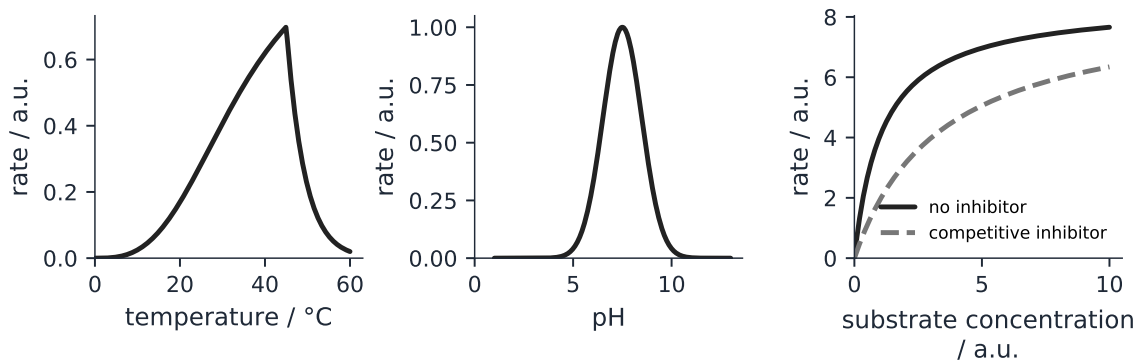
- **Diffusion** – net movement down a concentration gradient, no ATP.
- **Facilitated diffusion** – down the gradient through a channel or carrier protein.
- **Active transport** – against the gradient, powered by ATP, through carrier proteins that change shape.
- **Osmosis** – water moving from higher to lower **water potential** across a partially permeable membrane. Endocytosis and exocytosis move bulk material in vesicles.

For diffusion, Fick's law: rate is proportional to (surface area $\times$ concentration difference) $\div$ diffusion distance. Gas exchange surfaces answer all three: huge area, maintained gradient, and a wall one or two cells thick. This is why alveoli, capillaries and fish gills all look the way they do.

Proteins and enzymes

An amino acid carries an amino group, a carboxyl group, a hydrogen and an R group on one central carbon; two join by a **peptide bond** in a condensation reaction. The **primary structure** is the sequence; hydrogen bonds coil it into the **secondary**; further hydrogen, ionic and disulfide bonds fold the **tertiary**; separate chains assemble into the **quaternary** – haemoglobin is the classic example.

An **enzyme** is a protein whose **active site** fits one substrate shape. Binding is not rigid: the **induced-fit** model has both molecule and site flexing until complementary. This is why temperature and pH change rate – they act on the tertiary structure – and why the active site is specific.



Enzyme rate curves: temperature rises then collapses as the protein denatures above its optimum; pH shows a sharp optimum; substrate concentration saturates, and a competitive inhibitor is out-competed at high concentration.

Competitive inhibitors bind the active site and are outnumbered as substrate concentration rises; **non-competitive inhibitors** bind elsewhere and distort the site, so rate falls whatever the concentration.

Worked check. Q10 is quoted at 2.4: between 10 °C and 20 °C the rate is 2.4 times faster; between 45 °C and 55 °C the same factor means the denatured enzyme is collapsing at an increasing rate. Q10 only describes; it does not explain.

DNA, replication and the code

A **mononucleotide** is a pentose (deoxyribose or ribose), a phosphate and a nitrogenous base. Condensation between phosphate and sugar of neighbours makes a strand held by **phosphodiester bonds**. DNA is two antiparallel strands: A pairs with T (two hydrogen bonds), G with C (three), twisted into a double helix.

Replication is **semi-conservative**: helicase unzips the two strands, each is a template, and **DNA polymerase** DNA joins free nucleotides in the 5' to 3' direction. Meselson and Stahl grew bacteria first on heavy ^{15}N then light ^{14}N nitrogen; after one round of replication all the DNA was one intermediate density, after two rounds half was light – exactly what semi-conservative predicts and the alternatives forbid.

A gene is a sequence of bases coding for a sequence of amino acids. The code is a **triplet** code: three bases per amino acid. It is **non-overlapping** (read in successive groups), **degenerate** (most amino acids have several codons, so some mutations are silent), and carries start and stop codons.

Worked check. A gene of 300 base pairs coding a protein of 99 amino acids plus a stop codon: $99 \times 3 + 3 = 300$. ✓

From gene to protein – and when it goes wrong

Transcription: RNA polymerase binds the gene, uses one strand as template and builds **mRNA** RNA. Translation: in the cytoplasm, a **ribosome** reads the mRNA codons, and **tRNA** RNA molecules whose **anticodons** pair with those codons deliver amino acids in order; peptide bonds join them.

Mutations arise from replication errors. A **substitution** swaps one base: one amino acid changes, or nothing changes (degeneracy). An **insertion** or **deletion** adds or removes a base and shifts every reading frame after it – a **frameshift** – usually destroying the protein.

Cystic fibrosis is the course's model: deletion of three bases removes one phenylalanine from the CFTR protein, so chloride transport fails, mucus stays thick, and airways clog. The allele is recessive: carriers are healthy. **Genetic screening** can identify carriers, test embryos (pre-implantation genetic diagnosis) or foetuses (amniocentesis, chorionic villus sampling). Screening questions always carry ethics: who is tested, who is told, what happens to a positive result.

Check yourself

1. Explain why amylopectin releases glucose faster than amylose, referring to bonds and ends of chains.
2. Calculate the BMI of a 91 kg man 1.83 m tall and classify him.
3. During ventricular systole, which valves are open and which are shut, and why?
4. The oxygen saturation at 4 kPa falls from about 60 % to about 34 % when CO₂ rises. Explain the molecular cause.
5. Describe the clotting cascade in order, from endothelial damage to fibrin.
6. A study reports that people who eat more yoghurt have fewer heart attacks. List three checks you would make before believing a causal claim.
7. Explain why a competitive inhibitor lowers the rate at low substrate concentration but not at very high concentration.
8. Meselson and Stahl ruled out conservative replication. Which observation, after one round, did the job?
9. A frameshift mutation is usually worse than a substitution. Explain why, using the words triplet and degenerate.
10. Name two benefits and two risks of genetic screening for cystic fibrosis carriers.

Answers: 1 many 1,6-branch points give many chain ends for enzymes to attack simultaneously; 2 $91/1.83^2 = 27.2$, overweight; 3 atrio-ventricular shut, semilunar open, because ventricular pressure exceeds aortic but not atrial-from-above pressure; 4 hydrogen ions from CO₂ bind haemoglobin, changing its shape and lowering oxygen affinity (Bohr effect); 5 damage exposes collagen, platelets and damaged cells release thromboplastin, thromboplastin + Ca²⁺ converts prothrombin to thrombin, thrombin converts fibrinogen to fibrin, fibrin mesh traps cells; 6 sample size and

selection, confounders such as age/exercise/diet, blinded comparison, mechanism or dose–response; 7 the inhibitor competes for the active site, so at very high substrate concentration nearly every collision with the site is substrate; 8 after one round all DNA was a single intermediate band – conservative predicts one heavy and one light band; 9 a frameshift changes every triplet after the mutation, so the whole protein from that point is a different amino-acid sequence, while degeneracy lets many substitutions change nothing; 10 benefits – informed family planning, earlier treatment or lifestyle change; risks – anxiety, discrimination by insurers or employers, false positives or negatives.