

2 · Organisation

AQA · GCSE · Biology

Organisation: from cells to systems

Cells form tissues, tissues form organs, organs form systems – and the digestive system, blood and plants each show the pattern. This reference covers **AQA GCSE Biology 8461, topic 4.2 Organisation**.

How the exam treats this topic:

- Paper 1 carries Organisation. Required practicals: RP4 (food tests) and RP5 (pH and amylase rate).
- You must **evaluate** heart-disease treatments and lifestyle risk data, and interpret graphs and tables of risk factors.
- Recall exactly: the enzymes table, the heart's vessels, blood components, plant tissues, transpiration factors.

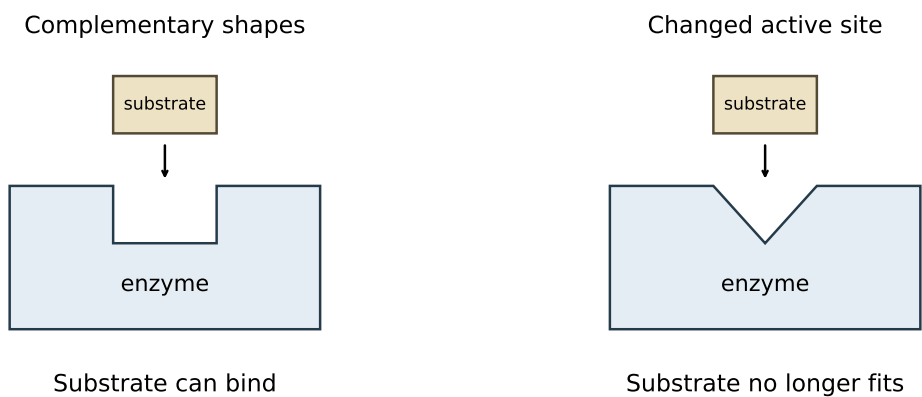
Principles of organisation

Cells → tissues → organs → organ systems → organism. A **tissue** contains cells with similar structure and function. An **organ** contains different tissues working together. **Organ systems** contain organs working together.

Digestive example: a muscle cell belongs to muscular tissue; the stomach also contains glandular tissue (secretes digestive juice) and epithelial tissue (covers surfaces). These tissues together form the stomach. The stomach, intestines and other digestive organs work together in the digestive system. Calling the stomach a tissue misses its several tissue types.

The human digestive system (4.2.2.1)

Enzymes are protein biological catalysts: they speed reactions without being used up. In the simplified **lock-and-key model**, a substrate fits a complementary **active site**. Products leave and the enzyme can act again. This is a shape model, not a drawing of real molecules.



Cold versus denatured: at low temperature, particles move more slowly, so there are fewer successful enzyme–substrate collisions; warming can increase the rate again. High temperature can change the enzyme’s shape permanently (**denaturation**), so the substrate no longer fits. pH changes can reduce activity by affecting the active site; do not claim every small move away from optimum causes permanent denaturation.

Enzyme	Production sites	Substrate → products
Amylase	Salivary glands; pancreas; small intestine	Starch → simple sugars
Protease	Stomach; pancreas; small intestine	Protein → amino acids
Lipase	Pancreas; small intestine	Lipids → glycerol + fatty acids

Amylase breaks starch into **simple sugars**; it does not directly turn starch into glucose. Digestive enzymes make small soluble products that cross the small-intestine wall into blood. Digestion is breakdown; absorption is movement into blood. Digestion products can build new carbohydrates, lipids and proteins, and glucose is used in respiration.

Bile is made in the **liver** and stored in the **gall bladder**. It is alkaline: neutralising stomach acid provides suitable conditions for lipase in the small intestine. It also **emulsifies** large fat globules into smaller droplets, increasing surface area for lipase. Bile is not an enzyme; emulsification does not itself make glycerol and fatty acids.

RP4: use separate portions of a prepared food sample for each test. Record reagent, treatment and observed colour; use known positive and negative controls to check the procedure.

Substance	Reagent / treatment	Positive result
Reducing sugar	Benedict's; hot water bath	Green/yellow/orange/brick-red
Starch	Iodine solution	Blue-black
Protein	Biuret reagent	Purple/lilac
Lipid	Sudan III; mix, allow to separate	Red-stained oil layer

Benedict's starts blue; iodine starts yellow-brown; Biuret starts blue. A negative result means **not detected by this test**, not proof that none is present. Benedict's does not test for every kind of sugar. Heating supplies energy for the Benedict's reaction; brick-red is not the only positive colour. Wear eye protection; use a water bath, not a flame under the food-test tube. Follow school handling instructions for reagents.

RP5: pH and amylase rate. Change pH with buffers; measure time for the starch endpoint, then compare relative rates. Use a water bath to control temperature and sample onto iodine **every 30 s**, as specified by AQA.

Reproducible sequence: place separate drops of iodine in a spotting tile. Equilibrate measured starch, amylase and buffer in the same water bath. Mix at the chosen pH and start the timer immediately. Every 30 s, transfer a fresh small sample of reaction mixture to a new iodine drop, using clean equipment so iodine is not carried into the reaction tube. Record the first sample that stays yellow-brown. Repeat the sequence with fresh reactants at each pH.

Keep temperature, enzyme/starch concentrations and volumes, buffer volume, total volume and sampling interval constant. Repeat each pH at least three times and calculate a mean. Choose pH values that span the possible peak; investigate smaller pH steps around the fastest tested condition. Shortest time identifies the fastest **tested** pH, not an exact universal optimum.

Teacher example (invented data, not a past-paper table): mean endpoint times at pH 5, 6, 7 and 8 are 180, 90, 60 and 120 s. The same initial starch quantity and endpoint make reciprocal time a useful relative-rate measure.

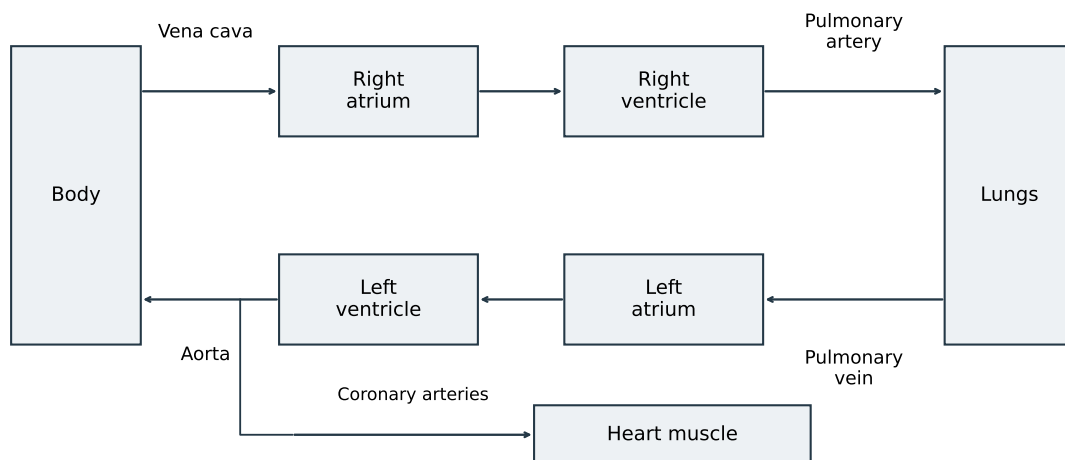
$$r = \frac{1}{t}$$

$$r = \frac{1}{60 \text{ s}} = 0.0167 \text{ s}^{-1}$$

At pH 6, $r = 1/(90 \text{ s}) = 0.0111 \text{ s}^{-1}$. The pH 7 reaction is 1.5 times as fast by this measure. Plot pH horizontally and relative rate (s^{-1}) vertically; a time graph instead has its **minimum** at the fastest tested pH. If starch still remains when observation ends, report "endpoint not reached"; do not invent an endpoint or use zero time. The 30 s sampling interval limits time resolution.

Original exam application, AQA June 2025 Paper 1H Q02.5 [4]: after 5 min the iodine stayed yellow-brown at 30 °C but became blue-black at 90 °C. At 30 °C no starch remained because amylase had broken it down; at 90 °C starch remained because amylase had denatured. Q02.6 [1] asks for a more precise best temperature: test additional temperatures between 10 and 60 °C. The four tested temperatures do not establish exactly 30 °C as the optimum.

The heart, blood vessels and blood (4.2.2.2–4.2.2.3)



Teacher flow model, not an anatomical layout: follow the arrows, not the position on this page. Body → vena cava → right atrium → right ventricle → pulmonary artery → lungs → pulmonary vein → left atrium → left ventricle → aorta → body. **Coronary arteries** branch from the aorta to supply heart muscle itself. Blood in the chambers does not directly supply all the muscle.

Double circulation: in one complete body–lung circuit blood passes through the heart twice. The right ventricle pumps to the lungs; the left pumps around the body at higher pressure and has a thicker muscular wall. Pressure/resistance, rather than “distance” alone, explains the difference. Valves prevent backflow; atria receive returning blood and ventricles pump blood out.

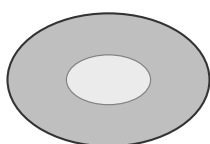
Arteries carry blood **away from** the heart; veins carry it **towards** the heart. The pulmonary artery carries deoxygenated blood; the pulmonary vein carries oxygenated blood, so oxygen content does not define an artery or vein. Cells in the **right atrium** form the natural pacemaker; an artificial pacemaker corrects an irregular rhythm.

Vessel	Structure → function
Artery	Thick muscular/elastic wall withstands high pressure; elastic recoil helps maintain flow
Vein	Wider lumen, thinner wall; valves reduce backflow at low pressure
Capillary	Wall one cell thick gives a short exchange path

Original AQA 2025 Q05.5 [1] needs “thick muscle/elastic layer”; “thick wall” alone is not enough for that mark. **2023 Q02.4 [2]** asks for two comparisons: thicker muscle and elastic layers, narrower lumen or absence of valves compared with veins. The narrower lumen is a structural comparison, not a complete explanation that it creates the heart’s pressure.

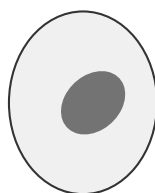
Lungs: air passes through trachea and bronchi to **alveoli**. Numerous alveoli provide large surface area; thin alveolar/capillary walls shorten the diffusion path; blood flow and ventilation maintain oxygen/carbon-dioxide gradients. Oxygen diffuses into blood; carbon dioxide diffuses into alveolar air.

Red blood cell



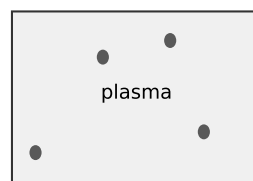
No nucleus
Biconcave shape
Binds oxygen

White blood cell



Has a nucleus
Defends against
pathogens

Plasma and platelets



Dots: platelets
Clotting limits
blood loss

Teacher cell models, not to scale. The red cell’s pale centre represents a depression, not a nucleus. **Blood** is a tissue: **plasma** carries dissolved glucose, amino acids, CO₂, urea and hormones, and distributes heat. **Red blood cells** have haemoglobin to bind oxygen, no nucleus (more room for haemoglobin) and a biconcave shape (large surface area for rapid exchange). **White blood cells** have nuclei; some engulf pathogens and others produce antibodies. **Platelets** are cell fragments involved in clotting, reducing blood loss and pathogen entry.

Coronary heart disease (4.2.2.4) and treatments

CHD: fatty material narrows the coronary arteries → reduced flow to heart muscle → reduced oxygen/glucose supply → less aerobic respiration and energy release → weaker contraction / fatigue. Exercise raises demand; restricted supply can contribute to chest pain. A percentage blockage does not imply the same percentage fall in flow.

Treatment	Benefit	Limitation / risk
Stent	Holds narrowed artery open; improves flow	Procedure risks; does not prevent new deposits
Statin	Lowers cholesterol; slows fatty deposits	Long-term medication; possible side effects
Replacement valve	Restores effective one-way flow	Surgery; mechanical valves need anticoagulants
Heart transplant	Replaces a failing heart	Donor shortage; rejection; immunosuppression
Artificial heart	Maintains pumping while awaiting transplant	Infection/clotting risk; device limitations

A faulty valve may not open fully (restricting flow) or may leak (allowing backflow). A stent treats a narrowed artery; a replacement valve treats valve disease. Transplant/artificial heart address severe pump failure, not every narrowed artery. Bypass surgery diverts flow around a blockage (accepted in the original 2023 Q02.9 scheme).

Worked evaluation, sheet Q3.2 [4]: a stent can restore flow promptly by holding the artery open, but its procedure has risks and it does not prevent future fatty deposits. Statins reduce cholesterol and slow further deposits, but need continued use and may cause side effects. The two act differently and may be used together. Choice depends on severity, urgency and patient circumstances; the question supplies no basis for claiming one is always best.

Original 2024 Q01.4–Q01.5 [1 + 2]: identify statins for reducing cholesterol; explain that a stent opens/holds open the artery, allowing more blood and oxygen/glucose to the heart muscle. “Unblocks it” alone misses the mechanism.

Original 2023 Q02.5 data: blockage 0, 10, 20, 50, 80%; flow 100, 64, 42, 8, 2 cm³/min. Describe a decreasing trend; these data are not a direct percentage-loss rule or a mathematical inverse-proportion law. **Q02.8 [6]** needs linked reasons from reduced supply through respiration/energy to contraction or symptoms. A list of separate symptoms is insufficient.

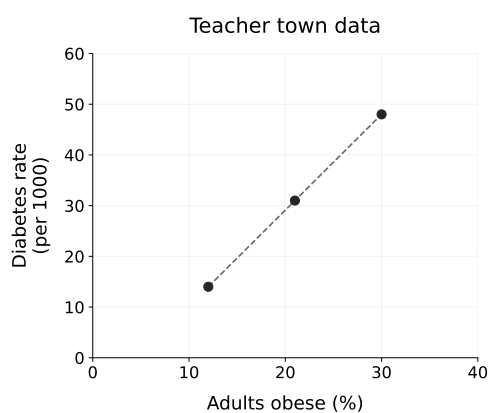
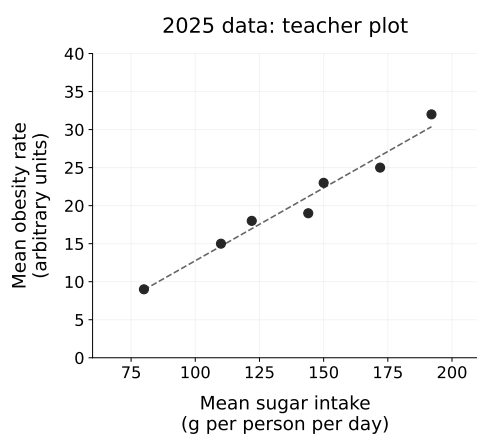
Health, lifestyle and cancer (4.2.2.5–4.2.2.7)

Health is physical and mental well-being. Communicable and non-communicable diseases, diet, stress and life situations can affect both. Disease interactions include immune defects increasing infection risk, some viruses triggering cancers, immune reactions following infection triggering allergies, and severe physical illness contributing to depression.

A **risk factor** is linked to an increased disease rate. It may be lifestyle or a substance in the body/environment. Increased risk does not mean every exposed person develops the disease.

Factor	Established risk / effect
Diet, smoking, exercise patterns	Cardiovascular disease
Obesity	Type 2 diabetes
Alcohol	Liver damage; brain effects
Smoking	Lung disease; lung cancer
Smoking / alcohol during pregnancy	Harm to unborn babies
Carcinogens, including ionising radiation	Cancer

Correlation and cause: a scatter plot shows an association, not its cause. An established causal risk factor such as obesity can contribute to Type 2 diabetes, but one three-town association does not isolate that effect from age, diet, activity, sampling or chance. Prior scientific evidence and the particular dataset are different sources of evidence. A known mechanism does not make every observed association causal.



Teacher redraw, not the original exam figure: left uses the exact AQA June 2025 Q02.2 table; right uses the teacher-authored sheet Q3.2 data. Dashed lines show fitted trends, not measured paths or proof of cause. The seven-country vertical axis is in **arbitrary units**, not percentages; the three-town diabetes axis is **per 1000**, not percent.

Reading a scatter plot: identify each axis and unit; use one point per paired observation; describe the direction with both variable names. A line of best fit represents the overall pattern and need not pass through every point or the origin. Original 2025 Q02.2 [4] requires a suitable vertical scale/label, five additional correct plots and a line of best fit. Q02.1 [1] accepts **Type 2 diabetes** as an obesity-related disease; do not give Type 1.

Worked teacher-town data (sheet Q3.2): A = 12% obese / 14 per 1000 diabetes; B = 21% / 31; C = 30% / 48. As the percentage obese rises, diabetes rate rises: positive correlation. Support the trend with two values using units.

$$R = \frac{d_C}{d_A}$$

$$R = \frac{48 \text{ per } 1000}{14 \text{ per } 1000} = 3.43$$

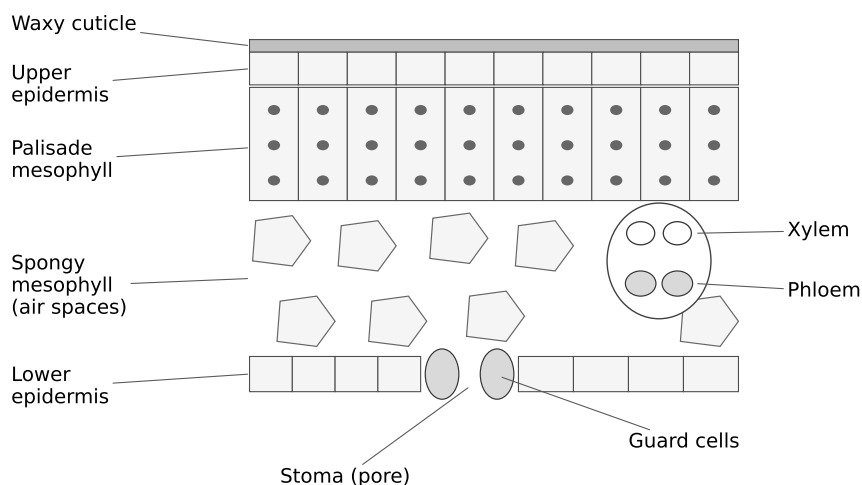
The rate is about **3.4 times** as high, not “roughly double”. The absolute increase is 34 cases per 1000, not 34 percentage points. Obesity is an established causal risk factor for Type 2 diabetes, but these three towns alone cannot prove that obesity caused the difference in their rates. Other factors and sampling must be considered.

Checking a survey claim (sheet Q3.3): compare similar groups and account for smoking, diet, exercise and age; these may affect both exposure and disease. Check sample size, selection and representativeness so a small/biased group does not stand for everyone. Check whether exposure preceded disease and whether findings repeat. Seek a plausible tested mechanism and independent review, but neither plausibility nor peer review alone proves causation. Each answer needs a check **and its reason**.

Cancer results from cell changes leading to uncontrolled growth and division. **Benign** tumours remain in one area, usually within a membrane, and do not invade other tissues. **Malignant** tumour cells invade nearby tissues and spread in blood to form secondary tumours. Compare containment/invasion and spread as separate features; size alone does not identify malignancy.

Smoking mechanism (sheet Q3.1): tobacco smoke contains **carcinogens**. These can damage/mutate DNA in lung cells; changes affecting division control can lead to uncontrolled division and malignant tumours. This explains increased risk, not certainty for every smoker or every mutation. Some cancers also have inherited genetic risk factors.

Plant tissues and transport (4.2.3)



Teacher model, not to scale: palisade dots represent chloroplasts. The leaf is an organ containing several tissues. Upper/lower epidermis protects; the waxy cuticle reduces water loss. Palisade mesophyll has many chloroplasts for photosynthesis; spongy mesophyll has air spaces for gas movement. **Guard cells** control **stomata**, balancing gas exchange and water loss. **Meristems** at growing root/shoot tips produce cells that can differentiate throughout plant life.

Original 2024 Q04.1–Q04.3: name palisade mesophyll, spongy mesophyll and meristem tissue in that order; lignin strengthens xylem; **translocation** is sugar transport. “Active transport” alone is not the name for movement through the phloem.

Feature	Xylem	Phloem
Cells	Dead; hollow, no cytoplasm	Living sieve tubes; some cytoplasm
Walls	Lignin-strengthened tubes	Perforated end walls (sieve plates)
Load	Water and mineral ions	Dissolved sugars
Direction	Roots → stems/leaves	Sources → growing/storage regions; can be up or down
Process	Transpiration stream	Translocation

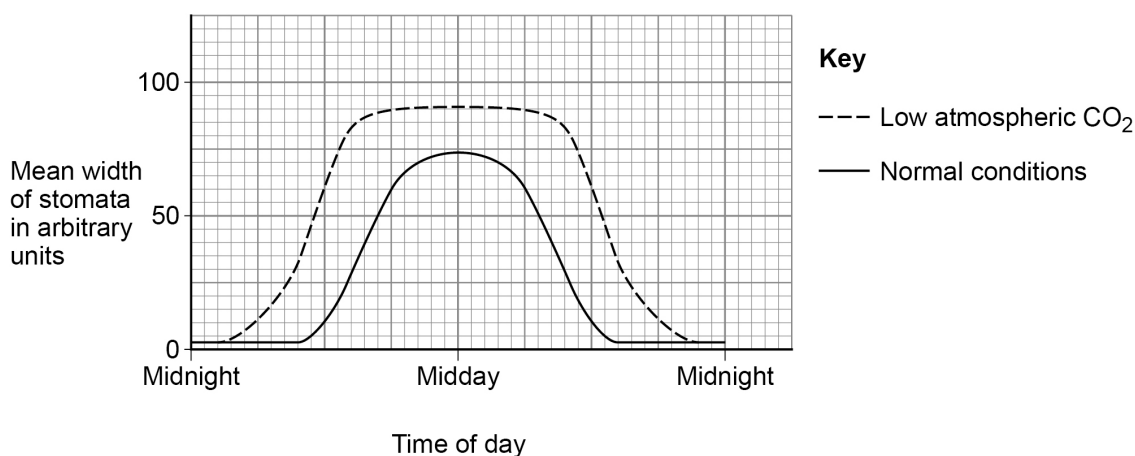
Worked comparison (sheet Q3.1 / original 2021 Q06.6 [6]): both are transport tissues with tube-like cells. Compare dead hollow lignified xylem with living phloem and sieve plates; then compare water/mineral transport upwards with dissolved-sugar transport to growing/storage tissues in either direction. A high-level original answer needs **both structure and function**, not six disconnected facts about only one tissue. “Both directions” describes transport in the tissue, not simultaneous opposite flow in every individual tube.

Root hairs: a large surface area supports uptake; water enters by osmosis through a partially permeable membrane. Mineral-ion uptake against its concentration gradient needs active transport and respiration energy. Original 2024 Q04.5 [5] extends the same logic to sugar loading: mitochondria → aerobic respiration → energy released → sugar moved low-to-high → active transport. This is a text-based teacher explanation; the paper’s referenced photograph is absent from the supplied PDF, so no identification of its missing label is inferred.

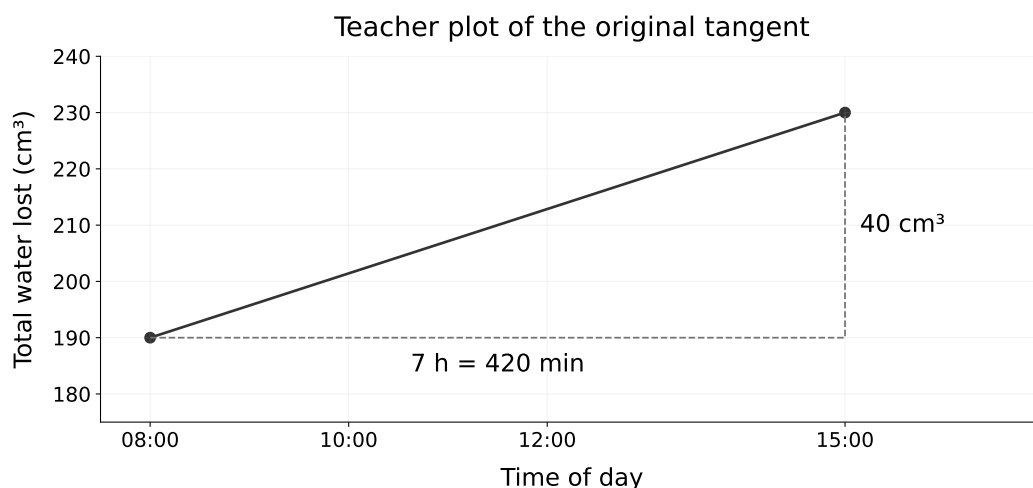
Transpiration : water evaporates from moist mesophyll cell walls into leaf air spaces, then water vapour diffuses through stomata into outside air. Water loss draws more liquid water through xylem in the transpiration stream. Roots, stem and leaves cooperate as a transport system. Keep evaporation (liquid → vapour) distinct from diffusion (vapour moving down its gradient).

Change	Usual effect, with other conditions controlled
Higher temperature	Faster evaporation / particle motion; rate rises
More air movement	Removes humid boundary air; steeper vapour gradient
More light	Stomata usually open more; rate rises
Higher humidity	Smaller vapour gradient; rate falls

These are expected effects under suitable water supply, not guarantees for every stressed plant. Original 2022 Q05.3: greatest rate in **warm, low-humidity** conditions. Sheet Q3.2: moving air removes humid air → maintains the water-vapour gradient → faster diffusion out. Sheet Q3.3: fewer upper-surface stomata can limit water loss from a more exposed surface while lower stomata still permit CO₂ uptake. This is a suggested advantage for the stated plant, not a law for every leaf orientation or species.



Original Figure 7 shows mean stomatal width in arbitrary units, not water-loss rate. Solid line = normal conditions; dashed = low atmospheric CO₂. Q05.4 [4]: near-closed stomata at night reduce water loss when little/no light is available for photosynthesis; wide opening around midday allows more CO₂ uptake for photosynthesis when light is greatest. Q05.5 [2]: in low CO₂, stomata are wider and open longer, allowing greater CO₂ entry for photosynthesis. Do not claim that low atmospheric CO₂ increases the concentration outside the leaf.



Teacher tangent plot: reproduces the given straight-line tangent's readings, not the original measured curve. Original 2021 Figure 10 plots **total water lost (cm³)** against clock time. Its height is cumulative volume; its gradient is rate. Q06.7 [4] asks for the tangent slope **at 12:00**, in cm³/min and standard form. Take two well-separated points on the tangent, even away from the contact point; do not use two points on the curved results line.

Known tangent readings: 08:00 / 190 cm³ and 15:00 / 230 cm³ (the latter is a point on the extended tangent, not a new measured result). Use $\Delta V = V_2 - V_1$ and $\Delta t = t_2 - t_1$ to calculate the changes:

$$\Delta V = 230 \text{ cm}^3 - 190 \text{ cm}^3 = 40 \text{ cm}^3$$

$$\Delta t = 15 \text{ h} - 8 \text{ h} = 7 \text{ h}$$

$$\Delta t = 7 \text{ h} \times 60 \text{ min/h} = 420 \text{ min}$$

$$r = \frac{\Delta V}{\Delta t}$$

$$r = \frac{40 \text{ cm}^3}{420 \text{ min}} = 0.0952 \text{ cm}^3/\text{min}$$

$$r = 9.52 \times 10^{-2} \text{ cm}^3/\text{min}$$

The official scheme uses 40/7 h or 40/420 min, allowing consistent tangent readings. Dividing the total volume by the clock time does not give the requested gradient. Q06.8

[2]: lower nighttime rate links mostly closed stomata to less light/lower temperature; it does not mean cumulative water loss falls overnight.

Potometer reasoning: bubble movement measures water uptake, a proxy for water loss, not an exact direct transpiration measurement (some water is retained/used). Use a water-filled sealed apparatus, avoid air entering the cut stem, allow acclimatisation, change one factor and control leaf area, time and the other environmental factors; repeat. Distance/time compares uptake rates in the same capillary; volume/time additionally needs its cross-sectional area. Do not claim every absorbed drop is immediately transpired.

Checklist before you call this topic done

- Order cells → tissues → organs → systems with one example each.
- Enzyme table: name, site, substrate, products; lock and key; denaturing.
- Bile: two functions with reasons; RP4 tests and colours; RP5 method.
- Label the heart with all five named vessels; double circulation; pacemaker.
- Artery/vein/capillary structure ↔ function; four blood components with adaptations.
- CHD: cause + each treatment's benefit and risk.
- Risk factors with causal mechanisms; benign vs malignant tumours.
- Plant tissues and their jobs; xylem vs phloem; four transpiration factors.