Hydration and nutrition

A2 · SL and HL

Equal body-mass changes do not necessarily mean equal sweat losses

Account for inputs, outputs and stored material

A2 covers water/electrolyte balance, fuelling for health and performance, and overlapping ATP-supply systems.

AHL: micronutrients, the gut microbiome, lactate accumulation and excess post-exercise oxygen consumption.

All numerical examples and plotted models are invented. Use supplied records; these are not individual drinking, diet or exercise prescriptions.

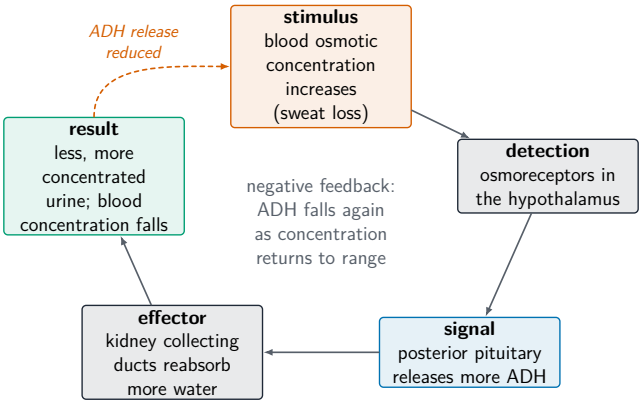
Follow material across the body boundary

Quantity / route	Process and interpretation
Inputs	Food, drink and metabolic water; **absorption** in small and large intestines
Outputs	Urine, faeces, respiratory water and skin losses
Electrolytes	Dissolved ions contribute to osmotic conditions and nerve/muscle function
Concentration	Solute relative to water; less total solute does not always mean a lower concentration

A compartment can lose solute yet become more concentrated

1. **Invented model.** 100 solute units / 10 water units $= 10$. Lose 10 solute and 2 water: $90/8 = 11.25$.
2. **Comparison.** Concentration rises 12.5% despite less total solute.
3. **Practice and limit.** $120/12 = 10 \to 108/9 = 12$: a 20% rise. These are accounting models, not clinical sodium measurements or complete body compartments.

Trace the stimulus, hormone-release site and target



The response tends to oppose the initiating concentration increase.

Separate hormone synthesis, release and target action

1. **Hypothalamus.** Detects osmotic changes and participates in thirst; neurons synthesize ADH/vasopressin.
2. **Posterior pituitary and kidney.** ADH is released from the posterior pituitary; increased action makes kidneys reabsorb more water, reducing urine water loss.
3. **Feedback.** Retention tends to oppose increased concentration. Blood-volume/pressure signals and other regulatory pathways also contribute; kidneys do not create water.

Water deficit and sodium concentration are different quantities

State / claim	Interpretation and limit
Dehydration	Loss of body water; not a synonym for every sodium abnormality
Hypernatraemia	Abnormally high blood sodium concentration
Hyponatraemia	Abnormally low blood sodium concentration; excessive hypotonic intake can contribute
More is always better	Unsupported: a sports drink does not automatically remove overdrinking risk

Define what the measure actually tells you

Indicator	Useful evidence and limitation
Body mass	Standardized net change; correct for recorded intake/output and consider clothing/error
Urine colour	Simple observation; pigments, medication, lighting and timing can confound
Concentration	State urine or blood and method; osmolarity uses volume, osmolality solvent mass
Thirst	Important cue; perception, access, conditions and timing affect interpretation

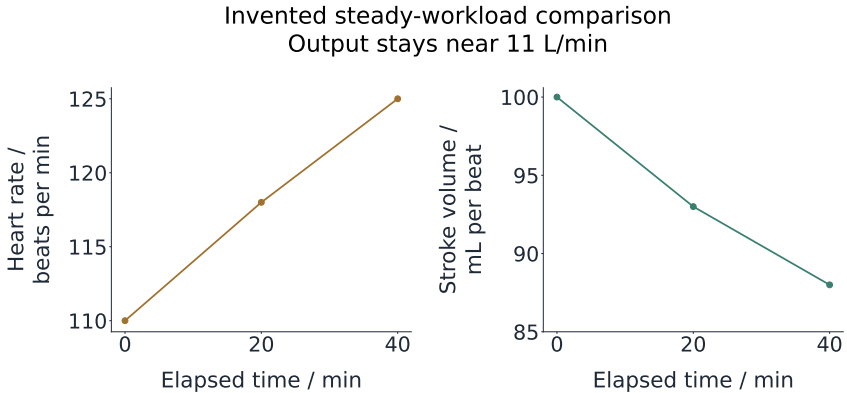
Recover sweat loss from the mass balance

1. **Given invented records.** 60.0 kg before, 59.6 after; drink 0.50 kg, urine 0.10 kg; duration 1.5 hours.
2. **Balance and rate.** Sweat $\approx 60.0 - 59.6 + 0.50 - 0.10 = 0.80$ kg. Rate $= 0.80/1.5 \approx 0.53$ kg per hour.
3. **Distinction.** Net mass fall is 0.40 kg, about 0.67% of initial mass. Add drink because it replaced mass; subtract urine as a separate output.

Convert the duration before calculating a rate

1. **Practice.** 55.0 → 54.5 kg; drink 0.40 kg; urine 0.20 kg; 45 minutes.
2. **Calculate.** Sweat ≈ 0.70 kg; 45 minutes = 0.75 hour; rate ≈ 0.93 kg per hour. Mass fall ≈ 0.91%.
3. **Limits.** Other respiratory/metabolic exchanges and clothing/balance error affect the estimate. Short-term mass loss does not establish fat loss; never induce dehydration for class data.

Interpret a rising heart rate at steady external workload



Changing heart rate alone does not establish a higher workload.

Check the product before claiming higher cardiac output

1. **Teaching model.** 110 beats per minute $\times$ 100 mL per beat $=$ 11.0 litres per minute. Later 125 $\times$ 88 gives the same output.
2. **Practice.** 120 $\times$ 90 and 135 $\times$ 80 both give 10.8 litres per minute.
3. **Mechanism.** Fluid loss and rising temperature can affect plasma volume, venous return and stroke volume. Output may be maintained or fall; the model is not universal compensation.

Visible sweat is not identical to successful cooling

Heat exchange depends on environment and the response

Temperature, humidity, airflow, clothing, exercise intensity and acclimatization affect heat exchange and losses.

Humidity can limit evaporation. Dripping sweat does less evaporative cooling than sweat that evaporates.

Drift can occur in thermoneutral or hot conditions. Compare matched workload and timing; one heart-rate reading does not diagnose hydration state.

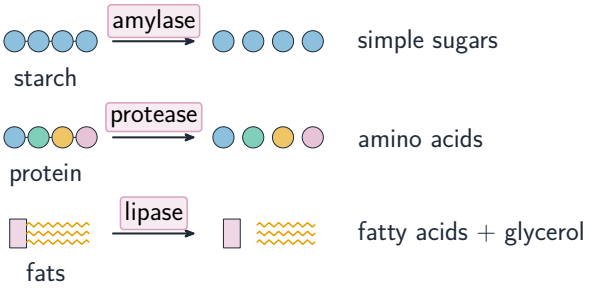
Keep chemical breakdown, crossing and transport distinct

Step	Example and boundary
Digestion	Carbohydrate enzymes form smaller sugars; proteases form peptides/amino acids; lipases form absorbable lipid products
Bile	Emulsifies lipid and supports **absorption**; it is not an enzyme
Absorption	Large area and thin epithelium support transfer; water/ions need not be digested like starch
Transport	Sugars/amino acids enter blood; much dietary lipid initially enters lymph after packaging

A food test detects a component rather than a health outcome

1. **Methods.** Benedict with approved heating: reducing sugars; iodine: starch; Biuret: protein; ethanol emulsion: lipids.
2. **Controls.** Use appropriate positive/negative controls and approved handling/disposal; keep ethanol away from flames.
3. **Inference.** A positive result does not measure the whole diet, prove uptake or show that a food causes disease.

Three digestive enzymes



Lipase: fats to fatty acids and glycerol

Fuel supply is only one macronutrient role

Macronutrient	Role and limitation
Carbohydrate	Supports rapid ATP supply; glycogen in liver and muscle serves different local/systemic roles
Lipid	Energy store and membrane roles; high energy per gram does not mean fastest ATP supply
Protein	Amino acids for tissue, enzymes and repair; can also contribute to energy metabolism
Context	Growth, age, body composition, training and individual circumstances affect needs

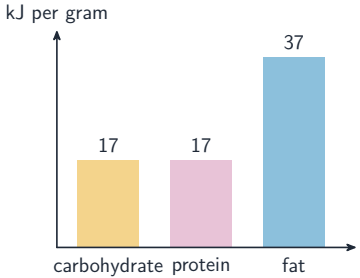
Use energy factors with an energy denominator

1. **Teaching factors.** Carbohydrate 17, protein 17 and fat 37 kJ per gram are approximate metabolizable factors, not ATP yields.
2. **Invented portion.** 60 g carbohydrate, 20 g protein, 10 g fat: 1,020 + 340 + 370 = 1,730 kJ.
3. **Fractions.** Fat energy share = 370/1,730 $\approx$ 21.4%; nutrient-mass share = 10/90 $\approx$ 11.1%. The questions and denominators differ.

Apply the same method to a different original portion

1. **Practice portion.** 40 g carbohydrate, 15 g protein and 8 g fat.
2. **Energy.** $680 + 255 + 296 = 1{,}231$ kJ; fat share $296/1{,}231 \approx 24.0\%$.
3. **Compare.** Mass share $8/63 \approx 12.7\%$. Digestibility, fibre and food-label methods can affect actual estimates; these portions are calculation examples.

Energy per gram



fat stores the most energy per gram;
carbohydrate supplies ATP faster;
protein builds and repairs first

Fat stores more than twice as much per gram

Storage and structural polysaccharides



amylose
(coiled)



amylopectin
(branched)



glycogen
(many branches)



cellulose
(straight chains)

> Glycogen's branches release glucose quickly

Match timing and availability to the task and person

A new product is not automatically a performance improvement

Before activity, meal timing and composition affect fuel availability and comfort. Poorly tolerated large, fat-rich or fibre-rich meals near activity can cause discomfort.

Longer/harder work can increase the value of carbohydrate availability. Brief activity does not change ATP systems instantly because a drink is swallowed.

Training and rest days both need physiological support. Match strategy to demands, age and individual context; control prior diet, fluid and workload in a comparison.

Energy availability is not whole-day net energy balance

1. **Defined invented model.** Availability = (intake − exercise expenditure) / fat-free mass.
2. **Calculate.** $(8{,}000 - 2{,}000)/50 = 120$ kJ per kilogram fat-free mass per day. At exercise expenditure 3,000 with intake unchanged: 100.
3. **Practice.** $(9{,}000 - 2{,}400)/55 = 120$. These values are not diagnostic cut-offs, intake targets or calculations including every daily expenditure.

Problematic energy shortage can impair health and adaptation

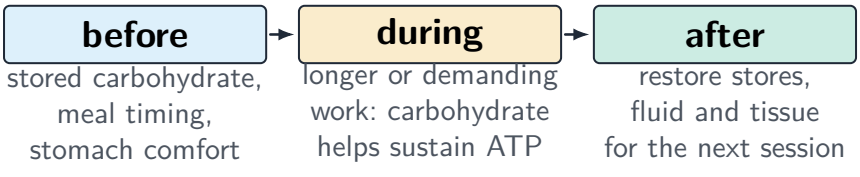
Do not diagnose RED-S from one number or a body-mass label

Problematic prolonged and/or severe low energy availability can contribute to relative energy deficiency in sport, RED-S/REDs.

Bone, hormonal/reproductive, immune, recovery and performance effects can occur in males and females; low body mass or a diagnosed eating disorder is not required.

Recording error, duration and multiple clinical findings matter. Qualified assessment is more complex than an arithmetic worksheet.

Fuelling across a session

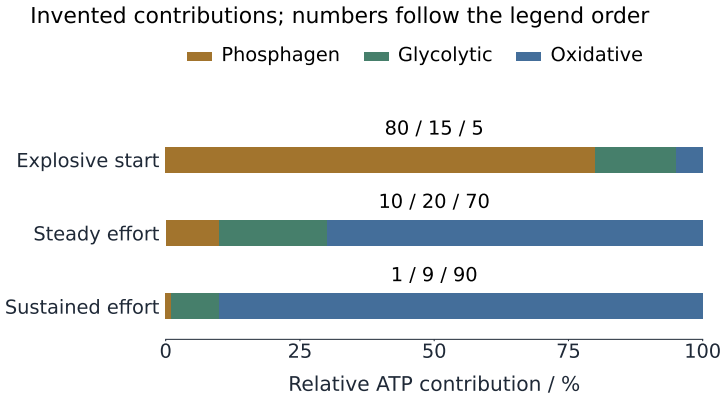


After: restore stores and recover

Compare supply rate and capacity without creating an on/off sequence

System	ATP-supply mechanism and limit
Phosphagen	PCr transfers phosphate to ADP; very rapid supply, small capacity; no direct oxygen requirement
Glycolytic	Carbohydrate breakdown in cytosol; rapid ATP; lactate formation regenerates oxidized NAD
Oxidative	Mainly mitochondrial, oxygen-dependent; carbohydrate/lipid fuels; sustained capacity, lower maximum rate

An energy continuum means changing relative contribution



Invented percentages are not fixed time cut-offs for named sports.

A relative share does not determine the absolute ATP used

1. **Explosive-start model.** Phosphagen/glycolytic/oxidative = 80/15/5%; all are represented. Oxidative metabolism supports phosphocreatine restoration in recovery.
2. **Sustained model.** 1/9/90%; a surge can still increase anaerobic contribution during an otherwise oxidative event.
3. **Context.** Intensity, duration, training, fuel and preceding work affect contributions. Lactate can be made with oxygen present and used as fuel; it is not simply waste.

Convert litres to millilitres before dividing by body mass

1. **Teaching comparison.** 3.6 litres per minute = 3,600 millilitres per minute. At 60 kg: 60; at 80 kg: 45 millilitres per kilogram per minute.
2. **Practice.** 3.0 litres per minute at 50 kg gives 60; at 75 kg gives 40.
3. **Meaning.** Changing the denominator changes the relative metric without necessarily changing absolute oxygen-delivery capacity. These comparisons do not justify a weight-loss target.

Maximum uptake is one endurance constraint among several

Economy compares cost at a matched submaximal speed

A maximal test and its protocol determine the reported maximum/peak; a demonstrated plateau and a highest observed test value are not automatically identical.

Age, body composition, physiological differences, genetics, training and lifestyle influence results with individual overlap. Sustainable intensity, pacing and conditions also affect endurance.

At the same speed, 2.4 $\rightarrow$ 2.1 litres per minute is a 12.5% lower oxygen cost; 2.0 $\rightarrow$ 1.8 is a 10% reduction. Lower submaximal cost does not by itself prove a higher maximum.

AHL: Link each nutrient to a limiting physiological process

Nutrient	Role and reasoning boundary
Iron	Haemoglobin/myoglobin support oxygen carriage and handling; extra iron is not automatic benefit
Calcium	Bone structure and signalling for contraction
Sodium/potassium	Osmotic and electrical conditions for fluid, nerves and muscle
Vitamins	Enable metabolic/tissue processes; do not supply food kilojoules like carbohydrate or fat

AHL: Adequate energy does not prove every nutrient is adequate

Assess a claim by naming the process and evidence

An adequate-energy intake can still lack a particular micronutrient; a vitamin-rich intake can still provide inadequate energy.

Identify the measured status and plausible limiting function. More supplement is not automatically better performance.

Laboratory findings and symptoms need professional interpretation. A class comparison is not a supplement prescription.

AHL: Association, microbial function and intervention are different evidence

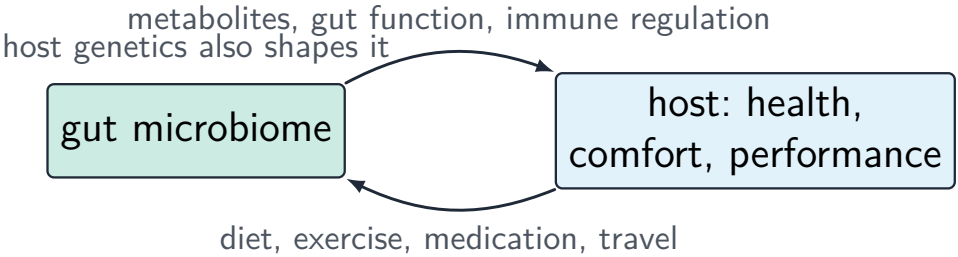
Host and microbial activity can influence each other

Genetics, diet, medication and lifestyle help shape gut microbial composition/activity. Metabolites can affect nutrient availability, gut comfort and physiological regulation.

Training itself and diet can explain differences between trained/untrained groups. Relative abundance is not an absolute cell count or direct measurement of function.

A controlled intervention needs specified strain/food, duration, prior diet, outcomes and variation. One microbial proportion does not prove improved endurance or universal probiotic benefit.

The gut microbiome

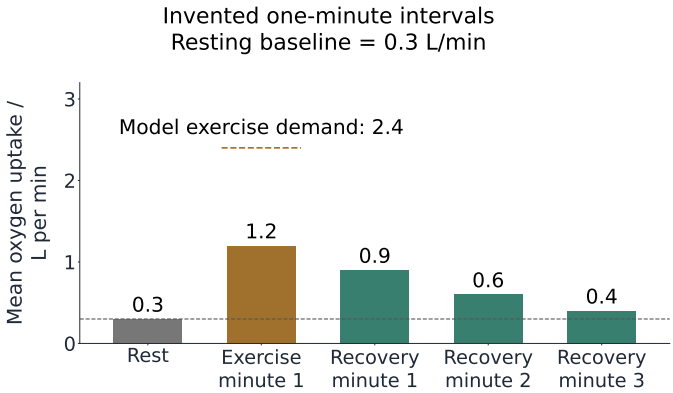


The influence runs both ways

AHL: A steady concentration can conceal rapid turnover

1. **Balance.** Lactate appearance and removal can both be nonzero and equal. A sustained concentration rise means a mismatch under the tested conditions.
2. **Invented stages.** Workload 100/150/200/250/300 W; lactate 1.0/1.2/1.8/3.4/6.0 millimoles per litre. Larger rises occur above 200 W.
3. **Inference.** Five short stages do not locate a unique maximum steady-state workload. Protocol and definition matter; oxidative ATP supply has not necessarily stopped.

AHL: Mark the baseline and define the integration intervals



Subtract resting use when calculating recovery oxygen excess.

AHL: Keep onset deficit separate from recovery uptake and excess

1. **Onset minute.** Oxygen-equivalent demand 2.4 minus mean uptake 1.2, times one minute: deficit estimate 1.2 litres.
2. **Three recovery minutes.** Mean rates 0.9, 0.6, 0.4 litres per minute give raw uptake 1.9 litres.
3. **Baseline subtraction.** Resting rate 0.3 for three minutes gives 0.9 litres. EPOC over the record $= 1.9 - 0.9 = 1.0$ litre; later excess may remain.

AHL: Recovery is more than removing a literal lactic-acid debt

Fast and slow components reflect overlapping processes

Fast recovery includes restoring oxygen stores and ATP/phosphocreatine.

Slower effects include temperature, hormones, ventilation/circulation, substrate processing and restoration of internal conditions. Lactate can be oxidized or used in other pathways.

EPOC depends on the activity and recovery conditions; it need not exactly equal onset deficit. Delayed soreness is not simply lactate retained for days.

Attempt the handout questions before checking results

State the boundary, units and denominator

Calculate concentration, sweat loss/rate, cardiac output, food energy, availability and oxygen metrics. Trace feedback and distinguish digestion from absorption.

Compare hydration measures and evaluate dietary evidence; 6 of 24 is a sample fraction, not causal proof.

AHL: explain micronutrient roles, microbiome evidence, lactate balance and baseline-subtracted EPOC.

Check the shared calculations and their interpretation

1. **Fluid and circulation.** Final model concentration 12, up 20%; sweat 0.70 kg, rate about 0.93 kg per hour; mass fall about 0.91%; both cardiac outputs 10.8 litres per minute.
2. **Fuel.** Portion 1,231 kJ; fat energy share about 24.0%, mass share about 12.7%. Defined availability 120 kJ per kilogram fat-free mass per day.
3. **Oxygen and evidence.** Relative values 60 and 40 millilitres per kilogram per minute; economy cost falls 10%; $6/24 = 25\%$. None alone establishes a clinical diagnosis or causal performance claim.

Check yourself

Connect physiological mechanisms to bounded evidence

Separate total solute from concentration, sweat from net mass change, energy share from mass share and relative from absolute oxygen uptake.

Trace ADH through hypothalamic synthesis, pituitary release and kidney action. ATP pathways overlap; a product label does not establish a benefit.

AHL: distinguish nutrient function, microbial association and intervention; steady lactate still permits turnover; EPOC requires a resting baseline.