Homeostasis

D3.3 · SL and HL

A changed environment does not require the same internal change

Follow regulated variables, feedback, signals and effectors

Shared D3.3 covers internal conditions, negative feedback, glucose regulation, diabetes and human thermoregulation.

AHL: kidney excretion and osmoregulation, filtration and reabsorption, medullary gradients, ADH and changing organ perfusion.

All numerical records are invented teaching models. They are not diagnostic cut-offs, patient records or results from an exam paper.

Name a variable and explain why regulation matters

Variable	**Connection to cellular function**
Core temperature	Reaction rates, membranes and protein structure
Blood pH	Protein interactions and enzyme function
Blood glucose	Supply for cellular metabolism
Osmotic concentration	Water movement and cell volume
Steady concentration	Can coexist with continuing input, removal and energy use

The internal environment is not the outside air

Regulation keeps conditions within suitable limits

Blood plasma and tissue fluid supply the immediate environment around cells. External conditions can vary more widely.

A set point is a regulatory reference. Suitable ranges, ordinary fluctuations, response delays and measurement uncertainty matter.

A model glucose reference of 5.0 mmol/L is not a universal personal target. Skin and core readings can differ.

Link a disturbance to an opposing response

1. **Above reference.** Detection and signalling increase effects that lower the variable.
2. **Below reference.** The response reverses direction and tends to raise the variable.
3. **Recovery.** As deviation declines, corrective drive generally declines. Negative means opposition, not a negative concentration.

One tissue can detect and signal; a response also takes time

A regulatory loop need not route every correction through the brain

Pancreatic endocrine cells can detect changes and adjust hormone secretion. Detector and signalling roles need not occupy separate organs.

Transport and cellular responses introduce delay; a peak or small overshoot can occur without loss of regulation.

Positive feedback amplifies a change, as in childbirth contractions. It completes a bounded event rather than stabilizing deviations in both directions.

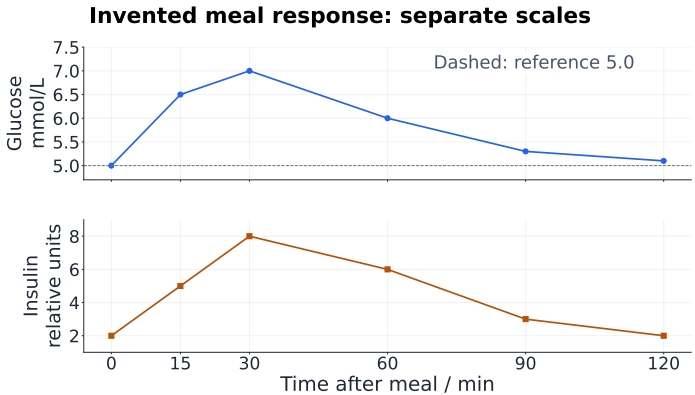
Raised glucose stimulates beta cells and target responses

1. **Detection and signal.** Beta cells release **insulin** into blood in response to raised glucose; target cells need appropriate receptors.
2. **Removal and storage.** Muscle/adipose GLUT4-mediated uptake increases; liver storage increases and glucose output decreases.
3. **Distinction.** Liver regulation is not the same GLUT4 mechanism. Insulin is a hormone regulating enzymes and transport, not the glycogen-forming enzyme.

Low glucose increases liver supply through glucagon

1. **Signal.** Pancreatic alpha cells increase **glucagon** secretion; blood carries it to targets.
2. **Liver.** Glycogen breakdown and production of glucose from appropriate non-carbohydrate substrates increase supply to blood.
3. **Boundaries.** Muscle glycogen mainly fuels the muscle. Basal hormone secretion can remain after corrective drive decreases; other hormones also influence glucose.

Compare temporal sequence using separate vertical scales



A timed association supports the model but does not isolate a cause.

Use the reference requested by the question

1. **Rise.** Model reference 5.0; peak 7.0 mmol/L: change 2.0 mmol/L; $2.0/5.0 \times 100 = 40\%$.
2. **Later fall.** $7.0 \rightarrow 5.1$: fall 1.9 mmol/L; $1.9/7.0 \times 100 \approx 27.1\%$ of the peak.
3. **Sampling.** Largest observed value is at 30 min. The true peak can lie between samples; a glucose-only trace does not measure **insulin**.

Temporal order is necessary for a proposed cause, but not sufficient

Ask what else changed and what was actually measured

Meal absorption, activity and other hormones can influence the trace. A return can follow the end of a disturbance without proving a named loop.

Standardize observation timing, activity, meal description and measurement method when evaluating supplied data.

Repeated readings from one person are not independent participants. Use supplied or approved observations rather than unapproved blood sampling.

Compare the physiological failure before the management response

Type	Failure and management principle
Type 1 failure	Usually autoimmune beta-cell loss; severe **insulin** deficiency
Type 2 failure	Resistance plus inadequate beta-cell compensation
Type 1 care	Usually replacement insulin, monitoring and nutrition/activity planning
Type 2 care	Nutrition/activity, medicines and sometimes insulin

Risk reduction and prevention are different claims

A risk factor changes probability, not certainty

Type 1 involves inherited susceptibility and immune/environmental influences. Sugar intake is not its simple cause; onset can occur at any age.

Family history, age, inactivity and excess body fat can increase type 2 risk. No single characteristic determines an individual outcome.

Appropriate activity, nutrition and weight management can reduce type 2 risk or delay onset. Lifestyle management does not replace missing insulin in type 1.

Correct the claim by naming the biological distinction

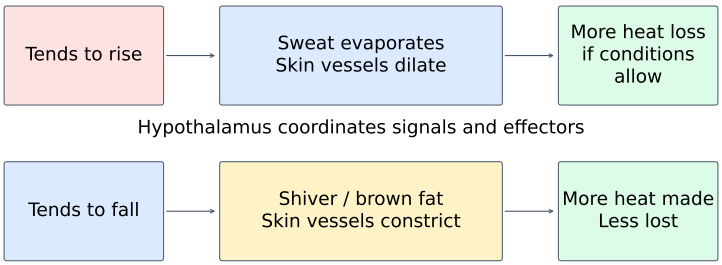
1. **Only children get type 1.** Onset can occur at any age; age alone does not determine mechanism.
2. **Type 2 never needs insulin.** Resistance can coexist with inadequate secretion; treatment may include **insulin**.
3. **Lifestyle guarantees prevention.** Risk factors are not certainties. General lifestyle measures do not reliably prevent type 1 autoimmunity; therapy can delay onset in selected high-risk people.

Place receptors, coordination and hormone sources correctly

1. **Detection.** Skin thermoreceptors provide peripheral information; hypothalamic mechanisms also respond to internal conditions.
2. **Rapid responses.** Neural pathways change sweat glands, skin arterioles and skeletal-muscle activity.
3. **Endocrine route.** Hypothalamic signals $\rightarrow$ anterior-pituitary TSH $\rightarrow$ thyroid thyroxin $\rightarrow$ altered metabolism. Pituitary does not secrete thyroxin.

Name the effector and the heat-balance consequence

Human temperature control: oppose the deviation



Physiology and behaviour can act together to oppose a deviation.

Separate altered skin flow from heat generation

Effector	How the response changes heat balance
Vasodilation	Increases skin blood flow, favouring outward transfer where conditions allow
Vasoconstriction	Reduces skin flow and heat loss near the core
Shivering	Repeated involuntary skeletal-muscle contractions raise heat production
Sweat	Evaporation removes energy; dripping is not the same process
Limit	Humid air limits evaporation; air hotter than skin can drive dry heat gain

Brown fat changes energy capture; raised hairs change insulation

Neither mechanism is the same as shivering

Brown-fat mitochondrial proton return can bypass ATP synthase, increasing energy released as heat rather than captured ATP; respiration continues.

Hair erection traps insulating air effectively in fur. Sparse human hair gives much less benefit; goosebumps are not a thick coat.

Clothing, shelter, shade, posture and activity change exchange. Brown fat is important in infants and can also be active in adults.

Calculate retained energy without inventing a temperature change

1. **Model rates.** Heat generation 160 J/s; heat loss 120 J/s.
2. **Retention.** $160 - 120 = 40$ J/s. Ten minutes $= 600$ s; $40 \times 600 = 24{,}000$ J $=$ 24 kJ.
3. **Limit.** Mass, effective heat capacity and changing rates are needed to infer a temperature rise. Steady temperature can coexist with large balanced flows.

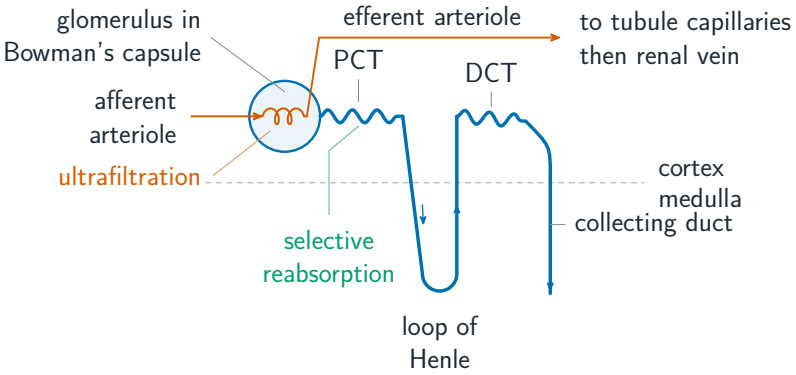
A temperature difference needs a comparable measurement

1. **Three repeats.** 36.8, 37.0, 37.2 degrees Celsius; mean 37.0.
2. **Replication.** One person measured three times remains one participant. Instrument and within-person variation can contribute.
3. **Method.** State site, device, timing, ambient conditions and activity. A changing skin reading is not automatically an equal core-temperature change.

Count particles and separate metabolic waste from digestive residue

Concept	Meaning and boundary
Excretion	Removal of metabolic waste, such as urea
Egestion	Removal of unabsorbed digestive material
Osmoregulation	Control of dissolved-particle concentration in body fluids
Units	Osmol/L counts particles, not only one ion species
Ideal NaCl model	0.10 mol/L $\rightarrow$ about 0.20 osmol/L after complete dissociation

Follow separate blood and tubular routes



Pressure-driven filtration precedes selective reabsorption.

The glomerular barrier does not decide nutritional usefulness

1. **Filter.** Glomerular hydrostatic pressure favours passage of water and small solutes into Bowman's capsule; cells and most large proteins remain in blood.
2. **PCT.** Microvilli supply area; pumps maintain sodium gradients that support useful-solute cotransport. Water follows appropriate gradients.
3. **Distinction.** Filtrate includes glucose as well as wastes. Not every reabsorbed substance is actively pumped; useful-solute transport has finite capacity.

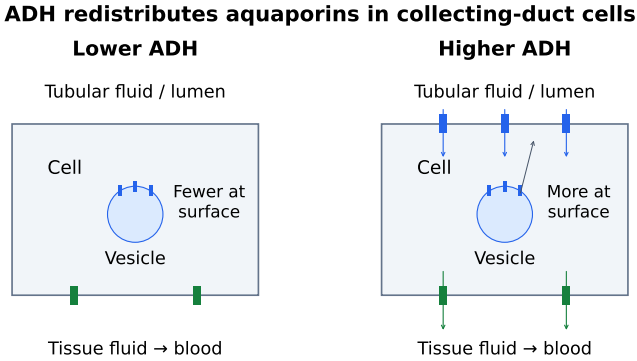
A large filtered volume is repeated processing

1. **Model.** 180 L water filtered per day; 1.5 L/day urine.
2. **Balance.** $180 - 1.5 = 178.5$ L/day reabsorbed; $178.5/180 \times 100 \approx 99.17\%$.
3. **Boundary.** This is not a 180 L/day drinking requirement. For a specified substance with no secretion, excreted amount = filtered − reabsorbed.

Connect segment permeability with collecting-duct reabsorption

1. **Ascending limb.** Relatively impermeable to water; sodium transport, including active transport in the thick segment, supports high medullary osmotic concentration.
2. **Descending limb.** Water can leave more readily; opposing flows and differing permeability support the gradient.
3. **Collecting duct.** Water reabsorption needs both a suitable gradient and membrane permeability. ATP does not directly pump water through aquaporins.

Find the membrane facing tubular fluid before placing aquaporins



ADH changes permeability; the osmotic gradient directs water.

Follow a rise in blood osmotic concentration to an opposing response

1. **Detect and signal.** Hypothalamic osmoreceptors respond; ADH synthesized in hypothalamic neurons is released from posterior pituitary.
2. **Target.** Aquaporin-2 moves from vesicles into the luminal membrane of collecting-duct cells. Water permeability rises.
3. **Response.** More water reabsorbed down the appropriate gradient; smaller, generally more concentrated urine output opposes raised blood concentration.

Lower ADH changes channels and water flow in the other direction

1. **Low osmotic concentration.** Reduced ADH drive favours return of luminal channels to intracellular vesicles.
2. **Consequence.** Less regulated water reabsorption; a larger volume of more dilute urine can result. Blood-volume and pressure signals also influence ADH.
3. **Conservation.** Urea excretion fixed at 0.60 mmol/hour: 0.120 L/hour gives 5.0 mmol/L; 0.030 L/hour gives 20 mmol/L. Same amount, less water.

Invented allocations illustrate different denominators

Region	Sleep / wakeful rest / vigorous exercise, L/min
Skeletal muscle	0.90 / 1.00 / 16.00
Gut	1.10 / 1.25 / 0.60
Brain	0.70 / 0.75 / 0.75
Kidneys	1.00 / 1.10 / 0.40
Other / total	0.80 / 0.90 / 2.25; totals 4.50 / 5.00 / 20.00

A smaller fraction can coexist with unchanged absolute flow

1. **Brain.** $0.75/5.00 = 15\%$ at rest; $0.75/20.00 = 3.75\%$ exercising; both 0.75 L/min.
2. **Muscle.** $1/5 = 20\%$; $16/20 = 80\%$; absolute flow increases sixteenfold.
3. **Kidneys and limits.** $1.10 \to 0.40$ is -0.70 L/min. Reduced flow does not mean zero flow or directly specify filtration or urine production; activity and sleep stage alter patterns.

Attempt the handout questions before checking these results

Trace a causal chain and state a bounded conclusion

Explain internal conditions, bidirectional feedback, glucose hormones, diabetes and temperature effectors.

Interpret the measured time course; calculate percentage changes and retained energy, then evaluate measurement and causation limits.

AHL: separate filtration/reabsorption, gradient/permeability, hormone synthesis/release and absolute flow/fraction of output.

Check the shared numerical results

1. **Glucose practice.** 4.8 $\rightarrow$ 6.0 gives $+1.2$ mmol/L, $+25\%$ of initial. 6.0 $\rightarrow$ 5.0 gives -1.0 mmol/L, a 16.7% fall from peak.
2. **Heat practice.** $180 - 130 = 50$ J/s retained; eight minutes $= 480$ s; energy 24,000 J $= 24$ kJ. Temperature rise needs further quantities.
3. **Temperature repeats.** Mean 37.0 degrees Celsius. Three readings are not three independent participants; compare measurement method and conditions.

AHL answers: water, urea and perfusion

1. **Water.** $150 - 1.2 = 148.8$ L/day reabsorbed, 99.2% of filtered volume.
2. **Urea.** 5.0 versus 20 mmol/L at the two flow rates; constant 0.60 mmol/hour excreted.
3. **Perfusion.** Brain 15% $\to$ 3.75% without absolute change; muscle 20% $\to$ 80%, sixteenfold absolute increase; renal change -0.70 L/min.

Check yourself

A response must connect the detector, target and corrective direction

Distinguish a hormone from an enzyme, a response delay from complete regulatory failure, and a percentage denominator from the measured value.

Separate heat energy from temperature change; explain environmental limits on physiological heat loss.

AHL: keep blood and filtrate routes separate; use both gradient and permeability to explain water reabsorption.