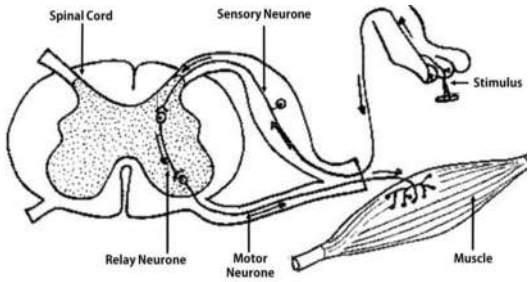
Responding to Stimuli

The Basics

- **Organisms are adapted to respond to external and internal stimuli.**
- **Receptors detect stimuli, and effectors respond.**
- **Plants can respond to light and gravity by IAA activity.**
- **The heart controls its own heartbeat, but can be helped by the brain when needed.**

Reflexes

- Simple reflexes allow an organism to move away from a harmful stimulus as quickly as possible.
- They consist of a receptor, sensory neuron, relay neuron and motor neuron that stimulate an effector.



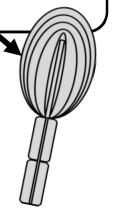
Experiment

Small invertebrates can be used to test movement. Choice chambers or mazes can be used to see how an animal moves towards or against a stimulus.

Pacinian corpuscle

Pacinian corpuscles are receptors that detect pressure:

- They **only** detect pressure – all receptors are specific to one type of stimulus.
- They have many layers, and when pressed, sodium channels in them are distorted.
- Distortion causes the influx of sodium, causes a flooding of positive charge in the corpuscle – termed a **generator potential** – which goes on to cause a nerve signal.



Kineses and taxes

- Movement either towards or away from a stimulus.

Kinesis is non-directional movement, while **taxis is directional movement.**

- Kinesis tends to be random, with animals moving around randomly until they are comfortable.
- Taxis can be imagined as a plant moving towards light.

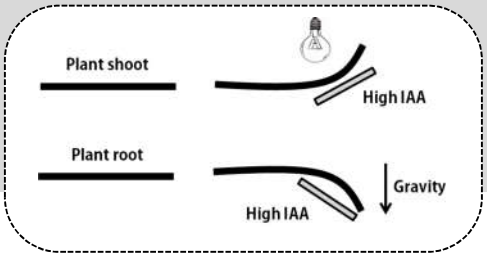
Tropism

- Tropism is a growth response to an external stimulus.
- In plants, this is typically divided into **photo**tropsim and **gravi**tropism.
- Tropisms can be positive (towards the stimulus) or negative (away from the stimulus).
- Tropism in plants is controlled largely by **growth factors**.

Indoleacetic acid (IAA)

Mainly involved in elongating plant parts – either roots or shoots.

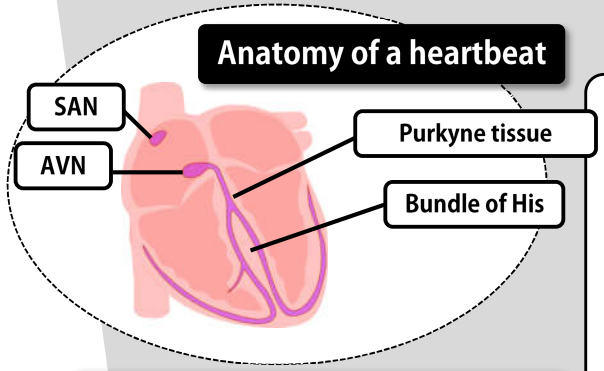
- IAA encourages growth, and accumulates on dark parts of the shoot.
  As a result, the shoot grows towards light.
- In roots, IAA is produced by root tip cells and actively transported along the root.
  The **amyloplast** is full of heavy starch and pulls the root down as a result of gravity.
  IAA inhibits elongation, causing the root to change direction and grow downwards.



Internal response – the heart

- The heart muscle is **myogenic** – it contracts of its own free will!
- Heart rhythm is controlled by the sinoatrial node (SAN).
- The SAN creates regular rhythms of electrical activity – a pacemaker.
- SAN passes signal through the atria but the ventricles are insulated by a layer of fibrous tissue.
- The atrioventricular node (AVN) detects electrical activity in the atria.
- AVN triggers activity in the Purkyne tissue. Signal is passed down bundles of His to base of ventricles, causing contraction and ejecting blood.
- Injury to these parts can have a significant impact on heart contraction, such as following a heart attack.

Anatomy of a heartbeat

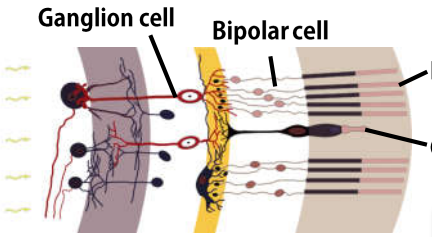


Exam tip... *Which takes longest to say?*

Parasympathetic slooooooows down the heart.
Sympathetic is quick to say – speeds up the heart.

Externally controlling the heart

- Sometimes, heart rate needs to change rapidly.
- The brain controls this – specifically the medulla.
- The medulla receives signals from receptors in the aorta, and carotid arteries.
- They can detect blood pressure and composition.
- There is an **acceleratory centre** and an **inhibitory centre**.
- As expected, acceleratory speeds up, inhibitory slows down.
- The nervous system controlling this is quite distinct.
- It is called the **autonomic nervous system**.
  It is split into two parts:
  1. The sympathetic part carries acceleratory signals.
  2. The **parasympathetic** part carries inhibitory signals.



The human retina

- Rods and cones contain pigments that absorb light, and result in generator potentials being formed.
- Rods detect low light levels, but cannot distinguish between colours.
- Cones are less sensitive, but are sensitive to colour. The human retina contains three types of cones, sensitive to red, blue and green light.
- Most cones are located in the centre of the retina, at the fovea. This area has the greatest visual acuity – the ability to see clearly.

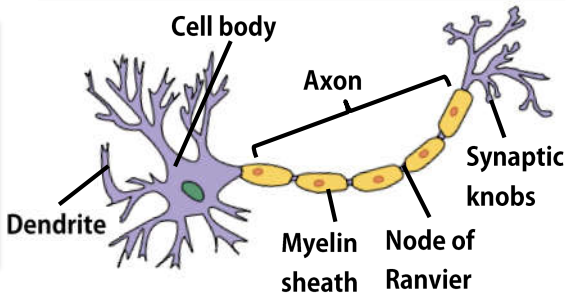
Word cloud

Kinesis
Taxis
Tropism
Gravitropism
Phototropism
IAA
Reflex
SAN/AVN
Pacinian corpuscle
Sympathetic
Parasympathetic

Nervous Control

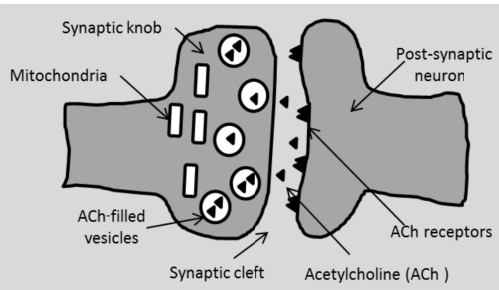
The Basics

- Neurons carry impulses across the body.
- Action potentials pass along neurons, and rely on charge differences inside and outside of cells.
- Myelin helps to speed up impulses by enforcing saltatory conduction.
- Neurons are separated by synapses which control where a signal is spread through the nervous system.



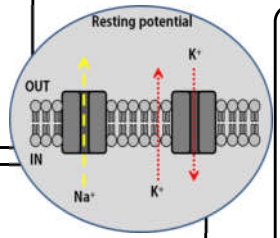
Why bother with synapses?

Synapses slow an impulse as diffusion is slower than the impulse. However, synapses keep control in the nervous system and ensure the impulse only moves in one direction – only the synaptic knob contains vesicles with neurotransmitters = ***unidirectionality***



Impulses

- Waves of electrical activity that pass along the neuron.
- Impulses are largely controlled by sodium-potassium pumps in the cell membrane.



Na^+/K^+ pumps

- Move 3 Na^+ out and 2 K^+ in, through an active process.
- Membrane permeable to K^+ which diffuses back out.
- More positive ions move out than are moved back in.
- **Inside is negatively charged relative to outside.**
- This is the **resting potential**, it measures **–70mV** in the neuron.

Stronger action potentials?

- A greater stimulus does not cause a greater action potential.
- Increasing the stimulus = increased frequency of action potentials.
- We say these impulses are **discrete**.
- Between each action potential, there is a break called a **refractory period**.
- This is the period where the neuron is repolarising and cannot be depolarised again.
- Similarly, you cannot have a 'small' action potential.
- Once the threshold potential is reached, an action potential is triggered – the **all-or-nothing principle**.

The Synapse

Neurons are not connected to each other – nor do they touch.
A small (~20 nm) synaptic cleft exists between them.
When a signal passes along a neuron, it reaches a synapse and releases neurotransmitters, such as acetylcholine (ACh).

- When an action potential reaches the synaptic knob, Ca^{2+} causes the vesicles to bind, and release ACh into the synaptic cleft.
- The post-synaptic membrane has receptors specific to ACh.
- They cause sodium channels to open.
- Na^+ enters the neuron, potentially triggering an action potential.

Action potential

When triggered by receptors on the membrane, a small change in the polarity of the neuron occurs. This must reach a **threshold potential**.

Once reached, Na^+ channels are opened, allowing Na^+ to flood back into the cell.

Rapidly, the inside of the neurone becomes positively charged (+30mV).

This is the **action potential**.

A positively charged neuron is considered **depolarised**.

- At peak depolarisation (+30 mV), the Na^+ channels close.
- **Repolarisation** causes K^+ channels to open, allowing positively charged ions to exit the cell, restoring the neuron's charge.
- More K^+ diffuses out than required.
- Period of **hyperpolarisation** where the polarity drops below resting potential.

Myelin and saltatory conduction

- Remember **myelin** – the fatty sheath around an axon?
- Myelin plays a crucial role in speeding up axon transmission.
- By covering the axon, ions can only cross the membrane where there is no myelin – at **nodes of Ranvier**.
- This means that the action potential 'jumps' from one node of Ranvier to the next, speeding the impulse.

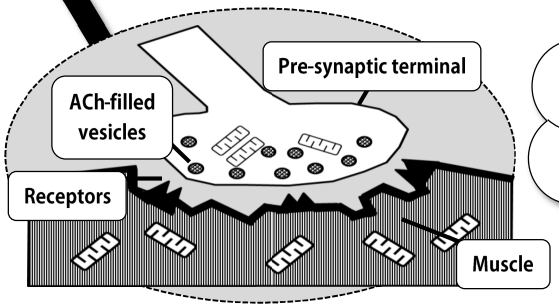
Summation and inhibition

- A single synapse acting on a neuron might not be enough to reach the threshold – multiple synaptic signals might be required – **summation**.
- If several impulses are released from the same neuron in a very short time, it is called **temporal** (for time) **summation**.
- If multiple different synapses empty on to a single post-synaptic neurone it is called **spatial summation**.
- Not all neurotransmitters cause action potentials – some can inactivate the post-synaptic neurone by releasing an inhibitor.
- These synapses are called **inhibitory** synapses.
- Inhibitory synapses are very important to develop fine or specific movements.

Exam tip – *'Saltatory' comes from the Latin word 'saltare', which means jump – just like the action potential in this kind of conduction.*

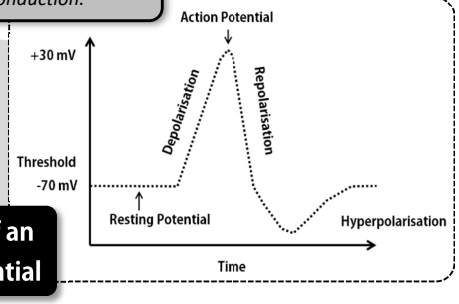
Neuromuscular junctions

A synapse can either link onto neurons or muscle fibres. All muscles respond to ACh, and when a synapse releases ACh onto a muscle, it is stimulated.



Impulse speed – *Besides being myelinated, thicker axons and increased temperature speed up nerve impulses*

Anatomy of an action potential



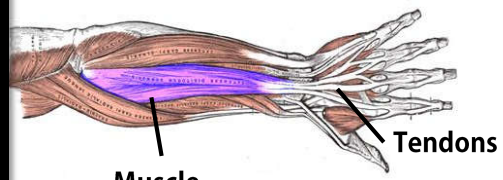
Word cloud

Impulse
Action potential
Refractory period
Resting potential
All-or-nothing principle
Myelin sheath
Sodium-potassium pump
Saltatory conduction
Node of Ranvier
Threshold potential
Depolarisation
Repolarisation

Muscles and Movement

The Basics

- Muscles work by pulling on an incompressible skeleton.
- Tendons transfer muscle pull on to bone.
- There are two types of muscle fibre: fast and slow.
- During contraction of muscle, thin filaments (actin) slide across thicker filaments (myosin).
- Calcium causes the contraction to start, and hydrolysis of ATP is vital for release of the filaments.
- Phosphocreatine provides ATP when stocks are low.

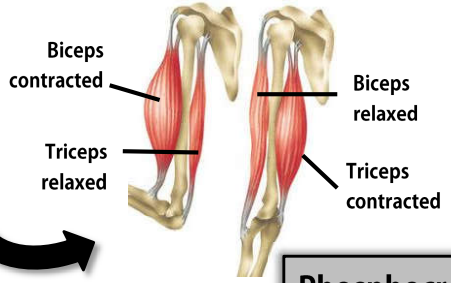


Myofibrils

- Thin filaments and thick filaments of fibrous protein make up the lines in a myofibril.
- The thin (grey) filaments are actin, the thick (black) filaments are myosin.
- The myosin are bound to the M-Line. They do not move.
- The actin filaments are bound to the Z-Line. They move during muscle contraction.
- The bands represent the filaments present. The I Band only contains actin – it looks light, while the A band contains actin and myosin and looks dark. The H zone only contains heavy chains, and is the halfway zone.

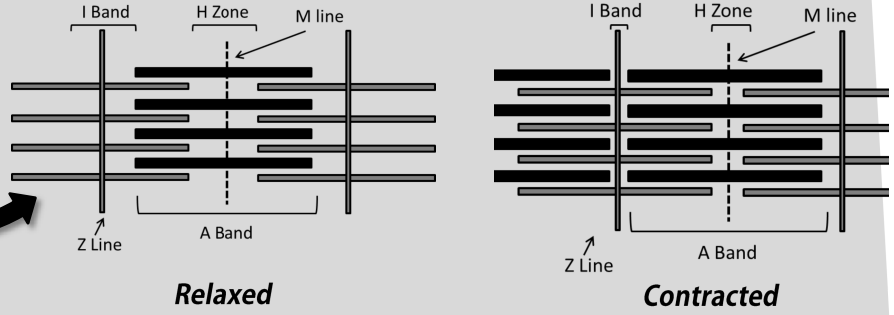
Making movements

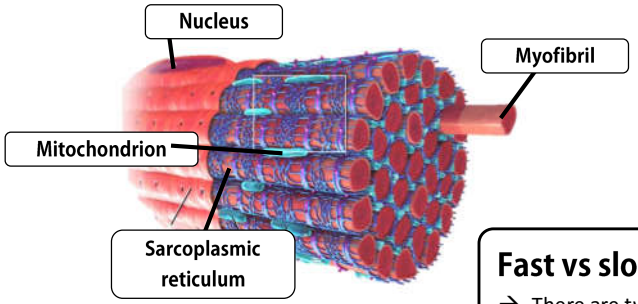
- Moving requires muscles to act on the skeleton and the two are connected by tendons.
- Muscles work in **antagonistic** pairs, which means as one muscle **contracts**, another muscle **relaxes** (consider the biceps and the triceps example).



Myofibrils – *how muscles contract*

- Fibres are made of many fibrils. On a light microscope they look stripy.
- Each fibril can be divided into distinct sections according to these stripes…





Phosphocreatine

Muscles only hold a small amount of ATP, and respiration can be too slow for some activity. However, muscles store phosphocreatine:

ADP + Phosphocreatine → ATP + creatine

This allows a muscle to produce a little more energy when ATP stocks are low.

Sliding-filament hypothesis

- During muscle contraction, **actin** and **myosin** filaments are believed to slide across each other.
- Evidence for this theory can be seen by looking at the **band pattern** of relaxed and contracted muscles.
- The **A band** constantly stays the same - the dark myosin does not move.
- The **I band**, however, shrinks as the actin slides across the myosin fibres during contraction.

Structure of a muscle

- Muscle cells are very different to all other body cells. They are called **muscle fibres** due to their long, fibre-like length.
- They also have many nuclei and the terms used for parts of the cell are different too.
- The cell membrane of a muscle cell is called **sarcolemma**, and the cytoplasm is called **sarcoplasm**.
- Each muscle cell is full of fibres called **myofibrils** (responsible for muscle contraction), and each myofibril is surrounded by **sarcoplasmic reticulum**.

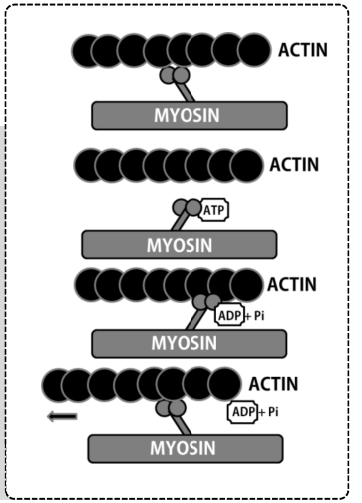
Fast vs slow

- There are two types of muscle fibre – **fast** and **slow**.
- The **slow** muscles in your **back** or your **neck** rarely move very quickly – they work to keep your head upright or your back straight for **long periods of time**.
- **Fast** muscle fibres dominate in your **legs** and **arms**, where short, **rapid** movements are needed.
- Fast fibres only have a small blood supply, and cannot work for a long time, while slow fibres have extensive blood supplies and can work successfully for extended times.

Actinomyosin bridges

It is slightly more complicated than filaments simply sliding!

- Myosin is dotted with '**heads**' along its length.
- The **sarcoplasmic reticulum releases Ca^{2+}** on to the filaments causing **tropomyosin** (on the actin) to reveal the myosin-binding sites.
- The myosin heads then bind to the actin sites to form **actinomyosin bridges**.
- The myosin head molecules then **bend**, pulling the actin filament. The bending movement causes an ATP molecule to become hydrolysed to ADP and Pi.
- A new **ATP** molecule then binds to the myosin molecule, **releasing** it from the actin, and allowing the heads to bind further along the actin.



Word cloud

Contraction
Antagonistic action
Myofibril
Sarcomere
Actin
Myosin
Tropomyosin
Phosphocreatine
A band
I band

Important! *ATP does not contract muscles – it causes a muscle to relax by releasing the myosin head from the actin, and allows active transport of Ca^{2+} in contraction.*

Homeostasis

The Basics

- The body must maintain internal conditions – this is done by homeostasis.
- Glucose levels are maintained by insulin and glucagon acting against each other.
- Adrenaline also controls glucose by a secondary messenger.
- Osmoregulation is by ADH from the pituitary controlling the activity of nephrons in the kidney.

Low blood glucose

- Detected by **alpha (α) cells** which secrete glucagon.
- Glucagon converts glycogen in the liver to glucose (**glycogenolysis**).
- Produce glucose from other substrates, such as glycerol and amino acids (**gluconeogenesis**).

Word cloud: Homeostasis, Glucose, β cells, α cells, Insulin, Glycogen, Glucagon, Glycogenesis, Gluconeogenesis, Glycogenolysis, Diabetes, Osmoregulation, Ultrafiltration, Reabsorption, Convoluted tubule, Loop of Henle, ADH, Aquaporin

Osmoregulation

Control of water potential of blood, by the pituitary gland and hypothalamus in the brain and the kidneys with neuronal and hormonal control.

Homeostasis – Systems for controlling internal

systems maintain homeostasis. Maintains normal pH, temperature, glucose, etc.

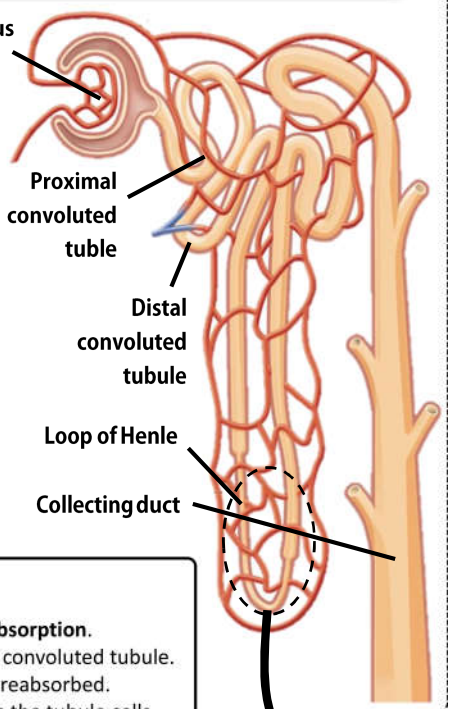
	Too high	Too low
Blood glucose	Respiration fails and cells die	Water potential of blood changes, causing cells to swell
Blood temperature	Enzymes begin to denature	Enzymes not as efficient
Blood pH	Enzymes begin to denature	Enzymes begin to denature

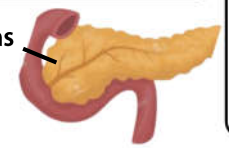
Adrenaline – secondary messengers

- Like glucagon, adrenaline causes a release of glucose into the blood.
- It binds to a **receptor**, and causes the activation of **adenyl cyclase**.
- Adenyl cyclase converts ATP to cyclic AMP (**cAMP**).
- cAMP binds to **protein kinase** and this converts **glycogen** to **glucose phosphate**.
- Not the adrenaline/glucagon but the cAMP that causes effect – this makes it a **secondary** messenger system.

The nephron

- Functional part of the kidney.
- Blood from the renal artery passes through an afferent tubule arteriole.
- Enters **glomerulus,** a ball of vessels at the start of the nephron.
- **High blood pressure** forces substances out of the blood at the glomerulus (**ultrafiltration**).
- Small substances removed from the blood.
- Reabsorbed at the **proximal convoluted tubule**.





Diabetes

Inability to regulate glucose efficiently.

	Type I Diabetes	Type II Diabetes
Typical patient	Starts early in life.	Normally adults.
Cause	Autoimmune – immune cells attack pancreas.	Often associated with obesity – lack of sensitivity to insulin.
Treatment	Insulin injection and dietary control.	Dietary control and exercise.

High blood glucose

High blood glucose levels are detected by the **β-cells** of the pancreas, located in the Islets of Langerhans. There, insulin is produced and enters the bloodstream, causing cells to move **glucose channels** to their surface. Fat cells, muscle cells and liver cells have the most glucose channels, and take up the most glucose:

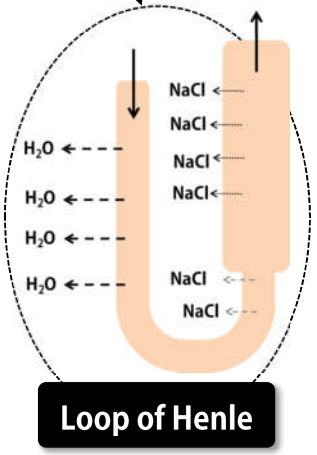
- Liver cells rapidly convert glucose to glucose phosphate, which is then converted to glycogen (**glycogenesis**).
- Muscle cells perform glycogenesis so that they have a ready supply for respiration.
- Fat cells manufacture fatty acids and glycerol from excess glucose.

Reabsorption

- Glucose, amino acids and inorganic ions need **reabsorption**.
- **Actively transported** into cells lining the proximal convoluted tubule.
- **Water potential** of the cells gets lowered – water reabsorbed.
- The useful substances **diffuse** into the blood from the tubule cells.

Loop of Henle

- **Descending limb** = **permeable** to water, but not Na^+ or Cl^-.
- Water passes out by **osmosis**.
- **Ascending** limb **impermeable** to water.
- NaCl **diffuses** out of lower part.
- NaCl **actively transported** from thick part.

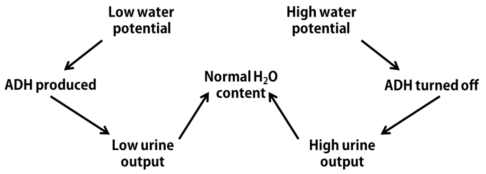


Experiment

Can identify sugar in urine with a colour-based test, such as Benedict's test. Colorimetry can be performed to identify the sugar concentration alongside a calibration curve of known concentrations.

Negative feedback

Much of homeostasis is controlled by negative feedback loops. It is a continuous loop of events that constantly modifies the internal environment.



Antidiuretic hormone

Water potential is ultimately controlled by ADH. Osmoreceptors in the hypothalamus sense the water potential of blood and control release of ADH from the posterior pituitary gland.

ADH and the distal convoluted tubule

Drop in water potential causes ADH release.

- ADH increases permeability of distal convoluted tubule and collecting duct.
- ADH binds to **aquaporins**, causing them to move to cell surface.
- Aquaporins allow water to cross membrane, reabsorbing water.
- Restoration of water potential inhibits ADH release.
- Fewer aquaporins, less water reabsorbed.