

CAIE Biology A-level

Topic 15: Control and coordination

Notes

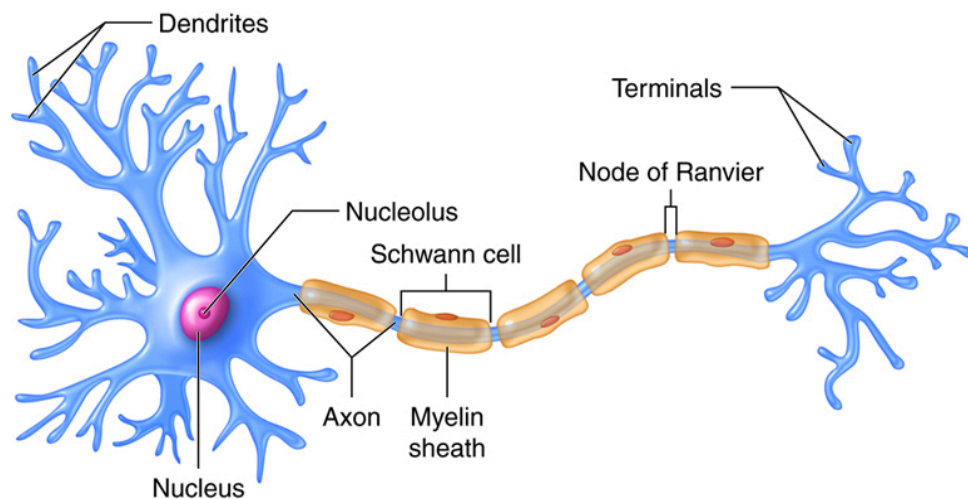
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Neuron structure

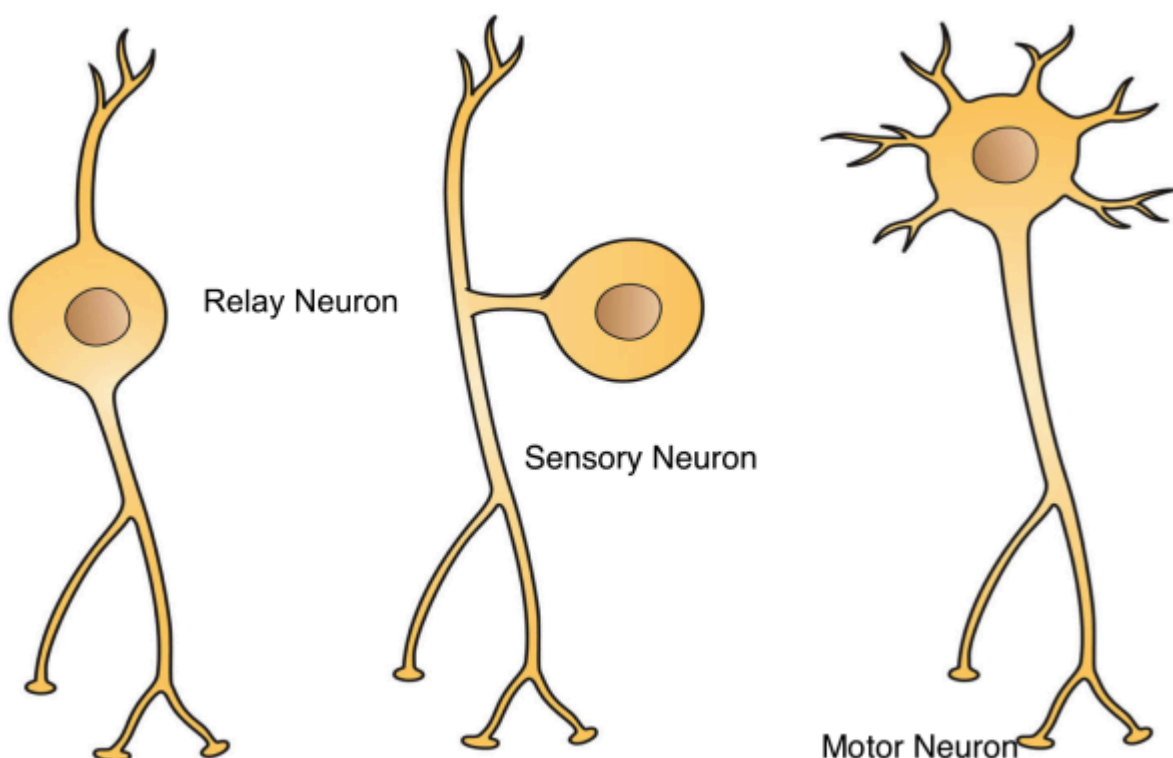
The nerve cells called **neurones** play an **important role in coordinating communication** within the nervous system. The **stimulus** is first detected by **sensory receptor cells** and transmitted to the sensory neurone.

The **structure of neurones is similar**, as they all have a **cell body composed of the nucleus** as well as **organelles such as mitochondria within the cytoplasm**. Apart from the essential components, they also contain extensions called **dendrites** involved in conducting impulses towards the cell body, as well as **axons** which conduct them in the opposite direction, that is away from the cell body.



There are three types of neurones—**sensory, motor, and relay**—which differ in function and in the position of the cell body.

- **Motor neurones** are involved in transmitting electrical signals from the central nervous system to muscles and glands in the body.
- **Sensory neurones** transmit impulses from receptors to the central nervous system.
- **Intermediate/Relay neurones** are located within the central nervous system and transmit the electrical impulses from sensory neurones to motor neurones.



The structure of neurones, such as the length of axons and the **polarised** nature of the neurone membrane in the resting state, enables the neurones to carry electrical impulses called **action potentials**.

The speed at which the electrical potential is carried can be increased with the help of the **myelin sheath**, which insulates axons. The myelin sheath is produced by **Schwann cells**. The mechanism by which the speed is increased is known as **saltatory conduction** where the action potential jumps between gaps in the myelin sheath called **nodes of Ranvier**.



Nerve impulse conduction

Nerve cells are **polarised in their resting state**. This occurs as a result of **an imbalance in the distribution of sodium and potassium ions**, thus giving the inside of the nerve cell a negative charge in comparison to the external environment. As a result of the polarisation, there is a difference in the voltage across the neurone membrane, with a value of -70 mV known as the **resting potential**.

- The resting potential is established and maintained by the **sodium-potassium pump**, which uses ATP to actively transport **three sodium ions** out of the neurone for every **two potassium ions** moved in. This creates a **steep concentration gradient for sodium**, as the membrane has extremely low permeability to **sodium ions**.
- The membrane is more permeable to **potassium ions than sodium ions**. While **potassium** ions are pumped into the cytoplasm, the membrane contains "leak" **potassium ion channels** that remain **open**, allowing potassium to **diffuse back out** of the neurone **down its concentration gradient**. This constant **loss of positive ions** results in a net **positive charge outside** the neurone and a **negative charge inside**, typically measured at **-70 mV** .

Upon stimulation, the neurone cell membrane becomes **depolarised**. This occurs as follows:

- Initially, a stimulus triggers the opening of some **voltage-gated sodium ion channels**, increasing the **membrane's permeability to sodium ions**. Sodium ions diffuse into the axon **down their electrochemical gradient**, making the **inside of the neurone less negative**—a process called **depolarisation**.
- If the potential difference reaches the threshold potential (**approximately -55 mV**), a positive feedback loop causes the remaining **voltage-gated sodium channels to open**. This results in a **rapid influx of sodium ions** until the potential difference reaches **$+30\text{ mV}$** .
- At $+30\text{ mV}$, the voltage-gated **sodium ion channels close** (become inactivated) and voltage-gated **potassium ion channels open**. Potassium ions **diffuse out** of the neurone **down their concentration gradient**, causing **repolarisation** of the membrane.
- Because these potassium channels are **slow to close**, an **excess of potassium ions leaves the cell**, causing the potential difference to become **more negative than the resting potential**; this is known as **hyperpolarisation**.
- Finally, the sodium-potassium pump **restores the ion distribution**, returning the neurone to its **resting potential of -70 mV** .

The action potential travels along the neurone as a **wave of depolarisation** where the **sodium ions move to the adjacent resting region**. Here, they **trigger a change in potential difference**, thus **stimulating another action potential**. There is a short period during which the neurone membrane cannot be excited as the sodium channels enter the recovery stage. This period is known as the **refractory period** and serves an important role in ensuring that the **action potentials can only pass in one direction as discrete signals**. It also **limits the number of impulses** that can be sent.



Action potentials in example

The following sequence of events describes how **human taste buds** can lead to an **action potential** being generated, and how the action potential can be subsequently **propagated** along the sensory neuron:

1. A dissolved food molecule (**tastant**) acts as a **stimulus** and reaches the taste pore to **bind to receptor proteins** on the membrane of a **chemoreceptor cell** in the taste bud. Binding of a food molecule **activates the receptor**, which either directly opens ion channels (e.g. salty \rightarrow Na^+ channels; sour \rightarrow H^+ entry) or triggers a **second-messenger pathway** that opens ion channels (e.g. sweet, bitter, umami).
2. In salty taste receptors, sodium ion channels open, causing **influx of Na^+** into the chemoreceptor cell. This produces a **generator potential in the chemoreceptor cell**. The **size of the generator potential** depends on the concentration of the tastant.
3. If the generator potential is large enough, it triggers **opening of voltage-gated Ca^{2+} channels** in the chemoreceptor cell.
4. The Ca^{2+} influx causes **vesicles to fuse with the presynaptic membrane**, releasing **neurotransmitters** into the **synapse** between the chemoreceptor cell and the sensory neurone.
5. Neurotransmitter binds to receptors on the **postsynaptic membrane** of the sensory neurone, opening **ligand-gated Na^+ channels**.
6. If enough Na^+ enters the sensory neurone, the membrane **depolarises** (to around -55 mV).
7. Once threshold is reached, **voltage-gated Na^+ channels open**, causing rapid **Na^+ influx** and producing the **action potential** in the sensory neurone.
8. The action potential then **propagates along the sensory neurone** towards the **central nervous system**, triggering adjacent voltage-gated channels.



Synapses

Synapses are junctions between two neurones.

The mechanism of synaptic transmission

- When an **action potential** reaches the presynaptic knob, the membrane **depolarises**, causing **voltage-gated calcium ion channels to open**.
- Calcium ions **diffuse** into the cytoplasm **down their concentration gradient**.
- The **increase in calcium ion** concentration triggers **synaptic vesicles** to move toward and **fuse with the presynaptic membrane**, releasing the **neurotransmitter** (such as acetylcholine) into the **synaptic cleft** via exocytosis.
- The neurotransmitter molecules **diffuse across the cleft** and **bind to specific receptor** proteins on the **postsynaptic membrane**. This binding causes chemically-gated (ligand-gated) **sodium ion channels to open**, allowing sodium ions to diffuse into the postsynaptic neurone.
- If the resulting **depolarisation** reaches the threshold potential, a **new action potential** is generated.

Excitatory vs. inhibitory synapses

The effect on the postsynaptic neurone depends on the type of neurotransmitter and receptor:

- **Excitatory Synapses:** Lead to **sodium influx and depolarisation**, making an action potential **more likely**.
- **Inhibitory Synapses:** Trigger the opening of **chloride ion channels or potassium ion channels**. The influx of chloride ions or efflux of potassium ions causes hyperpolarisation, making the membrane potential **more negative and harder to reach the threshold**.

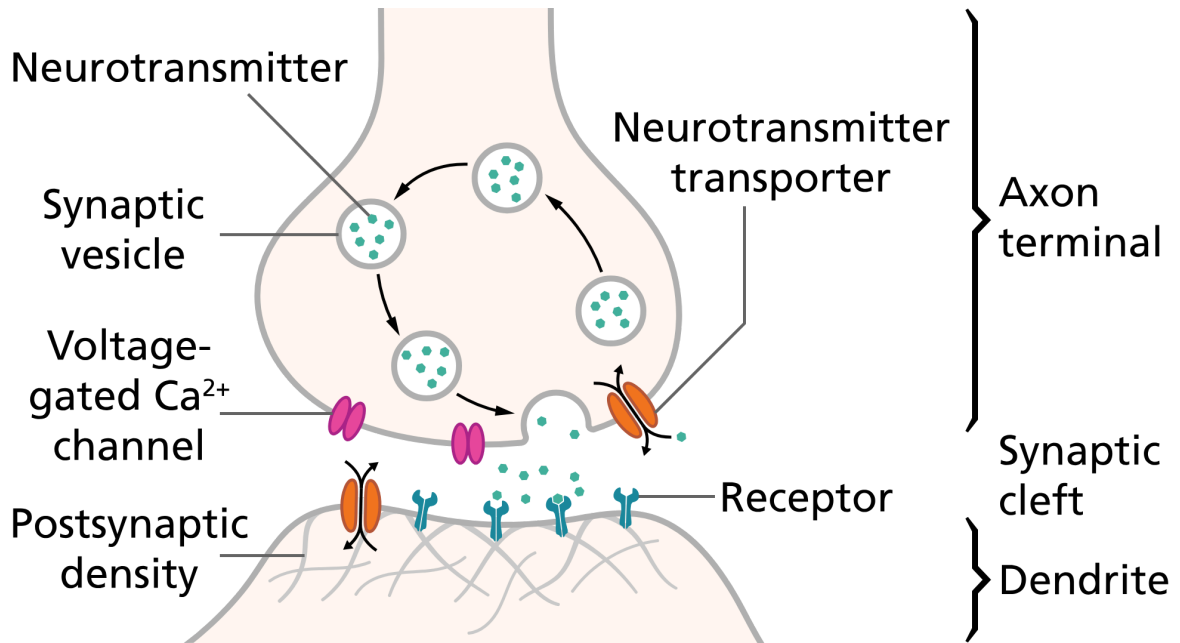
Termination of the signal and unidirectionality

To prevent permanent depolarisation and **overstimulation**, the neurotransmitter must be removed. In cholinergic synapses, the enzyme **acetylcholinesterase** hydrolyses acetylcholine into **choline and acetate**. These breakdown products are reabsorbed into the presynaptic neurone to be **resynthesised** into acetylcholine using ATP from mitochondria.

Synapses ensure unidirectional transmission of impulses because:

1. **Vesicles** containing neurotransmitters are only located in the **presynaptic** neurone.
2. **Receptors** for the neurotransmitter are only located on the **postsynaptic** membrane.





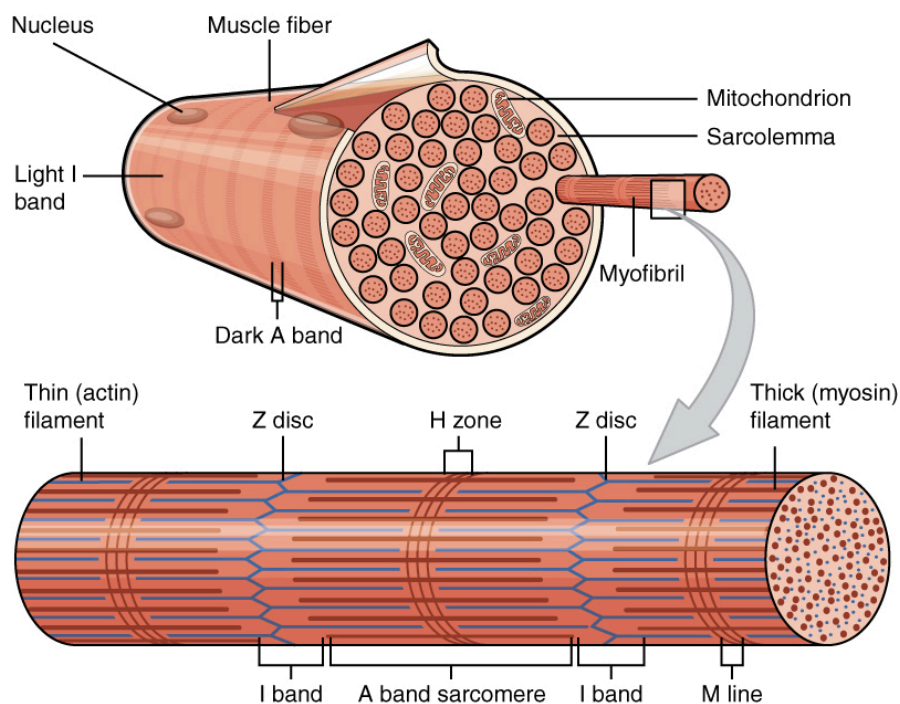
Muscles

Key words:

- **Tendons** – non-elastic tissue which connects muscles to bones.
- **Ligaments** – tough connective tissue with limited elasticity that joins bones together and stabilises joints.
- **Joints** – the area where two bones meet, allowing movement. They are made of fibrous connective tissue and cartilage.
- **Skeletal muscles** – muscles attached to bones. They are arranged in antagonistic pairs.
- **Antagonistic muscle pairs** – pairs of muscles which pull in opposite directions – as one muscle contracts, the other relaxes. **Flexors and extensors** are an antagonistic muscle pair such as triceps and biceps. When the triceps relax, the biceps contract to lift the arm.
- **Neuromuscular junction** – the junction between a motor neurone and a skeletal muscle fibre.

Striated muscle, also known as **skeletal muscle**, makes up most of the muscles in the body and is used for voluntary movement. It is made up of large bundles of long muscle fibres. They contain **myofibrils**: long, cylindrical organelles that are specialised for muscle contraction, made of **actin and myosin**. The cells also contain many nuclei and mitochondria to provide energy for movement.

Myofibril



The mechanism of muscle contraction

Upon the arrival of an action potential at the **neuromuscular junction**, the **sarcolemma** (muscle cell membrane) is depolarised. This depolarisation spreads through **T-tubules**, triggering the release of **calcium ions** from the **sarcoplasmic reticulum** into the sarcoplasm.

The formation of cross-bridges

1. **Troponin-Tropomyosin shift:** Calcium ions bind to **troponin** molecules, causing them to change shape. This pull moves the attached **tropomyosin** filaments away from the **myosin-binding sites** on the actin.
2. **Cross-bridge formation:** The exposed binding sites allow **myosin heads** to bind to the **actin**, forming **actomyosin cross-bridges**.

The sliding action (the power stroke)

3. **The power stroke:** The release of **ADP and inorganic phosphate** from the myosin head causes it to tilt (nod forward), pulling the actin filament over the myosin filament. This is known as the **power stroke**.
4. **Detachment:** A new molecule of **ATP binds** to the myosin head, causing it to change shape and **detach** from the actin filament.
5. **Re-cocking:** The enzyme **ATPase** (located on the myosin head) **hydrolyses** the ATP into ADP and inorganic phosphate. The energy released returns the myosin head to its original "high-energy" position, ready to bind further down the actin filament.

Relaxation

6. **Recovery:** When the stimulus stops, calcium ions are actively **transported** back into the sarcoplasmic reticulum. Troponin returns to its original shape, and **tropomyosin re-covers** the binding sites on the actin, preventing further cross-bridge formation.



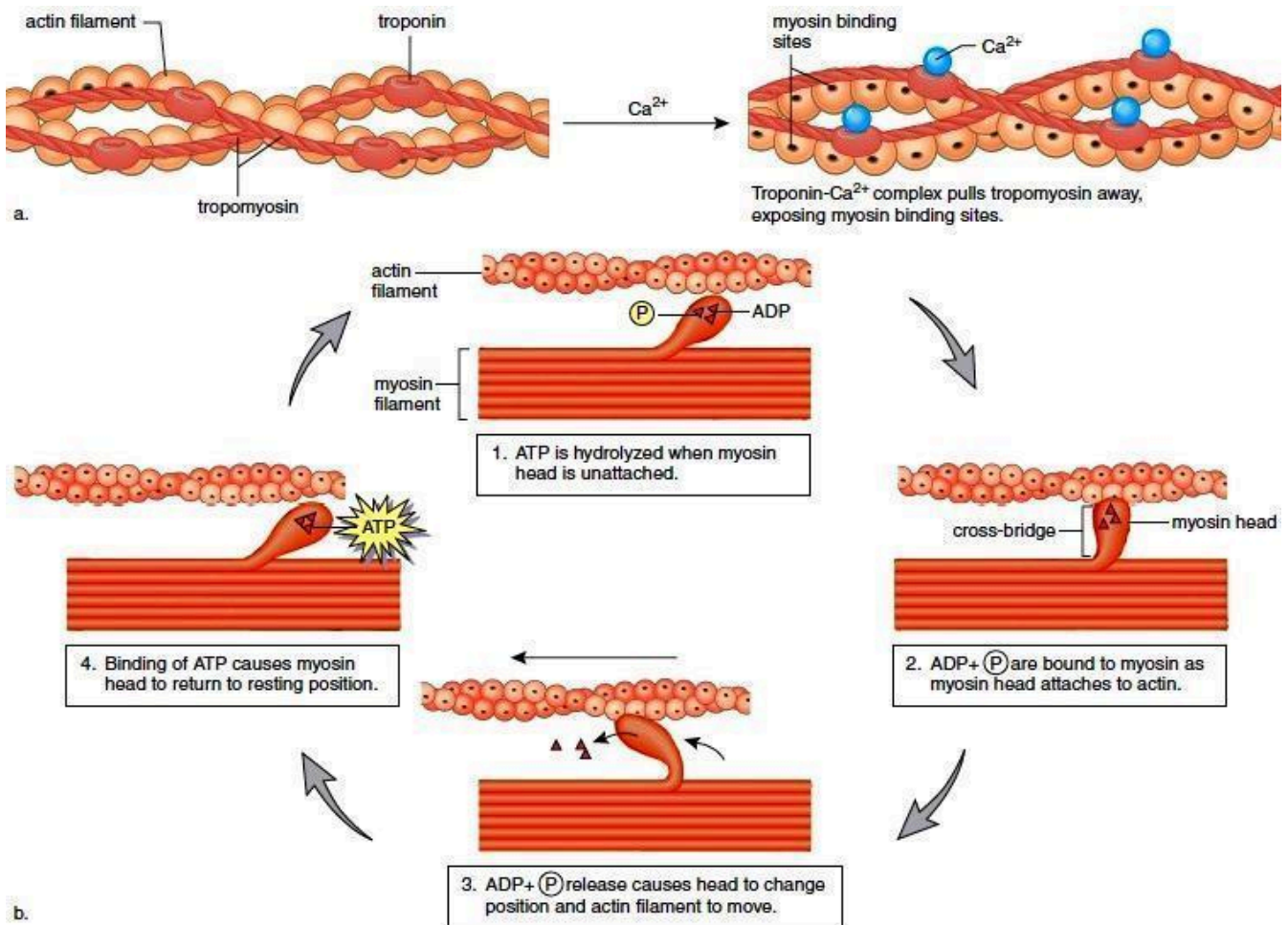


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Role of ATP in myofibril contraction:

- Allows **actomyosin cross-bridges to detach** and is hydrolysed so that the myosin can return to its original position (allows muscles to relax).
- Allows **reabsorption of calcium ions** via active transport.



The endocrine system

The endocrine system consists of endocrine glands that release hormones directly into the bloodstream.

Some hormones involved in the endocrine system are:

- **ADH (antidiuretic hormone)**
 - Involved in **osmoregulation** which is the maintenance of the water potential balance in the blood.
 - When the blood's water content is low, ADH is released by the posterior pituitary gland.
 - This increases the distal convoluted tubule and collecting duct walls' permeability to water.
 - Reabsorption of water from the tubules into the blood increases.
- **Glucagon**
 - Involved in the control of **blood glucose concentration**.
 - When glucose concentration is too low, glucagon is released from the alpha cells of the islets of Langerhans.
 - Stimulates hepatocytes to convert glycogen to glucose which diffuses out of hepatocytes into the blood.
- **Insulin**
 - Involved in the control of **blood glucose concentration**.
 - When glucose concentration is too high, insulin is released from the beta cells of the islets of Langerhans.
 - Stimulates hepatocytes to convert glucose into glycogen, which is stored in the liver.

The Endocrine System	The Nervous System
Uses hormones (chemical messengers)	Uses action potentials and neurotransmitters
Travel via bloodstream from endocrine glands	Travel via neurones
Slower response but long-lasting	Very fast response but short-lived
Act on any cells carrying specific receptors for a hormone, giving a system-wide effect	Act on specific cells connected by synapses



Control and coordination in plants

Plant growth responses can also be triggered by **plant growth regulators**.

Examples include:

- **Auxins** which promote cell elongation.
- **Gibberellins** which promote seed germination and stem growth.
- **Abscisic acid**, which inhibits seed germination and causes closing of stomata.

Auxins cause cell elongation by stimulating **hydrogen ion** transport into **cell walls**, lowering **pH** and activating **expansins**, which loosen cellulose microfibrils. Expansins are a special type of enzyme **involved in loosening the cellulose**. This makes **cell walls stretch to accommodate more water**, enabling the expansion and growth of cells.

When the shoot is illuminated from all sides, the auxins are distributed evenly and move down the shoot tip thus causing elongation of cells across the zone of elongation. Whereas **if the shoot is only illuminated from one side**, the auxins move towards the shaded part of the shoot thus causing elongation of the shaded side only (bending of shoot towards light).

Another example of control and coordination in plants is the rapid response of **Venus fly trap** to stimulation of hairs on the lobes of modified leaves. Closure of the trap is achieved through stimulation of the hairs which stimulate the release of calcium ions which in turn generates an action potential. Closing of the trap only occurs if several hairs are stimulated, and the trap seals when further stimulation of hairs occurs. When the trap is sealed, digestive enzymes are released to break down the insect.

