MASTER BIOLOGY

AQA Biology A-level

Module 6: Organisms respond to changes in their internal and external environments

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Survival and response

Key Words:

Stimulus – an internal or external change which can be detected by an organism. Only multicellular organisms such as animals can detect an internal change such as blood glucose level

Receptor - a receptor is a organ or specialised cell that detects changes caused by the stimulus

Response – An internal change or change in behaviour of an organism which occurs as a result of the stimulus

Taxes and Kinesis

A **taxis** is a response that involves **movement in a specific direction**. Positive taxis encompasses movement towards the stimulus and negative taxis refers to movement away from the stimulus. An example of positive chemotaxis is motile bacteria moving to an area where there is a **higher concentration of glucose**.

A **kinesis** is a response that involves **movement**, but this time in **random directions**. Both the **speed and frequency of direction change increase**. The response is carried out in order to increase the chance that the organism will enter different conditions more rapidly. An example is if you place a woodlouse in a dry area, it will speed up and change direction more frequently in order to increase the chance it enters a damp region, which woodlice prefer.

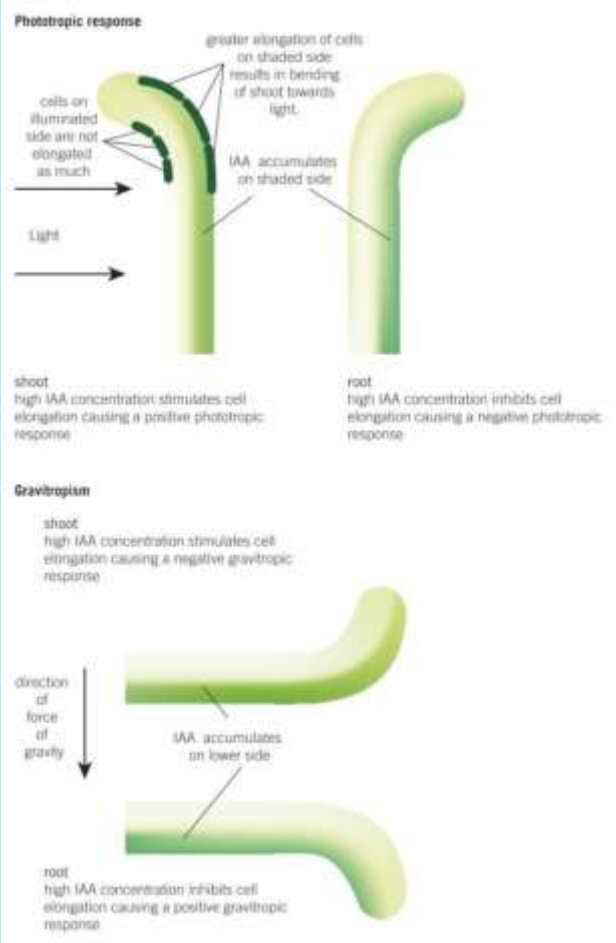
Plant response to stimuli

Plants respond to **external stimuli** to increase their chance of survival. For instance, they exhibit **tropisms**. A tropism is a **growth response** controlled by a **directional stimulus**.

An example of a tropism is **phototropism** where the direction of growth is determined by the **direction of light**. The shoots of the plant will grow towards the light, so we call shoots **positively phototropic.** The roots grow away from the light, deeper into the soil. Therefore, we refer to the roots as **negatively phototropic**.

Plant growth is controlled by **indoleacetic acid (IAA)**. This is a type of chemical which is called an **auxin**, that is often referred to as a plant hormone. It is produced in the tips and shoots of

flowering plants. The distribution of IAA around the plant controls tropisms. The main evidence for this is that if IAA is **unevenly distributed**, it causes **uneven growth** of the plant to occur.



When the shoot is illuminated equally from all sides, the auxins are **distributed evenly**. They diffuse down shoot tip thus causing **elongation of cells** across the zone of elongation. All of the cells in the shoot will elongate evenly as a result.

If the shoot is only illuminated from one side, the auxins move towards the **shaded part of the shoot.** This causes only the cells on the shaded side to elongate, causing bending of the shoot towards the light.

Gravitotropism

Gravitropism in roots works in the opposite way to phototropism, in that IAA has the opposite effect on elongation. In fact, IAA actually slows (inhibits) growth.

In the roots, gravity naturally pulls IAA down to the bottom side of the root, therefore IAA will build up on the lower side. In roots, IAA **inhibits growth**. This causes the cells on the upper

side to grow faster than those on the lower side. This will make the root to **bend downwards** and therefore reach deeper into the soil to find nutrients and water.

Reflexes

Reflexes are rapid automatic responses which aid in protecting an organism from harmful stimuli. Reflexes therefore allow an organism to **avoid danger** and give them a **survival advantage**. Reflexes are so fast because the electrical impulse does not go up to the brain, instead bypassing it. This means the brain does not have to process information and no decision needs to be made about what to do.

The reflex arc:

Stimulus –> Receptor –> Sensory Neurone –> Intermediate Neurone –> Motor Neurone –>Effector –> Response

Sensory Neurone - carries the nerve impulse **Motor Neurone** - carries the nerve impulse from the receptor to the spinal cord. from the spinal cord to the effector which can be a muscle or gland.

Intermediate Neurone - this is located entirely in the spinal cord and relays the nerve impulse from the sensory neurone to the motor neurone.

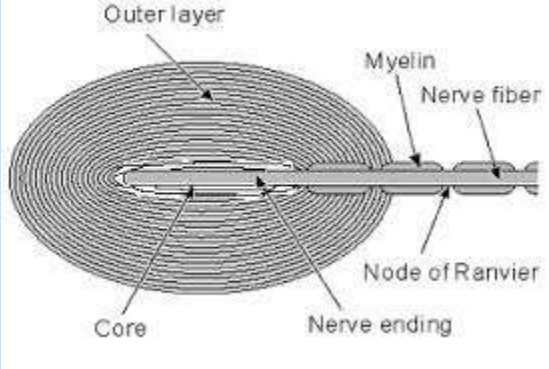
Receptors

Receptors detect changes in the internal and external environment. There are many types of receptors, each specific to a particular kind of stimuli, for instance photoreceptors detect changes in light. **Mechanoreceptors** such as the **Pacinian Corpuscle** detect mechanical stimuli such as pressure on the skin and vibrations.

Pacinian Corpuscle

Pacinian Corpuscles are located **deep in the skin**, and are mainly located on the fingers, soles of the feet and external genitalia but are also in joints, tendons and ligaments. Each Pacinian Corpuscle has one **sensory neurone.** The sensory neurone lies in the middle of the Pacinian Corpuscule and is surrounded by connective tissue arranged into layers called **lamellae**. There is a gel between each layer.

The Pacinian Corpuscle contains **stretch mediated sodium channels** in the cell surface membrane. When the skin, and therefore the Pacinian Corpuscule is not under pressure from touch, the sodium channels are **closed**. When something touches the skin, it bends the lamellae and the sodium ion channels become **deformed** and change shape. The channels opens, causing a **rapid influx of sodium ions** into the sensory neurone. This makes the membrane potential of the neurone more positive, causing the membrane to become **depolarised**. Much the same as the propagation of an action potential, if the membrane potential becomes positive enough, **generator potential** being created which goes on to create an action potential in the axon.

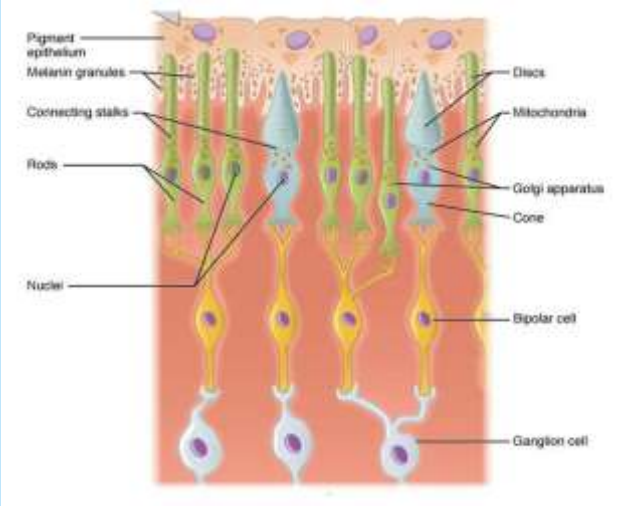


Photoreceptors

Photoreceptors are light receptors in the eye. The light enters the eye through the pupil and the amount of light entering is controlled by muscles in the **iris,** which is the coloured part of your eye. The lens of the eye focuses the light on the **retina** where the photoreceptors are located. The part of the retina that has a particularly high number of photoreceptors is the **fovea,** where light naturally hits the retina. The photoreceptors convert the light energy into nerve impulses, which travel down the **optic nerve** to the **brain**. The point where the optic nerve leaves the eye is known as **the blind spot** as there are no photoreceptor cells located there.

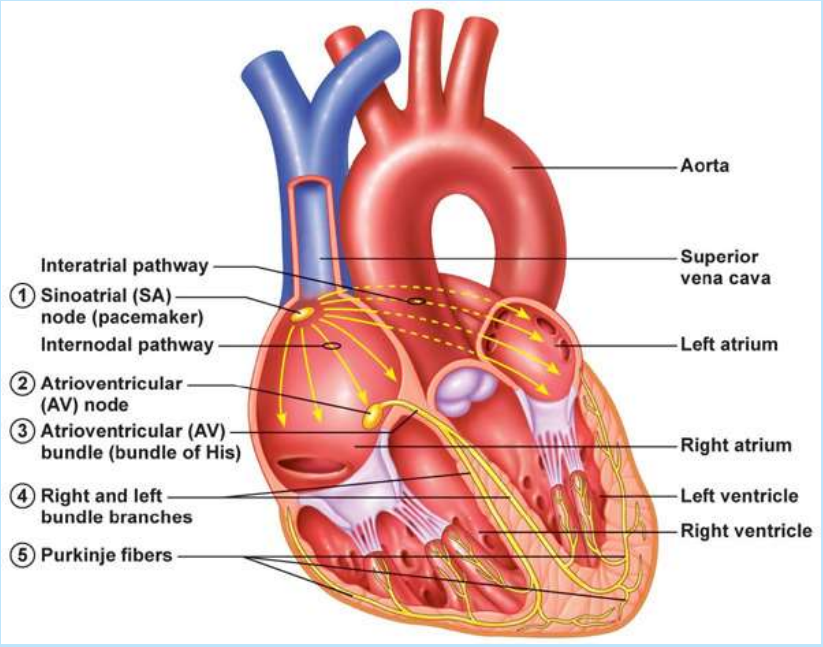
There are two types of photoreceptors in the retina, these are **cones** involved in **colour vision** and **rods** involved in **monochromatic** (black and white) vision. **Cone cells** are present in the greatest density in the fovea of the eye and contain a pigment called **iodopsin**. Cone cells require bright light in order to work and will not work in the dark. This is why it is difficult to differentiate colours in the dark. There are three different types of cone cell, each sensitive to the primary colours of light (red, green or blue). Cone cells provide **good visual acuity** because each cone cell makes one synapse with one **bipolar neurone** which connects to the optic nerve sensory neurone (ganglion cell). This is unlike rod cells below as many rod cells will synapse onto one bipolar neurone.

On the other hand, **rod cells** are mainly concentrated in the highest density outside of the fovea and contain the pigment **rhodopsin**. They are **very sensitive to light** and therefore are stimulated in **low light conditions**. Rod cells provide low visual acuity as more than one rod cell shares the same synapse with a bipolar cell. As a result multiple rods can contribute to the creation of a generator potential – this means less light is needed but acuity is sacrificed.



Contraction of the Heart

Due to the heart's ability to initiate its own contraction, it is referred to as **myogenic.** In the wall of the right atrium there is a region of specialised fibres called the **sinoatrial node** which is the **pacemaker** of the heart. This initiates a wave of **depolarisation** which causes the atria to contract at roughly the same time. The ventricles do not start contracting until the atria have finished due to the presence of tissue in the septum base of the atria which slows the conduction called the **atrioventricular node**. The wave then passes to ventricles via the **bundle of His** to the **apex** of the heart. The bundle of His branches into **Purkyne (or Purkinje) fibres** which carry the wave upwards on the either side of the heart (the outside walls). This causes the ventricles to contract from the bottom up, thus emptying them.



The Autonomic Nervous System

The autonomic (unconscious) nervous system is split up into 2 different divisions: the **sympathetic** and the **parasympathetic** nervous systems. Generally, the sympathetic nervous system is responsible for the **fight or flight response** ie. Getting the body ready to either run away or fight. In general, this means an **increase** in bodily functions such as heart rate and respiratory rate (breathing). On the other hand, the parasympathetic nervous system is responsible for **rest and digest** ie. Allowing the body to relax and bringing heart rate, blood pressure and respiratory rate back down once the fight or flight response is no longer needed.

The **sympathetic** nervous system utilises the neurotransmitter **noradrenaline** at its postganglionic synapses. The **parasympathetic** nervous system utilises **acetylcholine**.

Heart Rate Control

The sinoatrial node is connected to two nerves from the **medulla oblongata** in the brain. The **accelerator nerve**, which is a part of the **sympathetic nervous system**, delivers a higher frequency of impulses to the **SAN** to increase the **heart rate.** On the other hand, the **vagus nerve,** which is part of the **parasympathetic nervous system**, will deliver a **slower frequency** of impulses to slow down the heart rate.

Causes of increased heart rate:

- Increased pH caused by **high carbon dioxide** concentration in the blood, often due to exercise causing higher rate of respiration in the muscles. The drop in pH is detected by **chemoreceptors** located in **carotid arteries, aorta** and the **brain**. The receptors send impulses to the **medulla oblongata** more frequently via the **sympathetic pathway** as a result. The medulla oblongata will then send impulses more frequently to the sinoatrial node. This increases the heart rate. This consequently speeds up blood flow to the lungs where the CO2 can be expelled. It also means more oxygen-rich blood is delivered to the muscles to supply them for exercise.
- Blood pressure – If blood pressure drops, this results in a reduced delivery of oxygen to the tissues. This is monitored by **baroreceptors** in the carotid sinuses and in the aortic arch. If blood pressure drops, nerves from the carotid sinuses and the aortic arch will send impulses to the medulla oblongata via the **sympathetic pathway**. This then causes the medulla oblongata to send more frequent impulses via the sympathetic nervous system to the sinoatrial node. This causes it to create more action potentials and cause the heart to beat faster.
    - If the blood pressure rises, an **increased frequency of impulses** are sent from the medulla oblongata via the **parasympathetic pathway** to the SAN. This causes **heart rate to decrease**, lowering blood pressure.

When changes to blood pH and pressure have been corrected, impulses to maintain heart rate continue in their normal manner.

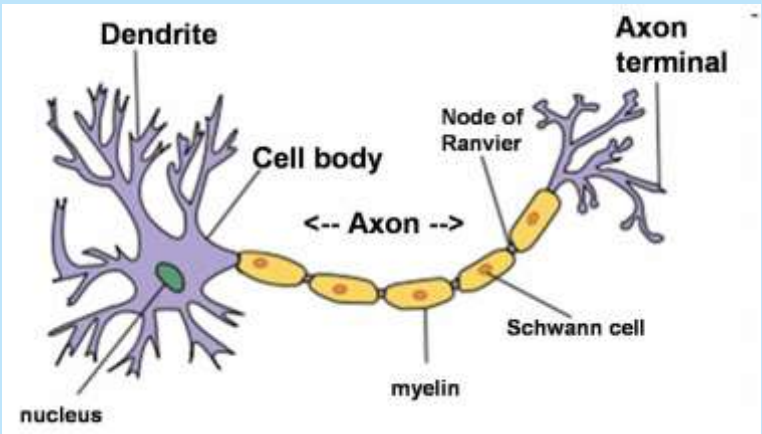
Nerve impulses

The nerve cells called neurones play an important role in coordinating communication within the nervous system.

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

The cell surface membrane of a neurone has what we call a **membrane potential**. This is the difference in charge between the inside of the cell and the outside of the cell. When these is a difference, we call this **polarisation**. Eg. If the difference in charge is -70mV, the membrane is polarised. If the difference in charge is 0, we say the membrane is **depolarised**.

All of this allows neurones to carry electrical impulses called **action potentials**.



Resting Potential

As previously mentioned, nerve cells are polarised in their resting state. This occurs as a result of an imbalance between **sodium ions** and **potassium ions**, thus giving the inside of the axon a negative charge in comparison to the external environment. The difference in the voltage across the axon membrane is **-70mV.** This is the voltage when the neurone is not doing anything and is at rest. As a result, it is known as the **resting potential.**

This **resting potential** is maintained using the **sodium potassium pump,** which relies on ATP to pump these ions against their concentration gradients. This pumps sodium ions out of the axon and potassium ions into the axon. This creates an electrochemical gradient with a higher concentration of sodium ions outside the axon. However, the potassium ions are able to move

back in by **facilitated diffusion** due to the presence of separate **potassium ion channels** which are mainly open, compared to sodium ion channels which are mainly closed. As a result of that, the outside of the axon is **positively charged** as there are more positively charged sodium ions outside than inside. For every three sodium ions that are pumped out of the axon, two potassium ions are pumped in. The pumping of ions requires the use of ATP as this is active transport.

Action Potential

When an action potential reaches the neurone it causes the axon membrane to be **depolarised** through a series of channels and ions movements. A step by step of how this happens can be found below:

1. The stimulus causes the **sodium ion channels to open**, making it more permeable to sodium ions.
2. These subsequently diffuse into the axon **down their concentration gradient**, as a result making the inside **less negative**
3. When the membrane potential has reached a charge of around -55mV (from -70mV before), it has reached **threshold potential.** If it does not reach this potential, an action potential will not be generated.
4. This causes sodium ion channels to open via **positive feedback**
5. This causes the potential to rise even further. Eventually, the membrane will reach 0 or even +10mV. We call this **depolarisation.** This is an action potential.
6. Once this has happened, the membrane must get back to its original resting membrane potential of -70mV. We call this **repolarisation**. The first step is that **sodium ions** that were open before **close**
7. Potassium ion channels **open.** The potassium ions diffuse out of the neurone down the concentration gradient and eventually **restore the resting potential**.
8. The closing of potassium ion channels is **slightly delayed** when it reaches -70mV, allowing the potassium movement out of the axon to keep going.
9. This leads to **hyperpolarisation** i.e. when the potential difference becomes more negative than the resting potential. This prevents another action potential being able to be generated for a little while. This is called the **refractory period**.
10. The resting potential is then achieved with the help of the **sodium-potassium pump** which returns the potential difference to the value of **-70mV.**

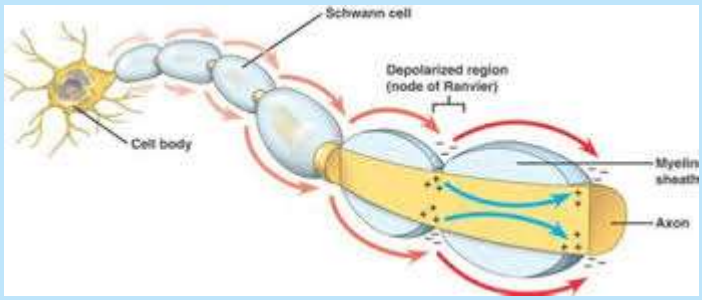
An easy way to remember the movement of ions during propagation of an action potential is
SIN AND POUT: Sodium IN and Potassium OUT

Movement of an Action Potential

An action potential must be able to travel along the long axon of an unmyelinated neurone and be passed to the next neurone. When an action potential arrives and causes depolarisation in the first section of the axon, this causes **sodium ion channels** in the next part of the neurone to open too. Whilst the first part of the axon is **repolarising**, the second part is starting to **depolarise.** This wave of depolarisation therefore continues along the axon until it reaches the synapse with the next neurone.

Myelin Sheath

Myelin sheath is a fatty covering found on axons of neurones. This fatty material does not conduct electricity and is therefore is an **insulator**. Not the entire axon is covered in this sheath, however. There are small areas which are uncovered axons. These are called **Nodes of Ranvier.** Therefore the action potential has to jump between these gaps in the myelin, which speeds up the travel of the action potential as they can skip large parts of the axon. The mechanism they do this by is called **saltatory conduction**.



Speed of an Action Potential

The speed of an action potential is affected by three main factors:

1. Presence or absence of myelin sheath - if an axon is myelinated then **saltatory conduction** can occur which is much faster than generating an action potential at every point along the axon.
2. Diameter of the axon - the **greater the diameter** of the axon the **faster the conduction**
3. Temperature – Higher temperatures mean **faster action potentials** as high temperatures mean the ions will diffuse more rapidly. It will also affect the **rate of respiration** and therefore the production of ATP needed in the sodium potassium pump.

Refractory Period

After an action potential has passed through an axon and the membrane has repolarised, there is a **short period** during which **the neurone membrane** cannot be excited again due to **hyperpolarisation.** This period is known as the **refractory period** and serves an important role in ensuring that an action potential can only pass in **one direction** as **discrete** signals.
Finally the **all-or-nothing principle** means that either an action potential is produced or it is not. A **threshold value** must be reached in order for an action potential to be created, with all action potentials being of the same strength.

Synaptic transmission

Synapses are **junctions** between two neurones. They have a greater role though than just transmitting signals from one neurone to another. These other roles include:

- Preventing action potentials from going in the **wrong direction**. They do this because the **neurotransmitter** is only made in the presynaptic neurone, with **receptors** only on the postsynaptic neurone
- Some synapses can be **inhibitory** and prevent the movement of action potentials. Most though are **excitatory**
- They can amplify the effects of low frequency action potentials **using summation**. More on this below:

Summation

There are two types of summation: **temporal** and **spatial**

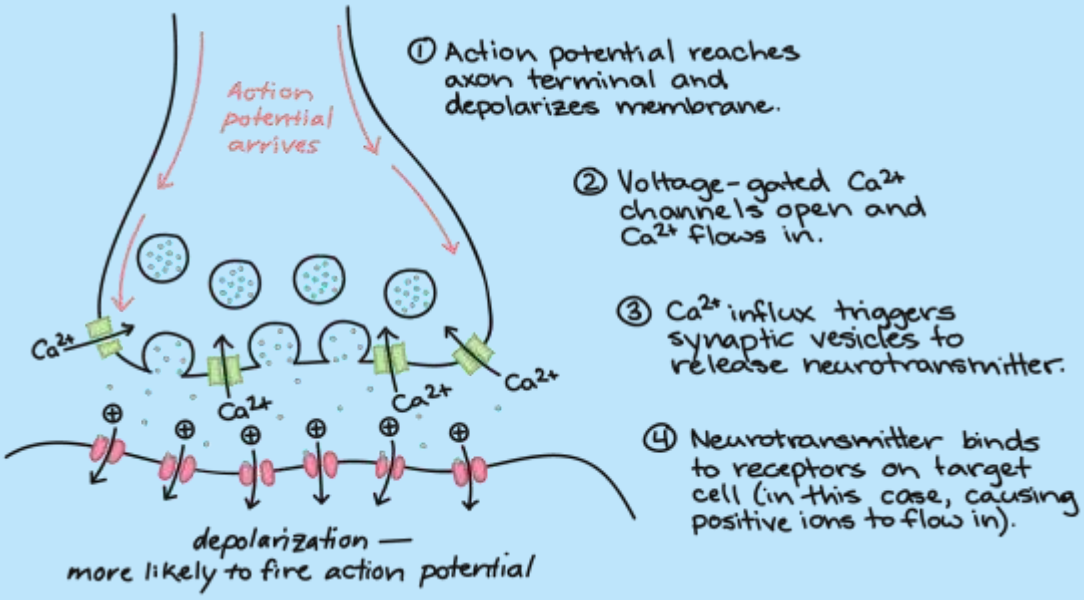
- Temporal - A single presynaptic neurone releases neurotransmitter many times, over a short period, causing threshold potential to be reached in the post synaptic neurone
- Spatial - multiple presynaptic neurones release neurotransmitter onto the postsynaptic neurone to reach the threshold value.

Synaptic Transmission

An action potential moves across a synapse from the pre-synaptic neurone to the post-synaptic neurone in the following way:

1. Upon the arrival of an action potential at the presynaptic neurone, the **presynaptic membrane depolarises** therefore causing **calcium ion channels to open**
2. Calcium ions **diffuse** into the presynaptic neurone down their concentration gradient. (Mentioning diffusion almost often gets you a mark on the exam and is often forgotten!)
3. The presence of calcium ions in the neurone causes the **fusion of synaptic vesicles,** filled with a particular **neurotransmitter** such as **acetylcholine,** with the presynaptic membrane
4. The neurotransmitter is then released into the **synaptic cleft**, that is the gap between the two neurones.
5. The neurotransmitter then **diffuses** across the synaptic cleft towards the post synaptic neurone
6. The neurotransmitter binds to its specific **receptors** located on the postsynaptic membrane
7. This stimulates the **opening of sodium ion channels** on the postsynaptic membrane. This allows **sodium ions** to enter the postsynaptic neurone down their concentration gradient by diffusion
8. This causes an action potential to be propagated in the postsynaptic neurone

9. The enzyme **acetylcholinesterase hydrolyses** acetylcholine in the synaptic cleft into **choline and ethanoic acid (acetyl).** These substances diffuse back across the synaptic cleft and back into the presynaptic neurone where it can be reassembled into acetylcholine and reused. This prevents continuous generation of an action potential in the post synaptic neurone.

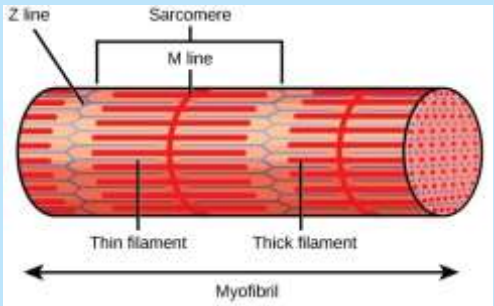


Skeletal muscles

Key words:

- **Tendons –** non-elastic tissue which connects muscles to bones.
- **Ligaments –** elastic tissue that joins bones together and determines the amount of movement possible at a joint.
- **Joints –** the area where two bones meet. They are consist of fibrous connective tissue and cartilage and allow body parts, such as limbs, to move
- **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, biceps contracts to lift the arm

Skeletal muscles are under **voluntary control** (somatic nervous system) and are attached to bones by ligaments and tendons. Muscle cells are grouped together to make a larger, stronger structure than can contract efficiently. Protein fibres called **myofibrils** run through these cells increasing their strength.

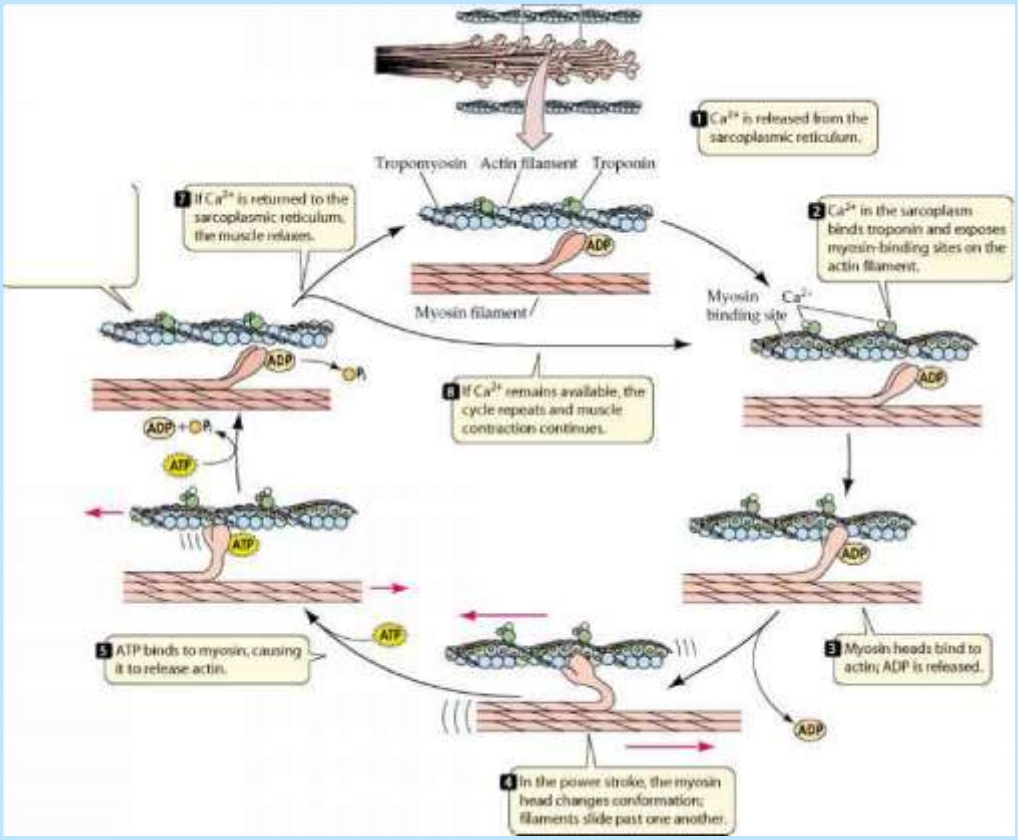


Myofibrils are made from **thick and thin filaments** which overlap in places to give a banded appearance. The thick filaments are made of **myosin** and the thin filaments are made of **actin**.

Two actin molecules are twisted together to make the thin filament. The myosin has **heads** which can attach to specific binding sites on the actin when the muscle is contracting.

When the muscle is at rest, another protein called **troponin** is wrapped around the filament and **prevents the binding of actin to the myosin head** by blocking the binding site. Actin can only bind to myosin head using a cross bridge when troponin moves out of the way.

Muscle Contraction



Step by Step

1. An action potential arrives at the muscle cell, releasing calcium ions into the sarcoplasm (the muscle cell version of the cytoplasm)
2. The calcium causes a **conformational change** in the tertiary structure (3D shape) of the troponin.
3. Troponin, which is **blocking the binding site** between actin and myosin, moves out of the way and allows a **cross-bridge** to form between actin and myosin head
4. The formation of the cross bridge causes the tertiary structure of the myosin head to change. As a result, it causes it to move, dragging the actin along with it. This is called the **power stroke**, which causes the actin to be moved **across** the myosin.
5. Once this has occurred, the myosin head releases the actin molecule
6. **The hydrolysis of ATP to ADP (**releasing energy and an inorganic phosphate) causes the myosin head to return to its previous position
7. If there is calcium still in the sarcoplasmic reticulum (ie. If the action potential has continued), this process can begin all over again, with myosin binding to another actin binding site further along the molecule. This is referred to as a **ratchet mechanism**.

Muscle contraction requires a large amount of ATP. A molecule of **ATP is hydrolysed** every time a myosin head moves and every time a calcium ion is pumped back into the endoplasmic reticulum where there is a high concentration of calcium ions.

Most of the ATP needed is produced through **aerobic respiration**. **Myoglobin** is a chemical similar to haemoglobin that is stored in muscles and can help to store a little more oxygen to be used for aerobic respiration.

Phosphocreatine is another molecule found in fast twitch muscle cells that can be used to supply the phosphate for ADP phosphorylation so that ATP can continue to be made.

Muscles are either slow or fast twitch, these are summarised below:

- **Slow twitch fibres** are specialised for **slow contractions** and are adapted to long periods of exercise such as marathon running therefore they do not fatigue quickly. They are adapted to aerobic exercise by having a **large store of myoglobin, a rich supply of blood vessels** and **many mitochondria.**
- **Fast twitch fibres** on the other hand are adapted for **rapid release of energy** during intense exercise such as sprinting – the contractions are intense and in short bursts. As a result they are adapted to this role by having **thick and numerous myosin filaments, a high concentration of glycogen, a high concentration of enzymes needed for anaerobic respiration** and **finally a store of phosphocreatine** so that ATP can be rapidly generated to provide energy.

Homeostasis and feedback mechanisms

Homeostasis allows an organism to maintain a **constant internal environment** within set points that are perfect for its survival. Homeostasis can be seen in the maintenance of many bodily functions, such as **body temperature, water potential, pH** and **blood glucose level**. These are kept constant despite changes in the **external environment** of the organism. Homeostasis is especially important in **body temperature** and **blood pH** because if these change too much then **enzymes** will become **denatured**.

This is achieved with **negative feedback** which counteracts any change in internal conditions to return internal conditions to the set optimal point.

In order for the negative feedback pathway to work, the following elements need to be present:

- Sensory receptors such as **temperature receptors** to detect **changes in internal conditions**. These receptors pass the message either via the **nervous or hormonal system** to the **effectors**
- Effectors such as the **liver or muscles** are the organs which are responsible for bringing about a response to restore the optimum conditions. An example is if **blood glucose** begins to fall, hormones such as **glucagon** are released to convert glycogen to glucose to bring it back to normal levels.

Another example of a control pathway is **positive feedback** which doesn't occur as often as negative and has an opposing effect. It increases the original change in the conditions. An example of positive feedback is the **dilation of the cervix during childbirth.**

Control of blood glucose concentration

The **concentration of glucose in blood** varies depending on food intake and **energy requirements**. It is important to keep the blood glucose concentration in the correct range of about 4-7mmols/L (70-99mg/dl). This is because:

- Glucose is needed for respiration to make ATP. If glucose isn't present, the organism will eventually die
- Glucose affects the water potential/osmotic potential of the blood. If the glucose is too high, this lowers the water potential and causes water to move out of cells via osmosis to counteract it. This causes severe dehydration and could also eventually lead to death.
- If the concentration of blood glucose is too high, it is **excreted in urine** and cannot be stored in the form of either glycogen or fat

There are two organs that are responsible for the control of blood glucose in the human body: the liver and the pancreas.

The **pancreas** has two main cells which help to control the blood glucose: **alpha** and **beta** cells. Alpha cells release **glucagon** when the blood glucose is too low (**glucagon** when the glucose is **gone**). Beta cells release **insulin** when the blood glucose is too high.

High Blood Glucose (Hyperglycaemia)

1. The rise in glucose concentration is detected by **beta cells** that are found in the **islets of Langerhans** in the **pancreas**.
2. **Insulin** is **secreted by beta cells**. This inhibits the action of **alpha cells** in the pancreas
3. Insulin travels in the blood to its target cells. There are 3 types of target cells: the **hepatocytes** in the **liver, fat cells** and **muscle cells.**
4. Binding of insulin to the **receptors** on the plasma membrane of these cells causes **adenyl cyclase** to convert **ATP into cAMP.**
5. **cAMP** activates certain enzyme controlled reactions in the cells to stimulate the **opening of glucose channels** in the cell surface membrane, thus causing more glucose to enter the cells
6. In the liver, the glucose which has newly entered the cells is converted to **glycogen.** This is called **glycogenesis**.
7. In the fat cells (adipocytes), the glucose is converted into lipids (fats)
8. In all cells, the respiratory rate increases in order to use glucose faster

Low Blood Glucose (Hypoglycaemia)

In a case where blood glucose concentration is too low:

1. **Alpha cells** in the **islets of Langerhans** in the pancreas detect a fall in blood glucose and secrete the hormone **glucagon**.
2. Glucagon secretion inhibits beta cell action and therefore inhibits release of insulin
3. Glucagon **stimulates hepatocytes** to convert **glycogen into glucose** by **hydrolysis**. This is called glycogenolysis.
4. Glucose diffuses out of hepatocytes into the blood
5. Cells use **fatty acids and amino acids for respiration** instead

The liver carries out three process in regulation of blood glucose:

1. **Glycogenesis** – making glycogen from glucose removed from the blood
2. **Glycongenolysis** – breaking down stored glycogen into glucose, which can be released into the blood.
3. **Gluconeogenesis** – synthesis of glucose from other molecules such as amino acids.

Adrenaline

Another way in which glycogen can be broken down into glucose to raise blood glucose levels is using the **secondary messenger adrenaline**. The process is outlined below:

1. **Adrenaline** is released from the **adrenal glands** in response to low blood glucose.
2. **Adrenaline** fuses to its **receptor** on the cell surface membrane of **liver cell** (hepatocyte). This receptor has parts outside and inside of the membrane as it spans the entire width of the membrane. The binding of adrenaline on the outside causes the part of the receptor on the inside to change shape (conformational change)
3. This activates the enzyme **adenyl cyclase** which **converts ATP to cyclic AMP (cAMP)**. This acts as a second messenger.
4. The **cAMP** then changes shape and activates **protein kinase enzyme** which catalyses the conversion of glycogen into glucose.

Diabetes

There are two types of diabetes:

Type 1 is **insulin dependent** diabetes and occurs **early in life** and results in loss of insulin production. This is caused by the immune system destroying its own **beta cells** in the pancreas. The beta cells therefore cannot make insulin any more. As a result, people with type 1 diabetes have to control their blood sugar level by self injecting insulin, with the dose matched to diet and exercise.

Type 2 diabetes is **not insulin dependent** and often appears **later on in life**. It can be caused by decreased insulin production or by **insulin receptors** on target cells becoming **unresponsive** to insulin. This is often caused by **obesity** and poor diet. This can be controlled by **diet manipulation and exercise**, although certain drugs can be used if this fails.

Control of blood water potential

There are two kidneys in the human body. The human kidney has the following general structure:

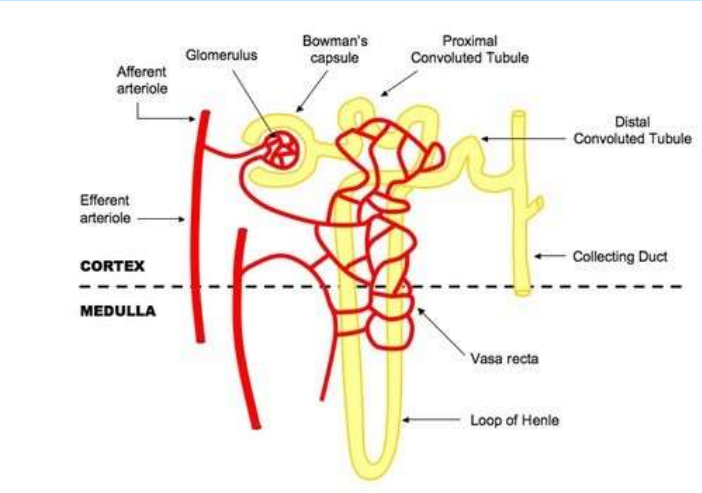
- An **outer fibrous capsule** that protects the kidney.
- A layer called the **cortex** made up of the Bowman's capsules, convoluted tubules and blood vessels

- The **medulla** which contains the bottom of the loops of Henle, collecting ducts and blood vessels
- The **renal pelvis**, which collects urine into the ureter.
- **Renal tubules** – these carry the ultrafiltrate from the glomerulus, where it was squeezed out of the blood at **high hydrostatic pressure**, to the ureter, where it exits the kidney and eventually leaves the body as urine. The order of kidney tubules and what is reabsorbed where is below:
    - Proximal convoluted tubule – **glucose** and amino acids, as well as some ions such as sodium, are reabsorbed back into the blood here. Almost all glucose and amino acid absorption should occur here. Water is reabsorbed too. Glucose is re-absorbed via co-transport with **sodium ions**.

    - Loop of Henle – this is a deep loop which descends from the cortex, where most of the tubules are, into the medulla. There is a **descending limb** and an **ascending limb.** This ascending limb is **impermeable to water**. The descending limb is where most water reabsorption occurs. In the ascending limb, lots of sodium and chloride ions are reabsorbed

    - Distal Convoluted Tubule – sodium reabsorption, mediated by the hormone **aldosterone** occurs here, along with some more water

    - Collecting Duct – minimal reabsorption occurs here, unless the body is dehydrated. If the body is dehydrated, the hormone **antidiuretic hormone** (ADH) is released and causes specialised water channels called **aquaporins** to be inserted into the cell surface membranes of the cells lining the collecting duct. This allows more water reabsorption back into the blood than would have otherwise occurred, causing **urine to become more concentrated,** as there is less water in it. There is more on this in the section entitled 'Concentration of Urine'.



Water Reabsorption

There is a 4 step process by which water is reabsorbed back into the blood by the kidneys, these stages are described below:

1. Ultrafiltration - the blood enters the kidney via the **renal artery**. This gives off an **afferent arteriole** which feeds blood into the **glomerulus**, which is a tangle of capillaries where the blood is at extremely high hydrostatic pressure. Water, ions, amino acids and glucose are **forced out** of the glomerulus through the **glomerular basement membrane**. **Large proteins** are too big to fit through and remain in the blood. The hydrostatic pressure to do this is aided by the **efferent arteriole** leaving the glomerulus being narrower than the **afferent arteriole** entering. The efferent arteriole can also constrict to increase pressure in the glomerulus if needed

2. Selective Reabsorption – the fluid containing ions etc that has been squeezed out of the blood and into the kidney tubule first enters the **proximal convoluted tubule** (PCT). In the PCT, **all glucose in the glomerular filtrate must be reabsorbed** back into the blood, as glucose is useful. Glucose is reabsorbed in the process of **co-transport** with **sodium.** It is carried out by **actively transporting sodium ions** from the epithelial cells to the blood, creating a **low concentration of sodium ions in the epithelial cells**. Sodium ions therefore consequently move in from the lumen of the proximal convoluted tubule by **facilitated diffusion,** bringing in glucose through co-transport. The glucose then **diffuses** into blood capillaries.

3. Loop of Henle - The loop of Henle acts as a **counter-current multiplier** due to the blood flow travelling in the opposite way to the ultrafiltrate in the loop of henle. It works to reabsorb water by a multi step process:
    a. To begin with **sodium ions** are **actively transported** out of the ascending limb (the second part of the tubule) into the blood **using ATP.** Water is **NOT** reabsorbed in the ascending limb as it is impermeable to water.
    b. This causes the water potential of the blood to fall. This blood flows backwards in the opposite way to the fluid in the tubule. Therefore, it then travels to the blood vessels surrounding the **descending limb** (the first part of the Loop of Henle).
    c. The blood in the blood vessels surrounding the descending limb now has a **low water potential**. The ultrafiltrate in the descending limb has a higher water potential than the blood. The descending limb **is** permeable to water. Therefore, water will leave the descending limb via osmosis, down a water potential gradient from a higher water potential in the descending limb, to a lower water potential in the blood.
    d. At the hairpin of the loop the water potential is at its lowest, where sodium ions are naturally diffusing out. They start to be actively transported when they reach the ascending limb

4. Distal Convoluted Tubule and the Collecting Duct - Water naturally moves out of the distal convoluted tubule and collecting duct by osmosis. The collecting duct runs parallel to the loop of Henle and therefore as you move down into the **medulla, ion concentration increases**.

Concentration of Urine

The permeability of the collecting duct can also be altered by hormones. Here is a step by step process of how this works:

1. **Osmoreceptors** in the **hypothalamus** in the brain detect changes in blood water potential.
2. When water potential of the blood falls, the receptor **shrinks**, therefore causing the hormone called **antidiuretic hormone (ADH)** to be released
3. This passes to the **posterior pituitary gland** where it is secreted into the blood
4. When it arrives at the kidney, it binds to receptors on the surface of the collecting duct and activates the enzyme **phosphorylase**
5. This causes vesicles containing **aquaporins**, which are a type of water channel, to be embedded in to cell surface membrane of the cells lining the collecting duct
6. This **increases water permeability** and therefore increases the amount of reabsorption of water into the blood. Urea can also be reabsorbed through these channels. Urea leaves the collecting duct into the cells, causing water to leave and be reabsorbed in the blood.