

Control and coordination (chapter 15):

- The mammalian nervous system is made up of central nervous system (brain and spinal cord) and peripheral nervous system (cranial and spinal nerves)
- Communication using nerve action potentials (brief changes in the distribution of electrical charge across the cell surface membrane, caused by rapid movement of Na^+ and K^+ ions) / impulses which travel along nerve cells (neurones) at very high speeds to their target cells
- Compare the nervous and endocrine systems as communication systems that co-ordinate responses to changes in the internal and external environment in mammals:
 - Differences:

		<i>nervous</i>		<i>endocrine</i>
1	communication	action potential / impulse	and	hormone ;
2	nature of communication	electrical (and chemical)	and	chemical ;
3	mode of transmission	neurone / nerve cell	and	blood ;
4	response destination	muscle / gland	and	target, organs / tissue / cells ;
5	transmission speed	fast(er)	and	slow(er) ;
6	effects	specific / localised	and	(can be) widespread ;
7	response speed	fast(er)	and	slow(er) ;
8	duration	short-lived / temporary	and	can be long-lasting / permanent ;
9	receptor location	on cell surface membrane	and	either on cell surface membrane or within cell ;

➤ Similarities:

10	cell signalling	both involve cell signalling ;
11	detail	both involve signal molecule binding to receptor ;
12	chemicals	both involve chemicals ;

- There are three different types of neurones:
 - ❖ Motor neurone:
 - Dendrites lead to cell body containing nucleus, many mitochondria and RER
 - Has one long axon with synaptic knobs at the end furthest from cell body
 - For some of its length, covered in myelin sheath made by specialised cells: Schwann cells
 - Has small uncovered areas of axon between Schwann cells: nodes of Ranvier
 - ❖ Sensory neurone:

- Dendrites at the ends of the long axon with synaptic knobs, with the cell body containing nucleus, many mitochondria and RER near the source of stimuli or ganglion
- For some of its length, covered in myelin sheath made by specialised cells: Schwann cells

❖ Relay neurone found in the central nervous system

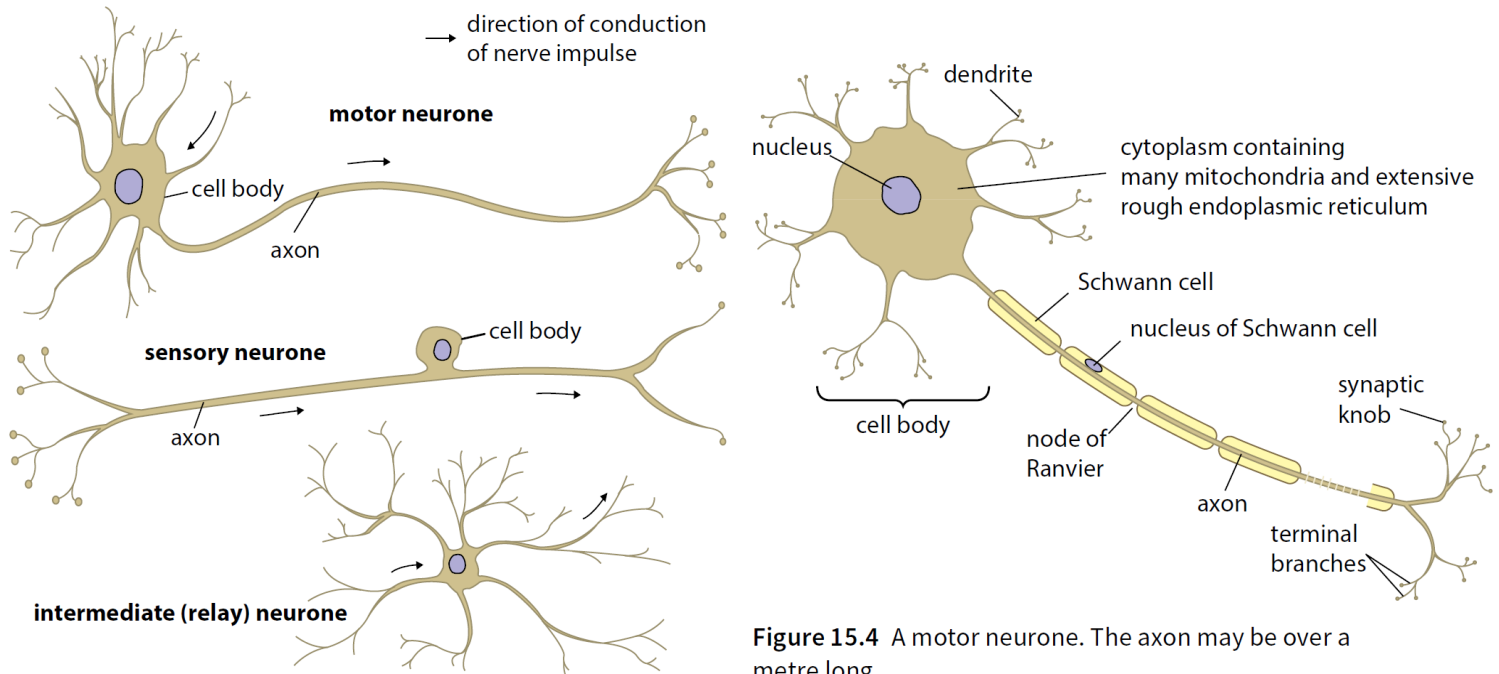


Figure 15.3 Motor, sensory and intermediate neurones.

Figure 15.4 A motor neurone. The axon may be over a metre long.

- The functions of sensory, relay and motor neurones in a reflex arc:
 - Sensory neurone – receives impulses from receptor
 - relay neurone – passes impulses on to motor neurone
 - motor neurone – sends impulses to the effector
- The transmission of action potential in a myelinated neurone and its initiation from a resting potential:
 - Initially at a resting potential (p.d. between -60 mV to -70 mV), as inside is slightly more negative than the outside, which is produced and maintained by sodium-potassium pumps in the cell surface membrane (3 Na^+ removed for every 2 K^+ brought in across the potential gradient by active transport)
 - Na^+ channels open and Na^+ enters the cell down their electrochemical gradient, where the p.d. becomes positive ($+40\text{ mV}$) due to depolarisation
 - Na^+ channels close and K^+ channels open, hence K^+ moves out of cell down their concentration gradient, hence p.d., becomes negative due to repolarisation
 - Local circuits occur
 - Myelin sheath acts as an insulator hence prevents movement of ions, hence action potentials / depolarisation occurs only at nodes of Ranvier, process called saltatory conduction (action potential jumps from node to node)
 - Process is a one-way transmission due to the refractory period
 - If the p.d. generated does not reach the threshold potential (between -60 mV and -70 mV), action potential does not occur

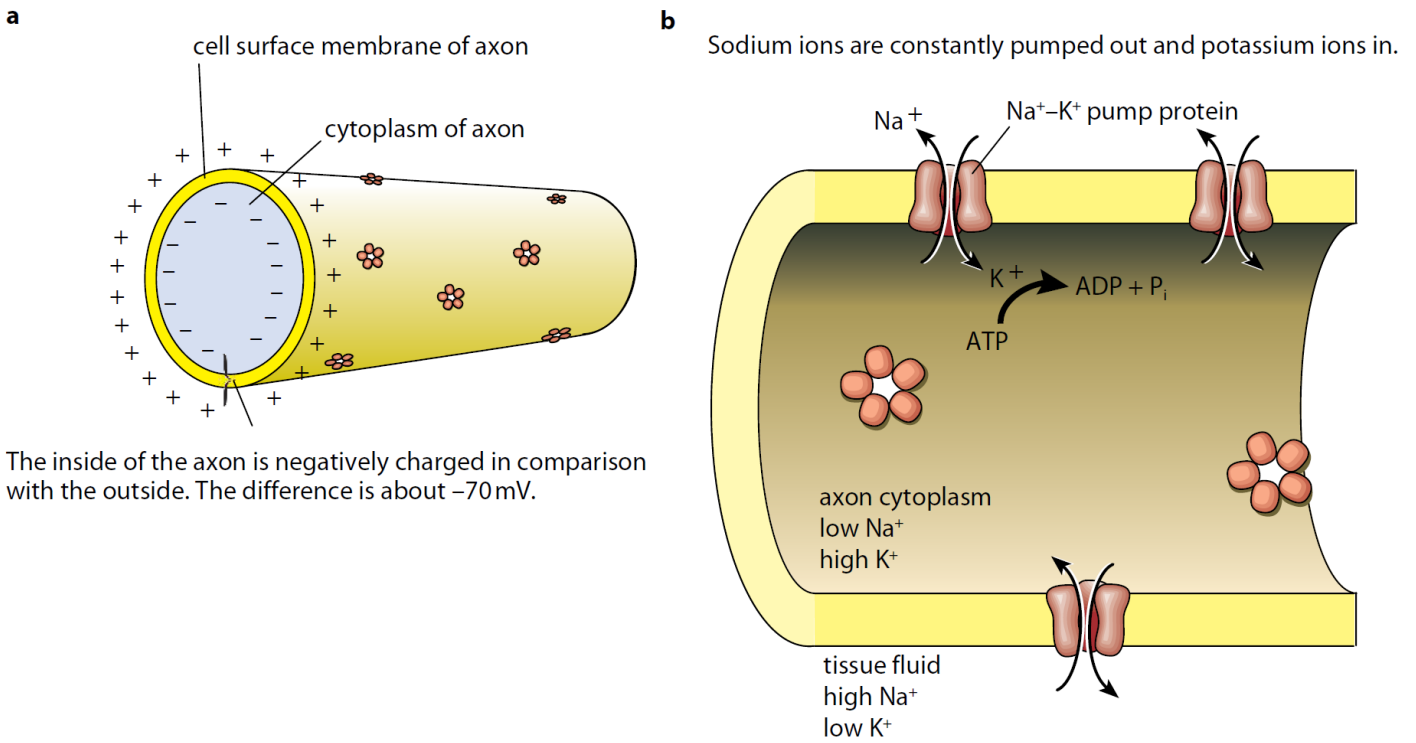


Figure 15.10 **a** At rest, an axon has negative electrical potential inside. **b** The sodium–potassium pump maintains the resting potential by keeping more sodium ions outside than there are potassium ions inside.

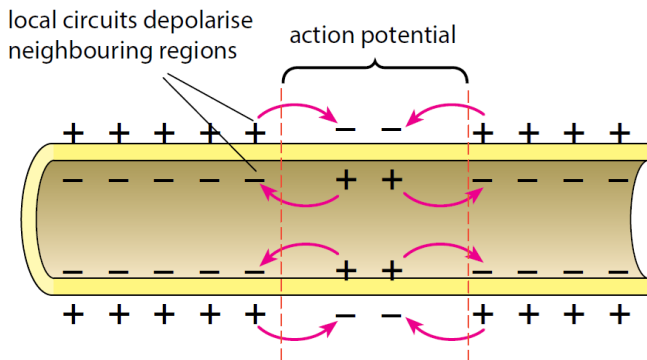


Figure 15.13 How local circuits cause an action potential to move along an axon.

- The length of the refractory period determines the maximum frequency at which impulses are transmitted along neurones, as the transferred region will still be recovering from the action potential it just had (sodium ion voltage-gated channels are ‘shut tight’ and cannot be stimulated to open, however great the stimulus)

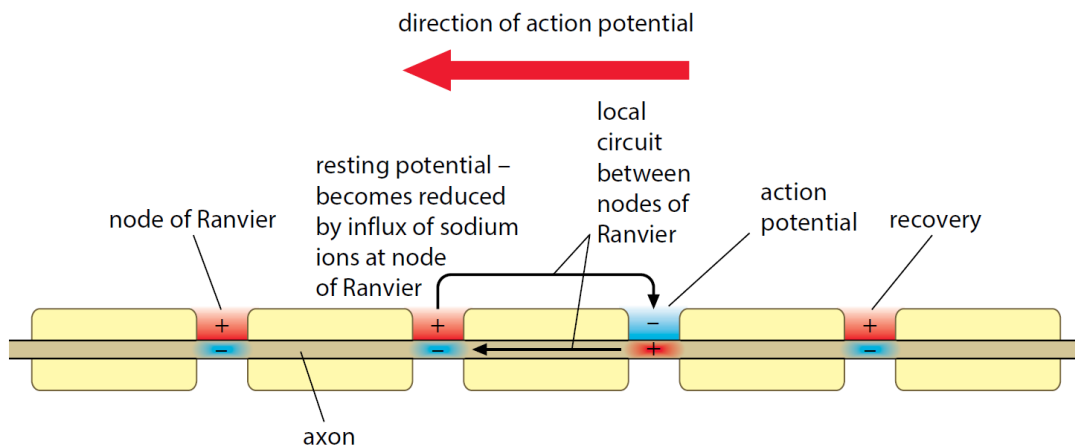


Figure 15.15 Transmission of an action potential in a myelinated axon. The myelin sheath acts as an insulator, preventing differences in potential across the parts of the axon membrane surrounded by the sheath. Potential differences can only occur at the nodes of Ranvier. The action potential therefore ‘jumps’ from one node to the next, travelling much more swiftly than in an unmyelinated axon.

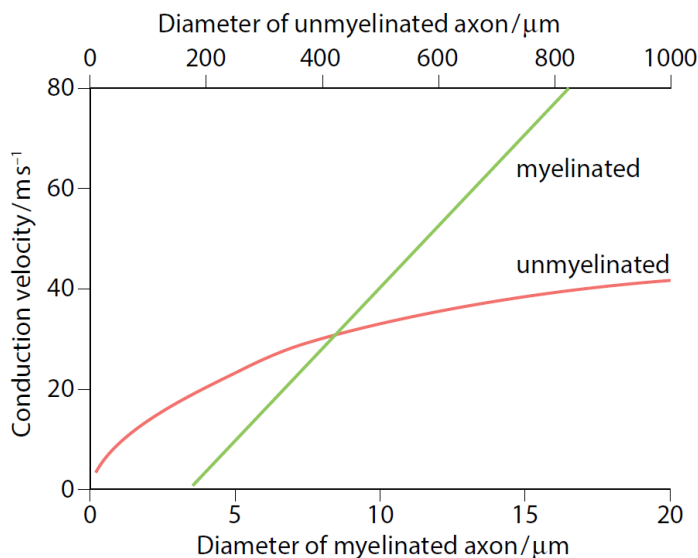


Figure 15.16 Speed of transmission in myelinated and unmyelinated axons of different diameters.

- The difference in strength of stimulus causes the different frequency of action potentials, where a strong stimulus produces a rapid succession of action potentials, each one following along the axon just behind its predecessor; a weak stimulus results in fewer action potentials per second; a strong stimulus stimulate more neurones than a weak stimulus
- The brain can therefore interpret the frequency of action potentials arriving along the axon of a sensory neurone, and the number of neurones carrying action potentials, to get information about the strength of the stimulus being detected. The nature of the stimulus, whether it is light, heat, touch or so on, is deduced from the position of the sensory neurone bringing the information. If the neurone is from the retina of the eye, then the brain will interpret the information as meaning 'light'
- **Sensory receptor cell:** a cell that responds and converts a stimulus into an electrical impulse by initiating an action potential (e.g. **chemoreceptor**)
- The roles of sensory receptor cells in detecting stimuli and stimulating the transmission of nerve impulses in sensory neurones (e.g. chemoreceptor cell found in human taste buds):
 - Chemicals act as a stimulus
 - Each chemoreceptors contain a specific receptor protein which detects a particular chemical
 - Sodium ions diffuse into cell via Microvilli, causing the membrane to depolarise, which increases the positive charge inside the cell known as receptor potential
 - Sufficient stimulation (above the threshold potential (below will only cause a local depolarisation of the receptor cell)) of Na^+ , stimulates opening of calcium ion channels, hence calcium ions enter cell, causing movement of vesicles containing neurotransmitter which is released exocytosis
 - Neurotransmitter stimulates action potential in sensory neurone
 - Chemoreceptors are transducers as they convert energy in one form into emergy in an electrical impulse in a neurone
 - All or nothing law where neurones either transmit impulses from one end to another or they do not
 - Chemoreceptor cells found in the papilla of the tongue

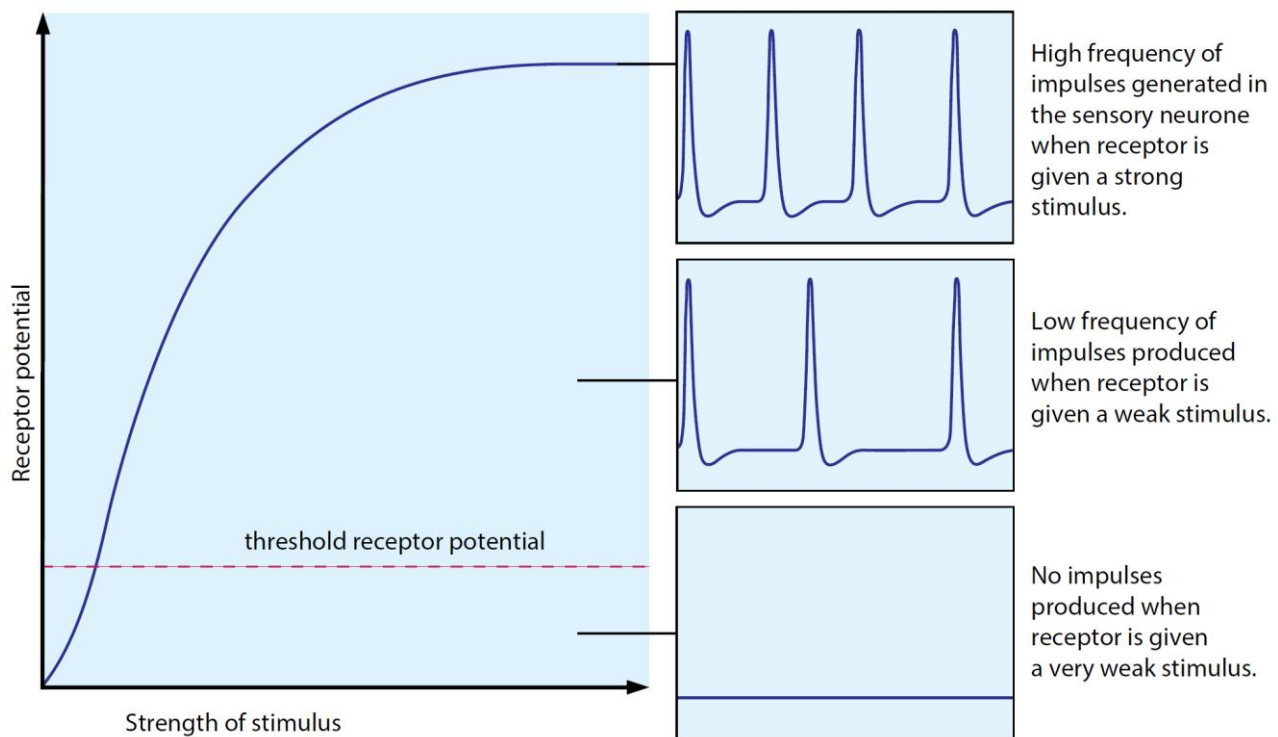


Figure 15.19 As the strength of a stimulus increases, the receptor potential also increases. If the receptor potential reaches the threshold, then impulses are sent along the sensory neurone at low frequency. Increasing the strength of the stimulus above the threshold increases the frequency of the impulses; it does not change their amplitude.

- The gap between two neurones is called **synaptic cleft**; the parts of the two neurones near the cleft, plus the cleft itself, make up a **synapse**
- An action potential occurs at the cell surface membrane of the presynaptic neurone
- Action potential causes the release of molecules of transmitter substance into the cleft
- The molecules of neurotransmitter diffuse across the synaptic cleft and bind temporarily on the postsynaptic neurone
- The postsynaptic neurone responds to all the impulses arriving at any one time by depolarising; sends impulses if the overall depolarisation is above its threshold
- Synapses that uses acetylcholine as its neurotransmitter is known as cholinergic synapses
- How a cholinergic synapse functions:
 - Action potential reaches presynaptic membrane causing depolarisation, and Ca^{2+} channels to open in the presynaptic membrane
 - An influx of Ca^{2+} ions (facilitated diffusion down its electrochemical gradient), stimulates vesicles containing ACh to move towards the presynaptic membrane and fuse with it
 - ACh released into synaptic cleft by exocytosis, and diffuses across the cleft
 - ACh binds to receptor proteins on the postsynaptic membrane; proteins change shape causing Na^+ channels to open
 - Na^+ facilitated diffusion into postsynaptic neurone causing depolarisation of the postsynaptic membrane creating an action potential, if the potential difference is above the threshold for that neurone
 - Acetylcholinesterase catalyses the hydrolysis of each ACh molecule into acetate and choline to prevent permanent depolarisation of the postsynaptic neurone; choline is taken back into the presynaptic neurone to be combined with acetyl CoA to form ACh once more and transported into the presynaptic vesicles
- Role of calcium ions in synaptic transmission:

- Enter the presynaptic neurone, and causes vesicles containing acetylcholine (ACh) to fuse with the presynaptic membrane, hence releasing ACh into the synaptic cleft (exocytosis)

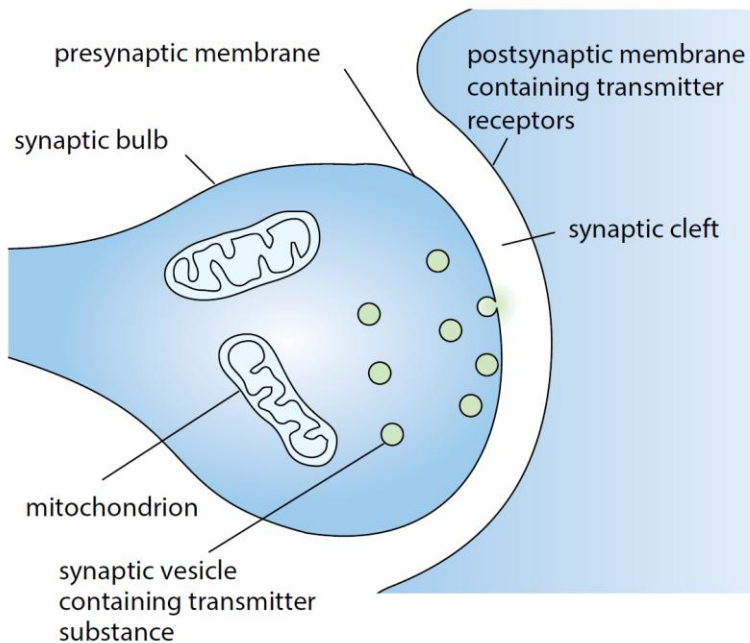


Figure 15.20 A synapse.

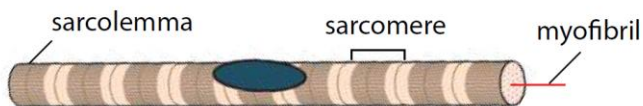
- Some insecticides have a similar structure to acetylcholine, hence affects the functioning of acetylcholinesterase by:
 - Acting as a competitive inhibitor – complementary to the active site – hence binds with the active site, this ACh is not broken down

source of poison	action of poison at synapse
A	blocks sodium channels
B	blocks calcium channels
C	prevents exocytosis
D	inhibits acetylcholinesterase

- ❖ A: Na^+ cannot enter post-synaptic neurone hence no depolarisation / action potential in the post-synaptic neurone
- ❖ B: Ca^{2+} cannot enter pre-synaptic neurone hence vesicles cannot move towards the pre-synaptic membrane
- ❖ C: ACh cannot be released into synaptic cleft
- ❖ D: ACh is not broken down hence continuous depolarisation / action potential, of post-synaptic neurone
- Ways in which the toxin (acts at cholinergic synapses) in cobra venom may cause muscle paralysis:
 - Binds to receptors on postsynaptic membrane, hence stops ACh from binding, inhibiting depolarisation, hence no action potentials / Na^+ ion channels stay shut
 - Stimulates ACh receptors causing continuous depolarisation / opens Na^+ ion channels

- Stops the release of ACh from presynaptic neurone
- Inhibits acetyl cholinesterase
- The roles of synapses in the nervous system:
 - Ensures one-way transmission as vesicles are only found in the presynaptic neurone, hence ACh will only be released from presynaptic neurone
 - Involved in memory and learning, where an increase in the number of synapses allows more interconnection of nerve pathways for memory, allowing a wider range of response
 - Allows interconnection of nerve pathways
- Striated muscle tissue makes up the many muscles in the body that are attached to the skeleton; only contracts when it is stimulated to do so by impulses that arrive via motor neurones; muscle tissue like this is described as being neurogenic, whereas the cardiac muscle in the heart is myogenic – it contracts and relaxes automatically, with no need for impulses arriving from neurones

Short length of muscle fibre



Highly magnified edge of muscle fibre

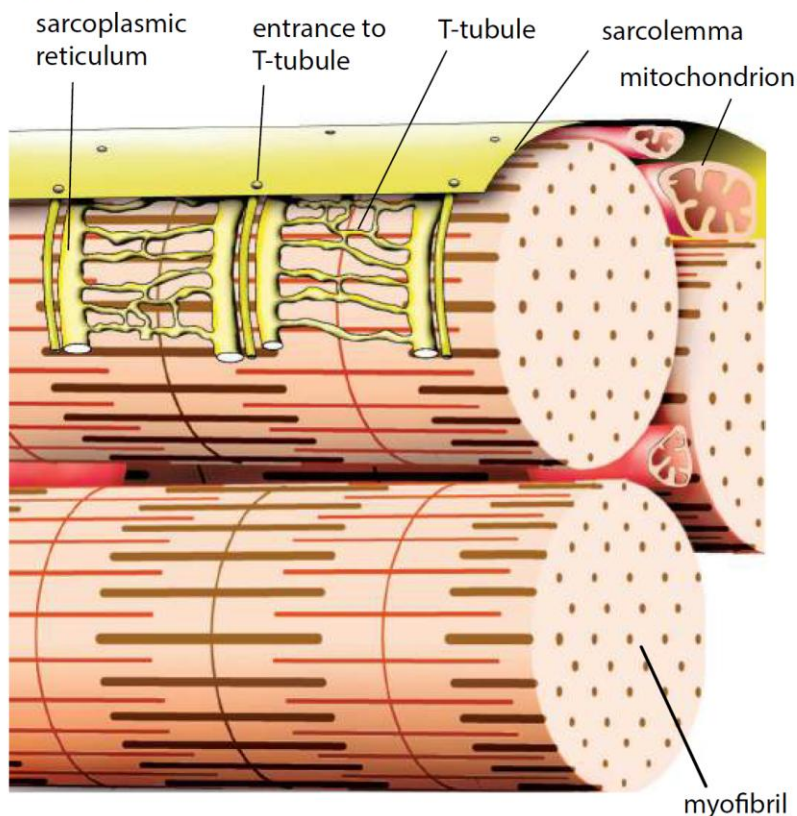


Figure 15.26 Ultrastructure of part of a muscle fibre.

- Multinucleate muscle fibres are called **syncytium** instead of cell; **sarcolemma** instead of cell surface membrane; **sarcoplasm** instead of cytoplasm; **sarcoplasmic reticulum (SR)** instead of endoplasmic reticulum; the deep infolding of the cell surface membrane into the interior of the muscle fibre is called **transverse system tubules** or **T-tubules**, which runs close to the sarcoplasmic reticulum

- Membranes of the sarcoplasmic reticulum have huge numbers of protein pumps that transport calcium ions into the cisternae of the SR
- The sarcoplasm contains a large number of mitochondria, packed tightly between the myofibrils to carry out aerobic respiration, generating the ATP that is required for muscle contraction
- Each myofibrils are made up of filaments (made from protein), where parallel groups of thick filaments (made up of **myosin**) lie between groups of thin ones (made up of **actin**), creating the stripes or striations
- The dark parts of the stripes, the A bands, correspond to the thick (myosin – fibrous protein with a globular head pointing away from the M line) filaments; the lighter parts, the I bands, are correspond to the thin filaments (2 chains made of many actins twisted together, consisting of another twisted fibrous protein – tropomyosin – and protein attached at regular intervals – troponin)
- The darkest parts of the A band are produced by the overlap of thick and thin filaments, while the lighter area within the A band, known as the H band, represents the parts where only the thick filaments are present
- The Z line provides an attachment for the actin filaments, while the M line does the same for the myosin filaments; the part of a myofibril between two Z lines is called a **sarcomere**

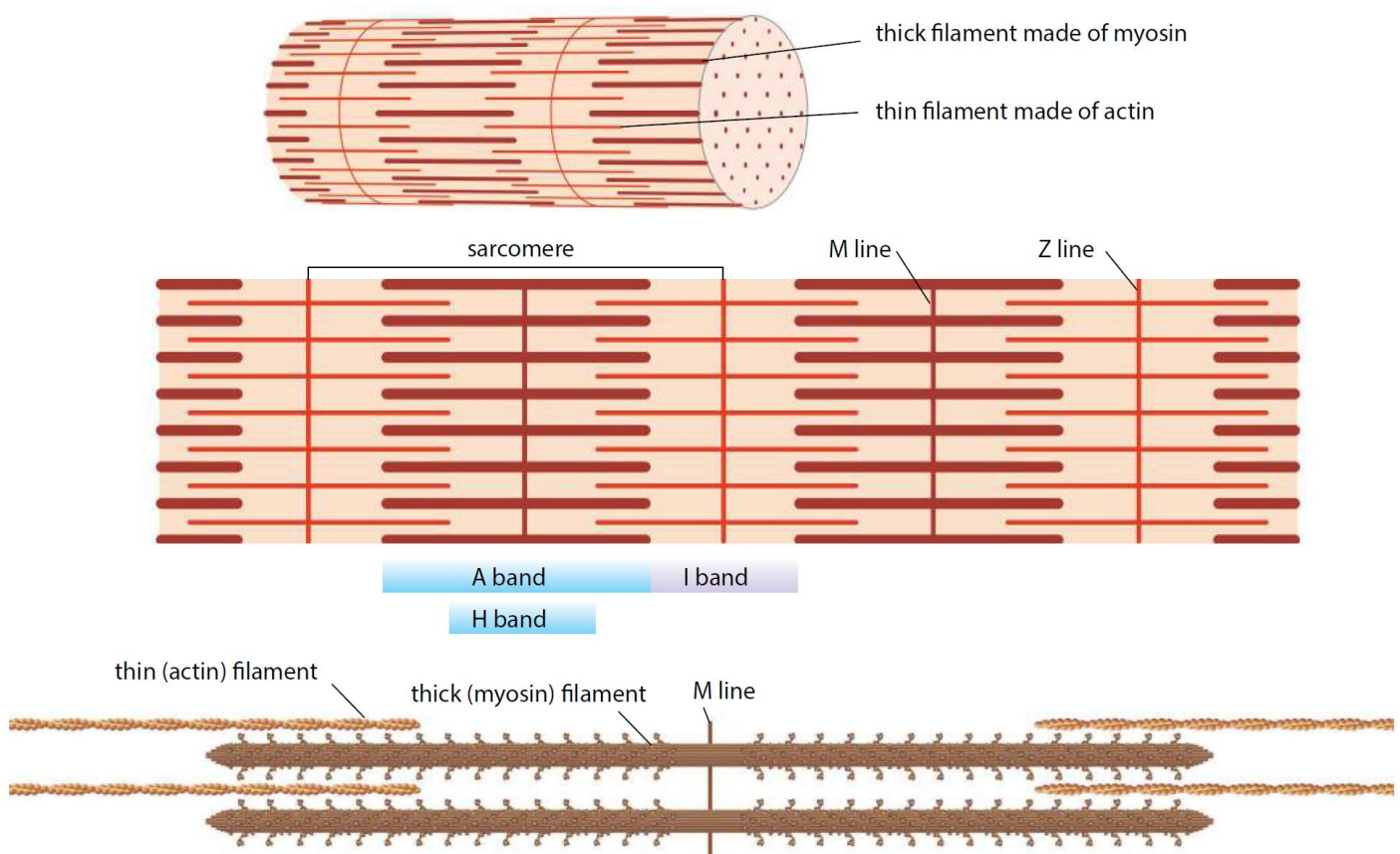
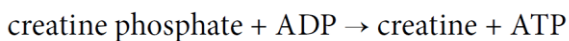


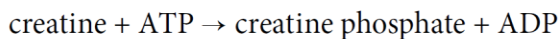
Figure 15.27 The structure of a myofibril.

- The sliding filament model of muscular contraction (shortening of I-band):
 - When sarcoplasmic reticulum (SR) depolarised
 - Calcium ion channels open
 - Tropomyosin covers / uncovers the myosin binding sites on actin; when calcium ions diffuse down a potential gradient through open channels from the SR to bind to troponin hence changes shape (so tropomyosin and troponin move away), allowing myosin to bind to actin, forming cross-bridges

- ATP hydrolysis ($\text{ATP} \rightarrow \text{ADP} + \text{P}_i$) causes myosin head (acts as ATPase) to tilt
- ADP and P_i detach and myosin head swings back / returns to previous position
- Actin is moved and power stroke occurs
- New ATP binds
- Myosin head detaches from actin and the cross-bridges break
- No ATP in striated muscle results to:
 - No pumping of calcium ions into SR
 - No detachment of myosin heads:
 - Hence no hydrolysis of ATP
 - Hence no cross bridge formation
 - Hence no power stroke / pulling of actin
 - Hence no recovery stroke / myosin head does not return to original position ;
- Stimulating contraction in striated muscle:
 - Action potential arrives resulting to the opening of Ca^{2+} channels in the presynaptic membrane, hence Ca^{2+} enters into the presynaptic neurone
 - Releasing vesicles containing acetylcholine to move towards and fuse to the presynaptic membrane
 - Acetylcholine released by exocytosis and diffuses across the cleft; binds to receptors on the sarcolemma
 - Na^+ channels open, hence Na^+ ions enter to depolarise the sarcolemma and generate action potential
 - Impulses then pass along the T-tubules towards the centre of the muscle fibre
 - Causing Ca^{2+} ions to bind with the troponin molecules
 - When there is no longer stimulation from the motor neurone, no impulses conducted along the T-tubules, hence the Ca^{2+} channels close and calcium pumps move Ca^{2+} back into stores in the SR; as Ca^{2+} leaves the binding sites on troponin, tropomyosin moves back to cover the myosin-binding sites, hence no more cross-bridges
- Muscles also have another source of ATP produced from creatine phosphate, keeping their stores in their sarcoplasm as their immediate source of energy



- Once energy demand has slowed down, ATP molecules produced by respiration can be used to 'recharge' the creatine



- Whereas if energy is still being demanded and there is no ATP spare to regenerate creatine phosphate, the creatine is converted to creatinine and excreted in urine

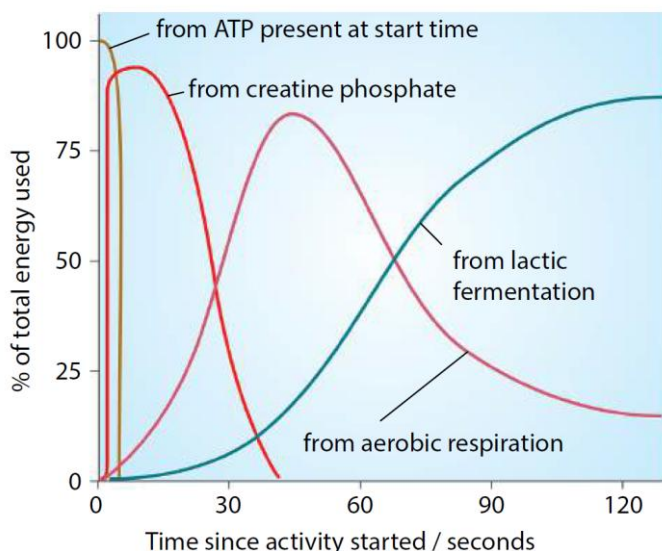


Figure 15.30 Energy sources used in muscle at high power output.

- The menstrual cycle – approximately 28 days:
 - Middle of this cycle, the female gamete is released in the oviduct, and if fertilisation occurs, the embryo develops in the uterus lining

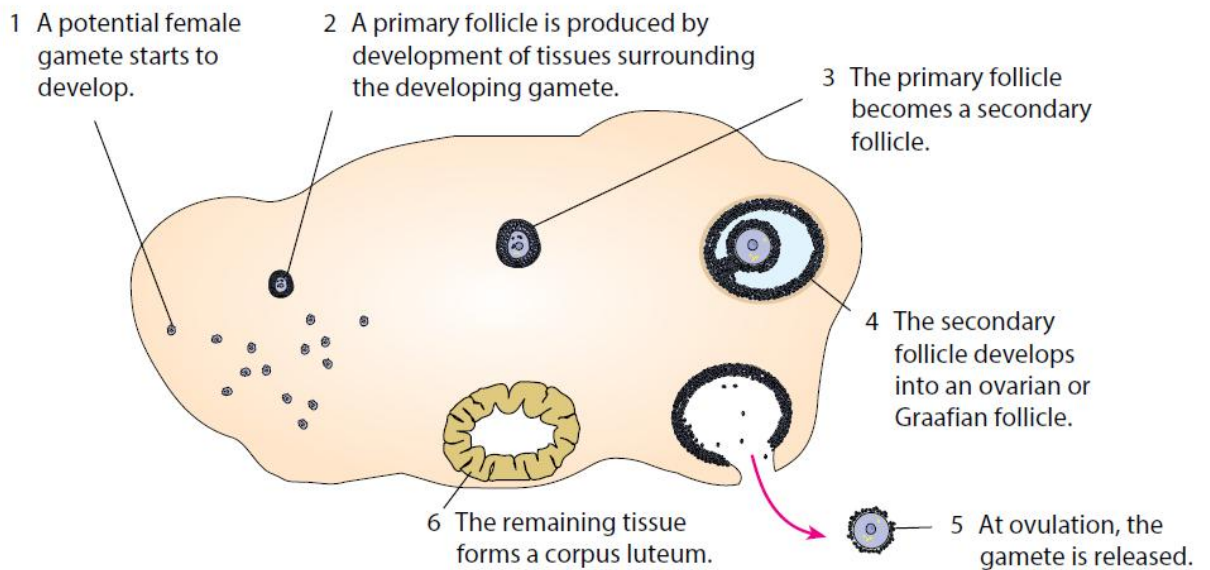


Figure 15.31 The ovary, showing the stages leading up to and following ovulation.

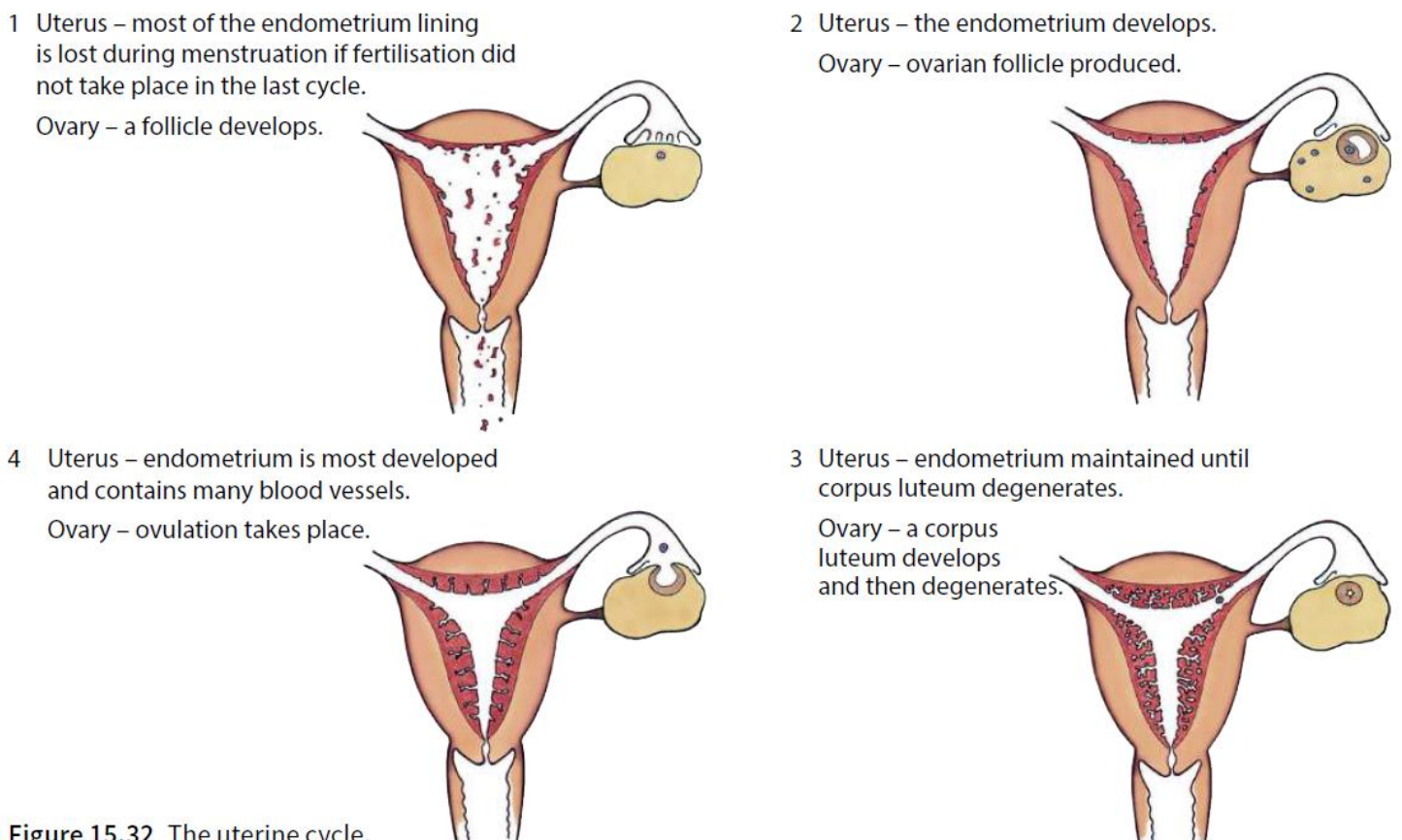


Figure 15.32 The uterine cycle.

- Coordinated by glycoprotein hormones released by the anterior pituitary gland – follicle **stimulating hormone** (FSH) and **luteinising hormone** (LH) which controls the activity of the ovaries – and ovaries (developing follicles secrete **oestrogen**,

whereas when the female gamete is released from the ovary at ovulation, the remains of the follicle (corpus luteum) secretes **progesterone**

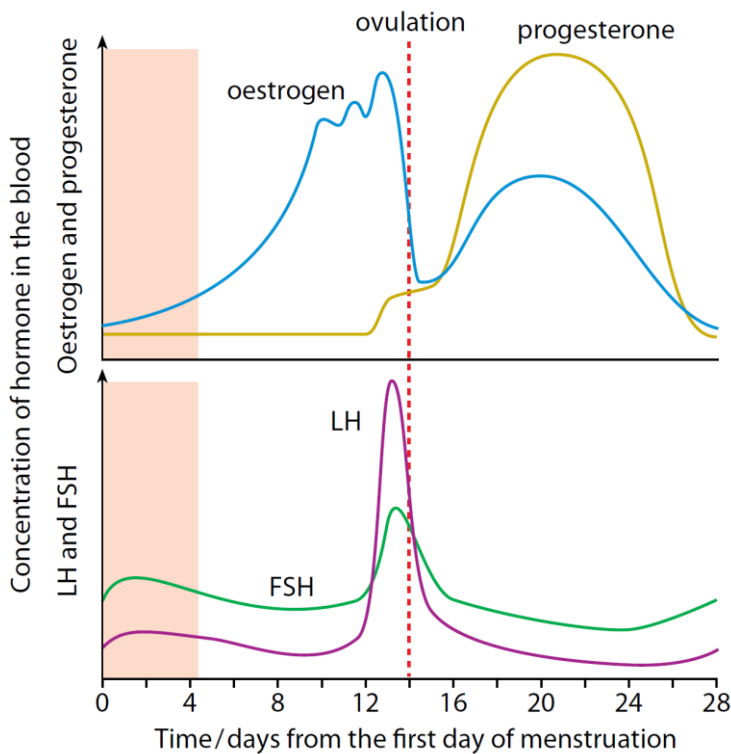


Figure 15.33 Changes in the concentrations of hormones in the blood during the menstrual cycle.

- FSH/LH, released by anterior pituitary during menstruation resulting to the development of Graafian follicle
 - In the ovary, due to the presence of LH and FSH, follicle cells secrete oestrogen
 - Oestrogen concentration rises for the first 12 days stimulating the endometrium to grow, thicken and develop numerous blood capillaries
 - The presence of oestrogen in the blood has a negative feedback effect on the production of LH and FSH, hence their concentrations decrease
 - Once the oestrogen levels have reached a high level (around day 14), it stimulates a surge in the secretion of LH and, to a lesser extent, of FSH; causes follicle to burst and shed its gamete into the oviduct (ovulation)
 - The follicle then collapses and form the corpus luteum, which secretes progesterone (maintains the lining of the uterus (endometrium) and inhibits the anterior pituitary from secreting FSH so no more follicle develop)
 - If no fertilisation, secretion of FSH/LH inhibited – as high levels of oestrogen and progesterone in the second half inhibit the secretion of FSH and LH – corpus luteum degenerates and progesterone concentration falls, resulting to the breakdown of the endometrium (menstruation)
 - Decrease also causes FSH to be secreted as anterior pituitary gland is no longer inhibited
- LH receptors located in the follicle cells, while FSH receptors located in the corpus luteal cells
 - Female mice without FSH receptors were sterile; they were found to have normal primary and secondary follicles in their ovaries, but no Graafian (ovarian) follicles or corpora lutea. Explain these observations:
 - Early follicle development is not dependent on FSH
 - With no FSH / no FSH receptors, follicle development stops

- With no FSH / no FSH receptors, Graafian / ovarian, follicle does not develop
- With no FSH / no FSH receptors, there is no ovulation
- No corpus luteum because these form from Graafian follicle / after ovulation
- Outline how the oestrogen/progesterone/combined contraceptive pill works to prevent pregnancy:
 - Synthetic hormones used as they do not get broken down quickly hence act for longer
 - Oestrogen / progesterone concentrations remain high, hence inhibits secretion of FSH and LH from anterior pituitary gland, where the presence of oestrogen in the blood has a negative feedback effect on the production of LH and FSH, hence their concentrations decrease, so Graafian follicle does not develop, thus inhibits ovulation
 - Thickens cervical mucus to prevent sperm from fertilising the egg
 - Thin endometrium prevent embryo implantation on endometrium
 - It is taken daily for 21 days; stops for 7 days to allow menstruation
- Discuss the biological, ethical and social implications of using this contraceptive pill:
 - ❖ Biological – negative
 - Rise in blood pressure
 - Nausea and headaches
 - Increased risk of breast cancer
 - Increase in STDs
 - ❖ Biological – positive
 - Regular menstruation
 - Reduced risk of developing, ovarian cancer
 - Reduced risk of uterine infections
 - ❖ Social / ethical – negative
 - Sexual freedom has led to more marriage breakdowns, hence more single parent families
 - Religious/cultural, objection
 - ❖ Social / ethical – positive
 - Reduction in unwanted pregnancies and abortions
 - Women have control over their fertility
 - Acts as a population control
- Venus fly trap:
 - Each trap consists of a pair of modified leaves joined by a midrib of hinge cells
 - The modified leaves have touch-sensitive hairs; if two hairs are touched within 20 seconds, or the same hair is touched twice in rapid succession, the trap closes
 - The surface of the lobes has many glands that secrete enzymes for the digestion of trapped insects
 - The touch of insects on the sensory hairs activates calcium ion channels in cells at the base of the hair to open, causing an influx in Ca^{2+} to generate action potential
 - Auxin increase triggered in hinge cells
 - H^+ ions pumped into the cell walls and calcium pectate 'glue' in cell wall dissolved
 - Ca^{2+} ions enter hinge cell
 - Water follows by osmosis down its water potential gradient
 - Hinge cells expand
 - Trap lobes, flip from convex to concave (change in elastic tension)
- Two types of plant growth regulators (hormones):

- Auxins: influence many aspects of growth including elongation growth which determines the overall length of roots and shoots; synthesised in the growing tips (meristems) of shoots and roots, where cells are dividing; actively transported from cell to cell
- Gibberellins: involved in seed germination and controlling stem elongation
- Role of auxin in cell elongation in plants:
 - Acid-growth hypothesis
 - Auxin stimulates proton pumps in the cell surface membrane
 - H^+ pumped into the cell wall by active transport, hence the pH of cell wall decreases, so pH-dependent enzymes activated (expansins) and loosen the bonds between cellulose microfibrils
 - Cell wall 'loosens' / becomes more elastic / able to stretch, hence more water enters the cell and turgor pressure increases, resulting to the expansion of the cell wall

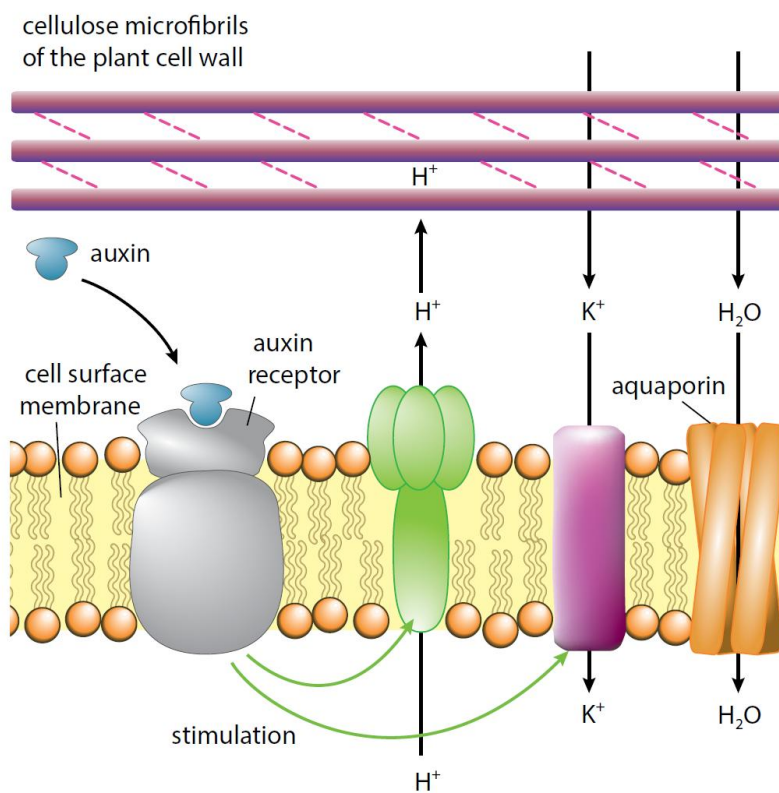


Figure 15.37 The binding of auxin to its receptor is thought to activate a membrane protein, which stimulates the pumping of protons out of the cell into the cell wall where they lower the pH and break bonds. Potassium ion channels are also stimulated to open leading to an increase in potassium ion concentration in the cytoplasm. This decreases the water potential so water enters through aquaporins.

- The role of gibberellin in the germination of wheat or barley:
 - Seed is initially dormant
 - When the seed absorbs water, embryo produces gibberellin
 - Gibberellin diffuses onto the aleurone layer and stimulate the cells to synthesise amylase, by the affecting the gene transcription of mRNA for amylase (breakdown of DELLA proteins which inhibit germination)
 - Amylase hydrolyses starch in the endosperm to soluble maltose molecules, which are converted to glucose and transported to the embryo for respiration (ATP for growth)
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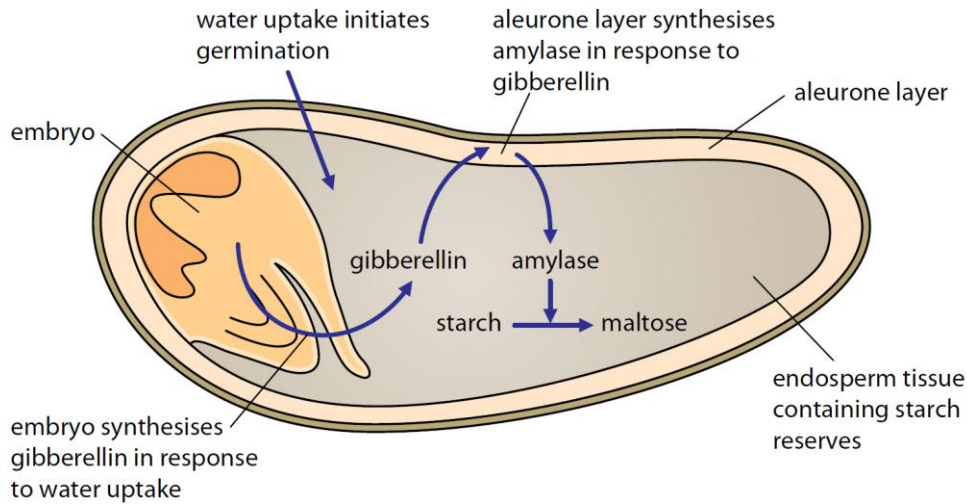


Figure 15.38 Longitudinal section through a barley seed, showing how secretion of gibberellins by the embryo results in the mobilisation of starch reserves during germination.

- The height of some plants is partly controlled by their genes, e.g. tallness in pea plants is affected by a gene with two alleles; if the dominant allele, **Le**, is present, the plants can grow tall, but plants homozygous for the recessive allele, **le**, always remain short
 - The dominant allele of this gene regulates the synthesis of the last enzyme in a pathway that produces an active form of gibberellin, **GA1**
 - Active gibberellin stimulates cell division and cell elongation in the stem, so causing the plant to grow tall
 - A substitution mutation in this gene gives rise to a change from alanine to threonine in the primary structure of the enzyme near its active site, producing a non-functional enzyme; this mutation has given rise to the recessive allele, **le**; hence homozygous plants, **lele**, are genetically dwarf as they do not have the active form of gibberellin