15 Control and co-ordination

All the activities of multicellular organisms require co-ordinating, some very rapidly and some more slowly. The nervous system and the endocrine system provide co-ordination in mammals. Similar co-ordination systems exist in plants.

15.1 Control and co-ordination in mammals

The nervous system provides fast communication between receptors and effectors.

Transmission between neurones takes place at synapses.

By the end of this topic you should be able to:

a) compare the nervous and endocrine systems as communication systems that co-ordinate responses to changes in the internal and external environment
b) describe the structure of a sensory neurone and a motor neurone
c) outline the roles of sensory receptor cells in detecting stimuli and stimulating the transmission of nerve impulses in sensory neurones
d) describe the functions of sensory, relay and motor neurones in a reflex arc
e) describe and explain the transmission of an action potential in a myelinated neurone and its initiation from a resting potential
f) explain the importance of the myelin sheath (salutatory conduction) in determining the speed of nerve impulses and the refractory period in determining their frequency
g) describe the structure of a cholinergic synapse and explain how it functions, including the role of calcium ions
h) outline the roles of synapses in the nervous system in allowing transmission in one direction and in allowing connections between one neurone and many others

The need for communication systems

The ability to detect change and to make responses is essential for the survival of living things. It is as much a feature of single-celled organisms as it is of flowering plants and mammals. We see this when an *Amoeba* detects a suitable food organism and captures it by phagocytosis.

Large and complex organisms detect changes in the external environment as well as changes within the body. They need to communicate this information to parts of the body where appropriate responses will be made.

We call changes that bring about responses **stimuli**. The stimulus is detected by a **receptor** and an **effector** brings about a response. Since response often occurs in a different part of the body, efficient internal communication is also essential. In mammals, internal communication involves both the **nervous** and **endocrine** (hormone-producing) systems. We will examine these next.

Introducing the nervous system

The nervous system is built from specialised nerve cells called **neurones**. Neurones are grouped together to form the **central nervous system**, which consists of the **brain and spinal cord**. To and from the central nervous system run nerves of the **peripheral nervous system**. Communication between the central nervous system and all parts of the body occurs via neurones in these nerves.

Specialised nerve cells (**neurones**) are organised into **central nervous system** and **peripheral nerves** linking **sense organs**, **muscles** and **glands** with the **brain** or **spinal cord**.

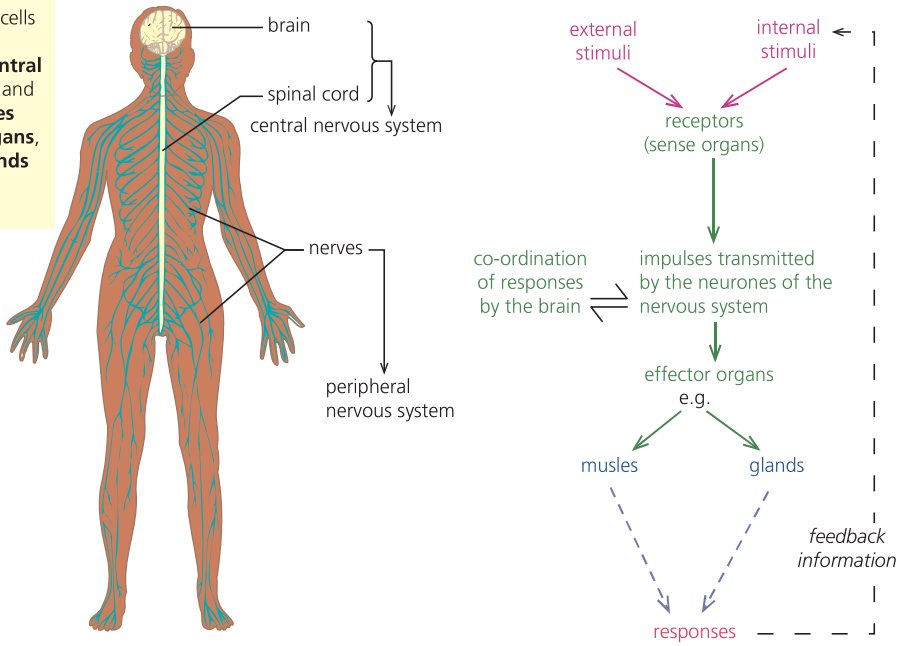


Figure 15.1 The organisation and co-ordination of the mammalian nervous system

Neurones – structure and function

Each neurone has a **cell body** containing the nucleus and the bulk of the cytoplasm. From the cell body, extremely fine **cytoplasmic processes** run. Many of these processes are very long indeed. They are specialised for the transmission of information in the form of **impulses**. Impulses are transmitted at speeds between 30 and 120 metres per second in mammals, so nervous co-ordination is extremely fast and responses are virtually immediate. Another feature of the nerve impulse is that it travels to particular points in the body. Consequently, the effects of impulses are localised rather than diffuse. (This makes communication by nerves different from that by hormones, as we shall see.)

The three types of neurones are shown in Figure 15.2.

Question

1 Compare motor, sensory and relay neurones by means of a concise table.

- **Motor neurones** have a cell body that lies within the brain or spinal cord. Many highly-branched cell processes extend from the cell body. These are called **dendrites**. Dendrites receive impulses from other neurones and conduct them *towards* the cell body. A single long **axon** transmits impulses *away* from the cell body. The function of a motor neurone is to transmit impulses from the central nervous system to effector organs, such as muscles or glands.
- **Relay neurones** (also known as inter-neurones) have numerous, short fibres. Each fibre is a thread-like extension of a nerve cell. Relay neurones occur in the central nervous system. They relay impulses to other neurones.
- **Sensory neurones** have a single long **dendron**, which brings impulses *towards* the cell body, and a single **axon** which carries impulses *away* from the cell body. Sensory neurones transmit impulses from receptors to the spinal cord or brain.

Surrounding the neurones there are different types of supporting cells called **glia cells**. These are also an important part of the nervous system. In the brain and spinal cord there are several types of glia cells. In the peripheral nervous system, the axons of many neurones are enclosed by glia cells called **Schwann cells**. These wrap themselves around the axon with many layers of cell surface membrane, forming a structure called a **myelin sheath** (Figure 15.2). The sheath consists largely of lipid with some protein. Between each pair of Schwann cells is a tiny, uncovered junction in the myelin sheath, called a **node of Ranvier**. The myelin sheath and its junctions help increase the speed at which impulses are conducted – a point we will return to shortly.

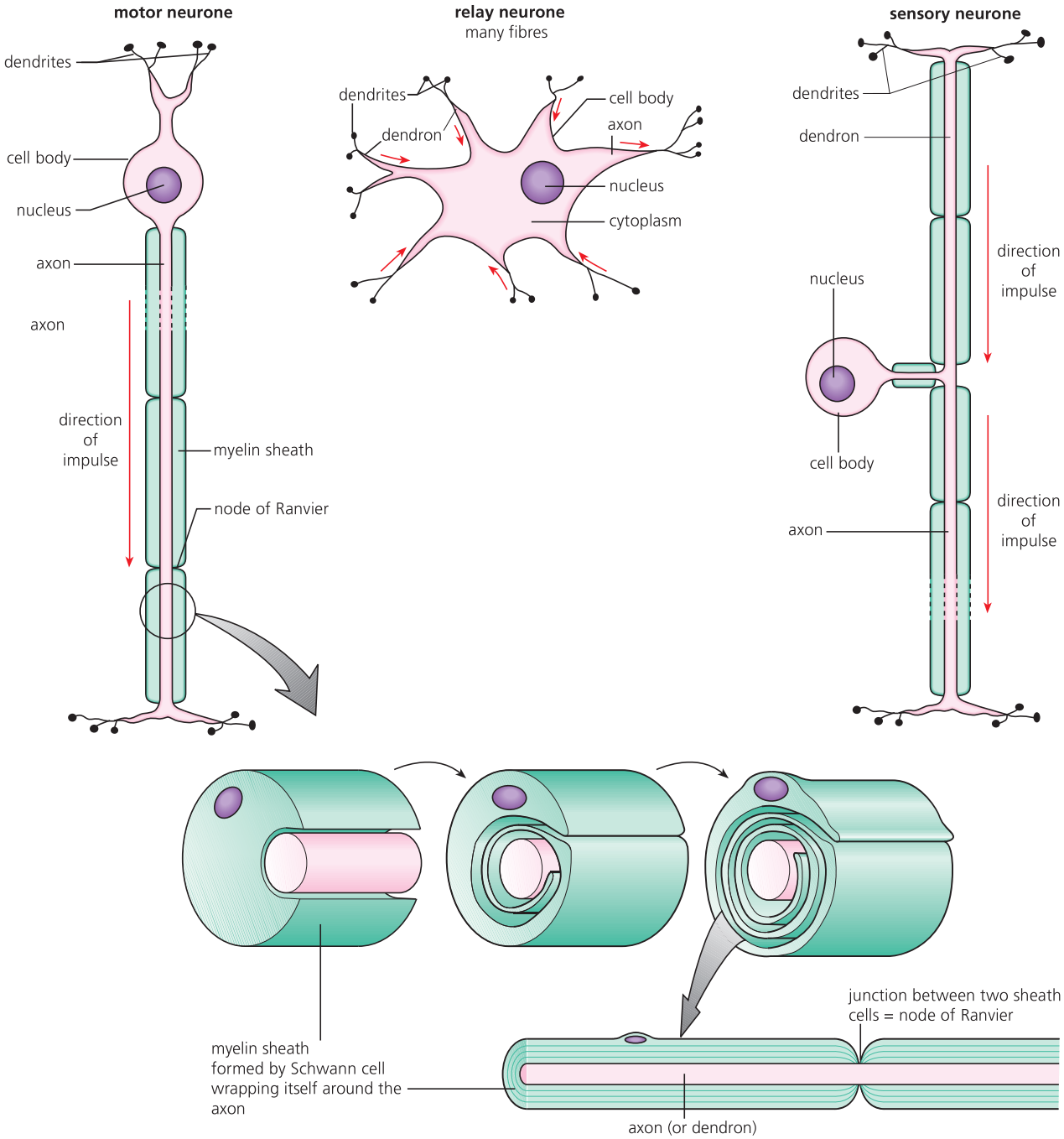


Figure 15.2 Neurones of the nervous system

Organisation among neurones – reflex arcs and reflex action

The transmission of nerve impulses is not a haphazard process. It involves organised pathways among the neurones. These pathways are called **reflex arcs**. The reflex arc consists of a **receptor,** which may be a sensory organ, connected by **neurones** to an **effector**, which may be a muscle or gland. A generalised reflex arc is shown in Figure 15.3.

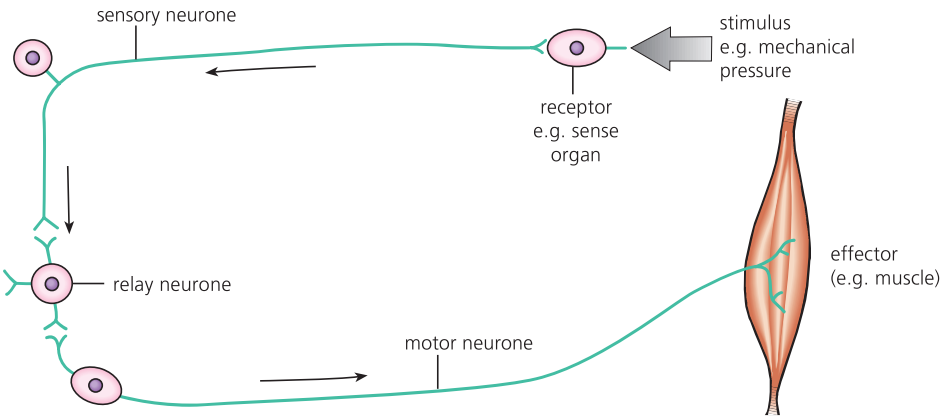


Figure 15.3 The layout of a reflex arc: the structural basis of reflex action

How does a reflex arc work?

The action begins when a sense organ detects a stimulus, which is a form of energy such as sound, light or mechanical pressure. This may become an impulse transmitted by a neurone that serves that sense cell. Once generated, the impulse is transmitted along dendrons and axons of a sequence of neurones of the reflex arc to an effector organ. When it arrives at the effector, the impulse causes a response – for example, it may cause a muscle to contract or a gland to secrete.

Extension

Reflex arcs and the nervous system

What has just been described is the simplest form of response in the nervous system. In mammals there is a complex nervous system. Many neurones connect reflex arcs with the **brain**. The brain contains a highly organised mass of relay neurones, connected with the rest of the nervous system by motor and sensory neurones.

With a nervous system of this type, complex patterns of behaviour are common, in addition to many reflex actions. This is because:

- impulses that originate in a reflex arc also travel to the brain
- impulses may originate in the brain and be conducted to effector organs.

Consequently much activity is **initiated** by the brain, rather than being simply a response to external stimuli. Also, reflex actions may be **overruled** by the brain and the response modified (as when we decide not to drop an extremely hot object because of its value).

So, the nervous system of mammals has roles in both **quick, precise communication** between the sense organs that detect stimuli and the muscles or glands that cause changes and in the **complex behaviour patterns** that mammals display.

Sensory receptors and the conversion of energy into impulses

All cells are sensitive to changes in their environment, but **sense cells** are specialised to detect stimuli and to respond by producing an impulse (an **action potential**). Specialised sense cells are called **receptors**. Different types of sensory receptors exist in the body.

Table 15.1 The sense organs of mammals

Sense data (form of energy)	Type of receptor	Location in the body
Mechanoreceptors: respond to mechanical stimulation		
light touch	touch receptors	mostly in dermis of skin
touch and pressure	touch and pressure receptors	dermis of skin
movement and position	stretch receptors, e.g. muscle spindles, proprioceptors	skeletal muscle
sound waves and gravity	sensory hair cells	cochlea and other parts of the inner ear
blood pressure	baroreceptors	aorta and carotid artery
Thermoreceptors: respond to thermal stimulation		
temperature change in the skin	nerve endings	dermis of skin
internally	cells of hypothalamus	brain
Chemoreceptors: responding to chemical stimulation		
chemicals in the air	sense cells of olfactory epithelium	nose
taste	taste buds	tongue
blood oxygen and carbon dioxide concentrations and pH	carotid body	carotid artery
osmotic concentration of the blood	osmoregulatory centre in hypothalamus	brain
Photoreceptors: respond to electromagnetic stimulation		
light	rod and cone cells of retina	eye

Question

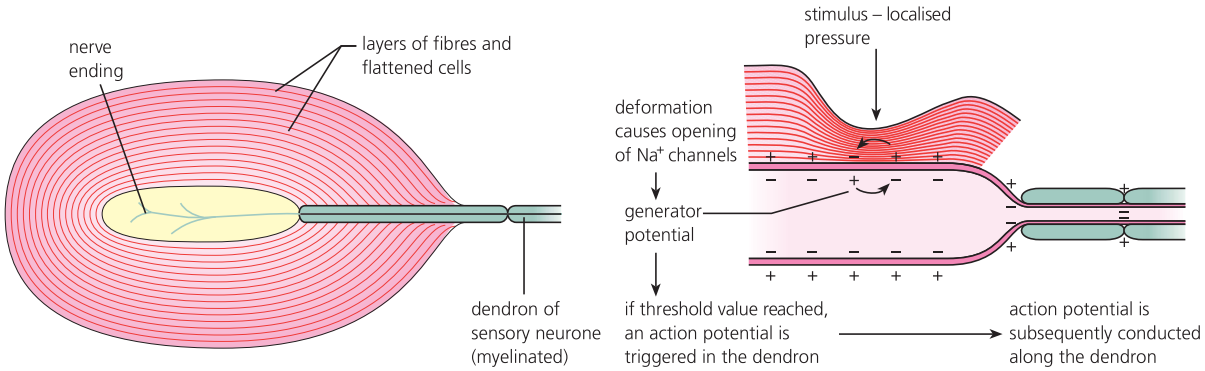
2 Use Table 15.1 to list the various types of stimuli (sense data) that originate from conditions within the human body that are detected by particular receptors.

The Pacinian corpuscle

At rest, the nerve ending within the Pacinian corpuscle maintains a resting potential of −70 mV, typical of the membrane of a neurone. When localised, strong pressure is applied to a Pacinian corpuscle it causes the layers of collagen of the capsule to be deformed. The outcome is a temporary change to the permeability of the membrane at the nerve ending. Sodium ions flow in, the membrane is depolarised and the interior starts to become less negative. The stronger the stimulus, the greater the depolarisation. If a threshold value is reached, it triggers an impulse in the sensory neurone that serves the sense cell.

The property of a sense cell is to transfer the energy of a particular type of stimulus into the electrochemical energy of an impulse, which is then conducted to other parts of the nervous system. The stimulus that the sense cell responds to is some form of energy, **mechanical**, **chemical**, **thermal** or **light** (**photic**). The sense organs of mammals are listed in Table 15.1 under headings of the stimuli they respond to.

Figure 15.4 The structure and function of a Pacinian corpuscle



Transmission of an impulse

An impulse is transmitted along nerve fibres, but it is *not* an electrical current that flows along the 'wires' of the nerves. Rather, the impulse is a momentary reversal in electrical potential difference in the membrane, caused by the rapid movements of sodium and potassium ions into and out of the axon.

The resting potential

A neurone that is not transmitting an impulse is said to be at rest. Actually, this not the case; during the 'resting' interval between the transmission of impulses, the membrane of a neurone actively creates and maintains an electrical potential difference between the inside and the outside of the fibre. The result is that, in a 'resting' neurone there is a potential difference between the inside and the outside of the fibre of about −70 mv. (The discovery of the resting potential came from the use of an external and internal microelectrode on an isolated axon, the amplification of the signal and its display on a cathode ray oscilloscope).

Two processes together produce the resting potential difference across the neurone membrane.

- There is **active transport** of potassium ions (K^+) in across the membrane and of sodium ions (Na^+) out across the membrane. The ions are transported by a sodium–potassium pump, with transfer of energy from ATP. So potassium and sodium ions gradually concentrate on opposite sides of the membrane. Of course, this in itself makes *no change* to the potential difference across the membrane.
- There is also facilitated diffusion of potassium ions out and sodium ions back in. The important point here is that the membrane is *far more permeable* to potassium ions flowing out than to sodium ions returning. This causes the tissue fluid outside the neurone to contain many more positive ions than are present in the cytoplasm inside. As a result, the inside becomes more and more negatively charged compared with the outside; the resting neurone is said to be **polarised**. The difference in charge, or potential difference, is about −70 mV. This is known as the **resting potential**. Figure 15.5 is a summary of how it is set up.

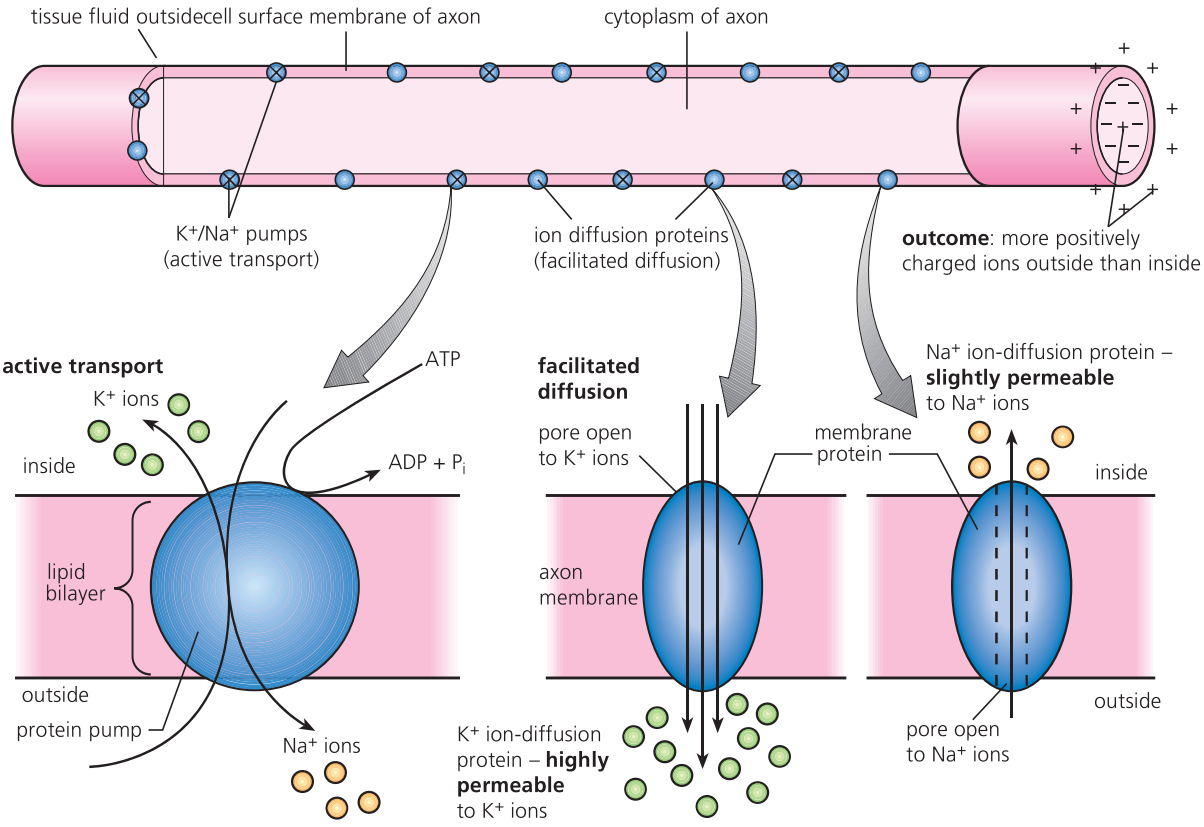


Figure 15.5 The establishment of the resting potential

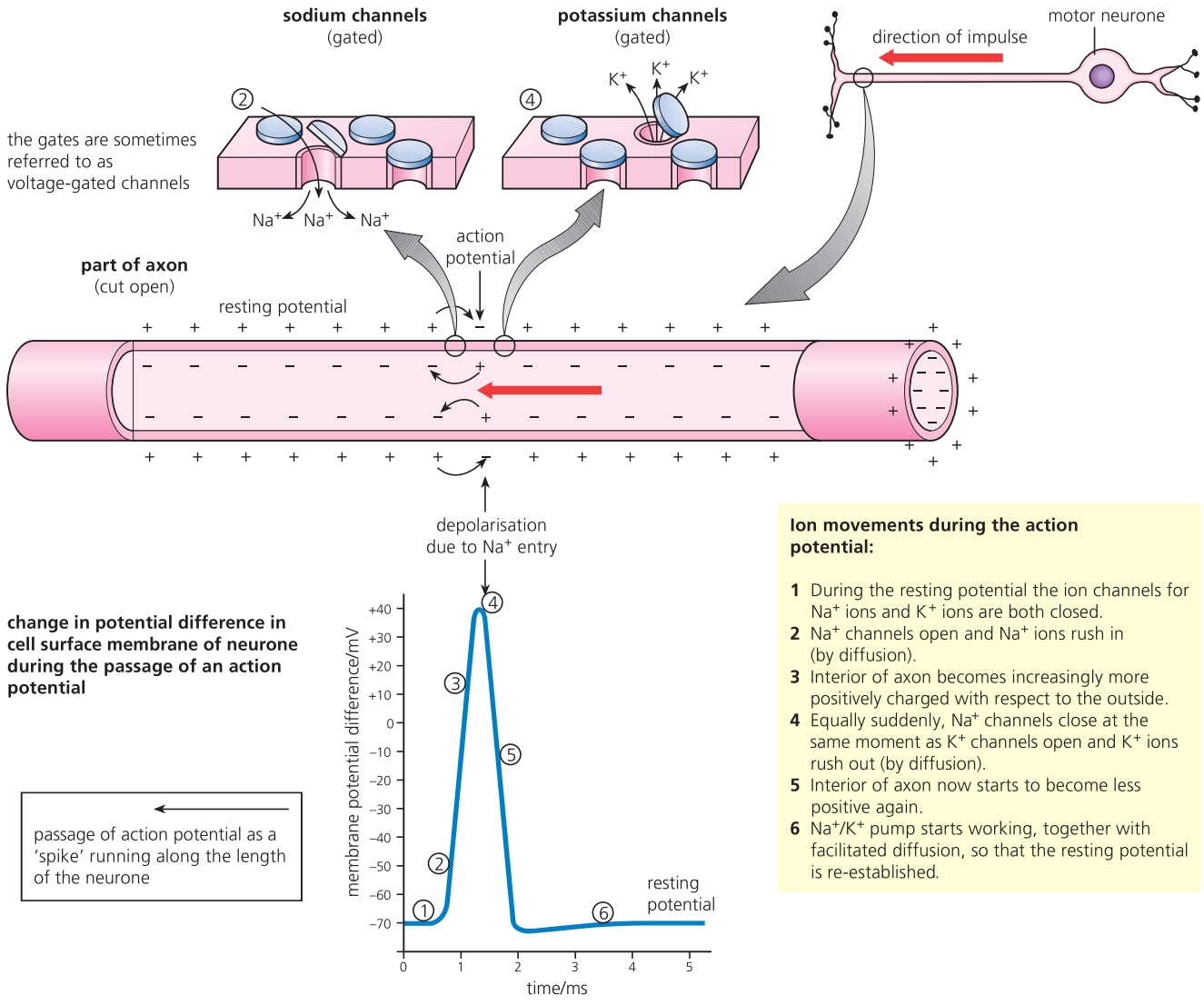
The action potential

The next event in a neurone, sooner or later, is the passage of an impulse. An impulse or **action potential** is triggered by a stimulus received at a receptor cell or sensitive nerve ending.

In the action potential, the energy transferred by this stimulus causes a temporary and local reversal of the resting potential. The result is that the membrane is briefly **depolarised** at this point. How does this happen?

The change in potential across the membrane occurs through pores in the membrane. They are called **ion channels** because they can allow ions to pass through. One type of channel is permeable to sodium ions and another to potassium ions. These channels are globular proteins that span the entire width of the membrane. They have a central pore with a gate which can open and close – they are **gated channels**. During a resting potential these channels are all closed.

The energy of the stimulus first opens the gates of the **sodium channels** in the cell surface membrane. This allows sodium ions to diffuse in, down their electrochemical gradient. This influx of sodium ions is very rapid indeed. So the cytoplasm inside the neurone fibre quickly becomes progressively more positive with respect to the outside. This charge reversal continues until the potential difference has altered from –70 mV to +40 mV. At this point, an action potential has been created in the neurone fibre.



Ion movements during the action potential:

1. During the resting potential the ion channels for Na^+ ions and K^+ ions are both closed.
2. Na^+ channels open and Na^+ ions rush in (by diffusion).
3. Interior of axon becomes increasingly more positively charged with respect to the outside.
4. Equally suddenly, Na^+ channels close at the same moment as K^+ channels open and K^+ ions rush out (by diffusion).
5. Interior of axon now starts to become less positive again.
6. Na^+/K^+ pump starts working, together with facilitated diffusion, so that the resting potential is re-established.

Figure 15.6 The action potential

The action potential then travels along the whole length of the neurone fibre. At any one point it exists for only two thousandths of a second (2 milliseconds), before the membrane starts to re-establish the resting potential. So action potential transmission is exceedingly quick – an example of **positive feedback**, in fact.

Almost immediately an action potential has passed, the sodium channels close and the potassium channels open. So potassium ions can exit the cell, again down an electrochemical gradient, into the tissue fluid outside. This causes the interior of the neurone fibres to start to become less positive again. Then the potassium channels also close. Finally, the resting potential of –70 mV is re-established by the action of the sodium–potassium pump and the process of facilitated diffusion.

Questions

3 Distinguish between positive and negative feedback.

4 What is the source of energy used to
   a establish the resting potential
   b power an action potential?

5 Why is an axon unable to conduct an impulse immediately after an action potential has been conducted?

The refractory period

For a brief period, following the passage of an action potential, the neurone fibre is not excitable. This is the **refractory period**. It lasts only 5–10 milliseconds in total. During this time, firstly there is a large excess of sodium ions inside the neurone fibre and further influx is impossible. As the resting potential is progressively restored, however, it becomes increasingly possible for an action potential to be generated again. Because of this refractory period, the maximum frequency of impulses is between 500 and 1000 per second.

The 'all or nothing' principle

Obviously stimuli are of widely different natures, such as the difference between a light touch and the pain of a finger hit by a hammer. A stimulus must be at or above a minimum intensity, known as the **threshold of stimulation**, in order to initiate an action potential at all. Either a stimulus depolarises the membrane sufficiently to reverse the potential difference in the cytoplasm (–70 mV to +40 mV), or it does not. If not, no action potential is generated. With all sub-threshold stimuli, the influx of sodium ions is quickly reversed, and the resting potential is re-established.

However, as the intensity of the stimulus increases the frequency at which action potentials pass along the fibre increases. (Note that individual action potentials are all of standard amplitude.) For example, with a very persistent stimulus, action potentials pass along a fibre at an accelerated rate, up to the maximum possible permitted by the refractory period. This means the effector (or the brain) recognises the intensity of a stimulus from the **frequency** of action potentials (Figure 15.7).

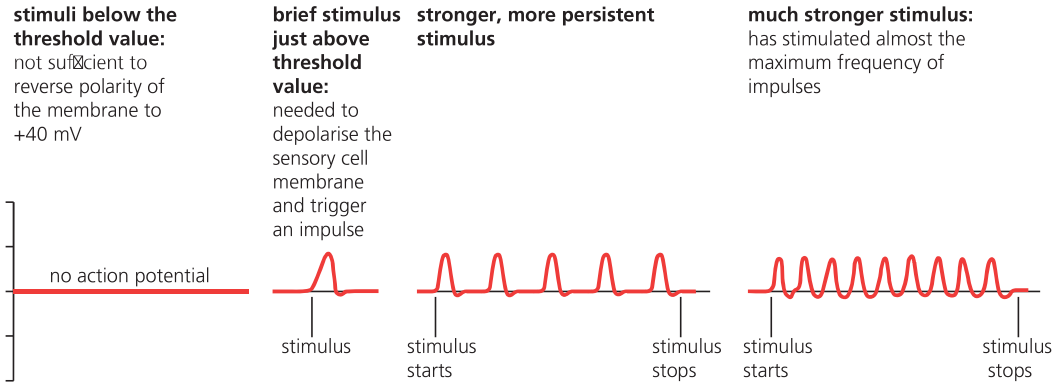


Figure 15.7 Weak and strong stimuli and the threshold value

Speed of conduction of the action potential

We noted earlier that the presence of a myelin sheath affects the speed of transmission of the action potential. The junctions in the sheath, known as the nodes of Ranvier, occur at 1–2 mm intervals. Only at these nodes is the axon membrane exposed. Elsewhere along the fibre, the electrical resistance of the myelin sheath prevents depolarisations. Consequently, the action potentials are forced to jump from node to node. This is called **saltatory conduction** (from the Latin *saltare* meaning 'to leap'). This is an advantage, as it greatly speeds up the rate of transmission.

Not all neurones have myelinated fibres. In fact, non-myelinated dendrons and axons are common in non-vertebrate animals. Here, transmission is normally much slower, because the action potential flows steadily, all along the fibres. However, among non-myelinated fibres it is a fact that large diameter axons transmit action potentials much more speedily than do narrow ones. Certain non-vertebrates like the squid and the earthworm have giant fibres, which allow fast transmissions of action potentials (not as fast as in myelinated fibres, however). We saw that the original investigation of action potentials was carried out on such giant fibres.

Question

6 Why do myelinated fibres conduct impulses faster than non-myelinated fibres of the same size?

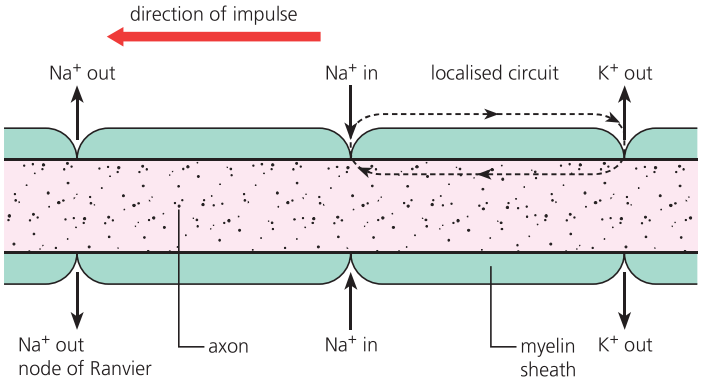


Figure 15.8 Saltatory conduction

Synapses – the junctions between neurones

Where two neurones meet they do not actually touch. A tiny gap, called a **synapse**, is the link point between neurones. Synapses consist of the swollen tip (**synaptic knob**) of the axon of one neurone (the **pre-synaptic neurone**) and the dendrite or cell body of another neurone (the **post-synaptic neurone**). Between these is the **synaptic cleft**, a gap of about 20 nm.

The practical effect of the synaptic cleft is that an action potential cannot cross it. Here, transmission occurs by particular chemicals, known as **transmitter substances**. These substances are all relatively small, diffusible molecules. They are produced in the Golgi apparatus in the synaptic knob and held in tiny vesicles before use.

Acetylcholine (**ACh**) is a commonly occurring transmitter substance. The neurones that release acetylcholine are known as **cholinergic neurones**. Another common transmitter substance is **noradrenalin** (released by **adrenergic neurones**). In the brain the commonly occurring transmitters are glutamic acid and dopamine.

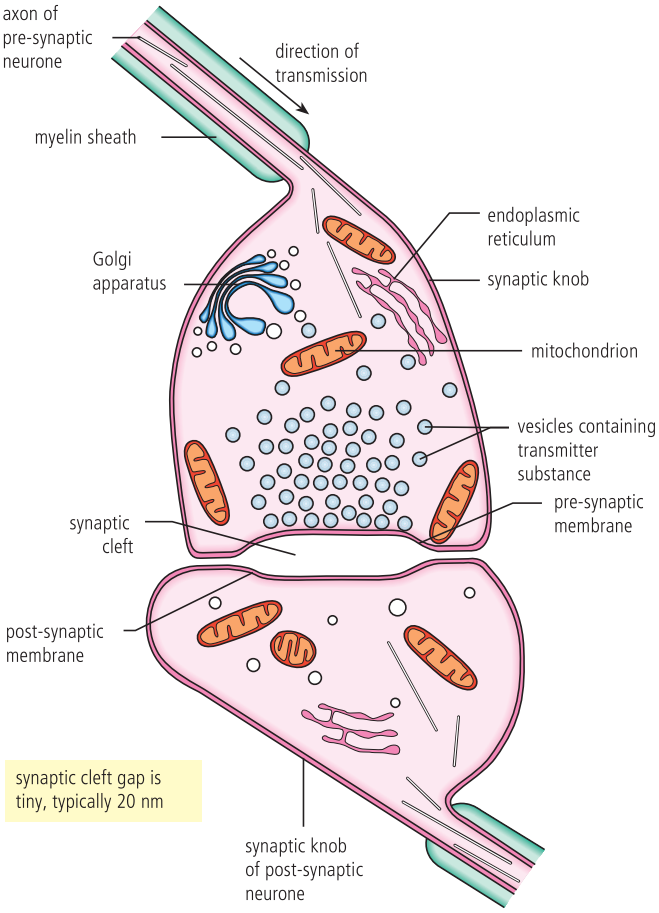
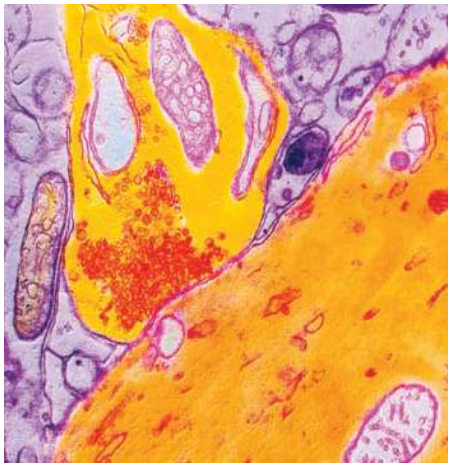


Figure 15.9 A synapse in section



TEM of a synapse (×100 000)

Steps to synapse transmission

It may help to follow the steps in Figure 15.10.

1 The arrival of an action potential at the synaptic knob opens **calcium ion (Ca^{2+}) channels** in the **pre-synaptic membrane**. Calcium ions flow in from the synaptic cleft.

2 The calcium ions cause **vesicles of transmitter substance** to fuse with the pre-synaptic membrane and they release a transmitter substance into the synaptic cleft.

3 The transmitter substance diffuses across the synaptic cleft and binds with a **receptor protein**. In the **post-synaptic membrane** there are specific receptor sites for each transmitter substance. Each of these receptors also acts as a channel in the membrane which allows a specific ion (sodium, potassium or some other ion) to pass. The attachment of a transmitter molecule to its receptor instantly **opens the ion channel**.

When a molecule of the transmitter substance attaches to its receptor site, sodium ion channels open. As the sodium ions rush into the cytoplasm of the post-synaptic neurone, **depolarisation** of the post-synaptic membrane occurs. As more and more molecules of the transmitter substance bind, it becomes increasingly likely that depolarisation will reach the **threshold level**. When it does, an **action potential** is generated in the post-synaptic neurone. This process of build-up to an action potential in post-synaptic membranes is called **facilitation**.

4 The transmitter substance on the receptors is quickly **inactivated**. For example, the enzyme cholinesterase hydrolyses acetylcholine to choline and ethanoic acid. These molecules are inactive as transmitters. This reaction causes the ion channel of the receptor protein to close and so allows the resting potential in the post-synaptic neurone to be re-established.

5 Meanwhile, the inactivated products of the transmitter re-enter the pre-synaptic knob, are **resynthesised** into transmitter substance and packaged for reuse.

Question

7 In the synaptic knob, what is the role of:
a the Golgi apparatus
b mitochondria?

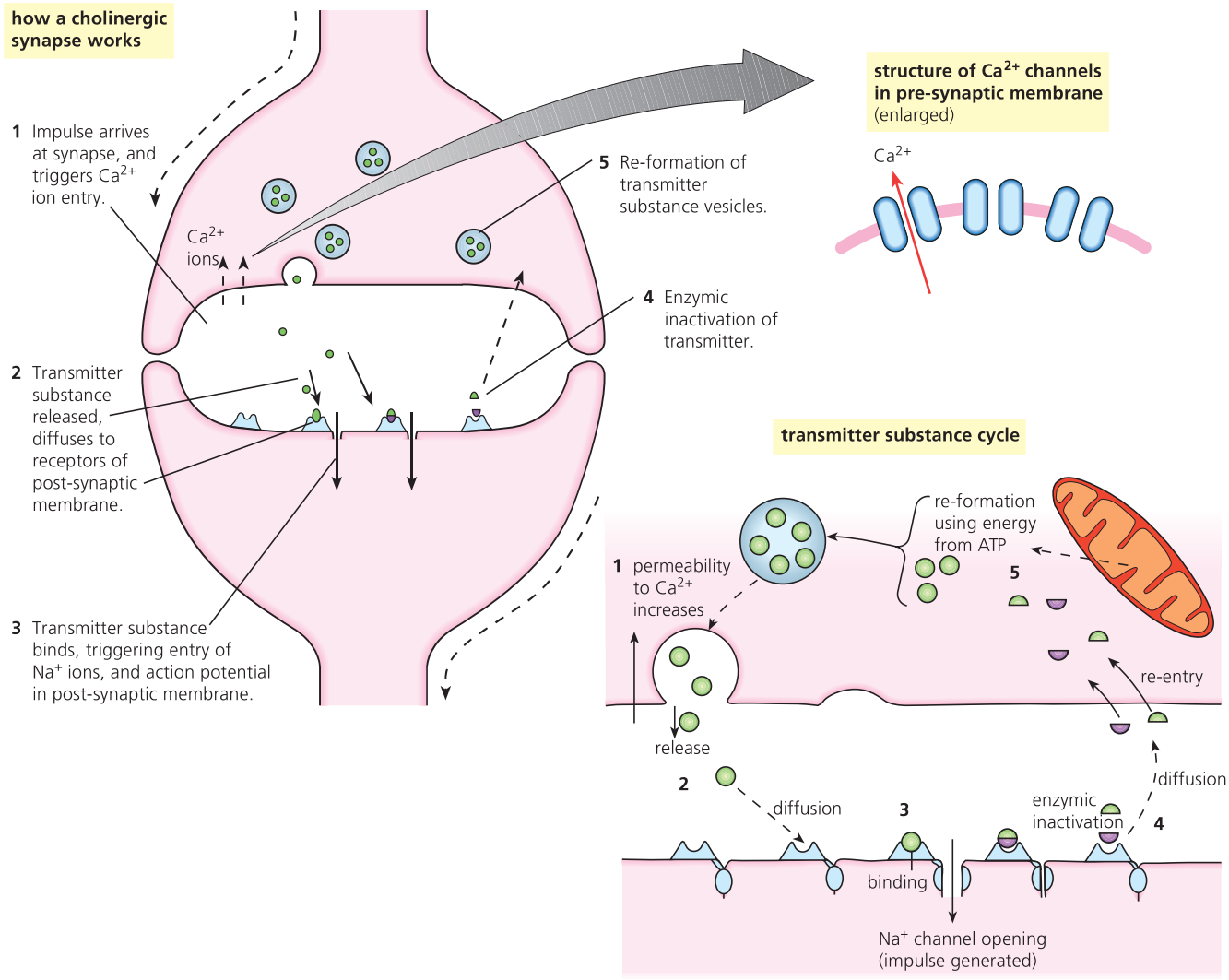


Figure 15.10 Chemical transmission at a synapse

Extension

An alternative type of synapse

In this section on synapses, it is an **excita-tory synapse** that has been described. That is, the incoming action potential *excited* the post-synaptic membrane and generated an action potential that was then transmitted along the post-synaptic neurone. Some synapses, however, have the opposite effect. These are known as **inhibitory synapses**.

Why have synapses between neurones?

Since synapses have the disadvantage of very slightly slowing down the transmission of action potentials, we may assume they also provide distinct advantages to the operation of nervous communication in organisms. In fact there are a number of advantages.

- They filter out low-level stimuli of limited importance.
- They protect the effectors (muscles and glands) from over-stimulation, since continuous transmission of action potentials eventually exhausts the supply of transmitter substances for a period of time. This is called synapse fatigue.
- They facilitate flexibility of response by the central nervous system, particularly by the brain.
- They allow integration of information since the post-synaptic neurone may receive action potentials from different types of neurone. Both excitatory pre-synaptic neurones and inhibitory pre-synaptic neurones exist. The post-synaptic neurone summates all the action potentials, thereby integrating action potentials from more than one source neurone or sense organ, for example.

Extension

Drugs may interfere with the activity of synapses

Some drugs **amplify** the processes of synaptic transmission, in effect they increase post-synaptic transmission. Nicotine and atropine have this effect.

Other drugs **inhibit** the processes of synaptic transmission, in effect decreasing synaptic transmission. Amphetamines and beta-blocker drugs have this effect.

15.1 *continued...* Neuro-muscular junctions and muscle contraction

Motor nerve endings make connections with muscle fibres and trigger muscle contraction.

By the end of this section you should be able to:

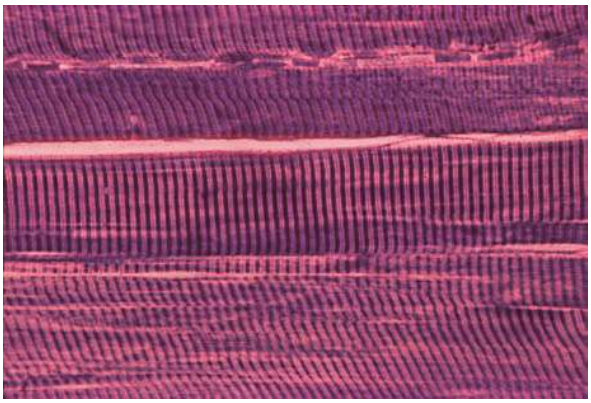
i) describe the roles of neuromuscular junctions, transverse system tubules and sarcoplasmic reticulum in stimulating contraction in striated muscle
j) describe the ultrastructure of striated muscle with particular reference to sarcomere structure
k) explain the sliding filament model of muscular contraction including the roles of troponin, tropomyosin, calcium ions and ATP

The structures of striated muscle

Skeletal muscle fibres appear striped under the light microscope, so skeletal muscle is also known as striated muscle. Striated muscle consists of bundles of muscle fibres (Figure 15.11). The remarkable feature of a muscle fibre is the ability to shorten to half or even a third of the relaxed or resting length. Actually, each fibre is itself composed of a mass of **myofibrils**, but we need the electron microscope to see this important detail.

The ultrastructure of skeletal muscle

Skeletal muscle fibres are multinucleate and contain specialised endoplasmic reticulum. By electron microscope we can see that each muscle fibre consists of very many parallel myofibrils within a plasma membrane known as the sarcolemma, together with cytoplasm. The cytoplasm contains **mitochondria** packed between the myofibrils. The sarcolemma infolds to form a system of transverse tubular endoplasmic reticulum (sometimes referred to as **T tubules**), and all parts of the **sarcoplasmic reticulum**. This is arranged as a network around individual myofibrils. The arrangements of myofibrils, sarcolemma and mitochondria, surrounded by the sarcoplasmic membrane, are shown in Figure 15.12.



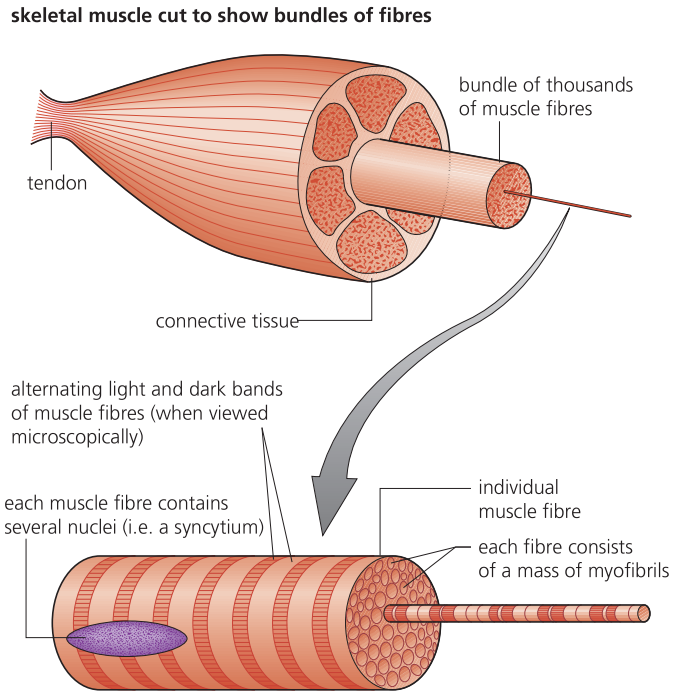
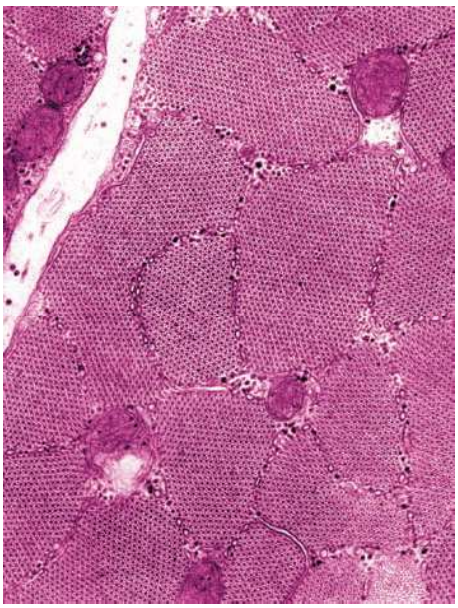


Figure 15.11 The structure of skeletal muscle

electron micrograph of TS through part of a muscle fibre, HP (x36 000)



stereogram of part of a single muscle fibre

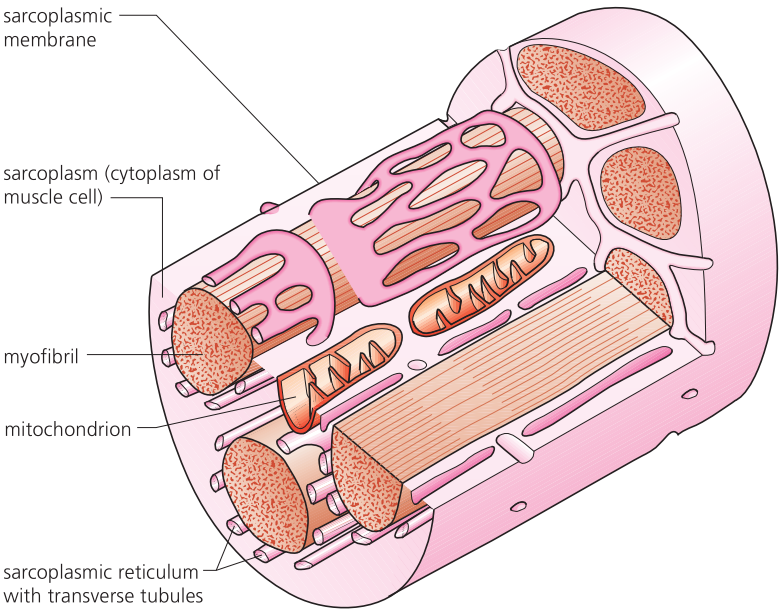


Figure 15.12 The ultrastructure of a muscle fibre

The striped appearance of skeletal muscle is due to an interlocking arrangement of two types of protein filaments, known respectively as **thick** and **thin filaments** – they make up the myofibrils. These protein filaments are aligned, giving the appearance of stripes (alternating **light and dark bands**). This is shown in the more highly magnified electron micrograph and interpretive drawing in Figure 15.13.

Electron micrograph of an individual sarcomere (×34 000)

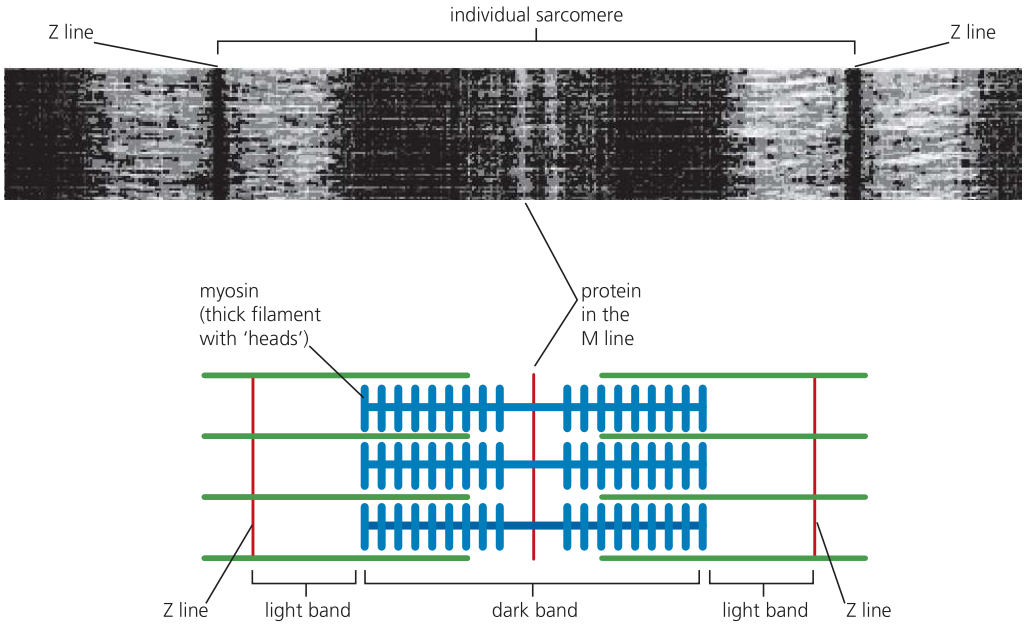


Figure 15.13 The ultrastructure of a myofibril

The thick filaments are made of a protein called **myosin**. They are about 15 nm in diameter. The longer, thin filaments are made of another protein, **actin**. Thin filaments are about 7 nm in diameter, and are held together by transverse bands, known as **Z lines**. Each repeating unit of the myofibril is, for convenience of description, referred to as a **sarcomere**. So we can think of a myofibril as consisting of a series of sarcomeres attached end to end.

How a motor nerve ending makes connection with a muscle fibre

Striated muscle fibres are innervated by a motor neurone nerve ending, at a structure known as a motor end plate or **neuromuscular junction**. This is a special type of synapse, but the transmitter substance is the familiar acetylcholine.

On arrival of an action potential at the neuromuscular junction, vesicles of acetylcholine are released and the transmitter molecules bind to receptors on the sarcoplasm (this is the plasma membrane of the muscle fibre). This triggers the release of calcium ions from the sarcoplasmic reticulum, into the cytoplasm around the myofibrils, via the T-tube system (Figure 15.SS). These calcium ions then remove the blocking molecules on binding sites of the actin filaments (see below). This starts the **contraction** process. When action potentials stop arriving at the muscle fibres, calcium ions return to the sarcoplasmic reticulum and the binding sites are again covered by blocking molecules.

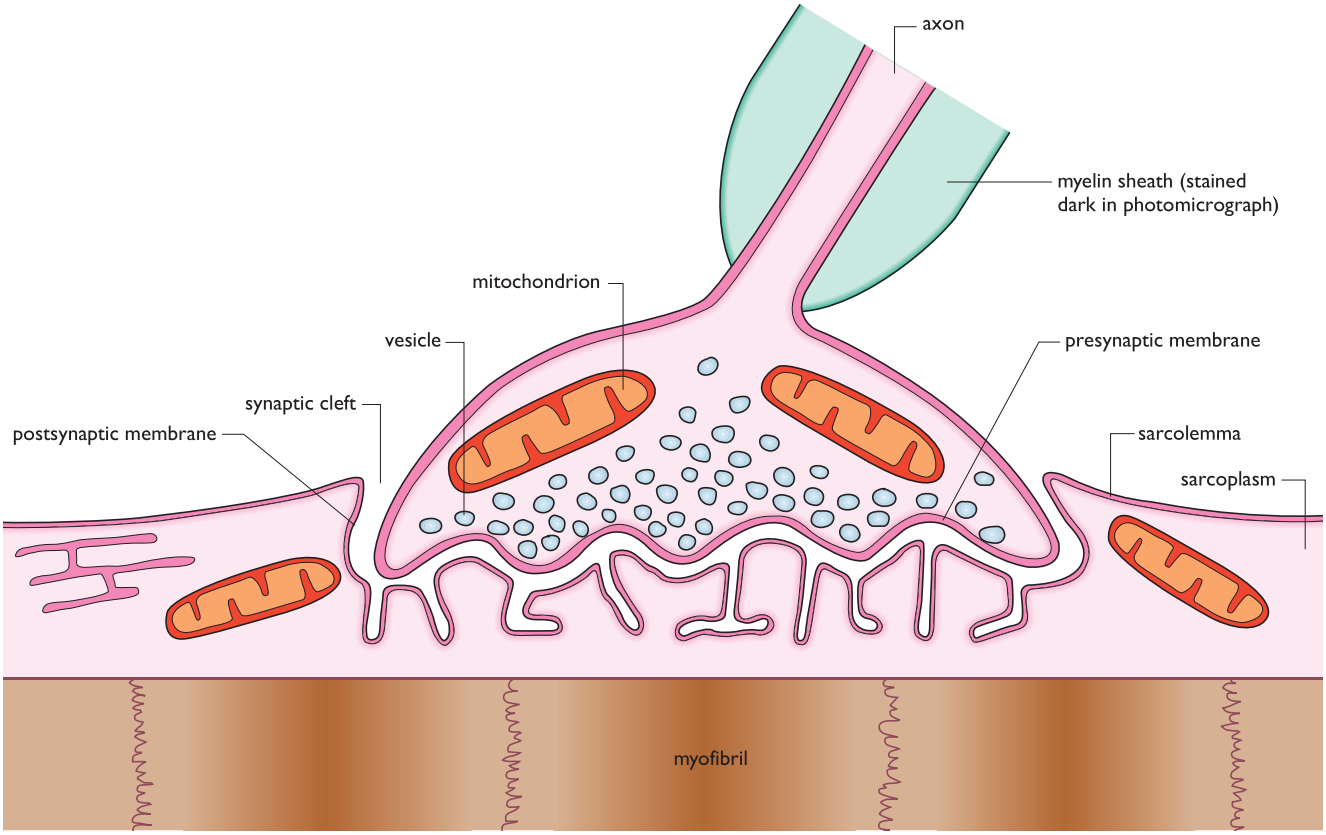


Figure 15.14 The structure of a neuromuscular junction

Question

8 Explain the relationship to a muscle of:
- **a** a muscle fibre
- **b** a myofibril
- **c** a myosin filament.

Skeletal muscle contracts by sliding of the filaments

When skeletal muscle contracts, the actin and myosin filaments **slide past each other**, in response to nervous stimulation, causing shortening of the sarcomeres (Figure 15.15). This occurs in a series of steps, sometimes described as a ratchet mechanism. A great deal of **ATP** is used in the contraction process.

Shortening is possible because the thick filaments are composed of many myosin molecules, each with a **bulbous head**, which protrudes from the length of the myosin filament. Along the actin filament are a complementary series of binding sites to which the bulbous heads fit. However, in muscle fibres at rest, the binding sites carry **blocking molecules** (a protein called **tropomyosin**), so binding and contraction are not possible.

Calcium ions play a critical part in the muscle fibre contract mechanism, together with the proteins tropomyosin and **troponin**. The contraction of a sarcomere is described in the following four steps.

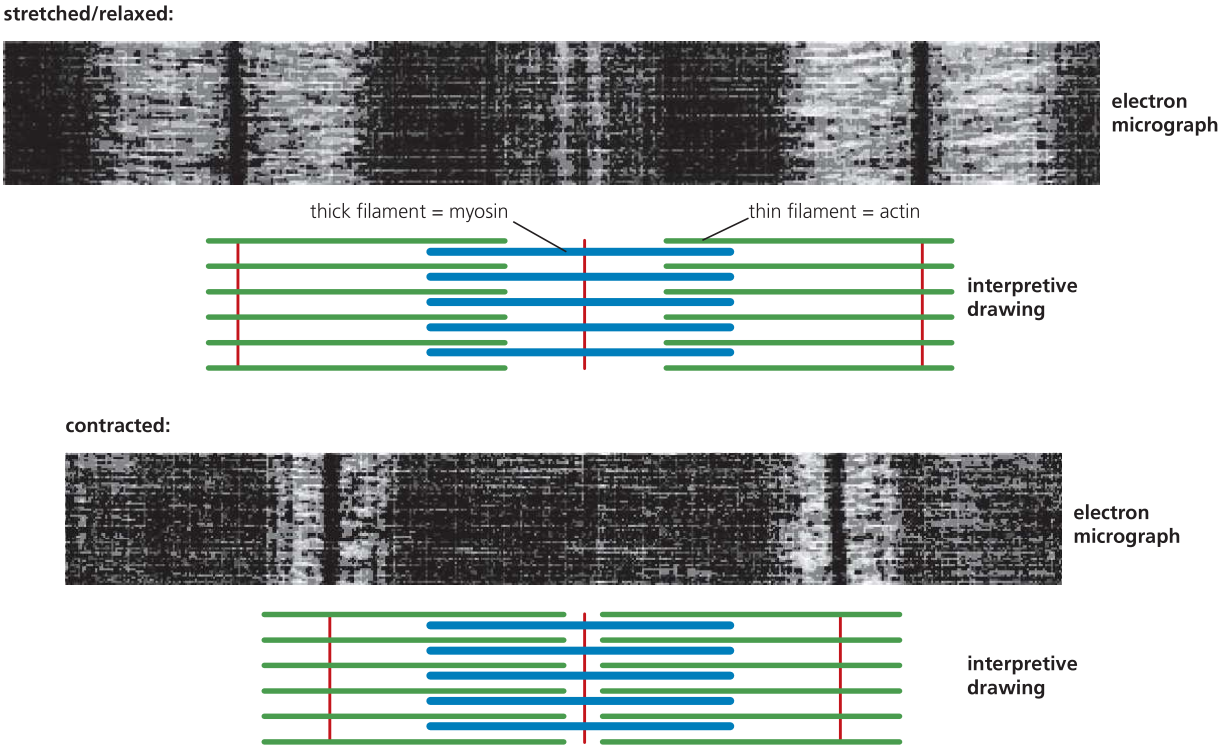


Figure 15.15 Muscle contraction of a single sarcomere

1 The myofibril is stimulated to contract by the arrival of an action potential. This triggers release of calcium ions from the sarcoplasmic reticulum, which surround the actin molecules. Calcium ions now react with an additional protein present (troponin) which, when so activated, triggers the removal of the blocking molecule, tropomyosin. The binding sites are now exposed.
2 Each bulbous head to which ADP and Pi are attached (called a **charged bulbous head**) reacts with a binding site on the actin molecule beside it. The phosphate group (P_i) is shed at this moment.
3 The ADP molecule is then released from the bulbous head, and this is the trigger for the **rowing movement** of the head, which tilts at an angle of about 45°, pushing the actin filament along. At this step, the **power stroke**, the myofibril has been shortened (contraction).
4 Finally, a fresh molecule of ATP binds to the bulbous head. The protein of the bulbous heads includes the enzyme ATPase, which catalyses the hydrolysis of ATP. When this reaction occurs, the ADP and inorganic phosphate (P_i) formed remain attached, and the bulbous head is now **'charged' again**. The charged head detaches from the binding site and straightens.

This cycle of movements is shown is Figure 15.16. The cycle is repeated many times per second, with thousands of bulbous heads working along each myofibril. ATP is rapidly used up, and the muscle may shorten by about 50% of its relaxed length. However, when the action potential stimulation stops, the muscle cell relaxes. Now, the filaments slide back to their original positions. Ion pumps in the sarcoplasmic reticulum pump calcium ions back inside, and so the calcium ion concentration surrounding the myosin filaments falls. Blockage of binding sites by tropomyosin is restored.

Arrival of nerve impulse releases Ca^{2+} from the sarcoplasmic reticulum. Ca^{2+} ions cause removal of blocking molecule from binding sites. Each myosin molecule has a bulbous head that reacts with ATP → ADP + P_i which remain bound.

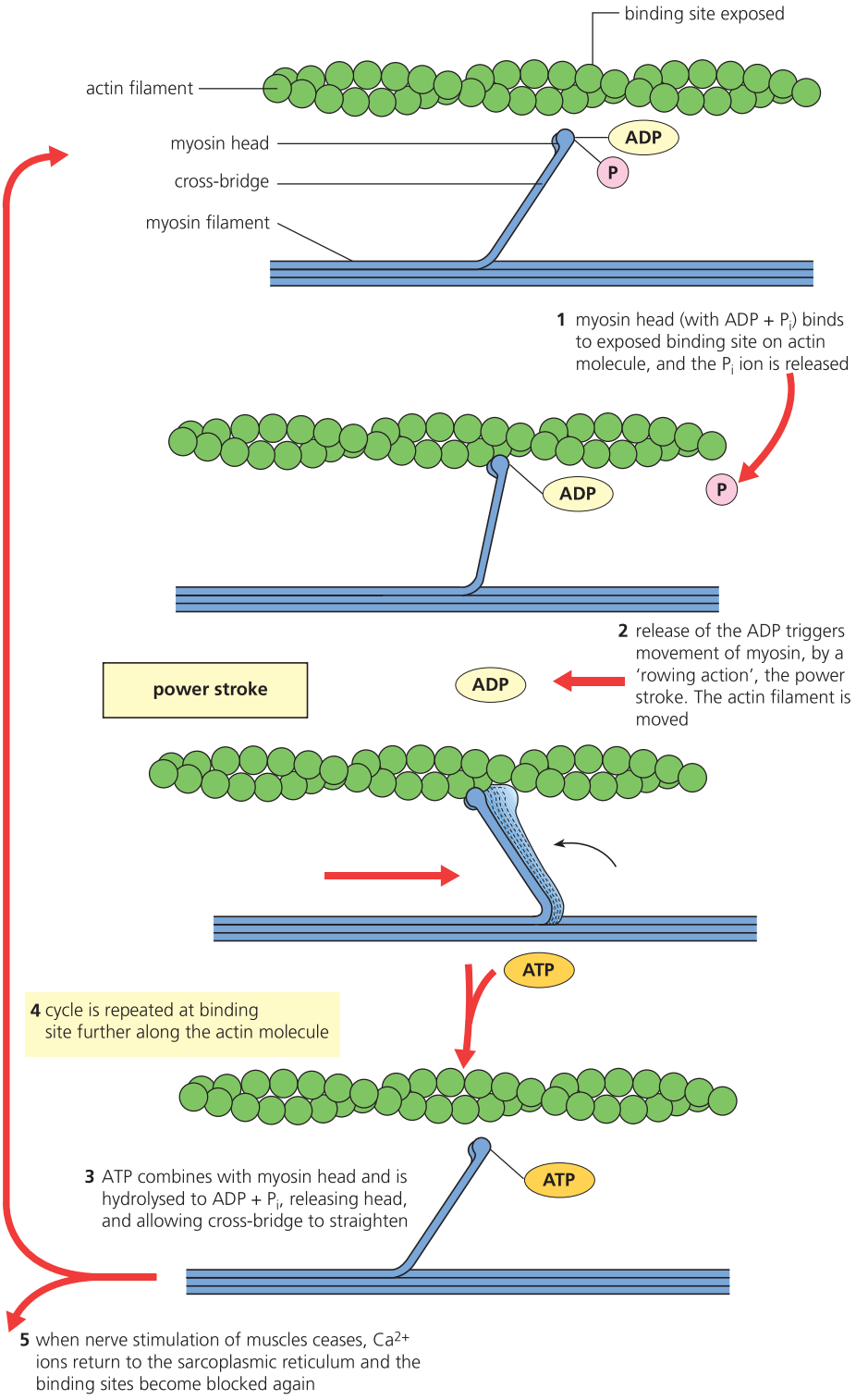


Figure 15.16 The sliding-filament hypothesis of muscle contraction

The mechanism of muscle contraction:

- The myofibril is stimulated to contract by nervous stimulation. This triggers release of calcium ions from the sarcoplasmic reticulum, around the actin molecules. Calcium ions react with the blocking molecules, removing them so **the binding sites are now exposed.**
- Each bulbous head to which ADP and Pi is attached (called a charged myosin head) reacts with a binding site on the actin molecule next to it. The phosphate ion (P_i) is shed at this moment.
- Then the ADP molecule is released from the myosin head. This is the trigger for the **'rowing movement'** of the head, which tilts by an angle of about 45°, pushing the actin filament along. At this step, **the power stroke**, the myofibril has been shortened (**contraction**).
- Finally, a fresh molecule of ATP binds to the myosin head. The protein of the myosin head includes the enzyme ATPase, which catalyses the hydrolysis of ATP. When this reaction occurs, the ADP and inorganic phosphate (P_i) formed remain attached. The myosin head is now 'charged' again. The charged head detaches from the binding site and straightens.

15.1 *continued*... The endocrine system

The endocrine system is a slower system that controls long-term changes. Fertility may be controlled by use of hormones.

By the end of this section you should be able to:

l) explain the roles of the hormones FSH, LH, oestrogen and progesterone in controlling changes in the ovary and uterus during the human menstrual cycle

m) outline the biological basis of contraceptive pills containing oestrogen and/or progesterone

Hormonal control – introducing the endocrine system

Endocrine gland: a gland containing specialised secretory cells that release a hormone into the bloodstream at a distance from the hormone's target organ.

Hormones are **chemical messengers**. They are produced and secreted from the cells in glands known as ductless or **endocrine glands**. These contain capillary networks and specialised secretory cells that make and release hormones. Hormones are released from the cells of the ductless gland, directly into the blood stream, when stimulated to do so. Hormones are then transported indiscriminately about the body via the blood circulation system, but they act only at specific sites, appropriately called **target organs**. Cells of the target organ possess specific receptor molecules on the external surface of their cell surface membrane to which the hormone molecules bind. A hormone typically works by triggering changes to specific metabolic reactions in their target organs. Although present in small quantities, hormones are extremely effective in the control and co-ordination of several body activities. The position of the main endocrine glands and the hormones produced are summarised in Figure 15.17.

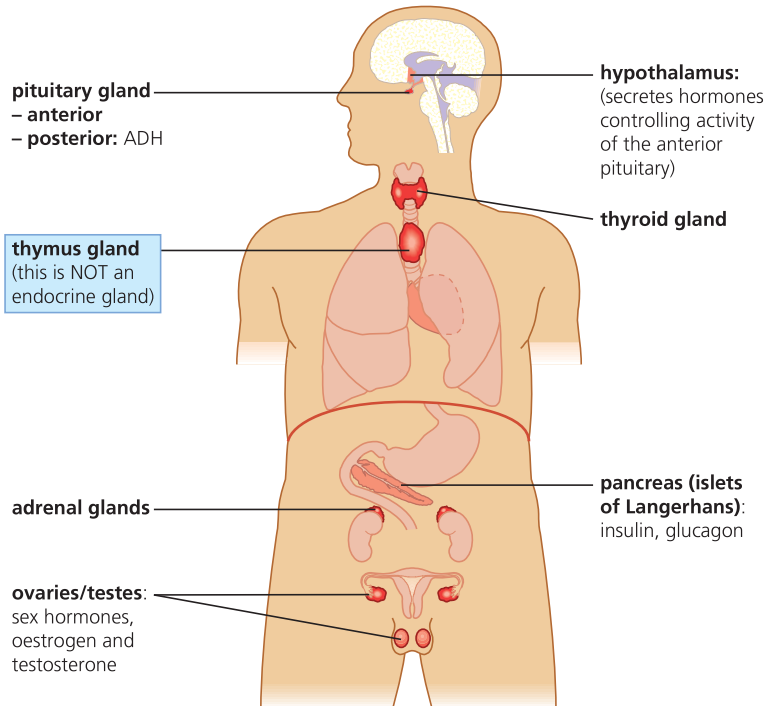


Figure 15.17 The human endocrine system

Hormones circulate in the bloodstream only briefly because in the liver they are continually being broken down. Any breakdown products no longer of use to the body are excreted in the kidneys. So, long-acting hormones must be secreted into the bloodstream continuously to be effective – and they are.

You can see immediately that hormonal control of body function is quite different from nervous control; the latter is concerned with quick, precise communication, whereas hormones mostly work by causing specific changes in metabolism or development, often over an extended period of time. However, these contrasting systems are co-ordinated by the brain. Our nervous system and hormones work together.

The structure of endocrine glands can be contrasted with that of other glands in our body which deliver their secretions through tubular ducts, such as the salivary glands in the mouth and sweat glands in the skin. These ducted glands are called exocrine glands. Their secretions pass out of the gland via ducts and they have altogether different roles in the body.

Question

9 Hormones, on secretion, are transported all over the body. Explain how the effects of hormones are restricted to particular cells or tissues.

The role of hormones in the menstrual cycle

We have seen that **hormones** are chemical messenger substances produced in the cells of the ductless or **endocrine glands**. From these, hormones are released into the blood and travel to all parts of the body. However, hormones have their effects on specific **target cells**.

The onset of **puberty** is triggered by a hormone secreted by a part of the brain called the **hypothalamus**. It produces and secretes a releasing hormone, the target organ of which is the nearby **pituitary gland**. Here it causes production and release of two hormones. These are called **follicle stimulating hormone (FSH)** and **luteinising hormone (LH)**. They are so named because their roles in sexual development were discovered in the female (although they do operate in both sexes).

The first effects of FSH and LH are the enhanced secretion of the sex hormones **oestrogen** and **testosterone** by the ovaries and testes. Then, in the presence of FSH, LH and the respective sex hormone(s), there follows sexual development of the body and the preparation of the body for its role in sexual reproduction. This process is now illustrated in the female reproductive system.

In the female, the secretion of oestrogen and another hormone, **progesterone**, do not occur at a steady rate; instead their secretion is a recurring (cyclical) event. Together with FSH and LH, the continually changing concentration of all four hormones brings about a repeating cycle of changes that we call the **menstrual cycle**. The menstrual cycle consists of two cycles, one in the **ovaries** and one in the **lining of the uterus** (the **endometrium**). The ovarian cycle is concerned with the monthly preparation and shedding of an egg cell from an ovary. The uterine cycle is concerned with the build up of the lining of the uterus. 'Menstrual' means *monthly*; the combined cycles take on average 28 days.

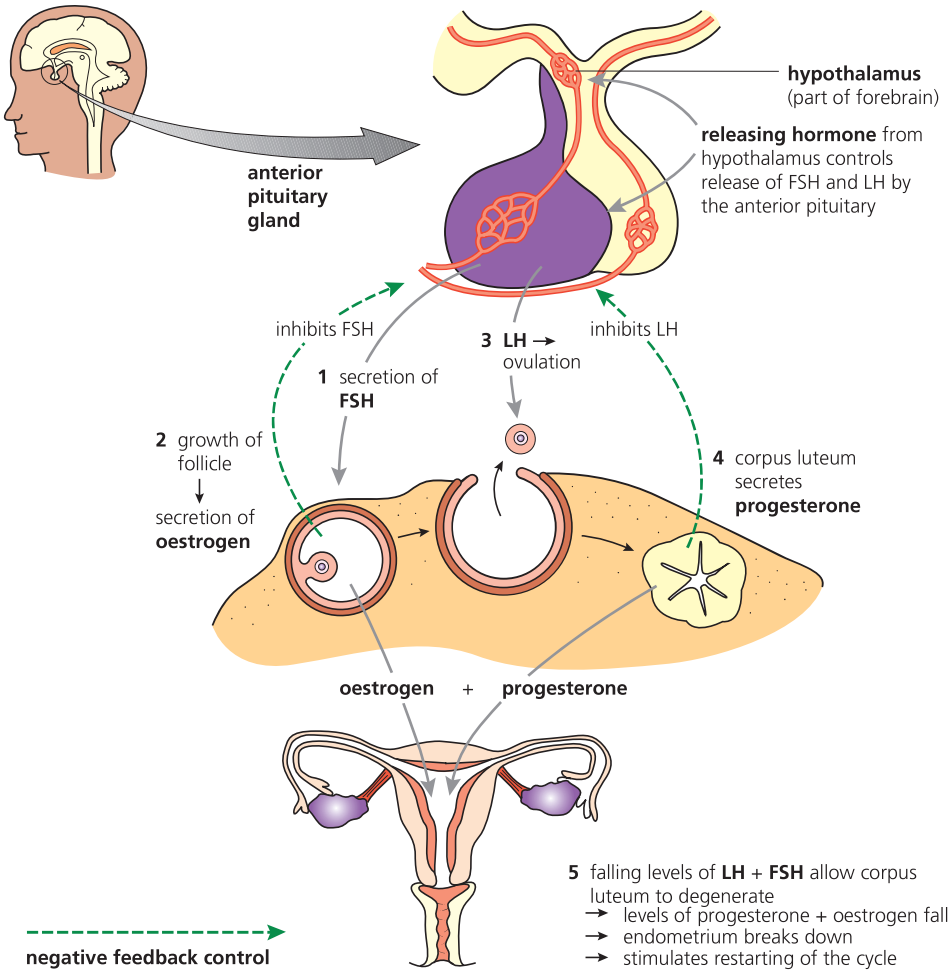


Figure 15.18 Hormone regulation of the menstrual cycle

Question

10 Explain what is meant by *negative feedback control*. Can you think how positive feedback might differ from negative feedback?

By convention, the start of the menstrual cycle is taken as the **first day of menstruation** (bleeding), which is the shedding of the endometrial lining of the uterus. The steps, also summarised in Figure 15.18 above, are as follows:

1 FSH is secreted by the pituitary gland, and stimulates development of several immature egg cells (in secondary follicles) in the ovary. Only one will complete development into a mature secondary oocyte (now in the Graafian follicle).

2 The developing follicle then secretes oestrogen. Oestrogen is a hormone that has two targets:
- in the uterus oestrogen stimulates the build up of the **endometrium**, which prepares the uterus for a possible implantation of an embryo, if fertilisation takes place.
- in the pituitary gland, oestrogen inhibits the further secretion of FSH. This prevents the possibility of further follicles being stimulated to develop. It is an example of **negative feedback control**.

3 Meanwhile, the concentration of oestrogen continues to build up, increasing to a peak value just before the mid-point of the cycle. This high and rising level of oestrogen suddenly stimulates the secretion of LH and, to a slightly lesser extent, FSH, by the pituitary gland. LH stimulates ovulation (the shedding of the mature secondary oocyte from the Graafian follicle) and the secondary oocyte is released from the ovary.

4 As soon as the ovarian follicle has discharged its oocyte, LH also stimulates the conversion of the vacant follicle into an additional, temporary gland, called a corpus luteum. The corpus luteum secretes progesterone and, to a lesser extent, oestrogen. Progesterone has two targets:
- in the uterus progesterone continues the build up of the endometrium, further preparing it for a possible implantation of an embryo, if fertilisation takes place.
- in the pituitary gland progesterone inhibits further secretion of LH, and also of FSH. This is a second example of **negative feedback control**.

5 The levels of FSH and LH in the bloodstream now decrease rapidly. Low levels of FSH and LH allow the corpus luteum to degenerate. As a consequence, the levels of progesterone and oestrogen also fall. Soon the level of these hormones is so low that the extra lining of the uterus is no longer maintained. The **endometrium breaks down** and is lost through the vagina in the first five days or so of the new cycle.

The falling levels of progesterone again cause the secretion of FSH by the pituitary and a new cycle is underway. The changing levels of the hormones during the menstrual cycle and their effects on the ovaries and the lining of the uterus are show in Figure 15.19.

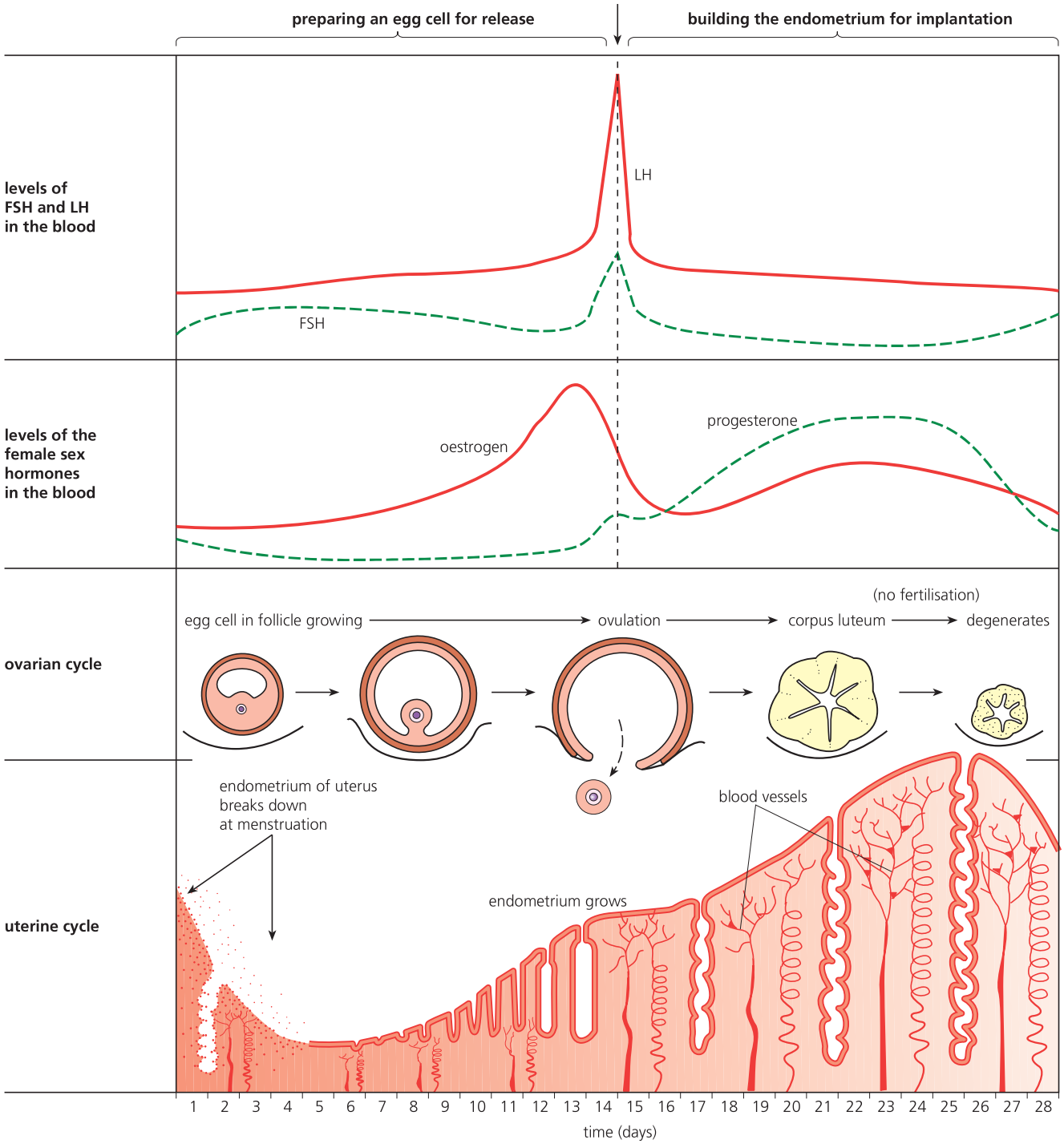


Figure 15.19 Changing levels of the hormones during the menstrual cycle and their effects on the ovaries and the lining of the uterus

Question

11 Identify the critical hormonal changes that:
- **a** trigger ovulation
- **b** cause degeneration of the corpus luteum.

Extension

If the egg is fertilised (the start of a pregnancy), then the developing embryo itself becomes an endocrine gland, secreting a hormone that circulates in the blood and maintains the corpus luteum as an endocrine gland for at least 16 weeks of pregnancy.

When eventually the corpus luteum does break down, the **placenta** takes over as an endocrine gland, secreting oestrogen and progesterone. These hormones continue to prevent ovulation and maintain the endometrium.

Controlling human reproduction

Conception is a process that starts with fertilisation and ends with implantation of an embryo resulting in **pregnancy**. **Contraception** allows control over human reproduction. There are many methods and they work in different ways. Some are more effective than others. One method is the contraceptive pill.

The biological basis of the contraceptive pill

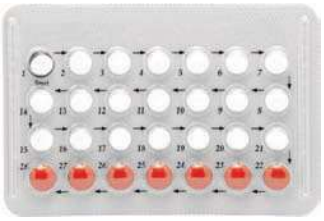


Figure 15.20 A commercial pack of 'the pill'

The combined oral contraceptive pill, more often known as 'the pill', contains two hormones. These are chemically very similar to the two natural hormones women produce in their ovaries – oestrogen and progesterone. The effect of the pill is to:
- prevent ovulation, the pill stops the ovaries releasing an egg each month
- thicken the mucus in the cervix making it difficult for sperm to reach the egg
- make the lining of the uterus thinner so it is less likely to accept a fertilised egg.

For the oral contraceptive pill to be effective, the woman must take one pill at the same time each day for 21 days. She then takes an inert pill (a placebo – containing an iron supplement, as a lot is lost in menstruation) for seven days, or she may take no pill at all. During this time menstruation occurs. This 'period' is usually lighter and shorter than is normally experienced.

In the body, the hormones from the pill prevent ovulation by decreasing the release of FSH and LH from the pituitary gland (without completely stopping their release). This restricts the growth of follicles in the ovaries and so a secondary oocyte does not grow and is not released. The endometrium still builds up in the uterus and then, later, breaks down again, but menstruation is shorter and lighter because the corpus luteum has not grown (in the absence of ovulation).

Nothing is without risk, but very many women who have access to it find the pill beneficial to their lifestyle, health and range of life choices. The likely benefits and possible risks are identified in Table 15.2.

Question

12 What do you understand by a 'placebo'?

Table 15.2 The risks and benefits of the pill

Risks	Benefits

• Some women develop nausea, headaches, tiredness and mood swings.
• It may trigger a rise in blood pressure, leading to an increased risk of thrombosis (blood clots are life-threatening.
• There is an increased risk of breast cancer, but this is only to a slight extent.
	• There is a reduced risk of:
	  • ovarian cysts
	  • cancer of the ovaries or uterus.
	• Menstruation is more regular for many, and pre-menstrual tension may be lessened.
	• The quality of the mucus plug in the cervix reduces the risk of bacterial entry to the uterus and therefore decreases the chance of infection.

Extension

The 'morning after' pill

This 'emergency' contraceptive medication contains synthetic progesterone that works in the body in the following ways:

- it delays ovulation or it inhibits ovulation if that event has not yet occurred
- it irritates the endometrium so that implantation does not occur.

Typically, the morning after pill is taken if a women has forgotten to take her combined oral contraceptive pill or if another contraceptive device has been omitted or used and failed (a condom may have broken). It may also be used if a woman has been raped.

In these 'morning after' situations, the sense of 'emergency' is a (quite natural) state of mind, rather than a biological fact. This is because fertilisation occurs in the oviduct, if at all. A fertilised egg then passes down to the uterus, a journey that takes 5–7 days. Only then can implantation in the wall of the uterus occur. So there is an interval of about 72 hours in which a pregnancy can be avoided. However, if fertilisation has taken place, then this particular contraceptive measure is a 'chemical abortion'.

Extension

The social and ethical implications of contraception

Overpopulation and the Earth's resources

The current human population explosion began at about the beginning of the Industrial Revolution, some 200 years ago and is ongoing. Birth rates are high in many communities and also for many there is an increasing life expectancy. This is most notable among people of the developed nations but it is a trend in many human populations (Figure 15.21).

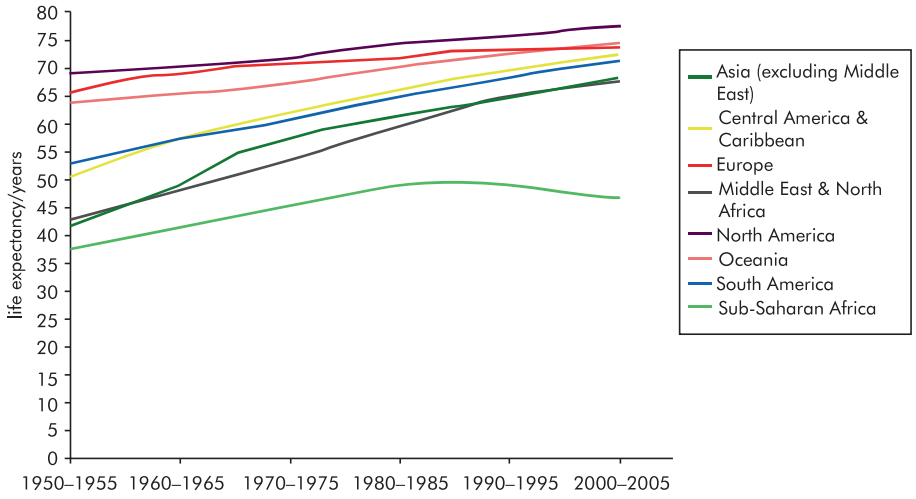


Figure 15.21 Life expectancy 1950–2005

People who make a study of human population growth (demographers) have predicted that by 2050 the Earth may have 10 billion human inhabitants – that is very many more than we currently have. The projections suggest that the world population will stabilise at this level.

Can such a total human population be sustained by the Earth's resources? Many people feel that it cannot. To them, the current destruction of habitats and devastation of biological diversity observed on all continents is directly or indirectly due to our attempts to provide for the lifestyles that existing humans seek (or seek to maintain). It is unacceptable to them that the environment is so abused. To these people, one immediate task is to reduce the birth rate. Contraception helps to make this possible.

15.2 Control and co-ordination in plants

Plant co-ordination systems involve rapid responses as in the case of the Venus fly trap, but also complex interactions between plant growth regulators, such as auxin and gibberellin.

Plants respond quite differently to different concentrations of plant growth regulators.

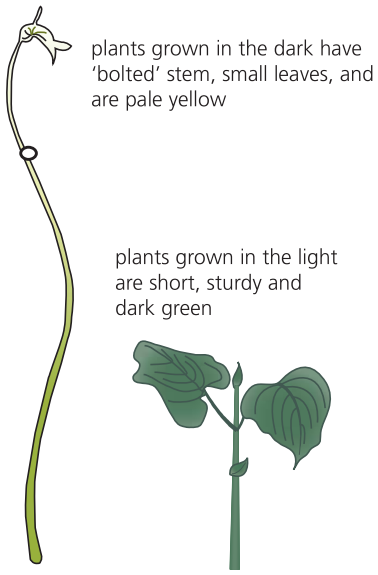
By the end of this section you should be able to:

a) describe the rapid response of the Venus fly trap to stimulation of hairs on the lobes of modified leaves and explain how the closure of the trap is achieved
b) explain the role of auxin in elongation growth by stimulating proton pumping to acidify cell walls
c) describe the role of gibberellin in the germination of wheat or barley
d) explain the role of gibberellin in stem elongation including the role of the dominant allele, *Le*, that codes for a functioning enzyme in the gibberellin synthesis pathway, and the recessive allele, *le*, that codes for a non-functional enzyme

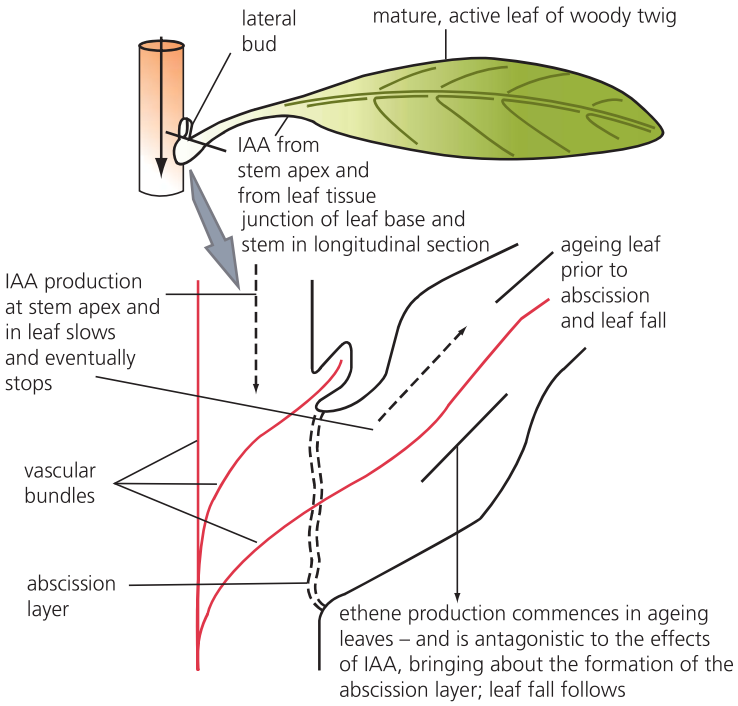
The responding plant – communication and control in flowering plants

Sensitivity is a characteristic of all living things, but the responses of plants and animals differ. Most plants are anchored organisms, growing in one place and remaining there, even if the environment is not wholly favourable. As a result, plant responses are often less evident than those of animals, but they are no less important to the plant. Plants are highly sensitive to environmental clues and external and internal stimuli, and respond to changes. Yet plants have no nervous system and no muscle tissue. Consequently, plant sensitivity may lead to responses less dramatic than those of animals. Rapid movements by plants are extremely rare (but see Figure 15.22c); plant responses are mostly **growth movements**. An example of this is the growth of young stems towards light. Another is the growth of the main root down into the soil in response to gravity. Occasionally, movements are due to turgor changes (Figure 15.22d). However, there is no doubt that plant responses are also precise and carefully regulated responses.

a) The response of a plant stem to light



b) Leaf fall in a woody plant of temperate climate



c) The response of the sensitive plant *Mimosa pudica* to touch

d) Closure of a leaf of the canivorous plant *Drosera rotundiflora*, to trap an insect

Figure 15.22 Examples of plant sensitivity and responses

Among the internal factors that play a part in plant sensitivity, the most important are the substances we call **plant growth regulators** and their effects. There are five major types of compound naturally occurring in plants that we call growth regulators. The effects of these chemicals are rather different from those of animal hormones (see Table 15.3).

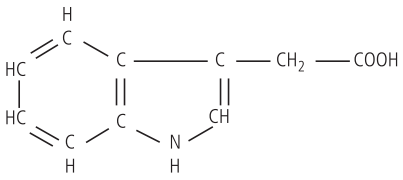
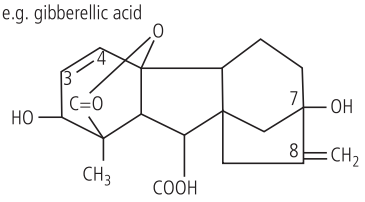
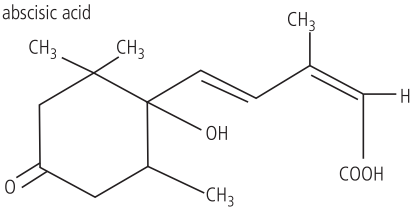
Plant growth regulators:

- occur in very low concentrations in plant tissues, so sometimes it is hard to determine their precise role
- are produced in a variety of tissues, rather than in discrete endocrine glands
- often interact with each other in controlling growth and sensitivity – some interact to reinforce each other's effects (synergism), others oppose each other's effects (antagonism)
- if they move from their site of synthesis (and not all do), they may diffuse from cell to cell, be actively transported or be carried in the phloem or xylem
- may have profoundly differing effects, depending on whether they are present in low or high concentration
- may have different effects according which tissue they are in or at different stages of development of a single tissue.

Question

13 What is the difference between growth and development?

Table 15.3 The plant regulators: discovery, roles and synthesis. The structural formulae show how chemically diverse these substances are – *they do not require memorising*.

Auxins, principally Indoleacetic acid (IAA)	
Discovery Initially by Darwin, investigating coleoptiles and their curvature towards a unilateral light source. Later by others, including the devising of a biological assay to find the concentrations of auxins in plant organs (since these are at low concentrations).	indole acetic acid (the principal auxin) 
Roles • Promotion of extension growth of stems and roots (at different concentrations) • Dominance of terminal buds • Promotion of fruit growth • Inhibition of leaf fall	
Synthesis At stem and root tips and in young leaves (from the amino acid tryptophan)	
Gibberellins (GAs)	
Discovery Initially, in dwarf rice plants, which, when infected with a fungus (*Gibberella* sp.), responded by growing to full height.	gibberellins e.g. gibberellic acid 
Roles • Promotion of extension growth of stems • Delay of leaf senescence and leaf fall • Inhibition of lateral root initiation • Switching on of genes to promote germination	
Synthesis In the embryos of seeds and in young leaves (except in genetically dwarf varieties) There are several types of gibberellin	
Abscisic acid (ABA)	
Discovery During investigation of bud and seed dormancy and in abscission of fruits	abscisic acid 
Roles • A stress hormone • Triggering of stomatal closure when leaf cells are short of water • Induction of bud and seed dormancy	
Synthesis In most organs of mature plants, in tiny amounts.	

Extension

Interactions of plant growth regulators

Sometimes plant growth regulators have their effects acting on their own, but in other situations, they interact with other growth regulators. When one substance enhances the effect of the other, this is known as **synergism**. For example, gibberellins (GAs) and indoleacetic acid (IAA) work synergistically in stem extension growth.

Alternatively, a growth regulator may reduce the effect of another. This situation is known as **antagonism**. Some important interactions involve the other growth regulators (cytokinins – their main role is in cell differentiation, and ethylene – its main role is in fruit ripening).

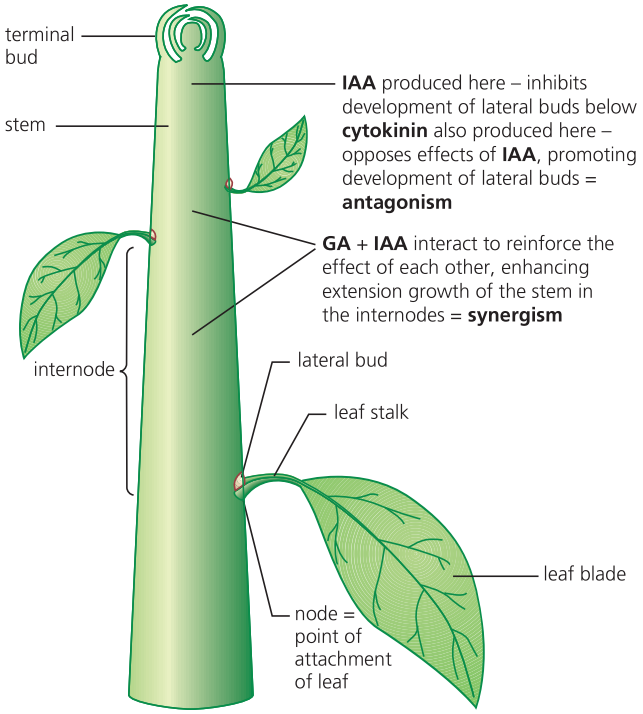


Figure 15.23 The interaction of growth regulators

Figure 15.24 Venus fly trap, with close-up view of leaves, one 'closed' with a 'prisoner'!

The Venus fly trap

The Venus fly trap (*Dionaea muscipula*) is a carnivorous plant, native to the subtropical wetlands of the East Coast of the USA.

Remember, plants typically obtain the ions they need for their metabolism by active uptake from the soil solution. However, in some habitats, the decay of dead organisms (by which the ions are released into the soil solution) may be very slow, and when they are released, the ions tend to be washed away by the high rainfall. In these environments carnivorous plants that can trap minibeasts such as flying insects prosper. They take their essential ions from the decaying or digested bodies of these victims, thus enabling them to survive in these unfavourable growing conditions where other plants often fail. The Venus fly trap is an example of this.

Look at Figure 15.24 now.

The margins of the leaves of this plant have short, stiff hairs, which when they are bent by an insect large enough to do so, trigger the two halves of the leaf to snap shut, trapping the insect. Then, short, glandular hairs on the inner leaf surface secrete hydrolytic protease enzymes that digest the soft tissue of the insect. Active uptake of the ions released then takes place into the leaf cells. Later, the trap reopens and the undigested matter is blown away.

The rapid closure of the Venus flytrap is one of the fastest movements observed in the plant kingdom. It occurs in the complete absence of a nervous system. It is due, at least in part, to an extremely sudden change in turgidity of cells in the hinge region of the leaf blade. However, how this rapid loss in turgor is brought about is not clear. Another hypothesis is a sudden acid-induced wall loosening in 'motor cells' of the leaf – implying that proton pumps are involved.

How auxin stimulates elongation growth

We have seen that auxin has a major role in the growth of the shoot apex, where it promotes the elongation of cells. Auxin has this effect on growth and development by direct action on the components of growing cells, including the walls. For example auxin-triggered cell elongation occurs due to the stimulating effect of auxin on proton pumps in the cell surface membranes of cells that lie beside the cellulose walls (Figure 15.25). The result is that the walls become acidic, and the lowered pH initiates the breakage of cross-links between cellulose microfibrils and binding polysaccharides that had helped to make the cellulose inflexible. The wall's resistance to stretching due to turgor pressure is decreased and elongation occurs.

Note that auxin transport across cells is polar, with its entry into the cell being passive (by diffusion), but its efflux is active (ATP-driven). Auxin efflux pumps set up concentration gradients in plant tissues, in fact.

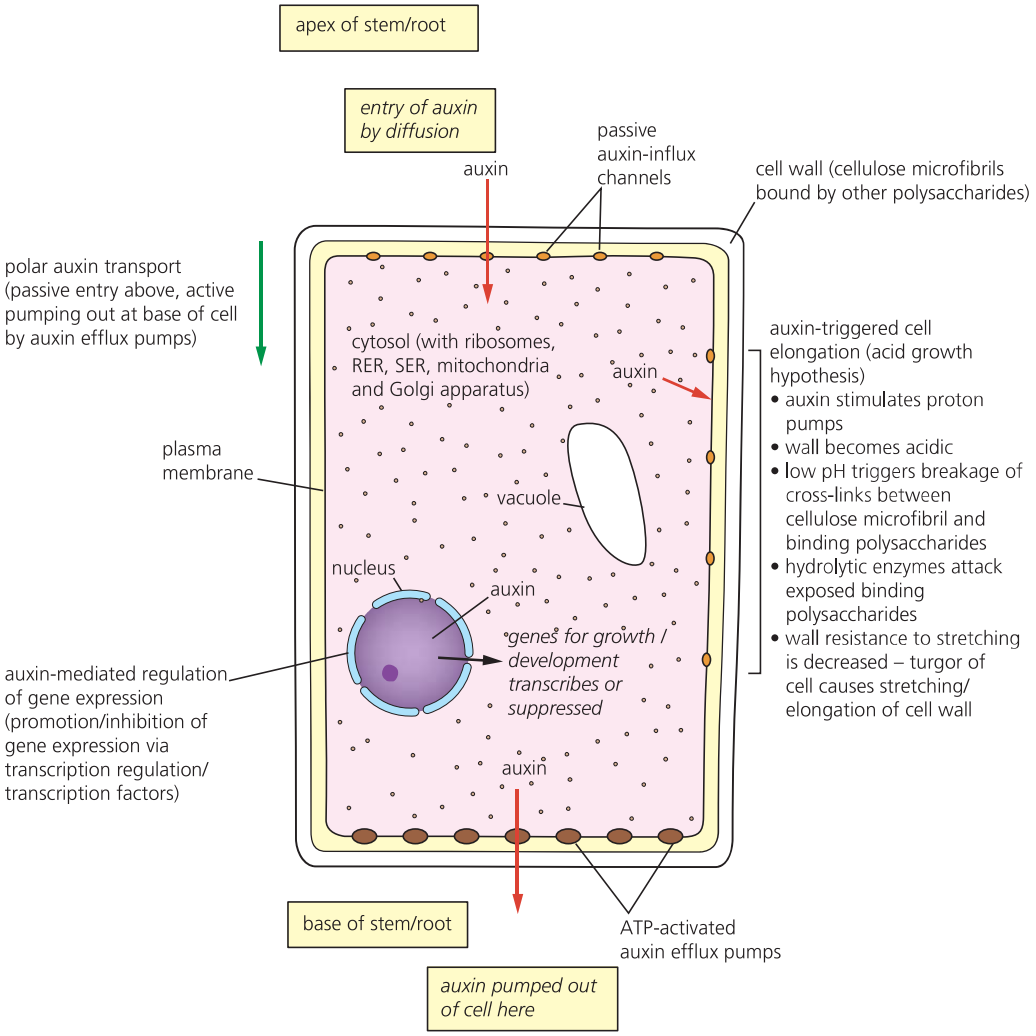


Figure 15.25 Auxin, proton pumps and cell elongation

The role of gibberellin in the germination of barley

Many higher plants have been found to contain **gibberellic acid (GA)**. In fact, several different forms of the molecule have been discovered and these are often referred to collectively as **gibberellins**. Gibberellins are formed in chloroplasts, in the embryo of seeds and in young leaves. Gibberellins are absent from dwarf varieties of pea plants, but when artificially supplied to dwarf varieties, they become tall.

The highest concentrations of gibberellins occur in fruits and seeds as they are formed and in seeds as they germinate. In the seed, gibberellins switch on genes that promote germination and go on to promote additional mitosis, thereby increasing the numbers of cells formed. Gibberellins also have a role in the mobilisation of stored food in germinating barley fruits, as illustrated in Figure 15.26, on the next page. During germination, the uptake of water activates the gene for gibberellic acid synthesis. This gibberellic acid then switches on the genes that control the synthesis of hydrolytic enzymes. Hydrolytic enzymes catalyse the mobilisation of the food reserves, causing the release of sugars, amino acids and fatty acids for germination and growth.

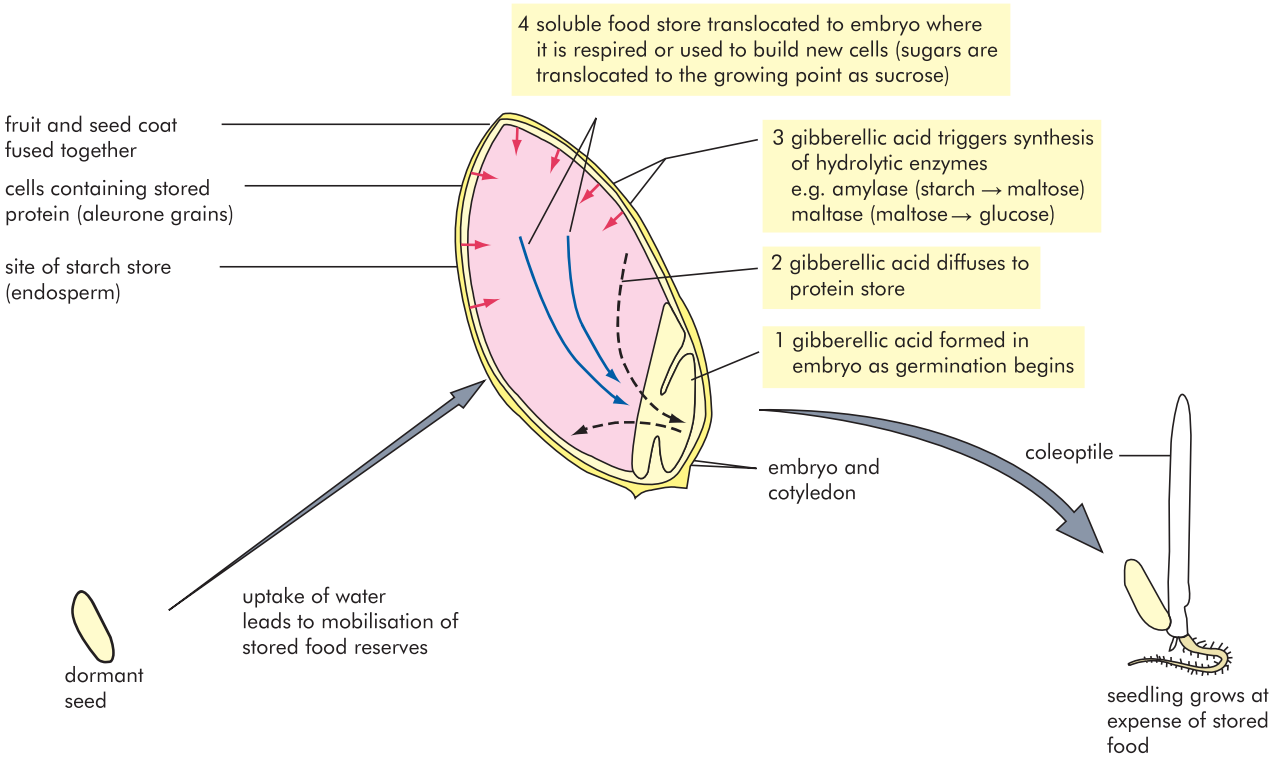


Figure 15.26 The role of gibberellic acid in the germination of barley fruit

The role of gibberellin in stem elongation – the gene involved

Gregor Mendel (1822–84) conducted experiments into the inheritance of a contrasting characteristics of the garden pea plant, *Pisum sativum*. For example, the height of stem in the garden pea plant, which may be either 'tall' (say about 48 cm), or 'dwarf' (about 12 cm). Ultimately, it was established that this characteristic is controlled by a single gene with two alleles.

We now know that this height difference between tall and dwarf pea plants is due to the ability (or otherwise) to convert a precursor molecule of gibberellin into an active form in the cells of

the pea plant. In fact, tall pea plants carry at least one allele (***Le***), which codes for a protein that functions normally in the gibberellin-synthesis pathway, catalysing the formation of gibberellin. That allele is 'dominant', so it may be present as a single allele or with two, but in either case, gibberellin will be present and the plant will be tall.

Dwarf pea plants carry two alleles (*le*) that code for a non-functional protein. This enzyme fails in the gibberellin-synthesis pathway. Plants with two recessive alleles remain dwarf, lacking the supply of gibberellin that enables extension growth in stem length to occur.

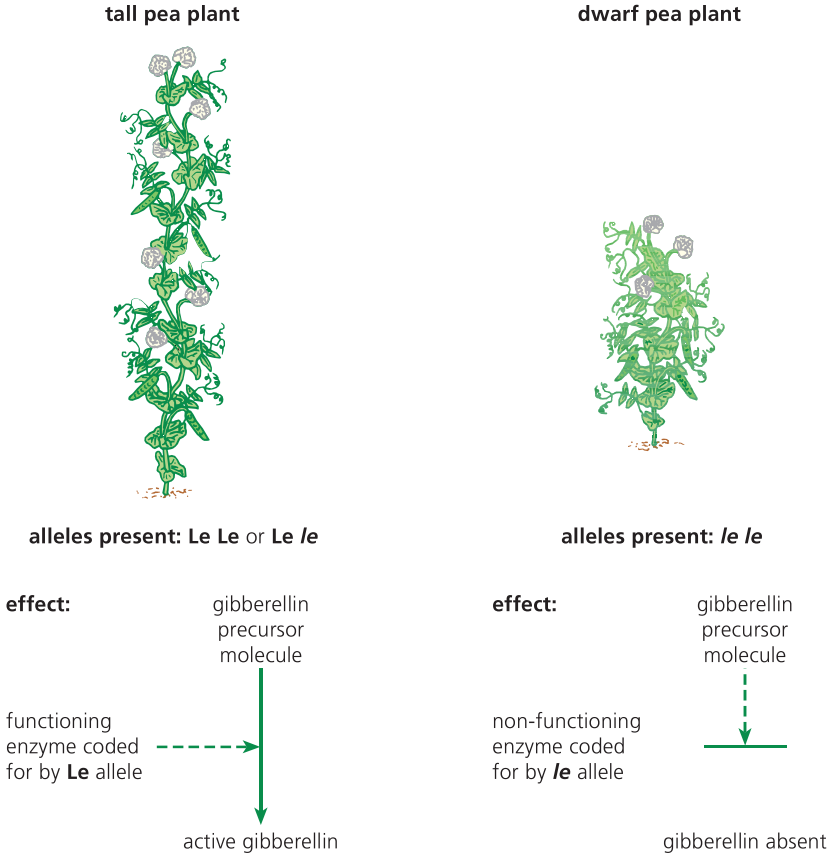


Figure 15.27 The role of specific alleles in the control of height in the garden pea

Summary

- The **nervous system** contains **receptors** (e.g. sense organs) linked to **effectors** (muscles or glands) by **neurones**, known collectively as **reflex arcs**. In mammals, reflex arcs are co-ordinated in the spinal cord and may be connected to the brain. Neurones are the basic units of the nervous system. The nerve fibres of neurones, the **dendron** and **axon**, are typically protected by a **myelin sheath**, which also speeds conduction of the impulse.
- An impulse or **action potential** is a temporary reversal of the electrical potential difference that is maintained across the membrane of the nerve fibres. Conduction is extremely fast and, after an action potential, there is a brief period when the fibre is not excitable (**refractory period**). Action potentials are transmitted between neurones across tiny gaps at **synapses**. Transmission here is chemical, involving diffusion of a specific transmitter substance.
- Receptors contain sensory cells or dendrites of sensory neurones that respond to stimuli with the production of action potentials. The **brain** receives and integrates incoming information from sensory receptors, sends impulses to effectors, stores information as a memory bank, and initiates activities in its own right.
- **Hormones** work with the animal's nervous system in the control and co-ordination of the body. Hormones are produced in endocrine glands, transported all over the body in the blood and affect specific target organs.

(continued)

Summary *(continued)*

- The **menstrual cycle** consists of two cycles, one in the **ovaries** and one in the **lining of the uterus**. The hormones controlling these cycle are **follicle stimulating hormone (FSH)** and **luteinising hormone (LH)** from the pituitary and **oestrogen** and **progesterone** from the ovaries. At the start, **FSH** stimulates **follicle development** to the point at which one eventually becomes an **ovarian follicle**. **Oestrogen** from this follicle triggers thickening of the **endometrium** and reduces the secretion of FSH. The rising levels of oestrogen cause **ovulation** and the empty follicle becomes a **corpus luteum**. The corpus luteum secretes progesterone. Build up of the endometrium continues until **fertilisation fails** to occur. The corpus luteum then breaks down, oestrogen and progesterone levels fall and **menstruation** follows.
- The **combined oral contraceptive pill** contains two hormones, chemically similar to oestrogen and progesterone. Provided the pill is taken daily for 21 days it:
  - prevents ovulation – the pill stops the ovaries releasing an egg each month
  - thickens the mucus in the cervix making it difficult for sperm to reach the egg
  - makes the lining of the uterus thinner so it is less likely to accept a fertilised egg.
- **Plant sensitivity** and co-ordination is mediated by **growth regulators** (including auxins, gibberellins and abscisic acid) which are different from animal hormones.
- Plant responses are mainly **growth responses**. For example, the tips of plant stems grow towards the light, and plant stems and root tips respond to gravity so the aerial shoot grows up and the roots down. Auxins (principally **indoleacetic acid**) maintain apical dominance of stem; gibberellins are active in stem elongation and in germination and abscisic acid is involved in the closure of stomata.

Examination style questions

1 Fig. 1.1 shows the changes in potential difference (p.d.) across the membrane of a neurone over a period of time. The membrane was stimulated at time **A** and time **B** with stimuli of different intensities.

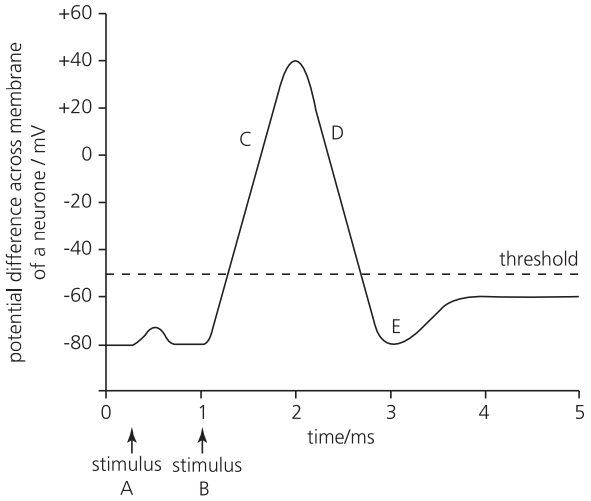


Fig. 1.1

a) Stimulus **B** resulted in an action potential. Describe what is occurring at **C**, **D** and **E**. [6]

b) Suggest why stimulus **A** did not result in an action potential being produced whereas stimulus **B** did. [2]

[Total: 8]

(Cambridge International AS and A Level Biology 9700, Paper 04 Q8 November 2007)

2 **a)** Outline the ways in which the endocrine **and** nervous systems carry out their roles in control and co-ordination in animals. [8]

b) Describe the part played by auxins in apical dominance in a plant shoot. [7]

[Total: 15]

(Cambridge International AS and A Level Biology 9700, Paper 41 Q9 June 2011)

3 **a)** The steroid hormones oestrogen and progesterone are secreted by the ovary. State precisely the sites of secretion of each. [2]

b) The most effective oral contraceptives for general use are the so-called combined oral contraceptives (COCs), which contain both oestrogen and progesterone.
Explain how COCs produce their effects. [4]

c) Describe two **social** implications of the use of contraceptives. [2]

[Total: 8]

(Cambridge International AS and A Level Biology 9700, Paper 04 Q2 June 2009)

4 **a)** By means of a labelled diagram show the structure of a synapse. [5]

b) Describe the sequence of steps by which an action potential crosses a cholinergic synapse. [10]

c) Outline the roles synapses play in the functioning nervous system. [5]

[Total: 20]