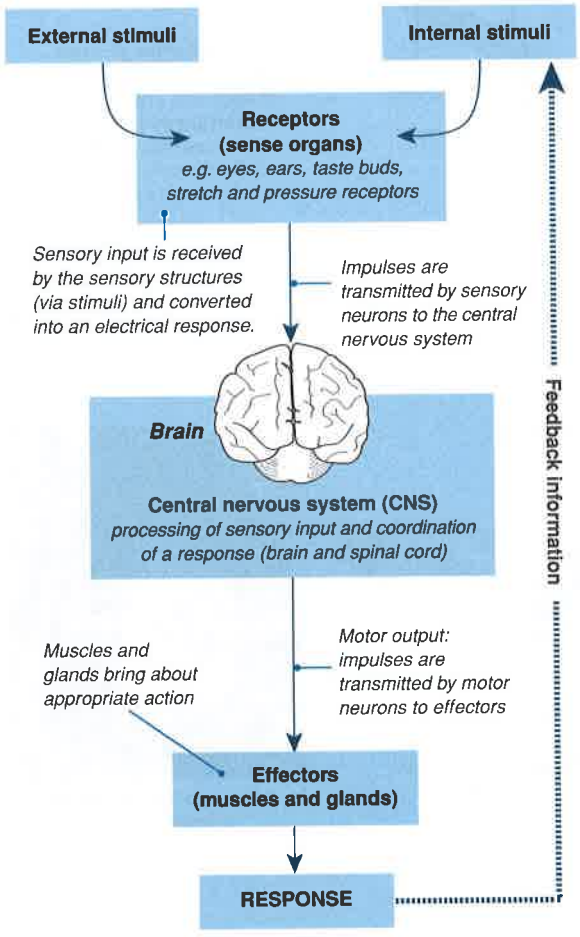
191 Nervous Regulatory Systems

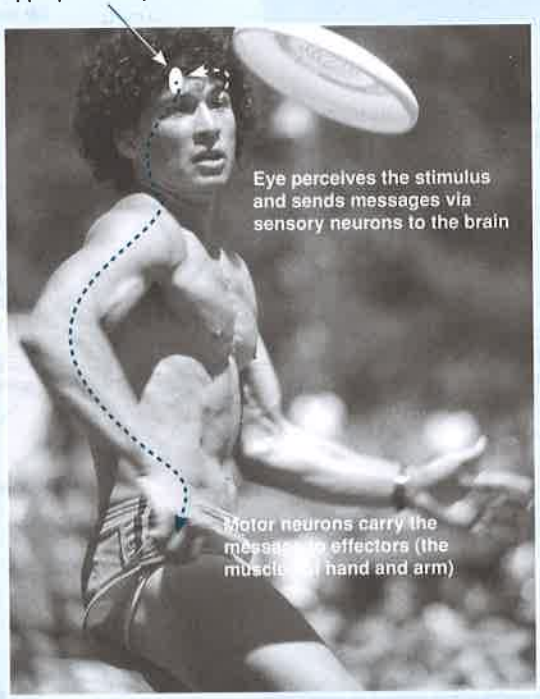
Key Idea: The nervous and endocrine systems work together to maintain homeostasis. Neurons of the nervous system transmit information as nerve impulses to the central nervous system, which coordinates appropriate responses to stimuli.

In humans, the nervous and endocrine (hormonal) systems work together to regulate the internal environment and maintain homeostasis in a fluctuating environment. The nervous system contains cells called **neurons** (nerve cells) which are specialized to transmit information in the form of electrochemical impulses (action potentials). The nervous system is a signalling network with branches carrying information directly to and from specific target tissues. Impulses can be transmitted over considerable distances and the response is very precise and rapid.

Coordination by the Nervous System

The vertebrate nervous system consists of the **central nervous system** (brain and spinal cord), and the nerves and receptors outside it (**peripheral nervous system**). Sensory input to receptors comes via stimuli. Information about the effect of a response is provided by feedback mechanisms so that the system can be readjusted. The basic organization of the nervous system can be simplified into a few key components: the sensory receptors, a central nervous system processing point, and the effectors which bring about the response (below).





In the example above, the frisbee's approach is perceived by the eye. The motor cortex of the brain integrates the sensory message. Coordination of hand and body orientation is brought about through motor neurons to the muscles.

Comparison of Nervous and Hormonal Control

	Nervous Control	Hormonal Control
Communication	*Impulses across synapses*	*Hormones in the blood*
Speed	*Very rapid (within a few milliseconds)*	*Relatively slow (over minutes, hours, or longer)*
Duration	*Short term and reversible*	*Longer lasting effects*
Target pathway	*Specific (through nerves) to specific cells*	*Hormones broadcast to target cells everywhere*
Action	*Causes glands to secrete or muscles to contract*	*Causes changes in metabolic activity*

1. Identify the three basic components of a nervous system and describe their role:

   (a) _____

   (b) _____

   (c) _____

2. Comment on the significance of the differences between the speed and duration of nervous and hormonal controls:

   _____

   _____

   _____

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192 Neuron Structure and Function

Key idea: Neurons are electrically excitable cells that are specialized to process and transmit information via electrical and chemical signals. Increased axon diameter and myelination both increase conduction speed along a neuron.

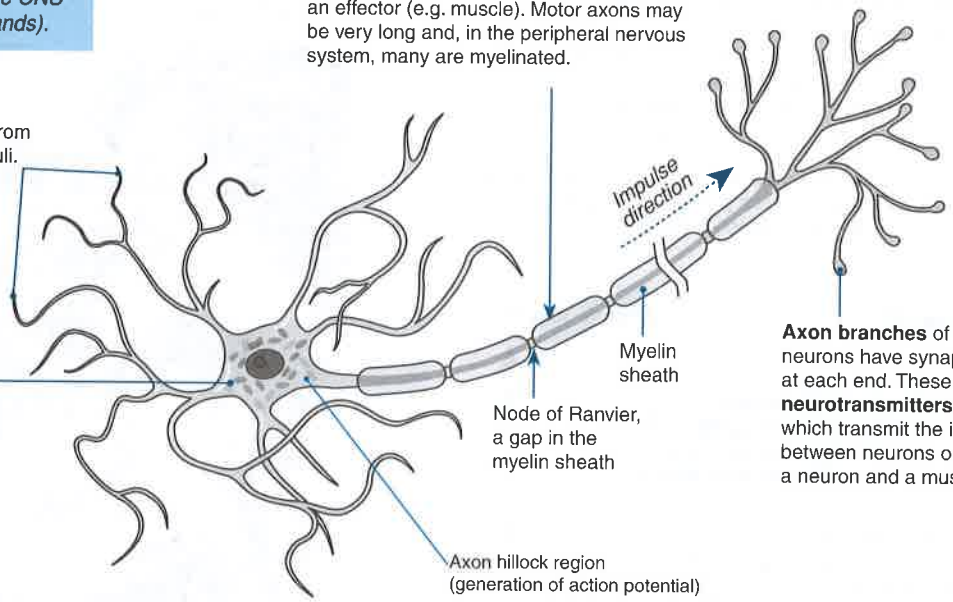
Neurons transmit information in the form of electrochemical signals from receptors (in the central nervous system) to effectors. Neurons consist of a cell body (soma) and long processes (dendrites and axons). Conduction speed increases with axon diameter and with myelination. Faster conduction speeds enable more rapid responses to stimuli.

Motor (efferent) neuron

Transmits impulses from the CNS to effectors (muscles or glands).

Axon: A long extension of the cell transmits the nerve impulse to another neuron or to an effector (e.g. muscle). Motor axons may be very long and, in the peripheral nervous system, many are myelinated.

Dendrites are thin processes from the cell body that receive stimuli.



Cell body or **soma** containing the organelles to keep the neuron alive and functioning.

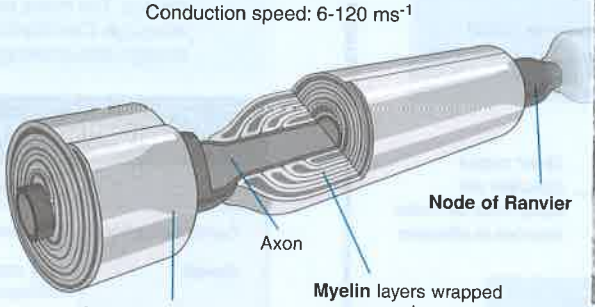
Axon branches of motor neurons have synaptic knobs at each end. These release **neurotransmitters**, chemicals which transmit the impulse between neurons or between a neuron and a muscle cell.

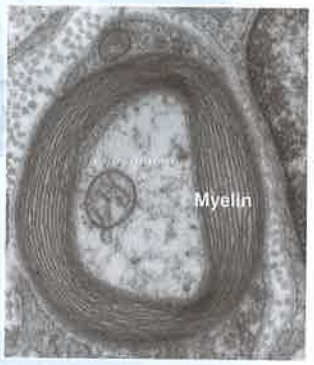
Where conduction speed is important, the axons of neurons are sheathed within a lipid and protein rich substance called **myelin**. Myelin is produced by oligodendrocytes in the central nervous system (CNS) and by Schwann cells in the peripheral nervous system (PNS). At intervals along the axons of myelinated neurons, there are gaps between neighbouring Schwann cells and their sheaths. These are called **nodes of Ranvier**. Myelin acts as an insulator, increasing the speed at which nerve impulses travel because it prevents ion flow across the neuron membrane and forces the current to "jump" along the axon from node to node.

Myelinated Neurons

Diameter: 1-25 µm

Conduction speed: 6-120 ms^{-1}





TEM cross section through a myelinated axon

Readnottaken

1. What is the function of a neuron? _____

_____

2. What factors increase the speed of conduction along a neuron? _____

_____

3. How does myelination increase the speed of nerve impulse conduction? _____

_____

_____

4. What is the advantage of faster conduction of nerve impulses? _____

_____

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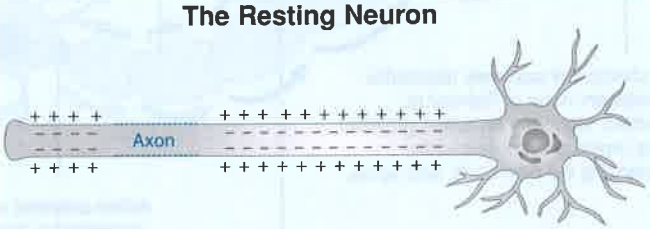
193 The Nerve Impulse

Key idea: A nerve impulse involves the movement of an action potential along a neuron as a series of electrical depolarization events in response to a stimulus.

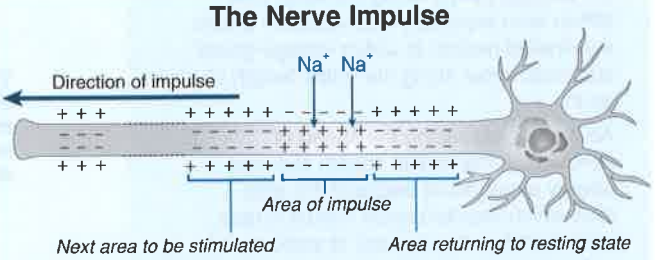
The plasma membranes of cells, including neurons, contain **sodium-potassium ion pumps** which actively pump sodium ions (Na^+) out of the cell and potassium ions (K^+) into the cell. The action of these ion pumps in neurons creates a separation of charge (a potential difference or voltage) either side of the membrane and makes the cells **electrically excitable**. It is this property that enables neurons to transmit electrical impulses. The **resting state** of a neuron, with a net negative charge inside, is maintained by the sodium-potassium pumps, which actively move two K^+ into the neuron for every three Na^+ moved out (below left). When a nerve is stimulated, a brief increase in membrane permeability to Na^+ temporarily reverses the membrane polarity (a **depolarization**). After the nerve impulse passes, the sodium-potassium pump restores the resting potential.

The Resting Neuron

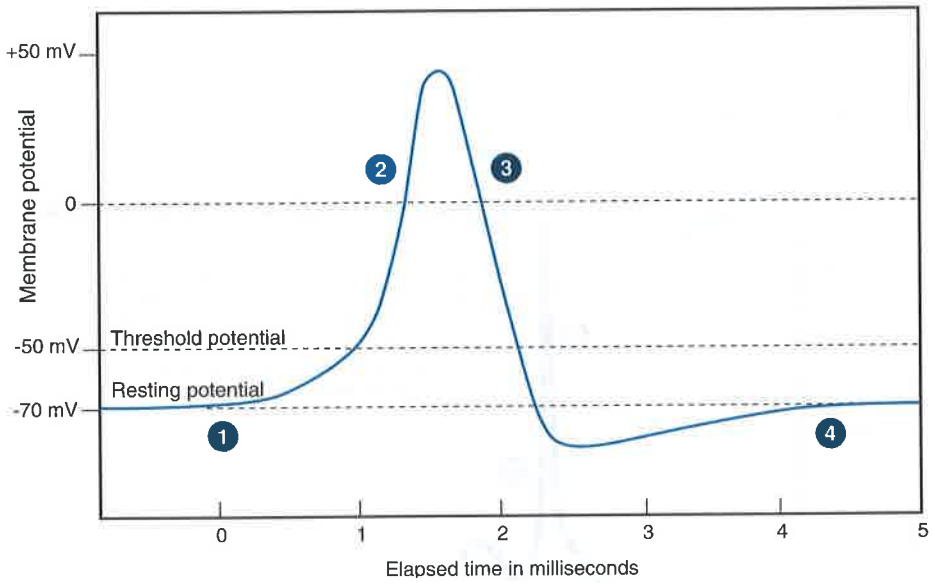


When a neuron is not transmitting an impulse, the inside of the cell is negatively charged relative to the outside and the cell is said to be electrically polarized. The potential difference (voltage) across the membrane is called the **resting potential**. For most nerve cells this is about -70 mV. Nerve transmission is possible because this membrane potential exists.

The Nerve Impulse



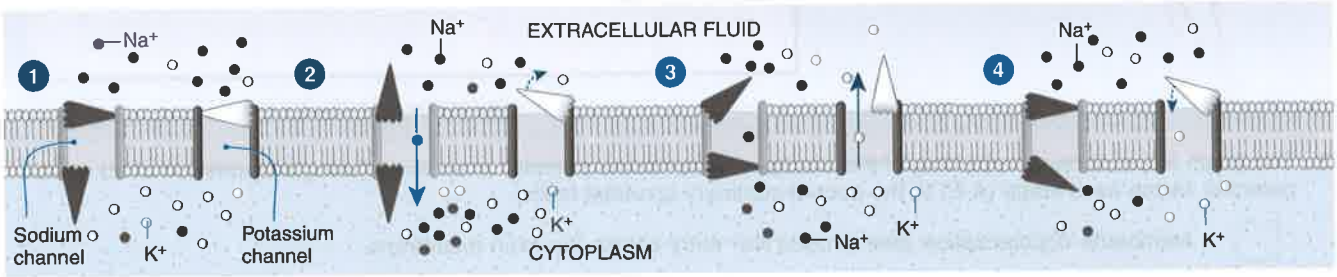
When a neuron is stimulated, the distribution of charges on each side of the membrane briefly reverses. This process of **depolarization** causes a burst of electrical activity to pass along the axon of the neuron as an **action potential**. As the charge reversal reaches one region, local currents depolarize the next region and the impulse spreads along the axon.



The depolarization in an axon can be shown as a change in membrane potential (in millivolts). A stimulus must be strong enough to reach the **threshold potential** before an action potential is generated. This is the voltage at which the depolarization of the membrane becomes unstoppable.

The action potential is **all or nothing** in its generation and because of this, impulses (once generated) always reach threshold and move along the axon without attenuation. The resting potential is restored by the movement of potassium ions (K^+) out of the cell. During this **refractory period**, the nerve cannot respond, so nerve impulses are discrete.

Voltage-Gated Ion Channels and the Course of an Action Potential



Resting state:
Voltage activated Na^+ and K^+ channels are closed.

Depolarization:
Voltage activated Na^+ channels open and there is a rapid influx of Na^+ ions. The interior of the neuron becomes positive relative to the outside.

Repolarization:
Voltage activated Na^+ channels close and the K^+ channels open; K^+ moves out of the cell, restoring the negative charge to the cell interior.

Returning to resting state:
Voltage activated Na^+ and K^+ channels close to return the neuron to the resting state.

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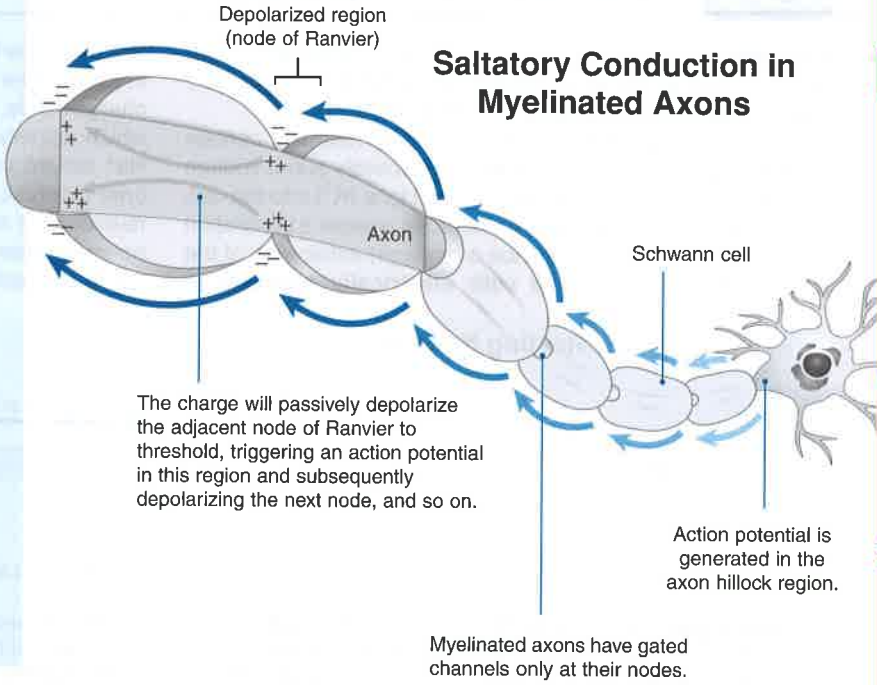
Axon myelination is a feature of vertebrate nervous systems and it enables them to achieve very rapid speeds of nerve conduction. Myelinated neurons conduct impulses by **saltatory conduction**, a term that describes how the impulse jumps along the fibre. In a myelinated neuron, action potentials are generated only at the nodes, which is where the voltage gated channels occur. The axon is insulated so the action potential at one node is sufficient to trigger an action potential in the next node and the impulse jumps along the fibre. This differs from impulse transmission in a non-myelinated neuron in which voltage-gated channels occur along the entire length of the axon.

As well as increasing the speed of conduction, the myelin sheath reduces energy expenditure because the area over which depolarization occurs is less (and therefore the number of sodium and potassium ions that need to be pumped to restore the resting potential is fewer).

Saltatory Conduction in Myelinated Axons



1. What is an action potential? _____

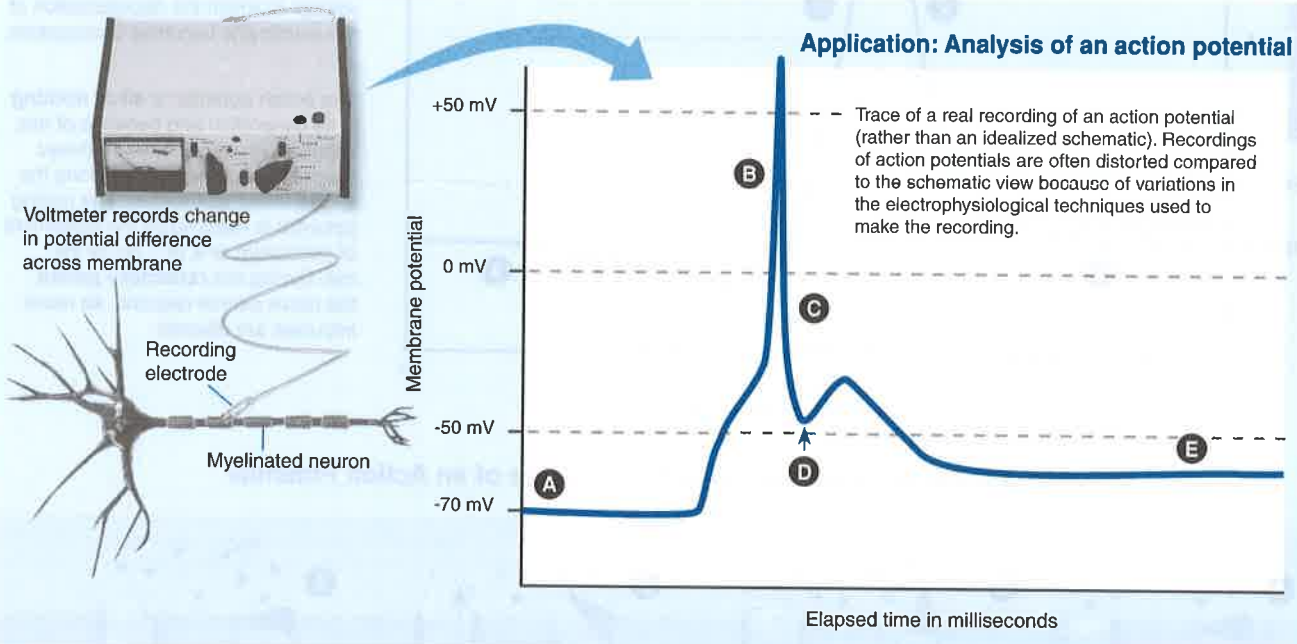
_____

2. (a) What occurs during saltatory conduction? _____

_____

(b) What influence does this have on conduction speed? _____

Application: Analysis of an action potential



3. The graph above shows a recording of the changes in membrane potential in an axon during transmission of an action potential. Match each stage (A-E) to the correct summary provided below.

- ☐ Membrane depolarization (due to rapid Na^+ entry across the axon membrane.
- ☐ Hyperpolarization (an overshoot caused by the delay in closing of the K^+ channels.
- ☐ Return to resting potential after the stimulus has passed.
- ☐ Repolarization as the Na^+ channels close and slower K^+ channels begin to open.
- ☐ The membrane's resting potential.

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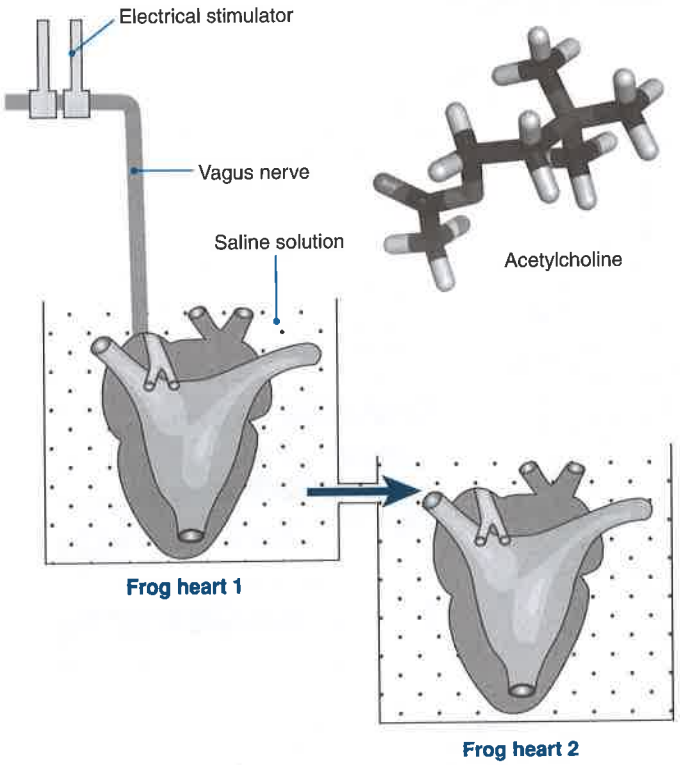
194 Neurotransmitters

Key idea: Neurotransmitters are chemicals that allow the transmission of signals between neurons.

Neurotransmitters are chemicals that transmit signals between neurons. They are found in the axon endings of neurons and are released into the space between one neuron and the next (the synaptic cleft) after depolarization or hyperpolarization of the nerve ending. Different neurotransmitters may produce different responses depending on their location in the body. They can be excitatory (likely to cause an action potential in the receiving neuron) or inhibitory (causing hyperpolarization) depending on the receptor they activate.

Neurotransmitters Carry Signals Between Neurons



Chemical signalling between neurons was first demonstrated in 1921 by Otto Loewi. In his experiment, the still beating hearts of two frogs were placed in connected flasks filled with saline solution. The vagus nerve of the first heart was still attached and was stimulated by electricity to reduce the heart's rate of beating. After a delay, the rate of beating in the second heart also slowed. Increasing the beating rate in the first heart caused an increase in the beating rate in the second heart. Electrical stimulus of the first heart caused it to release a chemical into the saline that then affected the heartbeat of the second heart. The chemical was found to be **acetylcholine,** now known to be a common neurotransmitter.

The Effect of Insecticides on Neurotransmitters

The discovery of neurotransmitters and how they work has allowed scientists to exploit their properties to develop useful applications, including insecticides. Insecticides are chemical substances used to control pest insect numbers. Many insecticides work by affecting the signalling of nerve cells by either blocking uptake of signalling molecules or facilitating the uptake of far greater amounts than normal.

Neonicotoid insecticides are a group of insectides which mimic the action of acetylcholine in synapses. They bind irreversibly to the postsynaptic nicotinic acetylcholine receptors causing the over-stimulation of the neuron, which results in death of the insect. The effects are cumulative, meaning the build up over time, so even at low doses they are fatal.

Neonicotoid insecticides are particularly effective against sucking insects, such as aphids (above), which cause large scale damage to many commercial crops.

1. What is the purpose of a neurotransmitter? _____

_____

2. (a) Name the neurotransmitter discovered by Loewi in his frog heart experiment: _____

(b) Why was there a delay before the second heart in the experiment reduced its beating rate? _____

_____

_____

_____

3. How do **neonicotoid insecticides** interact with chemical synapses? _____

_____

_____

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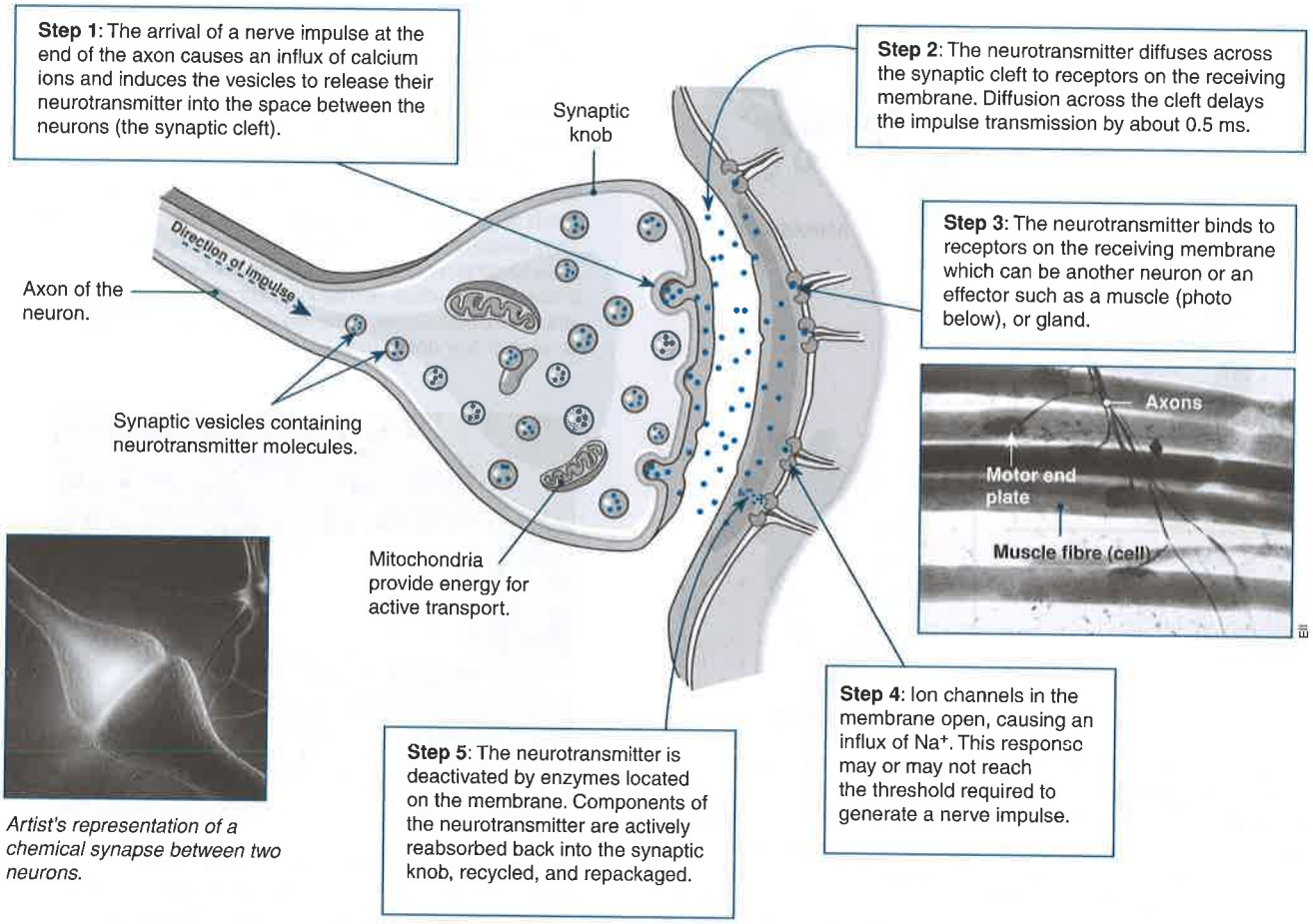
195 Chemical Synapses

Key idea: Chemical synapses are junctions between neurons, or between neurons and receptor or effector cells.

Action potentials are transmitted across junctions called **synapses**. Synapses can occur between two neurons, or between a neuron and an effector cell (e.g. muscle or gland). The axon terminal is a swollen knob, and a small gap (synaptic cleft) separates it from the receiving neuron. The synaptic knobs are filled with tiny packets of chemicals called **neurotransmitters**. The neurotransmitter diffuses across the gap, where it interacts with the receiving (post-synaptic) membrane and causes an electrical response. In the example below, the neurotransmitter causes a membrane depolarization and the generation of an action potential. Some neurotransmitters have the opposite effect and cause inhibition (e.g. slowing heart rate). Chemical synapses are the most widespread type of synapse in nervous systems.

The Structure of a Chemical Synapse



Artist's representation of a chemical synapse between two neurons.

1. What is a **synapse**? _____

2. What causes the release of neurotransmitter into the synaptic cleft? _____

3. Why is there a brief delay in transmitting an impulse across the synapse? _____

4. (a) How is the neurotransmitter deactivated? _____

   (b) Why is it important for the neurotransmitter substance to be deactivated soon after its release? _____

5. Suggest one factor that might influence the strength of the response in the receiving cell: _____

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196 Chemical Imbalances in the Brain

Key idea: Some mental disorders can be treated with drugs that act on the synapses in the brain.

Many types of mental illness result from disturbances to natural levels of specific neurotransmitters, and can lead to the failure of specific neural pathways. Scientists have used their knowledge of neurotransmitters and chemical synapses to develop drugs that either replace or boost levels of specific neurotransmitters and help treat specific brain disorders.

Parkinson's Disease

Patients with **Parkinson's disease** show decreased stimulation in the motor cortex of the brain. This results from reduced dopamine production in the substantia nigra region (right) where dopamine is produced. This is usually the result of the death of nerve cells. Symptoms, slow physical movement and spasmodic tremors, often don't begin to appear until a person has lost 70% of their dopamine-producing cells.

Treating Parkinson's Disease

Parkinson's disease is caused by reduced dopamine production and low dopamine levels in the brain pathways involved with movement. Treatments for Parkinson's have focused on increasing the body's dopamine levels. Dopamine is unable to cross the blood-brain barrier, so cannot be administered as a treatment. However, **L-dopa** is a dopamine precursor that can cross the blood-brain barrier and enter the brain. Once in the brain, it is converted to dopamine. L-dopa has been shown to reduce some of the symptoms of Parkinson's disease.

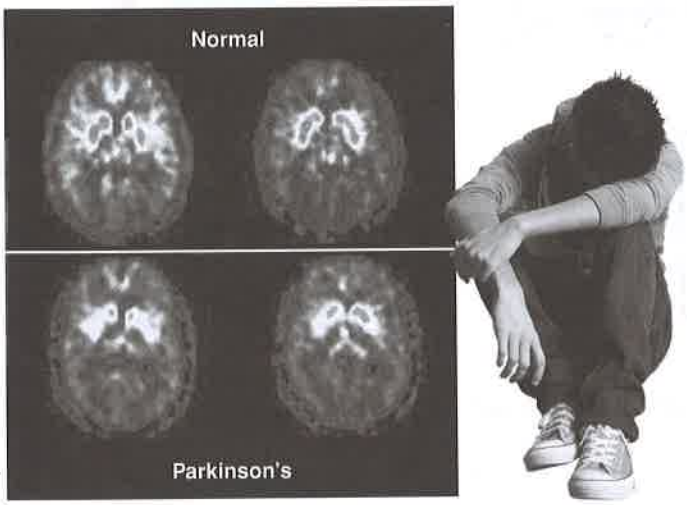
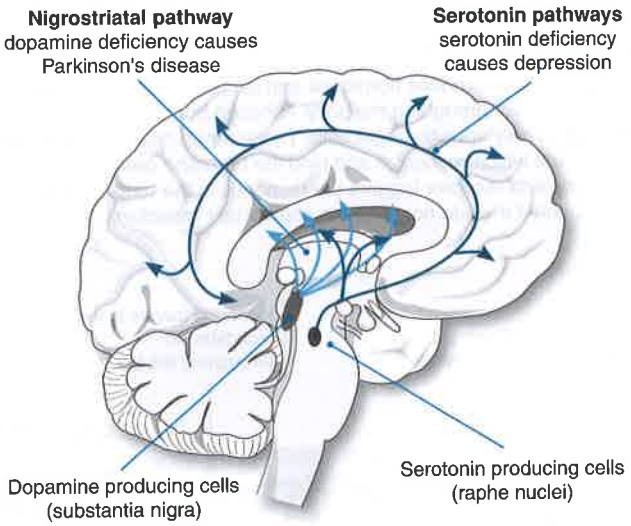


Image: NASA

Positron emission tomography (PET) measures the activity of dopamine neurons in the substantia nigra area of the brain. Parkinson's patients (lower panel) show reduced activity in the dopamine neurons compared with normal patients.



Depression

A person with **depression** (left) experiences prolonged periods of extremely low mood, including low self esteem, regret, guilt, and feelings of hopelessness. Depression may be caused by a mixture of environmental factors (e.g. stress) and biological factors (e.g. low **serotonin** production by the raphe nuclei in the brain, above).

Treating Depression

Recognition of the link between **serotonin** and **depression** has resulted in the development of **antidepressant drugs** that alter serotonin levels. Monoamine oxidase inhibitors (MAOI) are commonly used antidepressants that increase serotonin levels by preventing its breakdown in the brain. Newer drugs, called Selective Serotonin Re-uptake Inhibitors (SSRIs), stop serotonin re-uptake by presynaptic cells. This increases the levels of extracellular serotonin, making more available to bind to the postsynaptic cells, and stabilizing serotonin levels in the brain. SSRIs have fewer side effects than other antidepressants because they specifically target serotonin and no other neurotransmitters.

1. What role do neurotransmitters have in mental illness? _____

_____

_____

2. Describe the cause of the following diseases and describe how they can be treated using pharmaceuticals:

(a) Parkinson's disease: _____

_____

_____

(b) Depression: _____

_____

_____

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