

Impulses, synapses and receptors

Revision notes · Action potentials and saltatory conduction, cholinergic synapses, the pupil reflex, drugs at synapses, rods and rhodopsin, and habituation.

For Pearson Edexcel International AS Biology XBI11 and International A Level Biology YBI11. **A Level only** is IA2 content (Units 4 to 6), not needed for the International AS.

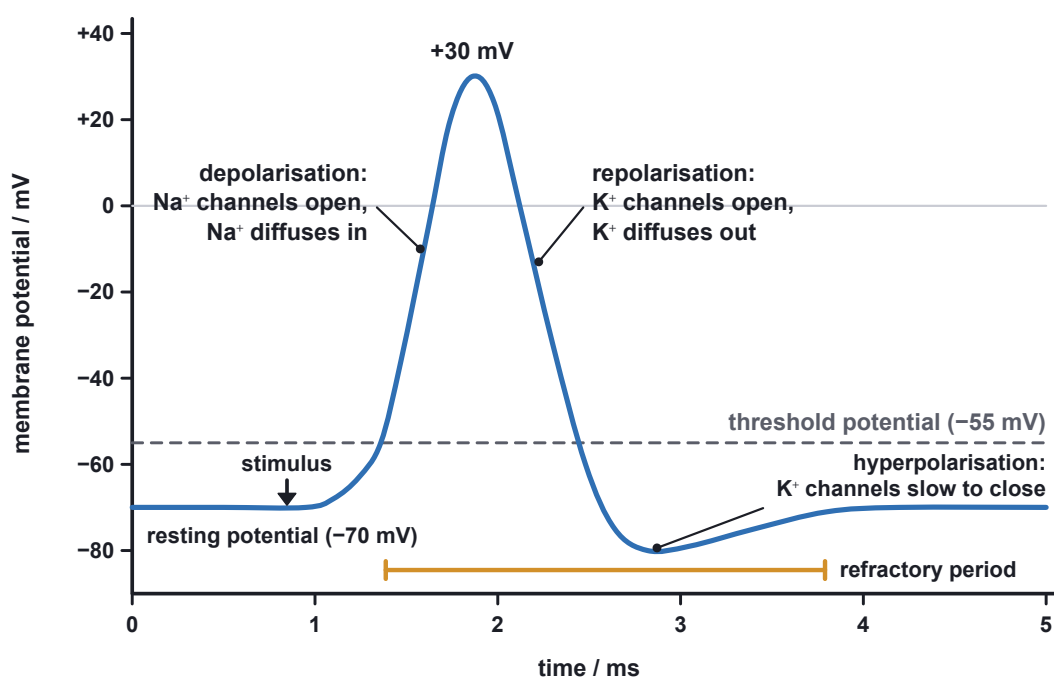
Impulses, synapses and receptors Spec 8.4-8.9 A Level only

Resting potential and action potentials Spec 8.4

At rest, the inside of an axon is negative relative to the outside. This **resting potential** is about -70 mV. It is set up by the **sodium-potassium pump**, which uses ATP to move 3 Na^+ out of the axon for every 2 K^+ moved in, and by **potassium ion channels** that leak K^+ out far more easily than Na^+ can leak in. The membrane is said to be **polarised**.

A stimulus that is strong enough opens some **voltage-gated sodium ion channels**. When the membrane potential reaches the **threshold** (about -55 mV), many more open. This is an **all-or-nothing** response: below threshold nothing happens, and above it every action potential has the same size.

1. **Depolarisation**: voltage-gated Na^+ channels open, so Na^+ diffuses in down its electrochemical gradient. The inside becomes positive, reaching about $+40$ mV.
2. At the peak the Na^+ channels close (they are inactivated) and voltage-gated **K^+ channels** open.
3. **Repolarisation**: K^+ diffuses out of the axon, so the inside becomes negative again.
4. **Hyperpolarisation**: the K^+ channels are slow to close, so the potential briefly falls below -70 mV. The K^+ channels then close and the sodium-potassium pump and K^+ leak channels restore the resting potential.



An **action potential**: depolarisation as Na^+ channels open, repolarisation as K^+ channels open, then hyperpolarisation.

During and just after an action potential the Na^+ channels are inactivated, so the region cannot respond to another stimulus. This **refractory period** means impulses travel in one direction only, do not overlap, and are separate events. The frequency of impulses, not their size, carries information about stimulus strength.

An action potential travels along the axon by **local circuits**. Na^+ entering one region diffuses sideways inside the axon, depolarising the next region to threshold, so its Na^+ channels open. The region behind is refractory, so the impulse moves forward.

Exam tip: Use the terms 'voltage-gated', 'down an electrochemical gradient' and 'permeability'. State which ion moves, in which direction, through which channel, and what happens to the potential.

Myelination and saltatory conduction Spec 8.5

In an unmyelinated axon the action potential must be regenerated at every point along the membrane, so conduction is slow (about 0.5 to 2 m s^{-1}). In a myelinated axon, the myelin sheath is an insulator, so ions can enter and leave only at the **nodes of Ranvier**. The voltage-gated Na^+ and K^+ channels are concentrated at the nodes.

1. An action potential at one node makes Na^+ enter and depolarise the node.
2. Local circuits carry the positive charge along inside the axon, through the myelinated section, to the next node.
3. The next node reaches threshold and an action potential forms there.
4. The impulse appears to **jump** from node to node. This is **saltatory conduction** (Latin saltare, to leap).

Because depolarisation occurs only at the nodes, conduction can reach about 100 m s^{-1} . Fewer ion channels need to open and fewer ions move, so less ATP is needed for the sodium-potassium pumps to restore the resting potential.

Common mistake: The impulse does not travel through the myelin. The action potential is generated at the nodes, and local circuits carry the depolarisation between them.

Conduction velocity

An impulse travels 0.45 m along a myelinated motor axon in 5.0 ms . Calculate the speed of conduction in m s^{-1} .

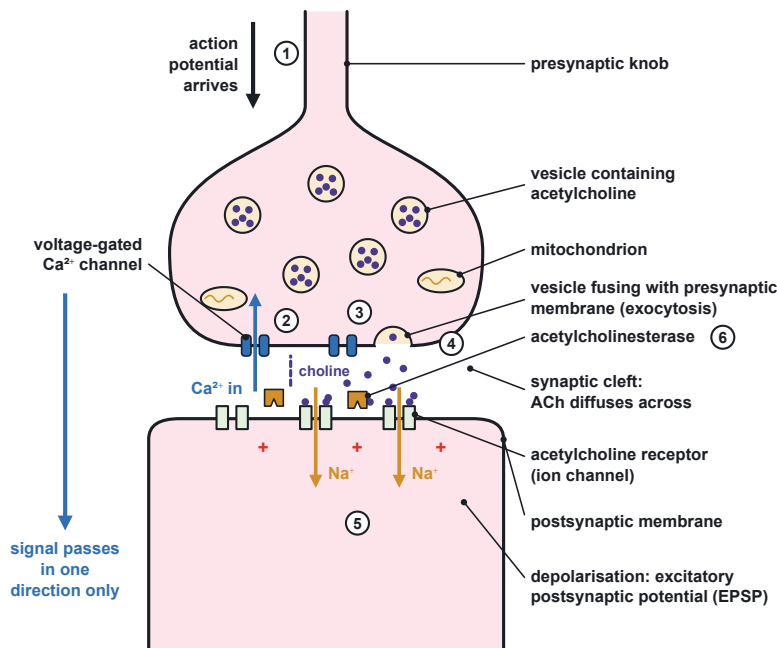
1. Convert time: $5.0 \text{ ms} = 5.0 \times 10^{-3} \text{ s}$
2. speed = distance $\div$ time = $0.45 \div 5.0 \times 10^{-3}$

Answer: 90 m s^{-1}

Maths skill: Convert to SI units before dividing: milliseconds to seconds, millimetres to metres.

Synapses and neurotransmitters Spec 8.6

A **synapse** is the junction between two neurones. A narrow gap of about 20 nm , the **synaptic cleft**, separates the **presynaptic knob** from the **postsynaptic membrane**. Impulses cannot cross the gap, so a chemical, a **neurotransmitter**, carries the signal. At a **cholinergic synapse** the neurotransmitter is **acetylcholine (ACh)**.



- ① action potential arrives and depolarises the presynaptic membrane
- ② voltage-gated Ca^{2+} channels open and Ca^{2+} diffuses into the knob
- ③ vesicles fuse with the presynaptic membrane and release ACh by exocytosis
- ④ ACh diffuses across the synaptic cleft and binds to receptors on the postsynaptic membrane
- ⑤ Na^{+} channels open and Na^{+} enters, depolarising the postsynaptic membrane; if the threshold is reached an action potential starts
- ⑥ acetylcholinesterase breaks ACh down into choline and ethanoic acid; choline is taken back into the knob and ACh is remade using ATP from the mitochondria

Transmission across a **cholinergic synapse**, in six numbered steps.

1. An action potential arrives at the presynaptic knob and opens voltage-gated **Ca^{2+} channels**. Ca^{2+} enters by facilitated diffusion.
2. Ca^{2+} makes vesicles containing ACh move to and fuse with the presynaptic membrane. ACh is released by **exocytosis**.
3. ACh diffuses across the synaptic cleft and binds to **receptors** on the postsynaptic membrane.
4. The receptors are linked to Na^{+} channels, which open. Na^{+} enters the postsynaptic neurone, so the membrane is depolarised.
5. If depolarisation reaches threshold, an action potential is set off in the postsynaptic neurone.
6. The enzyme **acetylcholinesterase** in the cleft hydrolyses ACh to choline and ethanoate (acetate), so the response stops. Choline is taken back into the presynaptic knob and used, with ATP from the mitochondria, to resynthesise ACh.

Synapses ensure **one-way transmission** because only the presynaptic knob has vesicles and only the postsynaptic membrane has receptors. A single impulse may not release enough ACh to reach threshold in the next neurone, so a synapse controls which signals are passed on.

Acetylcholine is one of many **neurotransmitters**; others include noradrenaline, dopamine and serotonin. Each binds only to receptors with a complementary shape. At an **excitatory synapse** the transmitter opens Na^{+} channels and depolarises the postsynaptic membrane. At an **inhibitory synapse** the transmitter (for example GABA) opens channels that make the postsynaptic membrane more negative (hyperpolarised), so an action potential is less likely. Whether the postsynaptic neurone fires depends on the combined effect of all the transmitter released onto it, from several synapses at once (spatial summation) or from impulses arriving in quick succession at one synapse (temporal summation).

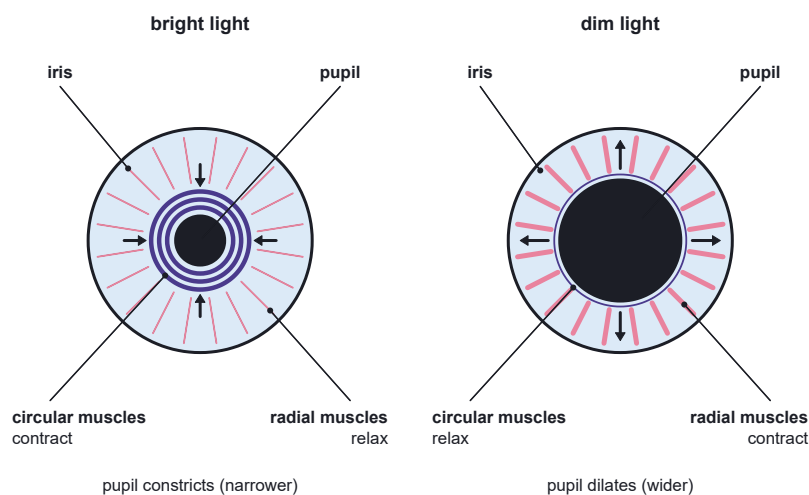
Exam tip: Name the ion (Ca^{2+} entering the presynaptic knob, Na^+ entering the postsynaptic neurone), the process (exocytosis, diffusion) and the enzyme that breaks down ACh. Each is a mark.

How the pupil dilates and constricts Spec 8.6

The **iris** contains two sets of muscle that act as effectors, controlling the size of the **pupil** and so how much light reaches the retina. They are **antagonistic muscles**: when one contracts the other relaxes.

Pupil responses		
	Bright light	Dim light
Circular muscles	Contract	Relax
Radial muscles	Relax	Contract
Pupil	Constricts (gets smaller)	Dilates (gets larger)
Purpose	Protects the retina (the light receptors) from damage by intense light	Lets more light reach the retina so rods can detect it
Control	Parasympathetic neurones release acetylcholine onto the circular muscles	Sympathetic neurones release noradrenaline onto the radial muscles

The pupil constriction in bright light is a reflex. Light receptors in the retina send impulses along the optic nerve to the brain, and motor neurones carry impulses to the muscles of the iris. This is rapid and automatic, so it protects the retina.



In bright light the **circular muscles contract** and the pupil constricts; in dim light the **radial muscles contract** and it dilates.

Common mistake: Do not say that the iris 'opens and closes'. The pupil is the hole; the muscles in the iris change its size.

Drugs and nerve impulse transmission Spec 8.7

A drug can stimulate (an **agonist**), block (an **antagonist**) or alter the amount of neurotransmitter, so it changes transmission of impulses along axons or across synapses.

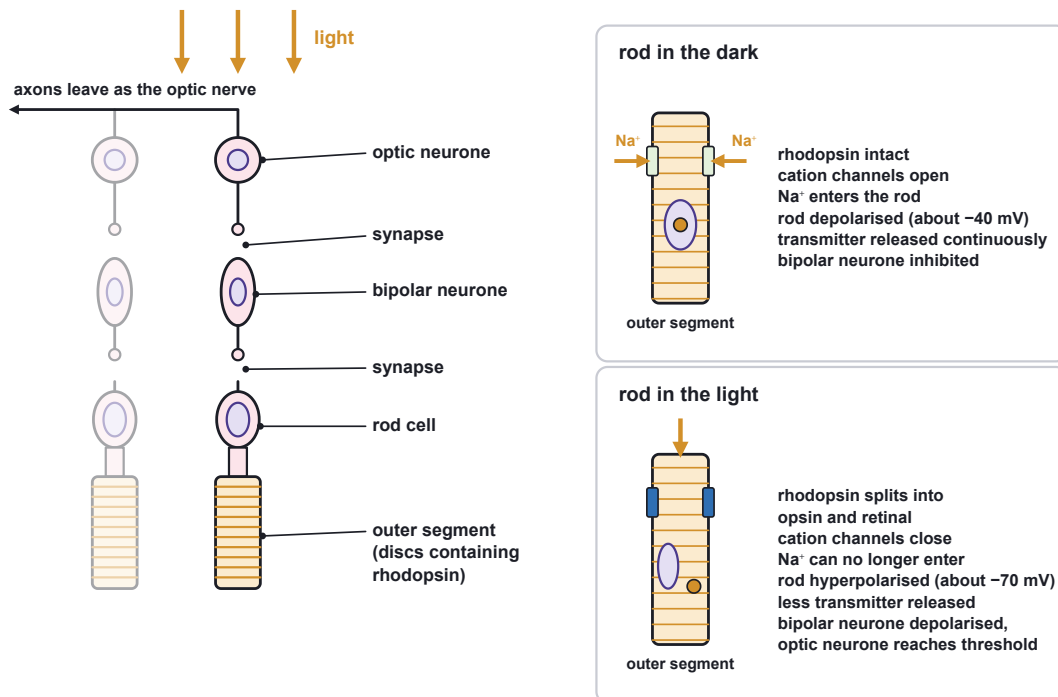
Drugs that affect nerve impulse transmission		
Drug	Action	Effect
Nicotine	Binds to acetylcholine receptors and mimics ACh, so Na ⁺ channels open and the postsynaptic neurone is depolarised. It is not broken down by acetylcholinesterase, so stimulation lasts longer.	Stimulates nerve impulses: raises heart rate and blood pressure; in the brain it also triggers release of dopamine, which contributes to addiction
Lidocaine	A local anaesthetic that blocks voltage-gated Na ⁺ channels in the axon membrane, so no depolarisation occurs and action potentials are not conducted.	Impulses from pain receptors do not reach the brain: numbness
Cobra venom alpha toxin	Binds very tightly (almost irreversibly) to acetylcholine receptors on the postsynaptic membrane, so ACh cannot bind and Na ⁺ channels do not open.	No depolarisation of the muscle: paralysis, including breathing muscles
L-DOPA	A precursor of dopamine that crosses the blood-brain barrier and is converted to dopamine in the brain. Dopamine itself cannot cross it.	Replaces dopamine lost in Parkinson's disease, reducing tremor and rigidity
MDMA (ecstasy)	Causes release of serotonin from presynaptic neurones and prevents its reuptake, so more serotonin stays in the synaptic cleft and acts on the postsynaptic membrane for longer.	Raised mood and energy; can cause overheating; serotonin stores become depleted

Exam tip: In a 'suggest how' question, name the target (receptor, channel or reuptake), say whether the drug mimics or blocks, and link it to the effect on depolarisation of the postsynaptic membrane.

Rods, rhodopsin and the optic neurone Spec 8.8

Rod cells in the retina detect light, particularly in dim conditions. Their outer segment contains the pigment **rhodopsin**, made of a protein, **opsin**, bound to **retinal**, a derivative of vitamin A.

In the retina, the rods synapse with bipolar neurones, which in turn synapse with the optic neurones whose axons form the optic nerve. The key point is that light causes hyperpolarisation, not depolarisation, of the rod cell.



Light passes through the **optic neurone** and **bipolar neurone** to the **rod cell**; in the light the rod hyperpolarises.

1. **In the dark:** cation channels in the rod membrane are open, so Na^+ continuously enters. The rod is partly depolarised (about -40 mV) and releases an inhibitory transmitter (glutamate) onto the bipolar neurone, so the bipolar neurone is hyperpolarised and does not pass a signal on.
2. **In the light:** absorbing light changes retinal from its cis form to its trans form, so rhodopsin breaks down into opsin and retinal (bleaching). Opsin activates a cascade of reactions in the membrane (a fall in cyclic GMP, which normally keeps the channels open), so the cation channels close.
3. Na^+ can no longer enter, but the sodium-potassium pump keeps removing it and K^+ continues to leave, so the rod becomes more negative: **hyperpolarisation** (about -70 mV).
4. The hyperpolarised rod releases less inhibitory transmitter, so the bipolar neurone is depolarised and releases transmitter onto an **optic neurone**.
5. The optic neurone reaches threshold and action potentials pass along the optic nerve to the visual cortex of the brain.

Rods are very sensitive because one photon can close many channels. Several rods synapse with one bipolar neurone, which makes rods sensitive in dim light but gives low visual acuity, and they do not distinguish colours.

Common mistake: The rod cell itself does not fire action potentials. The action potentials are formed in the optic neurone, after the bipolar neurone is depolarised.

Habituation Spec 8.9

Habituation is a form of simple learning in which an organism gradually stops responding to a stimulus that is repeated but has no good or bad consequence. For example, a bird stops flying off at a scarecrow, or you stop noticing the sound of a clock.

1. Each repeated stimulus causes fewer voltage-gated Ca^{2+} channels to open in the presynaptic knob.
2. Less Ca^{2+} enters, so fewer vesicles release neurotransmitter.
3. The postsynaptic membrane is less depolarised and the postsynaptic neurone is less likely to reach threshold.
4. The response (for example a muscle contraction) becomes smaller and may stop.

Habituation is useful because it stops an animal wasting energy on irrelevant stimuli and keeps its attention available for new stimuli that may signal food or danger. It differs from fatigue because a different stimulus restores the full response straight away.

Practical skill: Recommended additional practical: investigate habituation to a stimulus. Choose a response that can be scored, for example the withdrawal of a snail's tentacles when touched with a thin rod, or the number of times a stimulus causes a response in set intervals. Repeat the stimulus at a regular interval; record the response each time; control the strength, interval and position of the stimulus and temperature; test a different stimulus at the end to show it is not fatigue. Ethics: use minimal stimulation, handle animals gently and return them to their habitat.

Quick check

1. What is the resting potential of an axon, approximately?
2. Which ion enters the presynaptic knob and causes vesicles to release acetylcholine?
3. What does acetylcholinesterase do?
4. What is saltatory conduction?
5. What happens to a rod cell's membrane potential when light strikes it?

Quick check answers

Impulses, synapses and receptors

1. About -70 mV (the inside is negative relative to the outside).
2. Calcium ions (Ca^{2+}).
3. It hydrolyses acetylcholine to choline and ethanoate in the synaptic cleft, ending the stimulation.
4. Conduction in which the action potential jumps from one node of Ranvier to the next along a myelinated axon.
5. It becomes hyperpolarised, because rhodopsin splits and the cation channels close.