

Impulses, synapses and receptors What are nodes of Ranvier?	Impulses, synapses and receptors What is the resting potential of an axon and what sets it up?
Impulses, synapses and receptors What ion movement causes depolarisation?	Impulses, synapses and receptors What ion movement causes repolarisation?
Impulses, synapses and receptors What is the threshold potential and the all-or-nothing principle?	Impulses, synapses and receptors What is the refractory period and why does it matter?
Impulses, synapses and receptors Define saltatory conduction.	Impulses, synapses and receptors Why is saltatory conduction faster?

About -70 mV. The sodium-potassium pump (3 Na⁺ out, 2 K⁺ in) and K⁺ leak channels.

Gaps in the myelin sheath, about 1 mm apart, where the axon membrane is exposed; in a myelinated axon depolarisation happens only here.

K⁺ leaves the axon through voltage-gated potassium channels.

Na⁺ enters the axon through voltage-gated sodium channels.

Na⁺ channels are inactivated, so no new action potential forms. It makes impulses discrete and one-way.

About -55 mV. Below it no action potential forms; above it every action potential is the same size.

Depolarisation occurs only at nodes, so the impulse jumps between them rather than regenerating at every point.

The action potential jumps from node of Ranvier to node along a myelinated axon, by local circuits.

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Name the sequence at a cholinergic synapse.

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What does acetylcholinesterase do?

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Why are synapses one-way?

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What is the difference between an excitatory and an inhibitory synapse?

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How does the pupil constrict in bright light?

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How does the pupil dilate in dim light?

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How does nicotine affect synapses?

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How does lidocaine work?

Hydrolyses acetylcholine into choline and ethanoate in the synaptic cleft.

Ca^{2+} in, vesicles fuse, ACh released, ACh binds receptors, Na^+ in, postsynaptic depolarisation, acetylcholinesterase breaks ACh down.

Excitatory: transmitter depolarises the postsynaptic membrane (Na^+ in). Inhibitory: transmitter (for example GABA) hyperpolarises it, making an action potential less likely.

Only the presynaptic knob has vesicles of neurotransmitter and only the postsynaptic membrane has receptors.

Radial muscles contract and circular muscles relax.

Circular muscles contract and radial muscles relax.

It blocks voltage-gated sodium channels in axons, so action potentials are not conducted.

It binds to acetylcholine receptors and mimics ACh, depolarising the postsynaptic membrane.

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How does cobra venom alpha toxin work?

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Why is L-DOPA given in Parkinson's disease?

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How does MDMA act at synapses?

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Of what is rhodopsin made?

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What happens to a rod cell in the dark?

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What happens to a rod cell in the light?

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Define habituation.

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What is the mechanism of habituation?

It crosses the blood-brain barrier and is converted to dopamine, replacing the dopamine that is lacking.

It binds very tightly (almost irreversibly) to ACh receptors, blocking transmission to muscles and causing paralysis.

The protein opsin bound to retinal.

It increases the release of serotonin and prevents its reuptake, so more stays in the cleft.

Retinal changes from cis to trans, rhodopsin splits, cation channels close and the rod is hyperpolarised, releasing less transmitter.

Cation channels are open, Na^+ enters, the rod is depolarised and releases inhibitory transmitter.

Fewer Ca^{2+} channels open in the presynaptic knob, so less neurotransmitter is released.

A decrease in response to a repeated stimulus that is harmless.