

Q & A: Neuron depolarization, hyperpolarization, and action potentials

Question:

What causes the hyperpolarization and depolarization of membrane potential, and how does change in membrane potential trigger graded and action potentials for the transmission of signals?

Answer:

That's a great question! Here is a written explanation, with links to some videos that may also help you.

At rest, a typical neuron has a resting potential (potential across the membrane) of -60 to -70 millivolts. This means that the interior of the cell is negatively charged relative to the outside.

Hyperpolarization is when the membrane potential becomes more negative at a particular spot on the neuron's membrane, while depolarization is when the membrane potential becomes less negative (more positive). Depolarization and hyperpolarization occur when ion channels in the membrane open or close, altering the ability of particular types of ions to enter or exit the cell. For example:

The opening of channels that let positive ions flow out of the cell (or negative ions flow in) can cause hyperpolarization. Examples: Opening of channels that let **K** out of the cell or **Cl** into the cell.

The opening of channels that let positive ions flow into the cell can cause depolarization. Example: Opening of channels that let **Na** into the cell.

The opening and closing of these channels may depend on the binding of signalling molecules such as neurotransmitters (ligand-gated ion channels), or on the voltage across the membrane (voltage-gated ion channels).

Graded potentials

A hyperpolarization or depolarization event may simply produce a graded potential, a smallish change in the membrane potential that is proportional to the size of the stimulus. As its name suggests, a graded potential doesn't come in just one size – instead, it comes in a wide range of slightly different sizes, or gradations. Thus, if just one or two channels open (due to a small stimulus, such as binding of a few molecules of neurotransmitter), the graded potential may be small, while if more channels open (due to a larger stimulus), it may be larger. Graded potentials don't travel long distances along the neuron's membrane, but rather, travel just a short distance and diminish as they spread, eventually disappearing.

Action potential

Alternatively, a large enough depolarization event, perhaps resulting from multiple depolarizing inputs that happen at the same time, can lead to the production of an action potential. An action potential, unlike a graded potential, is an all-or-none event: it may or may not occur, but when it does occur, it will

always be of the same size (is not proportional to the size of the stimulus).

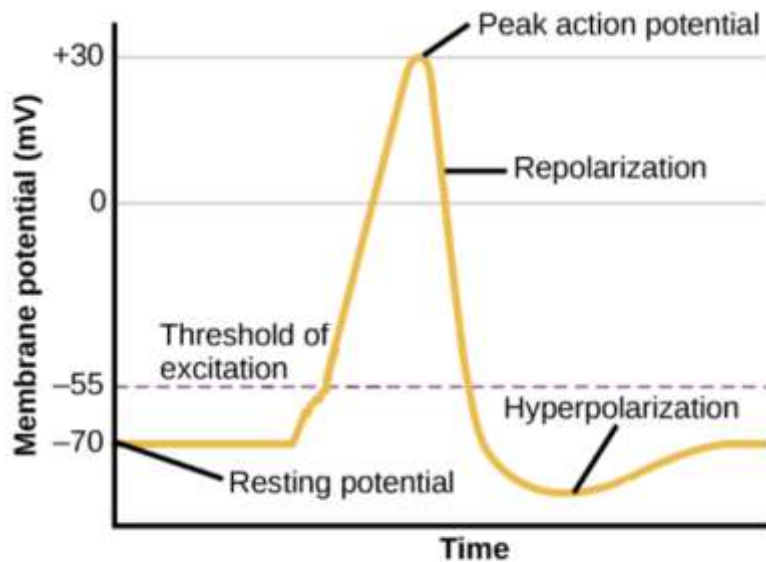


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An action potential begins when a depolarization increases the membrane voltage so that it crosses a threshold value (usually around -55 mV).

At this threshold, voltage-gated Na channels in the membrane open, allowing many sodium ions to rush into the cell. This influx of sodium ions makes the membrane potential increase very rapidly, going all the way up to about $+40$ mV. After a short time, the sodium channels self-inactivate (Close and become unresponsive to voltage), stopping the influx of sodium. A set of voltage-gated potassium channels open, allowing potassium to rush out of the cell down its electrochemical gradient. These events rapidly decrease the membrane potential, bringing it back towards its normal resting state.

The voltage-gated potassium channels stay open a little longer than needed to bring the membrane back to its resting potential. This results in a phenomenon called “undershoot,” in which the membrane potential briefly dips lower (more negative) than its resting potential.

Eventually, the voltage-gated potassium channels close and the membrane potential stabilizes at resting potential. The sodium channels return to their normal state (remaining closed, but once more becoming responsive to voltage). The action potential cycle may then begin again.

Transmission of a signal by action potentials

The cycle above is described for just one patch of membrane. However, an action potential can travel down the length of a neuron, from the axon hillock (the base of the axon, where it joins the cell body) to the tip of the axon, where it forms a synapse with the receiving neuron.

This directional transmission of the signal occurs for two reasons:

First, when one patch of membrane (say, right at the axon hillock) undergoes an action potential, lots of **Na** ions rush into the cell through that patch. These ions spread out laterally inside the cell and can depolarize a neighbouring patch of membrane, triggering the opening of voltage gated sodium channels and causing the neighbouring patch to undergo its own action potential.

Second, the axon potential can only travel in one

direction – from the cell body towards the axon terminal – because a patch of membrane that has just undergone one action potential is in a “refractory period” and cannot undergo another. The refractory period is primarily due to the inactivation of voltage-gated sodium channels, which occurs at the peak of the action potential and persists through most of the under-shoot period. These inactivated sodium channels cannot open, even if the membrane potential goes above threshold. The slow closure of the voltage-gated potassium channels, which results in undershoot, also contributes to the refractory period by making it harder to depolarize the membrane (even once the voltage-gated sodium channels have returned to their active state).

The refractory period ensures that an action potential will only travel forward down the axon, not backwards through the portion of the axon that just underwent an action potential.

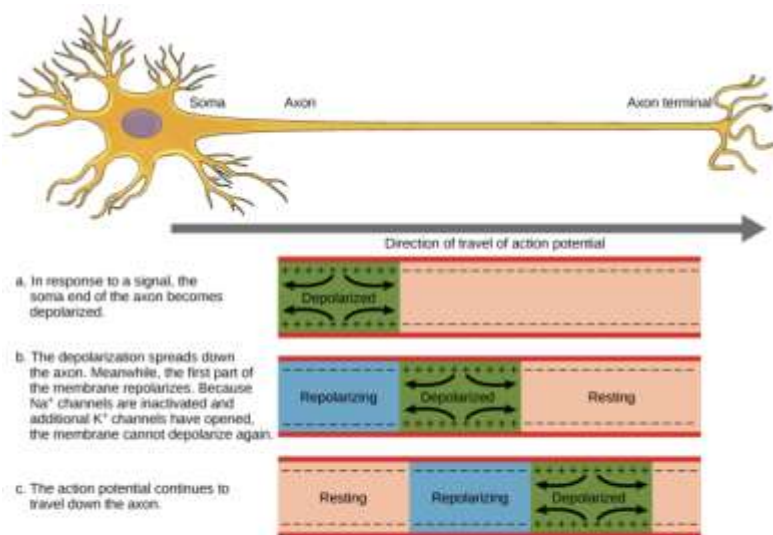


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When the action potential reaches the end of the axon (the axon terminal), it causes neurotransmitter containing

Vesicles to fuse with the membrane, releasing neurotransmitter molecules into the synaptic cleft (space between neurons). When the neurotransmitter molecules bind to ligand-gated ion channels on the receiving cell, they may cause depolarization of that cell, causing it to undergo its own action potential. (Some neurotransmitters also cause hyperpolarization, and a single cell may receive both types of inputs.)