Channel gating during an action potential

Channel gating is the opening - or activation - or closing – by either deactivation or inactivation – of the ion channels. In closed state, the ion channel is non-conducting, meaning that it's impermeable to ions and do not conduct electrical current. Ont he other hand, the open state means that the channel caconduct electrical current by allowing specific ions to pass through the plasma membrane, thus causing electircal current (and potential change) across the membrane.

Channel gating can be **caused** by various events, for example changes in voltage across the cell; drugs; hormones; changes in temperature; stretching or deformation of the cell membrane; addition of a phosphate group to the ion channel (phosphorylation); interaction with other molecules in the cell (e.g., G proteins), etc.

The **two main types** of gating happen in voltage-gated and ligand-gated ion channels. The stats in either can be activation, deactivation, inactivation and reactivation.

Activation in voltage-gated channels is caused by the ion channels' reaction the voltage differential across the membrane where parts of the channel domain act as voltage sensors. The membrane is depolarized, changes in electrostatic forces occour which moves the voltage-sensing domain. The result is the conformation of the channel and opening the pore changes.

In ligand-gated channels the transmembrane ion-channel protein opens to allow ions to pass through in response to the binding of a chemical messenger (ligand, eg. a neurotransmitter) to an extracellular receptor on or near the channel, causes conformational change, opens the pore and allows ion permeation.

Deactivation is the closing of the ion channel pore. In voltage-gated channels, voltage differntial returns to resting value and membrane potential becomes more negative, while ligand-gated channels the ligand dissociates from the cannel's receptor binding site.

Inactivation is when the flow of ions is blocked by a mechanism other than the closing of the channel – eg. the channel in open state stops allowing ions to flow through, or a channel in closed state may be preemptively inactivated to prevent the flow of ions. It always starts by depolarization off cell membrane, and ends with the restoration of resting potential. A typical example is called **ball and chain inactivation** (N-type or hinged lid inactivation) in voltage-gated channels. In this case, one or more of subunits of the channel have a ball domain located on its cytoplasmic N-terminus. This ball domain is electrostatically attracted to the inner channel domain, activating the ion channel. The inner channel domain is exposed, the chain folds and the ball enters the channel, ion permeation is allowed. Channel returns to closed state by blocking the channel domain, which causes the ball to leave the pore.

Action potential is the change in membrane potential from a resting value of about -70 mV to a peak of about +30 mV, and back to -70 mV again. It results from a rapid change in the permeability of the neuronal membrane to Na+ and K+ due to opening and closing of voltage-gated channels. Four phases take place, the first being **depolarization**, when sodium ion channels open due to an electrical stimulus and the charge inside is raised by Na+ ions. Next**, action potential** is produced when treshold is reached, and maximum response gets elicited. The third one is **repolarisation** during which raised charge inside makes Na+ channels close, K+ channels open and K+ ions move out (along the electrochemical gradient). Membrane potential falls and approaches resting potential. Hyperpolarisation makes up for **repolarisation** overshots during the refractory period.

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