Ion Channels Of Excitable Membranes Pdf Extra Quality Download

when the concentration of k+ inside the cell is greater than that in the extracellular fluid, the concentration gradient is very steep and the electrical force acts in a way that causes k+ to leak out of the cell. thus, a voltage-gated k+ channel is likely to be opened when the membrane potential is more positive than the equilibrium potential. the opening of a k+ channel allows k+ to flow out of the cell, thereby reducing the concentration gradient and the electrical force. the opening of a na+ channel has a similar effect, but it is much less effective because na+ is less abundant inside the cell than k+ and because na+ cannot leak out of the cell. thus, opening a na+ channel tends to depolarize the membrane and may trigger the opening of other channels. in contrast, the opening of cl- channels tends to hyperpolarize the membrane because cl- is much less abundant inside the cell than it is outside. thus, if a cl- channel opens when the membrane is more negative than the equilibrium potential, this tends to reduce the electrical force, thereby stopping the influx of positively charged k+ and na+ ions. these channels, then, are usually closed until the membrane potential reaches a value close to the equilibrium potential. the opening of the ion channels in the plasma membrane gives rise to the inward and outward currents that are recorded by patch-clamp. the outward current arises from the influx of na+ and k+ ions through the na+ and k+ channels, respectively. the inward current can be thought of as the equivalent of a battery, and the current through the membrane is the equivalent of the current in a battery. if the na+ and k+ channels were to remain open for an extended period, the current flowing through the membrane would eventually reach a steady state (figure 11-32). this current, however, is still very small and is usually not recorded; it can be detected by means of fast-response, high-gain recording equipment, such as that used to record the action potentials of nerve cells. the inward current is due to the flow of positively charged k+ ions through the channels, whereas the outward current is due to the movement of na+ ions out of the cell through the channels. thus, the inward and outward currents are often known as the transient and sustained currents, respectively. the membrane potential is the sum of the inward and outward currents, and it is the main electrical force underlying all cell function. it also determines the permeability of the membrane to specific ions and, therefore, the extent of ion exchange between the cell and its environment.

the available evidence suggests that na + channels inactivate spontaneously. however, it is more difficult to understand why a transient voltage-gated k+ channel should be able to remain open in the time interval between the opening and inactivation of a na + channel. to explain this, one must realize that voltage-gated k+ channels are made of four protein subunits. two of these subunits, alpha and beta, make up the conductance -gate. when this gate is closed, both na + channels and k + channels are effectively blocked. the other two subunits, alpha and delta, make up the sodium-channel-repeats -domain. the voltage-sensing domain of this fourth subunit is continuous with the voltage-sensing domain of the na + channel, so that opening the gate of the k + channel causes the voltage-sensing domain of the protein to enter the membrane as well. thus, there is a direct overlap between the electrical influence of a voltage applied to the membrane and the electric influence on a given k + channel. this suggests that a single k+ channel drains both the na + and the k + channels in a nerve axon terminal, and that the single opening of the gate of one channel causes the activation of the other channel. in fact, inactivation of the k+ channel tends to make the na + channel open further. therefore, the na + channel will stay open for a shorter time during the inactivation of the k + channel. the other channel type that can play a key role in the shaping of the action potential is the ca2+ -activated k + channel. this channel also has a short delay before the onset of its voltage sensitivity. this is partly because of the inactivation of the channels by the binding of calmodulin, which is structurally related to the na+ channel inactivation gate, to the membrane protein, and partly because calcium binding to these channels results in a displacement of the voltage dependence of the channels, so that the threshold for activation occurs at a less negative voltage.
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