



Functional Properties of HCN Channels Based on Multifaceted Features

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DESCRIPTION

Cation channels that open when the membrane potential is hyperpolarized are known as Hyperpolarization-activated and Cyclic Nucleotide-gated (HCN) channels. Noma and Irisawa first discovered these channels in the heart in 1976, and Brown, Difrancesco, and Weiss and his colleague went on to characterize them. HCN channels have a general structure that is similar to voltage-gated K⁺ channels. The conventional GYG sequence in the pore-forming region, the positively charged S4 section, and four subunits with six transmembrane segments make up HCN channels. HCN channels' K⁺ permeability, however, is permeable to Na⁺ and is less selective than that of normal K⁺ channels. HCN channels typically have a current reversal potential of roughly 30 mV. When it concerns to voltage-dependent gating, it has been seen that the S4 segment moves inward in response to hyperpolarization, but it is still unclear how the channel opens at the molecular level. For instance, extracellular Cl⁻ and extracellular K⁺ are necessary for the opening of HCN channels. Certain HCN channel isoforms have a cAMP-binding site that is located close to the C terminus, and cAMP alters the voltage dependency of activation in these channels. Phosphatidylinositol 4,5-bisphosphate (PIP₂), commonly known as an HCN channel modulator, alters the voltage dependence in a manner distinct from cAMP. HCN channels may perform the several jobs listed below because to these multifarious capabilities. In this study, we employ straightforward numerical simulations to attempt to comprehend the physiological implications of these functions.

The expression of HCN channels in heart, brain, taste buds, and pancreatic cells was previously shown in the literature. The electrophysiological role of HCN channels in the central nervous system is the main topic of this research. Whereas HCN2 is present almost everywhere in the brain, HCN1 isoform is primarily expressed in the cerebral cortex, hippocampus, and superior colliculus. HCN3 and HCN4 are expressed in specific areas of the brain. HCN channels are located in the dendrites, somas, and axon terminals of the neuron. There have been reports of expression in the axon terminals of Held and globus pallidus neurons. According to earlier studies, certain neurons

have unequal cell surface expression of HCN channels. In thalamic reticular neurons, where they are distributed almost equally, HCN channels are expressed more strongly in distal dendrites than in somas. A recent publication also mentioned isoform-specific localisation. For instance, developed rats' layer 5 pyramidal neurons of the HCN1 subtype express it abundantly in the dendrites, whereas the HCN2 subtype expresses it mostly in the soma. Epilepsy-like abnormal neuronal activity and environmental cues have an impact on the expression and kinetics of HCN channels. In several epilepsy models, there is a decrease in HCN channel expression.

Excitatory function

HCN channel currents depolarize and regulate the membrane potential close to the resting membrane potential because their typical current reversal potential is around 30 mV, which is higher than the usual threshold for the formation of action potential. A simulated neuron exhibits a hyperpolarized membrane potential when the conductance of HCN channels is blocked. In the superior colliculus of rats, for instance, blockage of HCN channels causes hyperpolarization. The EPSP amplitude is reduced despite HCN channel conductance. By depolarizing the membrane potential, this inhibitory impact is concealed. Furthermore, because inhibitory synaptic inputs hyperpolarizing conductance activates HCN channels to elicit depolarizing conductance, HCN channels act to reduce the impact of inhibitory synaptic inputs.

Inhibitory function

The membrane resistance decreases when the HCN channel conductance is activated. The influence of synaptic inputs on membrane potential is suppressed by this reduction. Postsynaptic potentials' amplitude and dynamics decrease and shorten as HCN channels become more active.

Modulatory function

The cortical and hippocampus neurons' synaptic inputs are normalized by dendritic HCN channels. In neocortical and hippocampal pyramidal neurons, the current density of the HCN

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channels increases with distance from the soma. Such dendritic HCN channels reduce the location dependence of synaptic inputs in hippocampal and neocortical pyramidal neurons and scale the EPSP amplitude measured at the soma. Computer simulation can replicate the effect using two models with gradient

and even HCN channel distributions. It's interesting to notice that both models exhibit identical current amplitudes and overlapping voltage dependence of HCN channels in this circumstance despite having differing HCN channel distributions.