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Gap junction channels and hemichannels in the CNS: regulation by signaling molecules.

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Abstract

Coordinated interaction among cells is critical to develop the extremely complex and dynamic tasks performed by the central nervous system (CNS). Cell synchronization is in part mediated by connexins and pannexins; two different protein families that form gap junction channels and hemichannels. Whereas gap junction channels connect the cytoplasm of contacting cells and coordinate electric and metabolic activities, hemichannels communicate intra- and extra-cellular compartments and serve as diffusional pathways for ions and small molecules. Cells in the CNS depend on paracrine/autocrine communication via several extracellular signaling molecules, such as, cytokines, growth factors, transmitters and free radical species to sense changes in microenvironment as well as to adapt to them. These signaling molecules modulate crucial processes of the CNS, including, cellular migration and differentiation, synaptic transmission and plasticity, glial activation, cell viability and microvascular blood flow. Gap junction channels and hemichannels are affected by different signaling transduction pathways triggered by these paracrine/autocrine signaling molecules. Most of the modulatory effects induced by these signaling molecules are specific to the cell type and the connexin and pannexin subtype expressed in different brain areas. In this review, we summarized and discussed most of the relevant and recently published information on the effects of signaling molecules on connexin or pannexin based channels and their possible relevance in CNS physiology and pathology. This article is part of the Special Issue Section entitled 'Current Pharmacology of Gap Junction Channels and Hemichannels'.