Modulation of gap junction channels and hemichannels by growth factors

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Abstract

Gap junction hemichannels and cell–cell channels have roles in coordinating numerous cellular processes, due to their permeability to extra and intracellular signaling molecules. Another mechanism of cellular coordination is provided by a vast array of growth factors that interact with relatively selective cell membrane receptors. These receptors can affect cellular transduction pathways, including alteration of intracellular concentration of free Ca^{2+} and free radicals and activation of protein kinases or phosphatases. Connexin and pannexin based channels constitute recently described targets of growth factor signal transduction pathways, but little is known regarding the effects of growth factor signaling on pannexin based channels. The effects of growth factors on these two channel types seem to depend on the cell type, cell stage and connexin and pannexin isoform expressed. The functional state of hemichannels and gap junction channels are affected in opposite directions by FGF-1 *via*protein kinase-dependent mechanisms. These changes are largely explained by channels insertion in or withdrawal from the cell membrane, but changes in open probability might also occur due to changes in phosphorylation and redox state of channel subunits. The functional consequence of variation in cell–cell communication*via* these membrane channels is implicated in disease as well as normal cellular responses.