# Carbon monoxide: A new player in the redox regulation of connexin hemichannels

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Carbon monoxide (CO) is a gaseous transmitter that is known to be involved in several physiological processes, but surprisingly it is also becoming a promising molecule to treat several pathologies including stroke and cancer. CO can cross the plasma membrane and activate guanylate cyclase, increasing the cGMP concentration and activating some kinases, including PKG. The other mechanism of action involves induction of protein carbonylation. CO is known to directly and indirectly modulate the function of ion channels at the plasma membrane, which in turn have important repercussions in the cellular behavior. One group of these channels is hemichannels, which are formed by proteins known as connexins (Cxs). Hemichannel allows not only the flow of ions through their pore but also the release of molecules such as ATP and glutamate. Therefore, their modulation not only impacts cellular function but also cellular communication, having the capability to affect tissular behavior. Here, we review the most recent results regarding the effect of CO on Cx hemichannels and their possible repercussions on pathologies. 

connexins

gap junction channels

gaseous transmitters

hemichannels

post-translational modification

redox potential

carbon monoxide

gap junction protein

ion channel

nitric oxide

brain ischemia

cell membrane

chemistry

gap junction

human

metabolism

oxidation reduction reaction

Brain Ischemia

Carbon Monoxide

Cell Membrane

Connexins

Gap Junctions

Humans

Ion Channels

Nitric Oxide

Oxidation-Reduction