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Metabolic modulation of carbon monoxide toxicity

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Abstract

Carbon monoxide (CO) gas is a product of the incomplete combustion of carbon-based fuels and substances. From a public health perspective, CO poisoning may be the cause of more than 50% of fatal poisonings in many industrial countries. The adverse effects of CO poisoning may be more widespread because of unreported situations and delayed neurologic effects, which may be linked to CO exposure. Chronic CO effects that are subtle, such as the adverse effects on vascular diseases, may increase the number of people at risk. The apparent role of CO as an important mediator of cell signaling is a paradox and may represent an example of hormesis, i.e. beneficial effects at low concentration but adverse effects at higher concentrations. Nevertheless, because CO can form ligands with iron (heme) and copper sites, the potential for metabolic intervention is likely. Furthermore, CO-induced oxidative stress opens the opportunity for modulating the adverse effects of CO with antioxidants (both water- and lipid-soluble compounds) and various factors involved with reducing oxidative stress. However, consideration must be given to the micro-environment in some situations that could potentially create more oxidation and subsequent metabolic damage if the combinations and concentrations of antioxidants are not correct, i.e. pro oxidant effects. Likewise, it is important that we take precautions in the development of antioxidant adjuvants to use with oxygen therapies in CO poisoning.

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