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Redox signaling in the heart and brain: the roles of nitric oxide and reactive oxygen species in disease and therapy

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Abstract

Nitric oxide (NO) and reactive oxygen species (ROS) are central to the pathophysiology of cardiovascular and neurological disorders, influencing intricate signaling pathways that manage vascular function, inflammation, and oxidative stress. NO, predominantly produced by NO-synthases, plays a vital role in maintaining vascular health by facilitating vasodilation and preventing platelet aggregation. However, its reaction with superoxide results in the formation of peroxynitrite, a highly reactive molecule that intensifies oxidative damage and impairs endothelial function. Elevated ROS levels, arising from sources like NADPH oxidases and mitochondrial activity, further heighten oxidative stress, driving the progression of conditions like atherosclerosis and neurodegenerative diseases. Therapeutic strategies aimed at restoring the balance between NO and ROS include the use of antioxidants to neutralize ROS, pharmacological methods to enhance NO bioavailability, and nanoparticle-based systems designed to address oxidative stress. Emerging research points to potential of targeting redox-sensitive pathways, such as the Keapl-NrfT axis, to slow disease progression. In neurological disorders, overproduction of ROS leads to neuroinflammation and neuronal apoptosis, which are central to conditions like Alzheimer's and Parkinson's disease. This review explores the complex relationship between NO and ROS in disease mechanisms, emphasizing cutting-edge therapeutic strategies that utilize redox signaling in cardiovascular and neurological conditions.

Keywords: Cardiovascular diseases; Hypoxic; Neurological diseases; Nitric oxide (NO); Oxidative stress; ROS pathways.

Conflict of interest statement