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Oxidative stress and pathophysiology of ischemic stroke: novel therapeutic opportunities.

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Abstract

Stroke is the second leading cause of death, after ischemic heart disease, and accounts for 9% of deaths worldwide. According to the World Health Organization [WHO], 15 million people suffer stroke worldwide each year. Of these, more than 6 million die and another 5 million are permanently disabled. Reactive oxygen species [ROS] have been implicated in brain injury after ischemic stroke. There is evidence that a rapid increase in the production of ROS immediately after acute ischemic stroke rapidly overwhelm antioxidant defences, causing further tissue damage. These ROS can damage cellular macromolecules leading to autophagy, apoptosis, and necrosis. Moreover, the rapid restoration of blood flow increases the level of tissue oxygenation and accountsfor a second burst of ROS generation, which leads to reperfusion injury. Current measures to protect the brain against severe stroke damage are insufficient. Thus, it is critical to investigate antioxidant strategies that lead to the diminution of oxidative injury. The antioxidant vitamins C and E, the polyphenol resveratrol, the xanthine oxidase [XO] inhibitor allopurinol, and other antioxidant strategies have been reviewed in the setting of strokes. This review focuses on the mechanisms involved in ROS generation, the role of oxidative stress in the pathogenesis of ischemic stroke, and the novel therapeutic strategies to be tested to reduce the cerebral damage related to both ischemia and reperfusion.

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