

The Role of Oxidative Stress in Traumatic Brain Injury Recovery

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Abstract

Globally, traumatic brain injury (TBI) is a leading cause of long-term neurological disability. After the immediate mechanical injury, secondary processes, particularly oxidative stress, lead to chronic neuronal injury, impaired neurogenesis, and poor recovery outcomes. Oxidative stress results of a disruption in the balance between reactive oxygen and nitrogen species. Oxidative stress causes lipid peroxidation, protein modification, neuroinflammation, and DNA damage. Both clinical and preclinical data implicate oxidative stress as a central mediator of poor TBI prognosis. This has made oxidative stress an emerging target for anti-TBI therapy. This review critically examines the oxidative stress mechanisms in TBI and evaluates the current evidence of safety and efficacy of antioxidant treatment for TBI. Common antioxidant therapies include direct scavengers of free radicals such as vitamins C and E, mitochondria-targeted antioxidants such as MitoQ, and endogenous antioxidant boosters such as N-Acetyl Cysteine (NAC). Some antioxidant therapies have shown some protective effect on TBI prognosis in animal models, but translation to clinical benefit in humans is still debatable. Some antioxidants have shown improvements in the survival of neurons and functional recovery based in *in-vivo* and *in-vitro* laboratory preclinical studies. Most antioxidant therapies have not undergone clinical testing in humans, with the exception of NAC, which has shown promising results. One randomized control has shown that, if given early, NAC results in a reduction of oxidative stress, a shorter ICU stay and higher scores on the Glasgow Coma Score. More, larger, randomized controlled trials are needed. Future directions include treatment, developing better biomarkers, and conducting large-scale clinical trials to identify effective treatment methods. Future studies should concentrate on specific therapy based on each patient's unique metabolic and genetic factors, as well as combination medicines that target multiple areas of secondary damage.