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## Oxidative/nitrosative stress and antidepressants: targets for novel antidepressants.

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#### Abstract

The brain is an organ predisposed to oxidative/nitrosative stress. This is especially true in the case of aging as well as several neurodegenerative diseases. Under such circumstances, a decline in the normal antioxidant defense mechanisms leads to an increase in the vulnerability of the brain to the deleterious effects of oxidative damage. Highly reactive oxygen/nitrogen species damage lipids, proteins, and mitochondrial and neuronal genes. Unless antioxidant defenses react appropriately to damage inflicted by radicals, neurons may experience microalteration, microdysfunction, and degeneration. We reviewed how oxidative and nitrosative stresses contribute to the pathogenesis of depressive disorders and reviewed the clinical implications of various antioxidants as future targets for antidepressant treatment.

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**KEYWORDS:** 2,3-dioxigenase; 5-HT; 5-hydroxytryptamine; ARE; ATP; Antidepressant; BDNF; CAT; COX; CREB; DNA; Depression; EFE; ER; FA; FST; GSK3; HPA; IDO; INF- $\gamma$ ; N-acetylcysteine; NAC; NAD; NO; NO synthase; NOS; Novel target; Nrf2; Oxidative/nitrosative stress; RNS; ROS; SOD; TST; adenosine triphosphate; antioxidant response element; brain-derived neurotrophic factor; cAMP-response element-binding-protein (CREB); catalase; cyclooxygenase; deoxyribonucleic acid; endoplasmic reticulum; ethyl ferulate (ethyl 4-hydroxy-3-methoxycinnamate); ferulic acid (4-hydroxy-3-methoxycinnamic acid); forced swimming test; glycogen synthase kinase 3; hypothalamic–pituitary–adrenal axis; interferon-gamma; nicotinamide adenine dinucleotide; nitric oxide; nuclear factor E2-related factor 2; reactive nitrogen species; reactive oxygen species; superoxide dismutase; tail-suspension test

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