International Journal of Pharmaceutical Research and Applications
Volume 10, Issue 6 Nov - Dec 2025, pp: 359-364 www.ijprajournal.com ISSN: 2456-4494

Impact of Oxidative Stress Modulation and Antioxidants on Mental Health

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Date of Submission: 10-11-2025 Date of Acceptance: 20-11-2025

ABSTRACT
Mental health disorders such as depression, anxiety, schizophrenia, and bipolar disorder are major contributors to global disability and socioeconomic burden. Although substantial research has improved understanding of their etiology, the molecular mechanisms underlying these conditions remain only partially defined. Emerging evidence identifies oxidative stress—an imbalance between reactive oxygen species production and antioxidant defense capacity—as a key factor in the development and progression of neuropsychiatric disorders. Disrupted redox homeostasis contributes to neuronal dysfunction, neuroinflammation, and apoptosis, thereby influencing disease onset and clinical outcomes.
Antioxidant systems, both enzymatic and non-enzymatic, play essential roles in maintaining neural integrity, and therapeutic strategies aimed at restoring oxidative balance have shown promise in alleviating psychiatric symptoms. In parallel, recent advances in bioengineering have positioned exosome-based therapies as novel acellular approaches for neuroprotection and regeneration. Bioengineered exosomes enriched with microRNAs, growth factors, and proteins can modulate oxidative and inflammatory pathways, promote angiogenesis, and enhance neuronal recovery following ischemic injury.
Together, these findings underscore oxidative stress as a central mechanism linking cellular dysfunction and mental illness, while exosome-based therapeutics represent a promising frontier for targeted neurorestorative interventions in neuropsychiatric and neurodegenerative disorders.
Key words: mental health, antioxidants, oxidative stress, neuron, biomarkers, ischemia, redox

INTRODUCTION

Mental health disorders—includingdepression, anxiety, schizophrenia, and bipolar disorder—are recognized as major contributors to global disability and impose a substantial socioeconomic burden (1). Despite advances in research, the precise mechanisms underlying these conditions remain only partially understood, creating the need to investigate additional biochemical processes that may shape their pathophysiology (2). Among these processes, oxidative stress—defined as an imbalance between the production of reactive oxygen species (ROS) and the capacity of antioxidant defence systems (3)—has emerged as a critical factor in the onset and progression of neuropsychiatric disorders.

Antioxidant systems, both enzymatic (4), such as superoxide dismutase, catalase, and glutathione peroxidase, and non-enzymatic, including glutathione, vitamin C, and vitamin E, play essential roles in preserving neural integrity and sustaining redox balance (5). Evidence from experimental and clinical research increasingly supports the therapeutic potential of antioxidant supplementation and modulation of oxidative stress (6), offering avenues for alleviating psychiatric symptoms and improving overall mental health outcomes. This review therefore seeks to critically examine the interplay between oxidative stress, antioxidant defences, and psychiatric illness, with emphasis on mechanistic insights (7), clinical findings, and emerging directions for pharmacological interventions (8).

The mechanisms underlying oxidative stress in the brain reflect a complex interaction of biochemical and cellular factors that make neurons particularly vulnerable to injury from ROS and reactive nitrogen species (RNS) (9). The brain's high oxygen consumption, enrichment with polyunsaturated fatty acids, and relatively weak antioxidant defences increase its sensitivity to oxidative insults (10). Under physiological conditions, ROS and RNS act as signalling molecules; however, when their production exceeds antioxidant capacity, they induce oxidative modifications of lipids, proteins, and nucleic acids (11). These alterations impair cellular function, trigger neuronal damage, and ultimately promote cell death (12).

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Specific neuronal populations, such as those in the hippocampal CA1 region and cerebellar granule cell layer (13), exhibit selective vulnerability, partly due to mitochondrial dysfunction, impaired ATP generation, inadequate DNA repair, calcium dysregulation (14), and excitotoxicity mediated by glutamate. Neuroinflammation can further amplify oxidative stress (15), creating a self-perpetuating cycle of cellular injury (16). Such damage disrupts synaptic plasticity, neurotransmitter release, and genomic stability (17), with downstream consequences for cognition and behaviour. These processes contribute not only to neurodegenerative conditions but also to psychiatric disorders (18), underscoring the central role of oxidative stress in brain health.

Oxidative stress is now recognized as a key component in the pathophysiology of psychiatric disorders (19), including schizophrenia, bipolar disorder, major depressive disorder (20), and anxiety disorders. Elevated markers of oxidative injury, such as lipid peroxidation products (21) like malondialdehyde and oxidized DNA, are consistently reported in these conditions. In schizophrenia and bipolar disorder, glutathione depletion and increased oxidative burden contribute to neuronal dysfunction and symptom severity. Depression has been associated with disturbances in redox balance (22), including ω-3 fatty acid depletion and elevated oxidative biomarkers. Anxiety disorders also show evidence of increased oxidative stress, with lipid peroxidation observed in disorders such as obsessive–compulsive disorder and panic disorder. The brain's intrinsic susceptibility—due to high oxygen use, lipid-rich composition, and limited antioxidant defenses (16)—helps explain this vulnerability. Redox imbalance interferes with neurotransmission, neuroinflammation, and cellular signalling, thereby shaping the clinical features (23) and course of mental illness (24). Interestingly, certain psychiatric medications also exhibit antioxidant properties, highlighting the therapeutic relevance of targeting oxidative stress.

The connection between psychological stress and cellular redox imbalance (25) provides further insight into the pathophysiology of mental health conditions. Stress activates the hypothalamic–pituitary–adrenal (HPA) axis (26) and the sympathetic nervous system, elevating cortisol and other stress mediators. These responses stimulate excess ROS production (27), tipping the balance toward oxidative stress. Resulting oxidative damage to lipids (28), proteins, and DNA undermines neuronal function and survival.

Psychological stress also weakens endogenous antioxidant defences (29), including enzymes such as superoxide dismutase, catalase, and glutathione peroxidase, further diminishing the brain's ability to neutralize ROS. This imbalance worsens oxidative damage in brain regions (30) governing emotion and cognition. Clinical studies consistently report heightened biomarkers of oxidative stress, including malondialdehyde and 8-oxo-2′-deoxyguanosine (25,29), in individuals exposed to chronic psychosocial stress.In addition, stress-related behaviours—poor nutrition, disrupted sleep, and substance use—exacerbate oxidative load (31). This bidirectional relationship between stress and oxidative imbalance (32) sustains neuronal injury and contributes to psychiatric symptoms, making oxidative stress modulation (33) a potential therapeutic target.

Therapeutic strategies aimed at mitigating oxidative stress show considerable promise in psychiatry. By preserving neuronal integrity and function (34), antioxidants can normalize biomarkers, reduce neuroinflammation, and restore neurotransmission (35), thereby improving outcomes in schizophrenia, bipolar disorder, major depression, anxiety, and obsessive–compulsive disorder. Conventional antipsychotics and antidepressants already exhibit partial antioxidant effects, while adjunctive agents such as N-acetylcysteine (NAC) (22,36) have demonstrated additional clinical benefits.

Supplementation with vitamins C and E, glutathione precursors (33), flavonoids, and polyunsaturated fatty acids has been associated with improvements in psychiatric symptoms and reversal of oxidative damage. Randomized controlled trials with NAC, for example, show reductions in depressive symptoms and functional gains in bipolar disorder (37). Similarly, the use of antioxidant vitamins (38) alongside psychotropic drugs correlates with improved anxiety and depression outcomes. Nonetheless, challenges persist regarding optimal dosing (39), long-term safety, and patient variability. Excessive supplementation may even disrupt physiological redox signalling (25,28,40). Rigorous trials are therefore essential to refine dosing strategies and ensure therapeutic efficacy.

Oxidative stress biomarkers are emerging as valuable tools for diagnosis and prognosis in psychiatry. In conditions such as schizophrenia, depression, bipolar disorder, and anxiety disorders (41), biomarker levels—including malondialdehyde and advanced oxidation protein products—correlate with symptom severity (42,43) and cognitive impairment (44). These markers can support biochemical profiling, guide personalized interventions (45), and monitor treatment responses. The antioxidant actions of psychotropic drugs (46) reinforce the therapeutic importance of oxidative modulation. Integrating oxidative biomarkers into psychiatric practice (47) could enhance diagnosis, improve monitoring, and potentially slow disease progression.

Despite this progress, key questions remain unresolved. It is still unclear whether oxidative stress is a cause or a consequence of psychiatric illness (48,49). Determining optimal antioxidant interventions, dosing regimens, and long-term benefits requires robust evidence from large, controlled clinical trials (36,50). Individual differences in genetics, lifestyle, and comorbidities (51) further complicate treatment. Future research should prioritize longitudinal studies, molecular investigations (19,52–54), and the development of precise biomarkers for specific oxidative pathways (55,56). Improved experimental models and translational approaches are critical to bridge laboratory discoveries with clinical applications.

Oxidative stress modulation represents a promising frontier in psychiatric research, offering new mechanistic insights into mental illness (57). Incorporating oxidative biomarkers into diagnosis and personalized treatment (58,59) and advancing antioxidant therapies as adjuncts (60) to existing medications could reshape psychiatric care. However, clinical validation and mechanistic clarification remain essential (61). Progress will depend on sustained interdisciplinary collaboration spanning molecular biology (62), clinical trials, and population studies to develop effective preventive and therapeutic innovations targeting oxidative stress in mental health.

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