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Publications 2026

OPEN ACCESS
ONLINE ISSN: 2992-5479
PRINT ISSN: 2992-605X

NEWPORT INTERNATIONAL JOURNAL OF PUBLIC HEALTH AND PHARMACY (NIJPP)

Volume 7 Issue 1 Page 117-122, 2026

https://doi.org/10.59298/NIJPP/2026/71117122

Neurotoxicity Associated with Systemic Inflammatory Disorders: Insights from Malaria, Diabetes, and Arthritis Models

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ABSTRACT

Systemic inflammatory disorders such as malaria, diabetes, and arthritis have long been recognized for their multi-organ effects, yet their impact on the central nervous system (CNS) remains an expanding area of research. Chronic inflammation and oxidative stress constitute the main pathological link between these systemic diseases and neurodegeneration. In malaria, inflammatory cytokine surges and oxidative injury disrupt the blood–brain barrier and trigger neuronal apoptosis. Diabetes-induced neurotoxicity arises from hyperglycemia-mediated oxidative stress, mitochondrial dysfunction, and microvascular impairment. Similarly, in arthritis, persistent systemic inflammation and oxidative stress alter neuronal metabolism and promote neuroinflammation. This review explores the molecular and cellular mechanisms underlying neurotoxicity in systemic inflammatory disorders, emphasizing the roles of reactive oxygen species (ROS), cytokine signaling, and microglial activation. It also discusses potential therapeutic interventions targeting redox and inflammatory pathways, including antioxidant therapy, cytokine modulation, and neuroprotective phytochemicals. Understanding the interplay between systemic inflammation and neural injury offers valuable insights into developing strategies for mitigating CNS complications in metabolic and infectious diseases.

Keywords: Neurotoxicity, oxidative stress, inflammation, malaria, diabetes, arthritis

INTRODUCTION

Neurotoxicity, defined as the adverse effect of chemical, biological, or metabolic insults on nervous tissue, is increasingly recognized as a consequence of systemic inflammatory disorders [1]. Traditionally, neurotoxicity has been linked to exposure to toxins or drugs, but recent research reveals that chronic inflammation in systemic diseases such as malaria, diabetes, and arthritis can induce comparable neuronal damage [2]. The mechanisms involve complex interactions between oxidative stress, cytokine signaling, mitochondrial dysfunction, and endothelial injury that ultimately compromise neuronal survival and cognitive function [3]. The central nervous system (CNS) is particularly vulnerable to oxidative damage due to its high oxygen consumption, abundant polyunsaturated fatty acids, and relatively limited antioxidant defenses [3]. When systemic inflammation persists, pro-inflammatory mediators penetrate or affect the blood–brain barrier (BBB), leading to microglial activation and neuroinflammation [4]. This state results in excessive ROS generation, neuronal apoptosis, and synaptic dysfunction, contributing to cognitive and behavioral impairments. This review synthesizes current evidence on the molecular mechanisms of neurotoxicity associated with systemic inflammatory disorders, using malaria, diabetes, and arthritis as representative models. It further explores shared redox–inflammatory pathways and discusses therapeutic strategies that can attenuate neurotoxic outcomes.

2. Oxidative Stress and Neuroinflammation: Mechanistic Overview

Oxidative stress arises when ROS generation exceeds the antioxidant defense capacity of cells. ROS such as superoxide anion, hydroxyl radical, and hydrogen peroxide can damage lipids, proteins, and nucleic acids, thereby

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impairing neuronal integrity [5]. The brain's limited regenerative capacity makes it highly susceptible to oxidative injury.

Inflammation further amplifies oxidative stress through cytokine-induced activation of NADPH oxidase, inducible nitric oxide synthase (iNOS), and mitochondrial dysfunction. Cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6) activate microglia and astrocytes, promoting neuroinflammatory cascades [6]. Activation of nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) and mitogen-activated protein kinases (MAPKs) enhances pro-inflammatory gene expression, while inhibition of the antioxidant transcription factor Nrf2 weakens cellular defenses [7]. Chronic oxidative and inflammatory stress leads to mitochondrial DNA damage, impaired ATP synthesis, and neuronal apoptosis [8]. These processes are central to the pathophysiology of neurodegenerative outcomes observed in systemic inflammatory diseases.

3. Neurotoxicity in Malaria

3.1 Pathophysiological Overview

Malaria, caused by Plasmodium species, remains a global health challenge with severe neurological manifestations, particularly in cerebral malaria [9]. Neurotoxicity in malaria arises from parasite sequestration in cerebral microvessels, immune activation, and oxidative stress [10]. During infection, the immune system releases high levels of cytokines such as TNF-α, interferon-gamma (IFN-γ), and IL-1β to combat parasitemia [11]. While this immune response is protective, excessive cytokine release disrupts the BBB, leading to cerebral edema and neuronal injury [12]. The oxidation of hemoglobin during parasite metabolism also generates ROS and free heme, which catalyze lipid peroxidation and damage neuronal membranes [13].

3.2 Role of Oxidative Stress and Inflammation

Studies show elevated markers of oxidative stress, including malondialdehyde (MDA) and nitric oxide (NO), in cerebral malaria [14]. These reactive species trigger neuronal apoptosis and synaptic dysfunction. Moreover, activated microglia and astrocytes produce pro-inflammatory mediators that exacerbate oxidative damage [15]. NF-κB activation in brain endothelial cells induces the expression of adhesion molecules, promoting sequestration of parasitized erythrocytes and leukocytes [16]. This leads to reduced cerebral perfusion and ischemia, further aggravating oxidative stress. Mitochondrial impairment and calcium dysregulation within neurons contribute to excitotoxicity and cell death.

3.3 Cognitive and Neurological Implications

Survivors of cerebral malaria often experience long-term cognitive deficits, learning disabilities, and behavioral impairments [17]. Experimental models reveal that oxidative damage to the hippocampus and cortex correlates with memory and attention deficits [18]. Thus, oxidative stress not only contributes to acute neurotoxicity but also to chronic neurocognitive sequelae.

4. Diabetes-Induced Neurotoxicity

4.1 Hyperglycemia and Oxidative Mechanisms

Diabetes mellitus, a chronic metabolic disorder characterized by hyperglycemia, is a well-documented cause of peripheral and central nervous system complications [19]. Prolonged hyperglycemia increases mitochondrial ROS generation, activates the polyol and hexosamine pathways, and promotes advanced glycation end-product (AGE) formation [20]. The interaction of AGEs with their receptor (RAGE) triggers oxidative stress and inflammatory responses in neural tissue [21]. Oxidative stress disrupts neuronal glucose metabolism, alters calcium homeostasis, and damages synaptic proteins essential for neurotransmission [22]. Elevated ROS levels also reduce neurotrophic factor availability, impairing neuronal survival and regeneration [22].

4.2 Neuroinflammation and Vascular Dysfunction

Inflammatory mediators such as TNF-α, IL-6, and monocyte chemoattractant protein-1 (MCP-1) are elevated in diabetic patients, indicating persistent neuroinflammation [23]. Endothelial dysfunction, induced by oxidative stress, compromises the BBB and permits infiltration of inflammatory cells into the CNS [24]. Activated microglia release additional ROS and nitric oxide, forming peroxynitrite radicals that further damage neurons [25].

4.3 Clinical Manifestations and Cognitive Decline

Diabetes-associated neurotoxicity manifests as diabetic neuropathy, impaired cognition, and increased risk of Alzheimer-like neurodegeneration [26]. Structural studies reveal brain atrophy, reduced synaptic density, and mitochondrial swelling in diabetic models. The link between insulin resistance and neuronal dysfunction, sometimes referred to as "type 3 diabetes," underscores the integral role of metabolic and redox disturbances in neurodegeneration [27].

4.4 Mitochondrial and Nrf2 Pathway Dysregulation

Mitochondrial impairment is a key factor in diabetes-related neuronal injury. Excess ROS damages mitochondrial DNA and inhibits ATP generation, leading to energy deficits [28]. Simultaneously, downregulation of Nrf2

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PRINT ISSN: 2992-605X
weakens antioxidant defenses, allowing oxidative and inflammatory cascades to progress unchecked [29]. Restoring Nrf2 activity through pharmacological agents or natural compounds has shown promise in mitigating diabetic neurotoxicity.

5. Neurotoxic Mechanisms in Arthritis

5.1 Systemic Inflammation and CNS Crosstalk

Arthritis, including rheumatoid arthritis (RA) and osteoarthritis (OA), is characterized by chronic systemic inflammation that extends beyond the joints. Elevated pro-inflammatory cytokines and oxidative stress markers in circulation have neurotoxic effects [30]. These systemic factors cross or influence the BBB, promoting microglial activation and neuroinflammation. Patients with chronic arthritis often experience fatigue, depression, and cognitive impairment, reflecting neuroinflammatory involvement. Experimental models indicate that inflammatory mediators such as TNF-α, IL-1β, and IL-17 alter neurotransmitter balance and promote oxidative neuronal injury [31].

5.2 Oxidative Stress and Microglial Activation

Excessive ROS in arthritis originates from activated immune cells and mitochondrial dysfunction. ROS stimulate NF-κB activation, leading to further cytokine release and amplification of neuroinflammation [32]. In parallel, oxidative stress impairs neuronal ion channels and synaptic plasticity, contributing to pain sensitization and cognitive deficits [33].

5.3 Neurodegenerative Consequences

Chronic inflammation in arthritis increases the risk of neurodegenerative disorders such as Alzheimer's and Parkinson's disease [34]. Amyloid beta aggregation and tau phosphorylation have been linked to systemic cytokine exposure and oxidative injury [35]. Thus, prolonged inflammation and oxidative stress in arthritis can accelerate neurodegenerative processes even in the absence of direct CNS infection or trauma.

6. Therapeutic Approaches and Neuroprotective Strategies

6.1 Antioxidant Therapy

Antioxidants form the cornerstone of neuroprotective strategies in oxidative stress–mediated conditions. Vitamins C and E, alpha-lipoic acid, and coenzyme Q10 scavenge free radicals, stabilize membranes, and restore redox balance [36]. Clinical studies indicate that antioxidant supplementation reduces oxidative biomarkers and improves cognitive function in diabetic and arthritic patients [37].

6.2 Nrf2 Activation and Redox Regulation

Pharmacological activation of the Nrf2 pathway enhances endogenous antioxidant capacity. Compounds such as sulforaphane, dimethyl fumarate, and curcumin induce Nrf2 translocation to the nucleus, upregulating detoxifying enzymes like heme oxygenase-1 and glutathione peroxidase [38]. Experimental evidence shows that Nrf2 activation protects against neuronal apoptosis in malaria, diabetes, and arthritis models [39].

6.3 Cytokine Modulation and Anti-Inflammatory Therapy

Targeting inflammatory cytokines has shown promising results in limiting neurotoxicity. Inhibitors of TNF-α and IL-6, as well as corticosteroids and selective cytokine modulators, reduce systemic and neural inflammation [40]. However, their use requires careful balance to avoid immunosuppression.

6.4 Mitochondrial Protection

Mitochondria-targeted antioxidants such as MitoQ and SS-31 peptides restore mitochondrial function, reduce ROS generation, and maintain membrane integrity [41]. Enhancing mitochondrial biogenesis through PGC-1α activation improves neuronal resilience.

6.5 Phytochemicals and Nutraceuticals

Natural compounds like resveratrol, quercetin, and catechins exhibit dual antioxidant and anti-inflammatory properties. These phytochemicals modulate signaling pathways including AMPK, PI3K/Akt, and JAK/STAT, contributing to neuronal survival and reduced oxidative stress [42]. Herbal extracts such as ginkgo biloba and green tea polyphenols have demonstrated neuroprotective effects in preclinical models of diabetes and arthritis [43].

6.6 Emerging Therapeutic Perspectives

Recent approaches explore combined therapies that target both systemic inflammation and neural oxidative stress. Nanocarrier-based delivery systems enhance the bioavailability of antioxidants and anti-inflammatory agents [44]. Gene therapy targeting Nrf2 or mitochondrial repair mechanisms also holds potential for future intervention.

CONCLUSION

Neurotoxicity in systemic inflammatory disorders such as malaria, diabetes, and arthritis arises from a complex interplay between oxidative stress, inflammation, and metabolic dysregulation. Excessive ROS generation, mitochondrial damage, and cytokine-mediated signaling collectively compromise neuronal integrity and cognitive function. Despite their different etiologies, these diseases share common redox–inflammatory pathways involving NF-κB activation, Nrf2 suppression, and BBB disruption. Therapeutic strategies that restore redox balance, protect

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PRINT ISSN: 2992-605X
mitochondrial function, and modulate inflammatory signaling have demonstrated promising neuroprotective potential. The use of antioxidants, Nrf2 activators, and phytochemical agents offers a multipronged approach to reducing neurotoxicity. As research advances, integrating these molecular insights into clinical practice may significantly improve neurological outcomes for patients suffering from systemic inflammatory diseases.

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CITE AS: Nassimbwa Kabanda D. (2026). Neurotoxicity Associated with Systemic Inflammatory Disorders: Insights from Malaria, Diabetes, and Arthritis Models. NEWPORT INTERNATIONAL JOURNAL OF PUBLIC HEALTH AND PHARMACY,7(1):117-122. https://doi.org/10.59298/NIJPP/2026/71117122