www.idosr.org Ivan, 2026

©IDOSR PUBLICATIONS

ISSN: 2579-079X

International Digital Organization for Scientific Research IDOSR JST/26/1220000
IDOSR JOURNAL OF SCIENCE AND TECHNOLOGY 12(2):15-21, 2026.
https://doi.org/10.59298/IDOSR/JST/26/122.1521

Pesticide-Induced Oxidative Stress and Neurodegeneration

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ABSTRACT

Pesticides are widely used in agriculture and pest control, yet mounting evidence links chronic exposure to adverse effects on human health, particularly the nervous system. One key mechanism through which pesticides exert neurotoxicity is oxidative stress-an imbalance between pro-oxidant and antioxidant systems that results in cellular damage. Pesticide-induced oxidative stress triggers lipid peroxidation, DNA damage, mitochondrial dysfunction, and neuroinflammation, processes implicated in the pathogenesis of neurodegenerative diseases such as Parkinson's disease, Alzheimer's disease, amyotrophic lateral sclerosis, and Huntington's disease. This review synthesizes current understanding of how pesticides cause oxidative damage in neural tissues, examines cellular mechanisms involved, and explores epidemiological and experimental evidence linking pesticide exposure to neurodegeneration. We further evaluate protective strategies, the role of genetic susceptibility, and future research directions to mitigate pesticide-related neurotoxicity.

Keywords: Oxidative stress, Neurodegeneration, Pesticides, Mitochondrial dysfunction, Neuroinflammation

INTRODUCTION

Pesticides encompass a wide range of chemical compounds designed to control pests in agriculture and households, including insecticides, herbicides, fungicides, and rodenticides[1]. While beneficial in increasing crop yields, preserving stored food, and controlling vectors of infectious diseases, many pesticides are biologically active compounds that may adversely affect non-target organisms[2]. Their widespread and sometimes indiscriminate use has resulted in environmental persistence, bioaccumulation, and chronic low-level human exposure through contaminated food, water, soil, and air. Occupational exposure among agricultural workers further increases the risk of toxic effects. Over the past several decades, epidemiological and experimental studies have increasingly linked long-term pesticide exposure with neurological impairments and elevated risk of neurodegenerative disorders[3]. These associations are particularly concerning given the progressive and often irreversible nature of neurodegeneration. A central mechanism underlying pesticide-induced neurotoxicity is oxidative stress, a pathological condition characterized by excessive production of reactive oxygen species (ROS) and reactive nitrogen species (RNS) relative to endogenous antioxidant defenses[4]. When the balance between oxidants and antioxidants is disrupted, oxidative damage accumulates in cellular macromolecules, impairing neuronal structure and function. The brain is especially vulnerable to oxidative stress for several reasons. It consumes a disproportionately high amount of oxygen relative to its size, contains abundant polyunsaturated fatty acids that are highly susceptible to lipid peroxidation, and possesses relatively limited antioxidant reserves compared to other organs. In addition, neurons are post-mitotic cells with limited regenerative capacity, making oxidative injury particularly consequential. This review examines how pesticides promote oxidative stress in neural tissue, highlights key chemical classes associated with neurotoxicity, and outlines the cellular mechanisms that contribute to neuronal vulnerability and degeneration.

2. Pesticides Commonly Linked to Neurotoxicity

Pesticides represent diverse chemical classes with distinct molecular targets, yet many converge on common oxidative and neurotoxic pathways[5]. Among those most frequently implicated in neurodegenerative outcomes are organophosphates, organochlorines, pyrethroids, and certain herbicides such as paraquat and rotenone. Organophosphates, including chlorpyrifos and malathion, exert toxicity primarily through inhibition of acetylcholinesterase, leading to excessive accumulation of acetylcholine at synapses[6]. Beyond acute cholinergic

toxicity, chronic low-dose exposure has been shown to induce oxidative stress, disrupt mitochondrial integrity, and impair neurodevelopmental processes. Organochlorines such as DDT and lindane are highly lipophilic and resistant to degradation, enabling them to accumulate in adipose tissue and neural membranes[7]. Their persistence in the environment and bioaccumulative nature increase the likelihood of long-term exposure. These compounds interfere with ion channels and mitochondrial function, promoting ROS generation and membrane destabilization[8]. Pyrethroids, including permethrin, act on voltage-gated sodium channels to prolong neuronal excitation. Although generally considered less persistent than organochlorines, accumulating evidence indicates that pyrethroids can enhance oxidative stress indirectly through increased neuronal activity and secondary mitochondrial strain[9]. Herbicides such as paraquat and rotenone have drawn particular attention due to their strong experimental links to Parkinsonian pathology. Rotenone directly inhibits mitochondrial complex I of the electron transport chain, while paraquat undergoes redox cycling to produce superoxide radicals. Both mechanisms result in substantial ROS production and selective dopaminergic neuronal injury. Collectively, these pesticides increase oxidative burden either by stimulating ROS generation, impairing mitochondrial respiration, or weakening endogenous antioxidant defenses, thereby precipitating oxidative damage in neural tissues.

3. Oxidative Stress: Mechanisms and Neural Vulnerability

3.1 Reactive Oxygen and Nitrogen Species

Reactive oxygen species include free radicals such as superoxide and hydroxyl radicals, as well as non-radical oxidants like hydrogen peroxide[10]. Reactive nitrogen species, including nitric oxide and peroxynitrite, further contribute to oxidative and nitrosative stress. Under physiological conditions, ROS and RNS are generated as byproducts of mitochondrial respiration and participate in intracellular signaling[11]. However, excessive accumulation leads to oxidation of lipids, proteins, and nucleic acids, altering their structure and function.

3.2 Antioxidant Defenses in the Brain

Neural cells rely on enzymatic antioxidants such as superoxide dismutase, catalase, and glutathione peroxidase to neutralize ROS[12]. Non-enzymatic antioxidants, including glutathione and vitamins C and E, provide additional protection. Despite these systems, the brain's antioxidant capacity is comparatively modest, rendering it susceptible to oxidative insults, particularly during chronic toxicant exposure.

3.3 Mitochondria as Targets

Mitochondria are both a primary source and a major target of ROS. Pesticides such as rotenone disrupt the electron transport chain, causing electron leakage and enhanced ROS production[13]. Damaged mitochondria generate less ATP, impair calcium homeostasis, and activate apoptotic signaling pathways. In neurons, which depend heavily on aerobic metabolism, mitochondrial dysfunction can rapidly compromise synaptic transmission and cell survival, amplifying the impact of oxidative stress[14].

4. Pathways of Pesticide-Induced Oxidative Stress

Pesticides induce oxidative stress through interconnected molecular and cellular mechanisms that ultimately compromise neuronal survival[15]. These processes are not isolated events but part of a complex cascade involving membrane damage, genomic instability, protein dysfunction, mitochondrial impairment, and chronic inflammation[16]. The cumulative impact of these alterations contributes significantly to progressive neurodegeneration.

4.1 Lipid Peroxidation

Reactive oxygen species readily attack polyunsaturated fatty acids within neuronal plasma membranes and intracellular organelle membranes, initiating lipid peroxidation[17]. This chain reaction generates highly reactive aldehydes such as malondialdehyde and 4-hydroxynonenal, which further propagate oxidative injury by forming adducts with proteins and DNA. Because neuronal membranes are rich in polyunsaturated lipids, particularly in synaptic terminals and myelin sheaths, they are especially susceptible to peroxidative damage[18]. Lipid peroxidation disrupts membrane fluidity and permeability, altering ion gradients essential for action potential generation and neurotransmitter release. Damage to synaptic membranes impairs vesicle fusion and receptor function, leading to defective synaptic transmission. In addition, oxidation of mitochondrial membranes compromises electron transport chain integrity, increasing electron leakage and further ROS production[19]. Thus, lipid peroxidation both results from and amplifies oxidative stress, creating a self-perpetuating cycle of neuronal injury.

4.2 DNA and Protein Damage

Oxidative stress also targets nuclear and mitochondrial DNA. ROS can induce base modifications, single- and double-strand breaks, and cross-linking[20]. One commonly observed lesion is 8-hydroxy-2'-deoxyguanosine, a marker of oxidative DNA damage. If not efficiently repaired, such lesions may lead to mutations, impaired transcription, and defective mitochondrial protein synthesis. Because neurons are long-lived cells with limited regenerative capacity, accumulated DNA damage can progressively impair cellular function[21]. Proteins are similarly vulnerable to oxidative modification. Oxidation of amino acid side chains, formation of carbonyl groups, and disruption of disulfide bonds can alter protein conformation and enzymatic activity. Key neuronal proteins,

including cytoskeletal components and metabolic enzymes, may lose structural integrity or become prone to aggregation[22]. Impairment of proteasomal and autophagic degradation pathways further exacerbates accumulation of damaged proteins. Over time, these molecular alterations compromise synaptic maintenance, axonal transport, and neuronal survival.

4.3 Neuroinflammation

Oxidative stress activates resident immune cells of the central nervous system, particularly microglia and astrocytes[23]. Upon activation, microglia release pro-inflammatory cytokines such as tumor necrosis factor-alpha and interleukin-1 beta, as well as additional reactive oxygen and nitrogen species. Astrocytes may also contribute by producing inflammatory mediators and altering glutamate homeostasis[24]. While acute neuroinflammation can serve protective functions, chronic activation becomes detrimental. Sustained inflammatory signaling enhances oxidative stress, disrupts the blood–brain barrier, and promotes excitotoxicity through excessive glutamate release[25]. This persistent inflammatory environment amplifies neuronal injury and establishes a vicious cycle in which oxidative stress and inflammation mutually reinforce each other. Such chronic neuroinflammation is increasingly recognized as a key contributor to progressive neurodegenerative processes associated with pesticide exposure.

5. Pesticide Exposure and Neurodegenerative Diseases

Growing epidemiological and experimental evidence links pesticide-induced oxidative stress with major neurodegenerative disorders[26-30]. Although causality is complex and multifactorial, oxidative mechanisms provide a biologically plausible bridge between environmental exposure and neuronal degeneration.

5.1 Parkinson's Disease (PD)

Parkinson's disease is characterized by progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta and accumulation of misfolded alpha-synuclein aggregates[27,31-34]. Dopaminergic neurons are particularly susceptible to oxidative stress due to dopamine metabolism, which itself generates reactive intermediates[28,35-38]. Epidemiological studies have consistently reported increased Parkinson's disease risk among individuals exposed to pesticides such as paraquat and rotenone. Rotenone inhibits mitochondrial complex I, impairing ATP production and promoting excessive ROS generation. Experimental models using rotenone replicate key features of Parkinsonian pathology, including dopaminergic neuron loss and motor deficits[29,39-42]. Paraquat, through redox cycling, produces superoxide radicals that selectively damage dopaminergic neurons. Oxidative stress also promotes alpha-synuclein misfolding and aggregation, further linking pesticide exposure to Parkinson's disease pathogenesis.

5.2 Alzheimer's Disease (AD)

Alzheimer's disease is marked by extracellular amyloid-beta plaques, intracellular neurofibrillary tangles composed of hyperphosphorylated tau, and widespread synaptic loss[30,43-46]. Oxidative stress is an early and prominent feature of Alzheimer's pathology. Chronic exposure to certain pesticides has been associated with cognitive decline and increased Alzheimer's disease risk in some population studies[31,47-50]. Organophosphates such as chlorpyrifos can enhance oxidative stress in cortical and hippocampal neurons, regions critical for memory and learning. Oxidative damage may accelerate amyloid-beta aggregation and tau phosphorylation, while mitochondrial dysfunction impairs neuronal energy metabolism[32,50-51]. These mechanisms suggest that pesticide-induced oxidative stress may contribute to both the initiation and progression of Alzheimer's disease.

5.3 Amyotrophic Lateral Sclerosis (ALS)

Amyotrophic lateral sclerosis involves degeneration of upper and lower motor neurons, leading to progressive muscle weakness and paralysis[33]. Oxidative stress and excitotoxicity are central features of ALS pathophysiology. Some occupational studies indicate elevated ALS incidence among agricultural workers exposed to pesticides, although results vary across populations. Pesticide-induced oxidative damage may exacerbate glutamate-mediated excitotoxicity and impair mitochondrial function in motor neurons[34]. Given that mutations in antioxidant-related genes are implicated in familial ALS, environmental oxidative stressors such as pesticides may act synergistically with genetic predispositions to increase disease risk.

5.4 Huntington's Disease (HD)

Huntington's disease is a hereditary neurodegenerative disorder caused by expanded CAG repeats in the huntingtin gene[35]. Although primarily genetic in origin, oxidative stress plays a significant role in neuronal dysfunction and striatal degeneration associated with the disease. Environmental toxicants, including pesticides, may intensify oxidative burden and mitochondrial impairment in vulnerable neurons[36]. Exposure to pro-oxidant pesticides could potentially accelerate symptom onset or worsen disease severity by compounding existing mitochondrial deficits and promoting protein aggregation[37]. While further research is needed, oxidative stress provides a plausible mechanistic link between environmental exposures and modification of Huntington's disease progression.

CONCLUSION

Pesticides contribute to oxidative stress in the nervous system through multiple mechanisms, including ROS overproduction, mitochondrial dysfunction, lipid peroxidation, and neuroinflammation. Evidence from cellular models, animal studies, and human research supports links between pesticide exposure and neurodegenerative diseases. While reducing exposure remains primary prevention, ongoing research into antioxidants, genetic susceptibility, and safer alternatives is vital. Protecting neurological health demands coordinated effort from scientists, clinicians, policymakers, and communities.

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CITE AS: Ivan Mutebi. (2026). Pesticide-Induced Oxidative Stress and Neurodegeneration. IDOSR JOURNAL OF SCIENCE AND TECHNOLOGY 12(2):15-21, 2026.
https://doi.org/10.59298/IDOSR/JST/26/122.1521