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Unraveling the Neurotoxic Effects of Heavy Metals: Insights into Neuroinflammation and Neurodegenerative Diseases

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DESCRIPTION

Heavy metals, including lead, mercury, and arsenic, are ubiquitous environmental pollutants that pose significant risks to human health, particularly to the delicate and intricate structures of the nervous system. Mounting evidence suggests that exposure to these toxic metals can induce neuroinflammation and neurotoxicity, contributing to the pathogenesis of neurodegenerative diseases. In this article, we delve into the mechanisms by which heavy metals exert their detrimental effects on the brain, shedding light on their role in neuroinflammation and neurodegeneration. Neuroinflammation, characterized by the activation of glial cells and the release of pro-inflammatory mediators in the central nervous system (CNS), is a common response to various insults, including heavy metal exposure. Upon entering the brain, heavy metals can disrupt the blood brain barrier, allowing their entry into the CNS and triggering inflammatory responses. Microglia, the resident immune cells of the brain, play a central role in orchestrating neuroinflammatory processes in response to heavy metal exposure. Activation of microglia leads to the production of pro-inflammatory cytokines, chemokines, and reactive oxygen species (ROS), promoting neuronal damage and dysfunction. Lead, a well-known neurotoxicant, has been implicated in the pathogenesis of neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD). Lead disrupts calcium homeostasis, impairs mitochondrial function, and induces oxidative stress in neurons, contributing to neuronal apoptosis and synaptic dysfunction. Moreover, lead exposure has been shown to promote the aggregation of amyloid-beta (Aβ) peptides and hyperphosphorylation of tau protein, key pathological hallmarks of AD. Mercury, another potent neurotoxic metal, exerts its effects through multiple mechanisms, including disruption of neurotransmitter signaling, inhibition of antioxidant defences, and induction of neuroinflammation. Methylmercury, the organic form of mercury found in contaminated seafood, readily crosses the blood-brain barrier and accumulates in the brain, where it interferes with synaptic transmission and neuronal development. Chronic exposure to methylmercury has been associated with cognitive deficits, motor impairments, and increased risk of neurodegenerative disorders such as PD and amyotrophic lateral sclerosis (ALS). Arsenic, a metalloid commonly found in drinking water and agricultural products, has emerged as a potential environmental risk factor for neurodegenerative diseases. Arsenic exposure can disrupt glutamate signaling, impair synaptic plasticity, and induce neuronal apoptosis in the brain. Additionally, arsenic-induced neuroinflammation has been implicated in the pathogenesis of AD, with studies demonstrating increased levels of pro-inflammatory cytokines and microglial activation in arsenic-exposed brains. The intricate interplay between neuroinflammation and neurodegeneration represents a complex and dynamic process driven by multiple factors, including heavy metal exposure. Chronic neuroinflammation perpetuates neuronal damage and exacerbates neurodegenerative processes, ultimately leading to progressive cognitive decline and motor dysfunction. Furthermore, heavy metals may synergize with other risk factors, such as genetic susceptibility and aging, to accelerate the onset and progression of neurodegenerative diseases. Understanding the mechanisms by which heavy metals induce neuroinflammation and neurotoxicity is crucial for developing targeted interventions to mitigate their adverse effects on brain health.

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CONFLICT OF INTEREST

The author states there is no conflict of interest.

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