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Uncovering the Link between Heavy Metals and Neurodegenerative Diseases: Exploring Complex Mechanisms

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DESCRIPTION

Heavy metals have increasingly been recognized for their role in promoting neuroinflammatory responses and contributing to the pathogenesis of neurodegenerative diseases such as Alzheimer's and Parkinson's. Advanced studies are shedding light on the intricate mechanisms through which these metals exacerbate neurological disorders. This article explores the latest research findings and implications of heavy metal exposure on neuroinflammation and the onset of debilitating conditions like Alzheimer's and Parkinson's diseases. Heavy metals such as lead, mercury, cadmium, and arsenic are pervasive environmental pollutants known for their toxicity to human health. They are widely distributed in the environment due to industrial activities, mining, and agricultural practices, posing significant risks to human populations, especially through air, water, and food contamination. Recent research has increasingly linked these metals to neurodegenerative diseases characterized by progressive deterioration of neurological function. Heavy metals can activate microglia, the immune cells of the central nervous system, leading to neuroinflammation. Activated microglia release pro-inflammatory cytokines such as interleukin-1β (IL-1β) and tumour necrosis factor alpha (TNF-α), as well as Reactive Oxygen Species (ROS). These inflammatory mediators contribute to neuronal damage and inflammation in the brain, which are central features of neurodegenerative diseases. Certain heavy metals, including lead and mercury, have been shown to disrupt the integrity of the Blood-brain Barrier (BBB). The BBB normally protects the brain by regulating the passage of substances between the bloodstream and the brain tissue. When compromised by heavy metals, the BBB allows toxic substances to enter the brain more easily, exacerbating neuroinflammation and neuronal damage. Heavy metals induce oxidative stress in brain cells by generating Reactive Oxygen Species (ROS) and impairing antioxidant defense mechanisms. Oxidative stress leads to damage of lipids, proteins, and DNA within neurons, contributing to neurodegeneration. The accumulation of oxidative damage over time is associated with the progression of neurodegenerative diseases. Heavy metals such as aluminium and copper have been implicated in the pathogenesis of Alzheimer's disease. Aluminium has been found in amyloid plaques and neurofibrillary tangles in the brains of AD patients, contributing to the aggregation of these pathological proteins. Copper dysregulation is associated with the formation of Reactive Oxygen Species (ROS) and oxidative stress, which are involved in neurodegeneration. The role of heavy metals, particularly manganese and iron, in Parkinson's disease has been extensively studied. Manganese exposure is associated with the development of a Parkinson's-like syndrome known as manganism, characterized by movement disorders and neurodegeneration. Iron accumulation in the substantia nigra region of the brain contributes to oxidative stress and dopaminergic neuronal degeneration, hallmark features of Parkinson's disease. Individual susceptibility to heavy metal-induced neurodegeneration may be influenced by genetic factors. Variations in genes involved in metal metabolism, oxidative stress response (e.g., superoxide dismutase, glutathione peroxidase), and neuroinflammatory pathways (e.g., cytokines) can modulate susceptibility to neurodegenerative diseases in response to heavy metal exposure. Advanced neuroimaging technologies such as Positron Emission Tomography (PET), Single-photon Emission Computed Tomography (SPECT), and Magnetic Resonance Imaging (MRI) play a crucial role in visualizing heavy metal accumulation in the brain. These techniques allow researchers to correlate metal distribution with disease progression and severity, providing insights into the mechanisms underlying heavy metal neurotoxicity.

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CONFLICT OF INTEREST

The author states there is no conflict of interest.

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