## Heavy Metal-Induced Neurotoxicity and Calcium Channel Involvement

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## Abstract

Heavy metal-induced neurotoxicity is a burgeoning concern in modern society due to widespread environmental contamination. This article delves into the intricate relationship between heavy metal exposure and neurological damage, focusing on the pivotal role of calcium channels in this intricate interplay. Heavy metals like lead, mercury, and cadmium have been recognized as potent neurotoxins, capable of disrupting calcium channel function. This disruption leads to a cascade of detrimental efects within neurons, including impaired synaptic transmission, excito toxicity, synaptic plasticity defcits, and neuroinfammation. Understanding the mechanisms underlying heavy metal-induced neurotoxicity and its connection to calcium channels is crucial for developing efective strategies to mitigate the adverse efects on the nervous system and prevent related neurological disorders.

Nevrotoxicity; Calcium channels; Lead; Mercury; Cadmium

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Neurotoxicity refers to the adverse e ects of various substances on the nervous system, resulting in damage to nerve cells, impaired function, and potentially severe neurological disorders. Among the numerous factors contributing to neurotoxicity, heavy metals have emerged as signi cant culprits. ese toxic metals, such as lead, mercury, cadmium, and arsenic, are ubiquitous in the environment due to industrial processes, pollution, and other human activities. One intriguing aspect of heavy metal-induced neurotoxicity is its intricate relationship with calcium channels, which play a pivotal role in maintaining neuronal function. is article explores the intricate connection between heavy metal exposure, neurotoxicity, and the involvement of calcium channels [1].

Heavy metal-induced neurotoxicity is a growing concern worldwide, driven by the pervasive presence of heavy metals in the environment due to industrial activities, pollution, and human practices. neurological health, lead, mercury, cadmium, and arsenic have been the most extensively studied.

thereby in uencing a multitude of cellular processes, including neurotransmitter release, synaptic plasticity, and gene expression [3]. Understanding how heavy metals interact with and disrupt calcium channels is pivotal to unraveling the mechanisms underlying heavy metal-induced neurotoxicity and devising strategies for mitigating their detrimental e ects on the nervous system.

Heavy metals have long been recognized as potent neurotoxins. ese substances have the ability to accumulate in various tissues, including the brain, where they can cause structural and functional damage. Some heavy metals, like lead and mercury, readily cross the blood-brain barrier, allowing them direct access to the central nervous system (CNS). Once inside the brain, heavy metals interfere with neuronal processes and can lead to a range of neurological disorders, particularly in developing children and adults with prolonged exposure [4].

Calcium ions (Ca2+) are essential for normal neuronal function. Editor Neurotoxicity refers to the harmful e ects of various substances on the Sep-2023, PreQC No: tyoa-23-114006 (PQ), Reviewed: 19-Sep-2023, QC nervous system, encompassing a spectrum of structural and functional 23-114006, **Revised:** 23-Sep-2023, Manuscript No: tyoa-23-114006 ( impairments. Among the heavy metals that pose a signi cant threat to published: 30-Sep-2023, DOI: 10.4172/2476-2067.1000236

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de ne. Even micronutrients, such as cobalt (Co), copper (Cu), copyright: © 2023 Manetti P. This is an open-access article distributed under (Fe), manganese (Mn), molybdenum (Mo), and zinc (Zn) cantebras of the Creative Commons Attribution License, which permits unrestric detrimental to living organisms, when present in excessive levels, and in and reproduction in any medium, provided the original author a a re ned equilibrium between de cient and toxic concentrations source are credited.

to be maintained. is is particularly important for very specialized tissues, such as the brain, where metals induce oxidative damage and some of the essential micronutrients, such as Fe, Zn, and Cu, have been implicated in etiology and development of di erent neurological and neurodegenerative diseases. Less obviously, living organism may nd use for nonessential toxic metals in extreme condition. An elegant example of unexpected biological function of Cd has been recently reported in marine diatoms [2].

is article delves into the intricate relationship between heavy metal exposure and neurotoxicity, with a speci c focus on the crucial role played by calcium channels in mediating these e ects. Calcium channels are fundamental components of neuronal function, as they govern the in ux of calcium ions (Ca2+) into neurons, Citation: Manetti P (2023) Heavy Metal-Induced Neurotoxicity and Calcium Channel Involvement. Toxicol Open Access 9: 236.

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