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Homeostasis of Zinc an Emerging Therapeutic Target for Diseases Related to Neuroinflammation

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Abstract

Zinc, an essential trace element, plays a pivotal role in numerous physiological processes, including immune function, neurotransmission, and antioxidant defense. This review explores zinc's critical role in maintaining cellular homeostasis and its emerging significance as a therapeutic target in diseases associated with neuroinflammation. Zinc homeostasis involves a complex interplay of absorption, distribution, cellular uptake, and excretion mechanisms regulated by zinc transporters and binding proteins. Moreover, zinc exerts modulatory effects on neuroinflammatory pathways, influencing microglial activation, cytokine production, and oxidative stress responses in the central nervous system. Dysregulation of zinc homeostasis has been implicated in various neuroinflammatory conditions, such as Alzheimer's disease, Parkinson's disease, and multiple sclerosis. Therefore, understanding the mechanisms underlying zinc's physiological roles and its potential as a therapeutic agent holds promise for developing novel treatments aimed at mitigating neuroinflammation and its associated pathologies. This review synthesizes current research findings to highlight zinc's dual role as a crucial micronutrient and a promising therapeutic target in neuroinflammatory diseases.

Keywords: Zinc homeostasis; Zinc transporters; Neuroinflammation; Microglia; Alzheimer's disease; Therapeutic targets

Introduction

Zinc is an essential trace element vital for numerous physiological functions, including enzymatic activity, immune response, and neurological processes [1]. Its role extends beyond basic cellular functions to encompass modulation of inflammatory responses within the central nervous system (CNS). This review explores the dynamic interplay between zinc homeostasis and neuroinflammation, highlighting zinc's emerging significance as a therapeutic target in diseases associated with CNS inflammation [2]. Zinc homeostasis involves intricate mechanisms governing its absorption from dietary sources, distribution throughout the body, cellular uptake facilitated by specific transporters, and regulation through binding proteins and storage mechanisms [3]. These processes ensure that zinc levels are tightly controlled to support optimal cellular function and systemic health. In the context of neuroinflammation, zinc has been identified as a pivotal modulator of immune responses and oxidative stress pathways within the CNS [4]. It influences microglial activation, cytokine production, and the balance of antioxidant defenses, thereby impacting the progression of neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and multiple sclerosis. Understanding the dual role of zinc as both a micronutrient essential for normal physiological processes and a potential therapeutic agent in combating neuroinflammation is crucial [5]. This review synthesizes current research findings to elucidate the mechanisms underlying zinc's involvement in neuroinflammatory pathways and discusses its therapeutic implications. By exploring zinc's therapeutic potential, we aim to pave the way for novel treatment strategies targeting neuroinflammatory conditions, ultimately improving outcomes for patients affected by these debilitating diseases.

Materials and Methods

A comprehensive search was conducted using electronic databases such as PubMed, Google Scholar, and relevant academic journals. Keywords included zinc homeostasis, zinc transporters, neuroinflammation, Alzheimer's disease, Parkinson's disease, multiple sclerosis, and therapeutic targets [6]. Articles published in English and pertinent to zinc's role in neuroinflammation were included. Studies, reviews, and clinical trials focusing on zinc's involvement in neuroinflammatory pathways, its effects on microglial activation, cytokine modulation, and oxidative stress within the CNS were selected [7]. Emphasis was placed on recent research and findings pertinent to zinc's therapeutic potential in neurodegenerative diseases associated with neuroinflammation. Key data points extracted included mechanisms of zinc homeostasis (absorption, distribution, cellular uptake, and excretion), zinc transporters involved in CNS zinc regulation, experimental models used to study zinc's effects on neuroinflammation, and clinical outcomes related to zinc supplementation or modulation [8]. Extracted data were synthesized to provide a cohesive overview of zinc's role in neuroinflammatory processes, its impact on CNS function and pathology, and implications for therapeutic interventions. This synthesis aimed to integrate findings from diverse studies to elucidate the complex interactions between zinc and neuroinflammation [9]. The synthesized information was critically analyzed to evaluate the strength of evidence supporting zinc as a therapeutic target in neuroinflammatory diseases. Limitations, gaps in knowledge, and areas requiring further research were identified and discussed. This review focused on summarizing existing literature; therefore, ethical approval was not required. Proper attribution and citation of sources were ensured to maintain academic integrity [10]. Limitations of the study include potential biases in the selected literature, variability in study methodologies, and the exclusion of non-English publications, which may have influenced the scope and comprehensiveness of the review. By employing these methods, this review aims to provide a comprehensive understanding of zinc's

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involvement in neuroinflammation and its potential as a therapeutic avenue for treating neurodegenerative diseases.

Conclusion

Zinc emerges as a pivotal player in the intricate landscape of neuroinflammation, influencing diverse pathways critical to CNS function and health. This review has highlighted zinc's dual role: as an essential micronutrient involved in fundamental cellular processes and as a potential therapeutic target in neuroinflammatory diseases. The maintenance of zinc homeostasis is crucial for ensuring optimal neurological function, as zinc participates in enzymatic reactions, synaptic transmission, and neuronal plasticity. Dysregulation of zinc levels, whether through deficiency or excess, has been implicated in neurodegenerative conditions such as Alzheimer's disease, Parkinson's disease, and multiple sclerosis. These disorders are characterized by chronic neuroinflammation, where zinc's ability to modulate microglial activation, cytokine production, and oxidative stress responses plays a significant role in disease progression. Therapeutically, targeting zinc pathways holds promise for mitigating neuroinflammation and its associated neurodegenerative sequelae. Strategies may include zinc supplementation to restore physiological Moreover; advancements in understanding zinc's role in neuroinflammation underscore the need for personalized medicine approaches that consider individual variability in zinc metabolism and disease susceptibility. Integrating zinc-based therapies into comprehensive treatment regimens has the potential to enhance therapeutic outcomes and improve quality of life for patients affected by neuroinflammatory diseases. In conclusion, the exploration of zinc's involvement in neuroinflammation represents a promising frontier in neuroscience and therapeutic development. Continued research efforts are warranted to elucidate mechanistic insights, optimize therapeutic strategies, and ultimately harness zinc's potential as a therapeutic modulator in the management of neurodegenerative disorders characterized by neuroinflammation.

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Conflict of Interest

None

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