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Editorial

The Role of Dendritic Cells in Alzheimer's Disease: Implications for Immunotherapy

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Introduction

Alzheimer's disease (AD) is a complex neurodegenerative disorder characterized by progressive cognitive decline, memory loss, and the accumulation of amyloid-beta plaques and neurofibrillary tangles in the brain. While the primary pathological features of AD have often been the focus of research, the role of the immune system, particularly dendritic cells (DCs), has garnered increasing attention. Dendritic cells are antigen-presenting cells that play a pivotal role in the immune response by processing and presenting antigens to T cells, thereby bridging innate and adaptive immunity. This essay explores the multifaceted roles of dendritic cells in Alzheimer's disease, including their involvement in immune dysregulation, their potential contribution to neuroinflammation, and their implications for therapeutic interventions.

Dendritic cells and immune response in alzheimer's disease

Dendritic cells are essential for maintaining immune homeostasis in the brain. In a healthy state, they facilitate the clearance of pathogens and dead cells while supporting tolerance to self-antigens. However, in Alzheimer's disease, the homeostatic balance is disrupted. Studies have shown that the presence of amyloid-beta proteins can lead to the activation of dendritic cells, prompting them to produce pro-inflammatory cytokines and contribute to a chronic inflammatory state in the brain [1]. This activation of DCs may contribute to the neuroinflammatory environment observed in AD, exacerbating neuronal damage and disease progression.

Interaction between dendritic cells and T cells

One of the critical roles of dendritic cells in the immune system is their ability to activate T cells. In AD, the interaction between DCs and T cells has significant implications. Research has indicated that DCs in the brains of AD patients display aberrant expression patterns of co-stimulatory molecules, leading to dysfunctional T cell responses [2]. This dysfunctional interaction can promote the polarization of T cells toward a pathogenic Th17 phenotype, which is associated with increased neuroinflammation and pathogenic processes in AD [3].

Moreover, the accumulation of amyloid-beta can impair dendritic cell maturation, leading to altered antigen presentation and cytokine production. As a result, T cell activation is often suboptimal, contributing to a failure in the clearance of amyloid plaques and further promoting the progression of neurodegenerative changes [4].

Dendritic cells and neuroinflammation

Neuroinflammation is a hallmark of Alzheimer's disease and is characterized by the activation of microglia and the presence of inflammatory mediators. Dendritic cells are found in the periphery and the central nervous system, where they modulate immune responses. In AD, their activation can lead to the exacerbation of neuroinflammation, contributing to neuronal injury.

Evidence suggests that DCs can interact with microglia, potentially amplifying inflammatory responses. The cytokines released from activated DCs can influence microglial behaviour, leading to increased production of pro-inflammatory cytokines and chemokines [5]. This interplay between dendritic cells and microglia highlights the dual role of DCs in both initiating and regulating inflammation in the context of Alzheimer's disease.

Therapeutic implications

Given the central role of dendritic cells in immune dysregulation and neuroinflammation in Alzheimer's disease, targeting these cells represents a promising avenue for therapeutic interventions. Modulating dendritic cell function could enhance antigen presentation, promote the activation of regulatory T cells, and curb the harmful inflammatory responses. Several strategies, including the use of immunomodulatory agents or dendritic cell vaccines, are being explored to restore immune balance and improve outcomes in Alzheimer's disease.

For example, promoting the maturation of dendritic cells and their ability to induce T cell tolerance may offer a strategy for reduc-

ing neuroinflammation and amyloid burden (Tzeng., *et al.* 2023). Furthermore, leveraging dendritic cells for targeted delivery of therapeutic agents to the central nervous system could enhance the efficacy of treatments aimed at ameliorating AD pathology.

Conclusion

In conclusion, dendritic cells play a multifaceted and significant role in the pathogenesis of Alzheimer's disease. Their involvement in immune dysregulation, T cell interaction, and neuroinflammation underscores the complexity of the disease and the immune response. As research continues to unveil the intricate relationships between dendritic cells and neurodegeneration, targeted immunotherapeutic strategies that modulate dendritic cell function may hold promise for the treatment of Alzheimer's disease.

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