Inflammation: A Major Target for Compounds to Control Alzheimer's Disease

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Abstract

Several hypotheses have been postulated to explain how Alzheimer's disease is triggered, but none of them provide a unified view of its pathogenesis. The dominant hypothesis based on build-ups of the amyloid-beta peptide has been around for longer than three decades; however, up to today, numerous clinical trials based on the amyloid postulates have been attempted, but all of them have failed. Clearly, the revisited tau hypothesis provides a better explanation of the clinical observations of patients, but it needs to integrate the cumulative observations on the onset of this disease. In this context, the neuroimmuno modulation theory, based on the involvement of inflammatory events in the central nervous system, accounts for all these observations. In this review we intend to emphasize the idea that neuroinflammation is a main target for the search of new therapeutic strategies to control Alzheimer's disease. Beyond mono-targeting approaches using synthetic drugs that control only specific pathophysiological events, emerging therapeutics views based on multi targeting compounds appear to provide a new pathway for Alzheimer's disease treatment.

Palabras clave

Palabras clave de autor: Alzheimer's disease; astrocytes; proinflammatory mediators; microglial cells; neuroinflammation; tau pathology
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