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Poster Presentation

Neuroinflammation in Alzheimer 's Disease

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Abstract

Alzheimer's disease (AD) is a neurodegenerative disorder and the most common form of dementia. Almost 47 million people suffer from dementia worldwide. AD accounts for approximately 60%–80% of all dementia cases. Three major pathologies characterize the disease: senile plaques, neurofibrillary tangles and inflammation. We review the literature on events contributing to the inflammation. Those inflammatory processes play a significant role in the pathophysiology of AD. Histopathologically is characterized by the presence of two major hallmarks, the intracellular neurofibrillary tangles (NFTs) and extracellular neuritic plaques (NPs) surrounded by activated astrocytes and microglia. The main component in the NP is the amyloid-β peptide (Aβ). Neuroinflammation is characterized by the activation of astrocytes and microglia and the release of proinflammatory cytokines and chemokines. Neuroinflammation is one of the main factors neurodegenerations. Study of the factors and pathways able to the first step of the inflammatory response induced to identify potential therapeutic targets through which to stop the progress AD. Evidence confirms that neuroinflammation, by neuronal, glial, and immune components, is a contributing cause of Aβ aggregation, tau hyper phosphorylation, and neuronal damage and death, so production of cytokines and pro-inflammatory molecules has initially a neuroprotective role, but subsequently becomes the cause of further neurodegeneration. Therefore, future studies must intensively investigate the intricate ways of the neuroinflammatory process and define the best time to control it, so it will be possible to achieve more focused therapeutic strategies in the hope of not only alleviating but also modifying AD progression.

Keywords: Alzheimer's disease, Neuroinflammation, Amyloid-β

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