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Apolipoprotein E and Alzheimer's Disease: Findings, Hypotheses, and Potential Mechanisms

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Abstract

Alzheimer's disease (AD) is a multifactorial neurodegenerative disorder that involves dysregulation of many cellular and molecular processes. It is notoriously difficult to develop therapeutics for AD due to its complex nature. Nevertheless, recent advancements in imaging technology and the development of innovative experimental techniques have allowed researchers to perform in-depth analyses to uncover the pathogenic mechanisms of AD. An important consideration when studying late-onset AD is its major genetic risk factor, apolipoprotein E4 (apoE4). Although the exact mechanisms underlying apoE4 effects on AD initiation and progression are not fully understood, recent studies have revealed critical insights into the apoE4-induced deficits that occur in AD. In this review, we highlight notable studies that detail apoE4 effects on prominent AD pathologies, including amyloid- β , tau pathology, neuroinflammation, and neural network dysfunction. We also discuss evidence that defines the physiological functions of apoE and outlines how these functions are disrupted in apoE4-related AD.

Keywords: Alzheimer's disease; A β ; apoE; biomarker; neural network deficit; neuroinflammation; tau.

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