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Exploring the Role of Mitochondrial Dysfunction in Alzheimer's Disease Progression

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Abstract: *Alzheimer's Disease (AD) is a progressive neurodegenerative disorder characterized by cognitive decline and memory loss, significantly impacting patients' lives and presenting a major public health challenge. Mitochondrial dysfunction has emerged as a critical factor in AD pathogenesis, playing a pivotal role in oxidative stress, energy deficiency, and cellular degeneration. This paper explores the mechanisms by which mitochondrial dysfunction contributes to AD progression, focusing on oxidative stress, amyloid-beta (Aβ) interactions, mitochondrial DNA mutations, and disruptions in mitochondrial dynamics. By examining recent research, this study sheds light on the connection between mitochondrial health and neurodegeneration, linking mitochondrial impairment with synaptic dysfunction, neuroinflammation, and cell death in AD.*

In addition to outlining pathological mechanisms, this paper reviews potential therapeutic strategies targeting mitochondrial pathways, including antioxidant therapies, mitochondria-targeted drugs, and gene therapy approaches. Emerging interventions, such as mitochondrial biogenesis enhancement and lifestyle modifications, are also discussed for their neuroprotective potential. This review concludes that targeting mitochondrial dysfunction holds promise for slowing or reversing AD progression, underscoring the need for continued research into mitochondrial-based treatments and biomarkers for early intervention..

Keywords: Alzheimer's Disease