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Role of β-Amyloid Aggregation in The Pathology of Alzheimer's Disease

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ABSTRACT

β-amyloid aggregation plays a central role in the pathology of Alzheimer's disease (AD), a progressive neurodegenerative disorder primarily affecting the elderly. β-amyloid peptides, derived from the amyloid precursor protein (APP), accumulate abnormally in the brain, forming insoluble plaques that are a hallmark of AD. These plaques disrupt cellular communication, leading to synaptic dysfunction and neuronal death. The aggregation of β-amyloid triggers a cascade of pathological events, including oxidative stress, inflammation, and the hyper phosphorylation of tau protein, which contributes to neurofibrillary tangles. This aggregation is also associated with mitochondrial dysfunction, further exacerbating neuronal damage. The spread of β-amyloid plaques in specific brain regions correlates with the severity of cognitive decline, highlighting their importance in disease progression. Despite extensive research, the exact mechanisms by which β-amyloid aggregation leads to neurodegeneration remain unclear, but its role in initiating and perpetuating the pathological processes in AD is well-established. Therapeutic strategies targeting β-amyloid aggregation, such as β-secretase inhibitors or monoclonal antibodies, have shown promise in reducing plaque burden, but their effectiveness in halting or reversing cognitive decline remains a significant challenge in the treatment of Alzheimer's disease.