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Name of the faculty: Dr. S. Amuthalakshmi

Journal Article: Innovative Approaches to Alzheimer's Treatment: Utilizing Tacrine Hybrids to Inhibit Amyloid Beta Aggregation as a Strategic Focus

Author's name: Angamuthu Prabakaran, **Amuthulakshmi Sivaperuman**, Ramalakshmi Natarajan, Nalini Calambur Nagarajan, Viswas Raja Solomon

Abstract:

Alzheimer's disease (AD) is a complex and progressive brain disorder marked by memory loss, cognitive decline, and behavioral changes. One of its defining features is the build-up of amyloid plaques, clumps of β-amyloid (Aβ) peptides, in the brain, along with the formation of neurofibrillary tangles. These Aβ peptides are generated when the amyloid precursor protein (APP) is cleaved by enzymes, with β-secretase (BACE1) playing a key role in the first step of this process. Because BACE1 starts the cascade that leads to harmful Aβ build-up, it has become an important target in the search for effective Alzheimer's treatments. As Aβ accumulates in neurons, it disrupts communication between brain cells and triggers oxidative stress, which worsens damage and accelerates disease progression. This is often exacerbated by imbalances in metal ions, such as copper and iron. While tacrine, an early acetylcholinesterase inhibitor, has shown benefits in managing AD symptoms, its limitations have led researchers to explore improved versions. One promising direction is the development of tacrine-based hybrid molecules. By combining tacrine with other chemical groups that have anti-β-amyloid (Aβ) effects, antioxidant properties, and metal-chelating properties, scientists aim to create compounds that target multiple aspects of the disease simultaneously. This review examines the emerging potential of tacrine hybrids, particularly

their capacity to inhibit BACE1 and prevent Aβ aggregation, providing new hope for more effective and disease-modifying therapies for Alzheimer's disease.

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