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Mitophagy in Alzheimer's disease and its potential as a therapeutic target

Accumulation of damaged mitochondria is a hallmark of age-related neurodegenerative disorders such as Alzheimer's disease (AD). Mitophagy, the selective autophagic degradation of damaged mitochondria, plays an essential role in maintaining neuronal health, and increasing evidence links its dysfunction to AD progression. This review elucidates the molecular mechanisms underlying mitophagy deficits and their interaction with amyloid- β (A β) and Tau pathology, which may create a vicious cycle of neuronal damage and death. Additionally, mitochondrial dysfunction exacerbates AD pathogenesis by activating the NLRP3 inflammasome, contributing to inflammation in neurons and microglia. Investigating the dual role of mitophagy in pathological ferroptosis further reveals its complex involvement in AD. By consolidating current insights, this study highlights mitophagy as a promising therapeutic target, emphasizing its potential role in developing novel, disease-modifying interventions for AD treatment and prevention.

Keywords: Alzheimer's disease; Mitophagy; Mitochondrial dysfunction; Neuroinflammation; Ferroptosis

Biography

I am a second-year Master's student at Shanghai Jiao Tong University School of Medicine, mentored by Dr. Bing Wang (Shanghai Sixth People's Hospital). My work centers on mitophagy and neuroinflammation in Alzheimer's disease. As co-first author, I published a narrative review in *Neurobiology of Disease*. At this meeting, I will share a concise framework and open questions from our synthesis, and I look forward to exchanging ideas and learning from this community.