

Dementia Journal Club



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Title: Twenty-two Years of Reddy Lab Research: Focus on Aging, Mitochondria, and Synapse

DATE: MAY 9TH, 2023; **TIME:** 12:30 PM EASTERN / 11:30 AM CENTRAL

LOCATION: VIRTUAL - **ZOOM** - **MEETING ID:** TBD **PASSCODE:** TBD

ABSTRACT Alzheimer's disease (AD) is a progressive neurodegenerative disease, characterized by memory loss and multiple cognitive impairments. AD is associated with multiple cellular changes, including deregulation of microRNAs, activation of glia and astrocytes, hormonal imbalance, defective mitophagy, synaptic degeneration, in addition to the accumulation of amyloid-beta (A β) plaques, phosphorylated tau (P-tau). Twenty-two years of research in the Reddy Lab revealed that impaired mitochondrial dynamics, defective mitochondrial biogenesis, synaptic damage, defective mitophagy leads to synaptic degeneration and cognitive dysfunction in AD. Our critical analyses of mitochondria and A β and P-tau revealed increased levels of A β and P-Tau, and abnormal interactions between A β and Drp1, P-Tau and Drp1 induced increased mitochondrial fragmentation and proliferation of dysfunctional mitochondria in AD neurons and depleted Parkin and PINK1 levels. These events ultimately lead to impaired clearance of dead and/or dying mitochondria in AD neurons. The purpose of my presentation is to highlight and discuss the recent research on mitochondria and synapses in relation to A β and P-tau, focusing on recent developments.