**Mitochondria and mitochondrial cascades in Alzheimer's disease**

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Viable hypotheses of Alzheimer's disease (AD) pathogenesis must account for its age-dependence; association with amyloid precursor protein, tau, and apolipoprotein E biology; connection with vascular, inflammation, and insulin signaling changes; and systemic features. Mitochondria and molecular phenomena influenced by mitochondria could potentially link these seemingly diverse characteristics. Mitochondrial biology can clearly initiate changes in pathways tied to AD and mediate the dysfunction and degeneration that ultimately produces the clinical phenotype. To some, a mitochondrial cascade hypothesis seems particularly straightforward. Aspects of this hypothesis still require extrapolation or leverage exploratory data, but knowledge accumulating over decades argue the validity of at least some of its tenets. Alternative AD hypotheses may yet come to account for mitochondria-related phenomena, but in the absence of this happening an overarching primary mitochondrial cascade hypothesis will continue to evolve and attract interest.

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