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Mitochondrial Dysfunction in Aging and its Implications for Neurodegenerative Diseases

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Opinion Article

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DESCRIPTION

Mitochondria are essential organelles in eukaryotic cells, responsible for energy production, metabolic regulation and maintaining cellular homeostasis. As the primary source of Adenosine Triphosphate (ATP), they play a major role in cellular activities such as ion balance, apoptosis, and cell signaling. However, with age, mitochondrial function tends to decline, contributing to the aging process and playing a significant role in the onset and progression of various age-related diseases, particularly neurodegenerative disorders. Mitochondrial dysfunction in aging is a central theme in the study of cellular degeneration and is associated with the pathophysiology of diseases like Alzheimer's, Parkinson's and Huntington's disease.

One of the most important factors in mitochondrial dysfunction is the accumulation of oxidative damage over time. Mitochondria produce Reactive Oxygen Species (ROS) as byproducts of oxidative phosphorylation, the process by which ATP is generated. While ROS are typically neutralized by cellular antioxidants, the constant production of ROS over time can overwhelm the cell's defense mechanisms, leading to oxidative stress. This stress damages mitochondrial components, including proteins, lipids and DNA. The mitochondrial genome is particularly susceptible to oxidative damage because it lacks the robust repair mechanisms found in nuclear DNA. Mutations in mitochondrial DNA (mtDNA) can impair mitochondrial function, leading to reduced energy production and further exacerbating oxidative damage, creating a vicious cycle that accelerates aging and the onset of disease.

The age-related decline in mitochondrial function also affects other critical processes in neurons. Mitochondria are vital for calcium homeostasis, which is crucial for maintaining synaptic activity and overall neuronal function. Impaired mitochondria are less efficient at buffering calcium, leading to elevated intracellular calcium levels. This dysregulation contributes to neuronal excitotoxicity, a process where excessive calcium influx triggers harmful cellular events,

including the activation of enzymes that break down cellular components, and eventually leads to cell death. Neuronal cells, particularly those in the brain, are highly dependent on mitochondria due to their high energy demands. When mitochondrial dysfunction occurs, neurons become more vulnerable to damage and degeneration.

Mitochondrial dysfunction is a key feature in neurodegenerative diseases like Alzheimer's, Parkinson's, and Huntington's. In Alzheimer's, amyloid-beta plaques and tau tangles accumulate, disrupting mitochondrial function and impairing mitochondrial trafficking within neurons, which affects cellular metabolism. In Parkinson's, mutations in genes like PINK1 and Parkin, essential for mitochondrial quality control, contribute to mitochondrial dysfunction and dopaminergic neuron loss. This leads to energy deficits and the buildup of damaged mitochondria, promoting neuronal death.

Similarly, in Huntington's disease, the toxic huntingtin protein, resulting from an expanded CAG repeat, disrupts mitochondrial dynamics, impairing processes like fission and fusion. This leads to mitochondrial fragmentation, loss of function, and increased oxidative stress, which accelerates neurodegeneration.

These insights into mitochondrial dysfunction offer potential therapeutic strategies. Compounds like coenzyme Q10, which enhances mitochondrial electron transport, and antioxidants that reduce oxidative stress are being explored. Additionally, approaches to promote mitochondrial biogenesis or facilitate the removal of damaged mitochondria through autophagy are under investigation. While promising, challenges remain in developing effective treatments due to the complexity of mitochondrial involvement in these diseases.