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The Role of Mitochondrial Genetics in Aging and Neurodegenerative Disorders

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Introduction

Mitochondria, often referred to as the "powerhouses" of the cell, play a crucial role in energy production, cellular metabolism, and survival. Unlike other organelles, mitochondria possess their own genome, known as mitochondrial DNA (mtDNA), which encodes essential components of the oxidative phosphorylation system. Mutations and alterations in mitochondrial genetics have been strongly linked to the processes of aging and the onset of neurodegenerative disorders. Because the brain is highly energy-dependent, disruptions in mitochondrial function directly impact neuronal survival and cognitive performance. Understanding mitochondrial genetics provides critical insights into the mechanisms of aging and the pathology of diseases such as Alzheimer's, Parkinson's, and Huntington's disease [1].

Description

Aging is strongly associated with a gradual decline in mitochondrial efficiency and accumulation of mtDNA mutations. Reactive oxygen species (ROS), generated as byproducts of oxidative phosphorylation, damage mtDNA over time, leading to impaired respiratory chain activity and reduced ATP production. This progressive decline contributes to cellular senescence and tissue dysfunction, hallmarks of aging. Unlike nuclear DNA, mtDNA lacks robust repair mechanisms, making it more vulnerable to oxidative damage. Consequently, the accumulation of mitochondrial mutations is thought to be a key driver of age-related physiological decline and increased susceptibility to degenerative diseases [2].

In neurodegenerative disorders, mitochondrial dysfunction is a central pathological feature. In Alzheimer's disease, mtDNA mutations and altered dynamics impair energy metabolism, exacerbate oxidative stress, and accelerate amyloid-β and tau pathology. Similarly, in Parkinson's disease, mutations in genes regulating mitochondrial quality control—such as *PINK1* and *PARKIN*—lead to defective removal of damaged mitochondria (mitophagy), resulting in neuronal death in the substantia nigra. Huntington's disease is also linked to mitochondrial abnormalities, where mutant huntingtin protein disrupts mitochondrial energy production and calcium homeostasis. These insights highlight the critical role of mitochondrial genetics in maintaining neuronal function and resilience [3].

Recent advances in molecular biology and genetics are paving the way for potential therapeutic interventions targeting mitochondrial dysfunction. Strategies include antioxidant therapies to reduce oxidative stress, gene-editing tools to correct pathogenic mtDNA mutations, and mitochondrial replacement therapies that transfer healthy mitochondria into affected cells. Additionally, lifestyle interventions such as caloric restriction, exercise, and mitochondrial-targeted supplements (e.g., coenzyme Q10, NAD+ precursors) have shown promise in enhancing mitochondrial health and slowing neurodegenerative processes. These approaches represent an evolving frontier in combating aging and neurological decline through manipulation of mitochondrial genetics [4,5].

Conclusion

Mitochondrial genetics lies at the intersection of aging and neurodegeneration, influencing both the pace of biological

decline and the vulnerability of neurons to disease. Mutations in mtDNA, impaired energy production, and dysfunctional quality-control mechanisms collectively contribute to cellular aging and the progression of disorders such as Alzheimer's and Parkinson's disease. By targeting mitochondrial genetics and function, researchers aim to develop innovative therapies that extend healthspan and preserve cognitive function. As our understanding deepens, mitochondria may hold the key not only to unraveling the mysteries of aging but also to developing effective treatments for some of the most challenging neurodegenerative diseases

Acknowledgement

None.

Conflict of Interest

None.

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