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Commentary

Mitochondrial Dynamics and their Impact on Human Health and Disease

Elias Romero*

Department of Biochemistry, University of Barcelona, Barcelona, Spain

DESCRIPTION

Mitochondria, long referred to as the powerhouses of the cell, have in recent years emerged as far more than static energy factories. They are dynamic, versatile organelles that undergo constant remodeling, fusion, and fission to meet the metabolic and physiological needs of the cell. Their functions extend beyond Adenosine Triphosphate (ATP) production to include calcium buffering, apoptosis regulation, Reactive Oxygen Species (ROS) generation, and biosynthesis of key metabolites. Increasingly, mitochondrial dynamics the balance between fusion and fission are recognized as critical determinants of cellular homeostasis, development, and disease. The ability of mitochondria to adapt structurally and functionally in response to stress, nutrient availability, and signaling cues positions them as central hubs of human physiology. Dysregulation of mitochondrial dynamics has been implicated in neurodegeneration, cancer, cardiovascular disease, metabolic disorders, and aging, making this an area of intense investigation in biology and medicine.

Fusion allows mitochondria to mix their contents, diluting damaged components and maintaining functional integrity, while fission enables segregation of dysfunctional mitochondria for degradation by mitophagy. This interplay ensures a healthy mitochondrial network capable of responding to cellular demands. Proteins such as Mitofusins (MFN1 and MFN2) and Optic Atrophy Protein 1 (OPA1) mediate fusion, while Dynamin-Related Protein 1 (DRP1) and FIS1 regulate fission. Mutations or dysregulation of these proteins often result in profound physiological consequences. For instance, mutations in *MFN2* are associated with Charcot-Marie-Tooth disease type 2A, a neurodegenerative disorder characterized by peripheral nerve degeneration. Similarly, altered DRP1 activity contributes to neuronal cell death in conditions such as Alzheimer's disease and Huntington's disease. These findings underscore the delicate balance of mitochondrial dynamics and its significance for human health.

The central nervous system, with its high energy demands and sensitivity to ROS, is particularly vulnerable to mitochondrial dysfunction. In Parkinson's disease, for example, mutations in *PINK1* and Parkin impair mitophagy, leading to accumulation of defective mitochondria that contribute to neuronal degeneration. Studies in experimental models show that promoting mitochondrial fusion or enhancing mitophagy can mitigate disease progression, highlighting potential therapeutic avenues. In Alzheimer's disease, altered mitochondrial dynamics contribute to synaptic dysfunction and amyloid-beta accumulation. Strategies aimed at restoring mitochondrial balance may therefore provide neuroprotective effects. Beyond neurodegeneration, mitochondrial dynamics play roles in neural development and plasticity, influencing axonal growth, synapse formation, and learning processes.

In cancer, mitochondrial remodeling supports the metabolic reprogramming that is a hallmark of tumor cells. While normal cells primarily rely on oxidative phosphorylation for energy, cancer cells often adopt glycolysis, even in the presence of oxygen, a phenomenon known as the Warburg effect. However, mitochondria remain crucial for biosynthetic processes and survival under metabolic stress. Altered dynamics in cancer cells promote resistance to apoptosis, enhance proliferation, and support metastasis. For instance, DRP1-mediated fission is often upregulated in cancer, and inhibiting fission can impair tumor growth in preclinical models. These findings highlight mitochondria as potential targets for anticancer therapies, though the challenge lies in selectively modulating dynamics in tumor cells without disrupting normal tissues.

Cardiovascular diseases also reflect the critical importance of mitochondrial dynamics. Cardiomyocytes, reliant on efficient ATP production, are particularly sensitive to mitochondrial dysfunction. During ischemia-reperfusion injury, excessive fission and ROS generation contribute to cell death, while enhancing fusion or promoting mitophagy confers cardioprotection. Experimental therapies targeting DRP1 inhibition or MFN2 activation are under exploration for treating heart disease. Similarly, mitochondrial dynamics influence vascular smooth muscle cell behavior, contributing to atherosclerosis and hypertension. These observations underscore

Correspondence to: Elias Romero, Department of Biochemistry, University of Barcelona, Barcelona, Spain, E-mail: elias.romero@ub.edu

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the systemic influence of mitochondrial health across different organs.

Metabolic disorders such as obesity and type 2 diabetes further illustrate the role of mitochondrial adaptability. In skeletal muscle and adipose tissue, impaired fusion-fission balance contributes to insulin resistance, altered lipid metabolism, and chronic inflammation. Mitochondrial dysfunction in pancreatic beta cells disrupts insulin secretion, exacerbating hyperglycemia. Lifestyle interventions such as exercise and caloric restriction improve mitochondrial function and dynamics, providing evidence that targeting mitochondrial health can restore metabolic balance. Nutritional factors including omega-3 fatty acids, polyphenols, and certain vitamins also modulate mitochondrial biogenesis and activity, supporting integrative approaches to disease management.

Aging represents perhaps the most universal manifestation of mitochondrial decline. Over time, cumulative damage, reduced mitophagy, and impaired dynamics lead to dysfunctional mitochondria that contribute to tissue degeneration, sarcopenia, cognitive decline, and frailty. The free radical theory of aging, though simplified, highlights the role of ROS in damaging mitochondrial DNA and proteins, accelerating senescence. Interventions such as caloric restriction, exercise, and pharmacological agents like resveratrol or NAD^+ precursors have been shown to preserve mitochondrial function and extend lifespan in model organisms. Translating these findings to humans remains a major scientific pursuit, with the promise of extending health span and mitigating age-related diseases.