Perspective

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Mitochondrial Dysfunction and Oxidative Stress: Emerging Mechanisms in Cellular Pathophysiology

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Introduction

Mitochondria, often termed the “powerhouses” of the cell, are essential organelles responsible for producing Adenosine Triphosphate (ATP) through oxidative phosphorylation, thereby sustaining vital cellular functions. Beyond energy generation, mitochondria also regulate cell death, calcium signalling, redox balance, and biosynthetic pathways, making them central to cellular homeostasis. However, when mitochondrial function is compromised, it triggers a cascade of metabolic and signalling disturbances that contribute to the pathogenesis of numerous diseases. Mitochondrial dysfunction is closely linked to oxidative stress, a condition characterized by excessive production of Reactive Oxygen Species (ROS) that overwhelm the cell’s antioxidant defences. Together, these processes form a vicious cycle mitochondrial damage increases ROS levels, and ROS further deteriorate mitochondrial integrity. This interplay between mitochondrial dysfunction and oxidative stress has been recognized as a fundamental mechanism underlying the development of various pathophysiological conditions, including neurodegenerative diseases, metabolic syndromes, cardiovascular disorders, and cancer [1].

Description

Mitochondria are not only energy-producing organelles but also dynamic regulators of cellular signalling and apoptosis. Under normal physiological conditions, mitochondria generate ROS as by-products of oxidative phosphorylation. Low to moderate ROS levels act as signalling molecules, regulating processes such as cell proliferation, autophagy, and immune responses. However, excessive ROS generation triggered by environmental toxins, genetic mutations, or metabolic stress can damage mitochondrial DNA (mtDNA), lipids, and proteins, leading to impaired respiratory chain activity. Such damage disrupts ATP synthesis, alters mitochondrial membrane potential, and triggers the release of pro-apoptotic factors like cytochrome c, ultimately promoting cell death [2].

In neurodegenerative diseases such as Parkinson’s and Alzheimer’s, impaired mitochondrial dynamics namely fission and fusion imbalances contribute to neuronal loss and synaptic failure Likewise, in metabolic disorders, defective mitochondria contribute to insulin resistance and lipid accumulation, exacerbating cellular stress and inflammation. The connection between mitochondrial dysfunction and oxidative stress extends beyond isolated organelle damage it represents a systemic failure of cellular resilience. When antioxidant systems such as superoxide dismutase (SOD), glutathione peroxidase, and catalase are unable to neutralize ROS, oxidative damage spreads to other cellular compartments, leading to genomic instability and chronic inflammation [3].

This oxidative burden activates signalling pathways like NF-κB and MAPK, which further drive the expression of pro-inflammatory and pro-apoptotic genes. In cardiovascular diseases, mitochondrial oxidative stress leads to endothelial dysfunction, reduced nitric oxide bioavailability, and cardiac remodelling. Emerging research also implicates mitochondrial-derived damage-associated molecular patterns (DAMPs) in triggering immune responses, linking mitochondrial distress to inflammatory pathologies therapeutically; strategies aimed at restoring mitochondrial function and redox balance are gaining traction [4,5].

Conclusion

Mitochondrial dysfunction and oxidative stress are intricately intertwined processes that lie at the heart of many chronic and degenerative diseases. Their bidirectional relationship creates a self-perpetuating cycle of cellular damage that compromises energy metabolism, signalling, and survival. As research continues to unravel the molecular underpinnings of mitochondrial pathology, it is increasingly evident that targeting mitochondrial health represents a promising therapeutic frontier.

Acknowledgement

None

Conflicts of interest

None

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