Mitochondria: Nexus of Health, Disease, Treatment

Hye-Jin Park
Department of Cellular Energy Systems, Yonsei University, Seoul, South Korea

*Corresponding Authors**

Hye-Jin Park
Department of Cellular Energy Systems, Yonsei University, Seoul, South Korea
E-mail: hyejin.park@yonsei.ac.kr

Received: 01-Jul-2025; **Accepted:** 08-Aug-2025; **Published:** 08-Aug-2025

Introduction

This review highlights the critical role of mitochondrial metabolism in driving cancer development and progression. It discusses how alterations in mitochondrial function, including glycolysis and oxidative phosphorylation, contribute to tumor growth, metastasis, and drug resistance. The article also explores various therapeutic strategies targeting mitochondrial metabolism as a promising avenue for cancer treatment[1].

This article delves into the critical role of mitophagy, the selective degradation of damaged mitochondria, in maintaining cardiac homeostasis. It discusses how impaired mitophagy contributes to various heart diseases, including ischemia-reperfusion injury, heart failure, and cardiomyopathy. The authors highlight the potential of targeting mitophagy pathways for therapeutic interventions in cardiovascular disorders[2].

This review provides a comprehensive overview of mitochondrial biogenesis, the process by which new mitochondria are formed. It details the intricate molecular mechanisms, including key transcription factors like PGC-1α, and their regulation. The article also discusses the implications of dysregulated biogenesis in various diseases and explores potential therapeutic strategies aimed at modulating mitochondrial content and function[3].

This article explores the expanding landscape of human diseases linked to mitochondrial DNA (mtDNA) mutations, moving beyond classical mitochondrial encephalomyopathies. It discusses how mtDNA mutations contribute to a wide range of conditions, including neurodegenerative disorders, cancer, and metabolic diseases, emphasizing the diverse clinical manifestations and the complex interplay with nuclear genome[4].

This review examines the crucial role of mitochondrial quality control mechanisms—including mitochondrial dynamics, biogenesis, and mitophagy—in maintaining neuronal health. It highlights how dysregulation of these processes in the brain contributes significantly to the pathogenesis of various neurodegenerative diseases, such as Alzheimer's and Parkinson's. The article proposes that targeting these pathways could offer therapeutic avenues[5].

This paper discusses the indispensable role of mitochondrial metabolism in the aging process and the development of age-related diseases. It emphasizes how alterations in mitochondrial function, including oxidative phosphorylation, fatty acid oxidation, and nutrient sensing, directly influence longevity and susceptibility to conditions like neurodegeneration and metabolic syndrome[6].

This article explores the dynamic nature of mitochondria, focusing on the processes of mitochondrial fusion and fission and their crucial roles in maintaining cellular homeostasis. It details how imbalances in these dynamics contribute to the development and progression of various diseases, including neurodegenerative disorders, cardiovascular diseases, and metabolic syndromes[7].

This article provides an in-depth look at mitochondrial reactive oxygen species (ROS), detailing their generation pathways within the mitochondria and their dual roles as signaling molecules and damaging agents. It discusses how dysregulation of mitochondrial ROS contributes to various pathophysiological conditions, including inflammation, aging, and chronic diseases, and highlights potential therapeutic strategies[8].

This review elucidates the multifaceted roles of mitochondria in modulating innate immune responses and inflammation. It explains how mitochondria act as signaling platforms, releasing DAMPs and regulating metabolic reprogramming in immune cells, thereby influencing host defense and inflammatory disease pathogenesis. The authors discuss therapeutic opportunities targeting mitochondrial pathways to control aberrant immune responses[9].

This article explores mitochondrial transplantation as an innovative therapeutic approach for mitochondrial diseases and other conditions characterized by mitochondrial dysfunction. It discusses the current progress, challenges, and future prospects of delivering healthy mitochondria into damaged cells or tissues to restore cellular energy production and function[10].

Description

Mitochondria are central to cellular function, with their metabolic processes critically impacting disease states. For instance, mitochondrial metabolism, including glycolysis and oxidative phosphorylation, is a key driver in cancer development, influencing tumor growth, metastasis, and drug resistance. Targeting these metabolic pathways shows promise for cancer therapy [1]. Beyond cancer, mitochondrial metabolism also serves as a central hub in the aging process and the pathogenesis of age-related diseases.

Cite this article: Park H,(2025) Mitochondria: Nexus of Health, Disease, *Treatment. jmcb* 07: 170. DOI: 1037532.jmcb.2025.7.3.170

Alterations in mitochondrial function, such as oxidative phosphorylation, fatty acid oxidation, and nutrient sensing, directly influence longevity and susceptibility to conditions like neurodegeneration and metabolic syndrome [6].

Maintaining mitochondrial health relies on robust quality control mechanisms. Mitophagy, the selective degradation of damaged mitochondria, is vital for cardiac homeostasis, and its impairment contributes to various heart diseases, including ischemia-reperfusion injury, heart failure, and cardiomyopathy. Exploring mitophagy pathways offers potential therapeutic interventions in cardiovascular disorders [2]. Mitochondrial biogenesis, the formation of new mitochondria, involves intricate molecular mechanisms and key transcription factors like PGC-1α. Dysregulated biogenesis has implications across various diseases, suggesting therapeutic avenues by modulating mitochondrial content and function [3]. The dynamic processes of mitochondrial fusion and fission are also crucial for cellular homeostasis. Imbalances in these dynamics contribute to the development and progression of diverse conditions, including neurodegenerative disorders, cardiovascular diseases, and metabolic syndromes [7].

The interplay of mitochondrial quality control mechanisms—including dynamics, biogenesis, and mitophagy—is especially critical for maintaining neuronal health. Dysregulation of these processes in the brain contributes significantly to the pathogenesis of various neurodegenerative diseases, such as Alzheimer's and Parkinson's. Targeting these pathways could offer promising therapeutic avenues for these conditions [5]. Furthermore, the expanding landscape of human diseases linked to mitochondrial DNA (mtDNA) mutations highlights their contribution to a wide range of conditions, extending beyond classical mitochondrial encephalomyopathies to include neurodegenerative disorders, cancer, and metabolic diseases. This emphasizes the diverse clinical manifestations and the complex interplay with the nuclear genome [4].

Mitochondrial reactive oxygen species (ROS) are integral to both cellular signaling and as damaging agents. An in-depth look at their generation pathways within mitochondria reveals their dual roles. However, dysregulation of mitochondrial ROS contributes to various pathophysiological conditions, including inflammation, aging, and chronic diseases. Identifying potential therapeutic strategies to modulate ROS levels is an active area of research [8]. Beyond ROS, mitochondria also elucidate multifaceted roles in modulating innate immune responses and inflammation. They act as signaling platforms, releasing damage-associated molecular patterns (DAMPs) and regulating metabolic reprogramming in immune cells, thereby influencing host defense and inflammatory disease pathogenesis. Therapeutic opportunities exist to control aberrant immune responses by targeting mitochondrial pathways [9].

Given the broad involvement of mitochondrial dysfunction in disease, innovative therapeutic strategies are emerging. Mitochondrial transplantation, for example, is explored as a novel approach for mitochondrial diseases and other conditions characterized by mitochondrial dysfunction. This therapy discusses the current progress, challenges, and future prospects of delivering healthy mitochondria into damaged cells or tissues to restore cellular energy production and function, offering a promising therapeutic direction [10].

Conclusion

Mitochondria play a crucial and multifaceted role in cellular health and disease. Their metabolic functions, encompassing glycolysis and oxidative phosphorylation, are central to cancer development, influencing tumor growth, metastasis, and drug resistance. Beyond metabolism, mitochondrial quality control mechanisms are vital. Mitophagy, the selective degradation of damaged mitochondria, is essential for cardiac homeostasis, with impairments contributing to heart failure and other cardiovascular diseases. Mitochondrial biogenesis, the process of forming new mitochondria, also impacts various conditions when dysregulated. The dynamic balance of mitochondrial fusion and fission is critical for cellular health; imbalances lead to neurodegenerative disorders, cardiovascular issues, and metabolic syndromes. Mitochondrial DNA (mtDNA) mutations are implicated in a broad spectrum of human diseases, extending beyond traditional mitochondrial encephalomyopathies to include neurodegenerative, cancer, and metabolic conditions. Furthermore, mitochondria are major sources of reactive oxygen species (ROS), which act as both signaling molecules and damaging agents, contributing to inflammation, aging, and chronic diseases. These organelles also modulate innate immune responses, acting as signaling platforms and influencing inflammatory disease pathogenesis. Given their pervasive involvement in disease, therapeutic strategies targeting mitochondrial pathways, including mitochondrial transplantation, represent promising avenues for treating diverse conditions characterized by mitochondrial dysfunction.

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