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# Generation of Mitochondria and its Biological Role of Neurodegenerative Diseases

**Anitha Homann***

*Department of Pharmacy, Osmania University, Hyderabad, Telangana, India*

## Description

Mitochondria are key cellular organelles that regulate a variety of vital physiological processes in animals' bodies. Mitochondria, as a primary oxygen consumer, consumes nearly all of the oxygen inhaled. Mitochondrial energy is needed to contract both voluntary and involuntary muscles [1,2]. Mitochondria effectively create energy that is necessary for practically all sorts of cellular processes. Additionally, the generated energy is needed to maintain ionic gradients across the plasma membrane, which are necessary for excitability of excitable cells, secreted material accumulation in vesicles, and vesicle fusion and cycling, which are required for neurotransmitter secretion [3]. As a result, a mitochondrial functional disorder can result in a variety of problems, including changes in tissue functions that can lead to disease, as well as substantial deficiencies in tissue function that can result in disability or death. In fact, mitochondria are required in the bodies of animals not only to supply energy, but also to sustain other physiological processes of cells, such as cellular calcium signalling, and any flaw in this process can lead to disease and dysfunction [4,5].

The mitochondrial respiratory chain is a very efficient system. It catalyses alternating one-electron oxidation-reduction processes, predisposing each electron carrier to oxygen side reactions. Under normal physiological settings, mitochondria have been proven to be the predominant intracellular generator of reactive oxygen species. According to estimations, reactive oxygen species production accounts for 1-2% of total daily oxygen consumption, and a woman weighing 60 kg generates 160-320 mmol of free radicals per day via cellular respiration, whereas a man weighing 80 kg generates almost 215-430 mmol per day. Neurodegenerative diseases are a diverse category of illnesses with distinct clinical signs and genetic causes. The disorders are characterised by the progressive loss of neuronal systems that are physiologically or physically related. Neurodegenerative disorders such as Parkinson's disease and Huntington's disease are common instances. Despite this diversity, mitochondrial dysfunction is thought to be a unifying underlying process that plays a role in several forms of neuronal degeneration. Mitochondrial malfunction has a wide range of negative effects on cellular functioning, including decreased energy production, cellular calcium buffering, phospholipase and protease activation, nitric oxide synthase activation, and reactive oxygen species formation. As a result, they play a critical role in ageing and can interact directly with a number of particular proteins implicated in hereditary types of neurodegenerative illnesses. Furthermore, a growing body of evidence suggests that mitochondrial dysfunction is associated to neurodegenerative diseases. However, our knowledge of the various routes through which mitochondrial malfunction affects physiological functioning is limited. The major goal of this paper is to summarise new research on the involvement of mitochondria in reactive oxygen species and their negative impact on neurodegenerative disease progression.

Neurodegenerative diseases are life-threatening illnesses that are quickly growing among the elderly, with the number of cases globally rapidly increasing. Patients, their families, and society all bear a significant health burden as a result of these diseases. The mechanisms involved in the pathogenesis and progression of neurodegenerative disorders are still being unravelled. However, certain molecular explanations underlying the development and pathophysiology of these diseases have been hypothesised, with functional abnormalities of mitochondria and oxidative stress being two main pathways thought to be implicated in disease progression. Reactive oxygen species are produced in brain tissues as a result of mitochondrial abnormalities, dopamine metabolism, and inflammatory neurons. As a result, future research should focus on protective biological mechanisms that contribute to the control of these biological processes. Various therapeutic techniques, such as restoring mitochondrial functions and lowering oxidative damage, have been developed based on the findings of earlier investigations. Despite promising results in model species, several clinical trials have failed to demonstrate an impact on disease progression. The failures of the tactics examined thus far should serve as a roadmap for future, more innovative strategies.

## Conflict of Interest

None.

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***Address for Correspondence:** Anitha Homann, Department of Pharmacy, Osmania University, Hyderabad, Telangana, India, E-mail: anitha_h@yahoo.com*



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