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Short Communication

# Mitochondrial Dysfunction Development and Its Impact on Parkinson's Disease

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## DESCRIPTION

Mitochondrial dysfunction is a key factor in the pathophysiology of Parkinson's Disease (PD), affecting both random and familial cases. This dysfunction is caused by a variety of reasons, including bioenergetic abnormalities, mutations in mitochondrial and nuclear DNA, changes in mitochondrial dynamics, and the presence of mutant mitochondrial proteins. The loss of dopaminergic neurons in the substantia nigra, which is a characteristic of Parkinson's disease, is strongly associated with mitochondrial dysfunction. Understanding the mechanisms behind mitochondrial failure in Parkinson's disease is critical for developing therapeutic techniques to address these faults and potentially slow the disease's progression, providing for future treatments [1].

Parkinson's Disease (PD) is a chronic and progressive neurological condition that affects around 1% of people aged 60 and up. It is defined by the loss of dopaminergic neurons in the substantia nigra pars compacta, which causes motor symptoms such as tremors, stiffness, and bradykinesia. However, the pathophysiology of Parkinson's disease is complex and involves multiple factors, such as genetic, environmental, and mitochondrial malfunction [2-5].

Mitochondria are vital organelles that provide energy, maintain calcium homeostasis, and regulate apoptosis. They are dynamic structures that experience fusion and fission events, which are critical to their function and integrity. Mitochondrial dysfunction has been linked in the etiology of various neurodegenerative illnesses, including Parkinson's.

The role of mitochondrial malfunction in Parkinson's disease was first hypothesized in the 1980s, when it was discovered that PD patients had lower activity of mitochondrial respiratory chain complex I in the substantia nigra. Since then, numerous investigations have validated the involvement of mitochondrial dysfunction in Parkinson's disease etiology.

There is evidence that mitochondrial dysfunction leads to Parkinson's disease (PD). To begin, mutations in genes encoding mitochondrial function proteins such as PINK1, Parkin, and DJ-1 have been linked to familial forms of Parkinson's disease. PINK1 and Parkin are involved in mitophagy, a process that selectively degrades damaged mitochondria, whereas DJ-1 plays a role in mitochondrial respiration and Reactive Oxygen Species (ROS) detoxification.

Second, investigations with neurotoxins that selectively target complex I of the mitochondrial respiratory chain, such as rotenone and Mitochondrial Permeability Transition Pore (MPTP), have been shown to cause dopaminergic neuron degeneration and motor impairments in animal models. These toxins disrupt mitochondrial function by blocking complex I activity, resulting in the buildup of ROS and oxidative stress [6-8].

Third, postmortem studies have revealed that Parkinson's disease patients had lower mitochondrial respiratory chain complex I activity and increased oxidative damage in the substantia nigra. Furthermore, electron microscope investigations have shown that PD patients have aberrant mitochondrial shape and distribution in afflicted brain areas.

One putative method by which mitochondrial malfunction contributes to Parkinson's disease development is the creation of oxidative stress. Mitochondria are the primary generator of ROS in cells, and defective mitochondrial activity can result in an increase in ROS generation, which can damage cellular components such as DNA, proteins, and lipids.

Increased oxidative stress has been reported in damaged brain regions in Parkinson's disease patients, and many markers of oxidative damage, such as protein carbonyls, lipid peroxidation products, and DNA oxidation products, have been found in the substantia nigra. In addition, decreased activity of antioxidant enzymes such as superoxide dismutase and glutathione peroxidase has been seen in PD patients, worsening oxidative damage.

Another way mitochondrial failure may contribute to Parkinson's disease development is by inducing apoptosis. Mitochondria regulate apoptosis, and failure in this mechanism can lead to cell death [9,10].

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In Parkinson's disease, the accumulation of damaged mitochondria and the accompanying release of pro-apoptotic proteins such cytochrome c can activate the intrinsic apoptotic pathway. This can cause the activation of caspases, which are proteases that cleave cellular components, eventually leading to cell death.

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