Hindawi

Parkinson's Disease

Special Issue on

Mitochondria: Key Organelle in Parkinson's Disease

CALL FOR PAPERS

Parkinson's disease (PD) is the second most common neurodegenerative disorder, characterized pathologically by loss of dopaminergic neurons in the substantia nigra pars compacta. As a consequence, PD patients show symptoms such as tremor, rigidity, or bradykinesia, but also nonmotor features (depression, sleep disorders, etc.). The etiology of PD is still unknown, involving genetic and environmental factors; however, mitochondrial dysfunction plays a central role in PD pathogenesis. In this regard, several PD-related proteins (PINK1, Parkin, DJ-1, LRRK2, and α-synuclein) are linked to mitochondria.

Mitochondria are highly dynamic organelles involved in essential cellular functions, including energy production, calcium homeostasis, metabolism of amino acids and lipids, mtDNA replication, programmed cell death, etc. Moreover, mitochondrial homeostasis is tightly regulated by several pathways, including mitochondrial biogenesis, remodeling (fusion/fission), and clearance of damaged mitochondria by autophagy (mitophagy), among others. Importantly, mitochondria and endoplasmic reticulum (ER) establish contact sites to maintain several of these pathways. Dopaminergic neurons are particularly sensitive to mitochondrial dysfunction, so alterations of these processes lead to a compromised cellular status.

Better understanding of mitochondrial quality control is essential in nondivisible cells, such as neurons, to prevent cell death and development of age-related neurodegenerative disorders (e.g., PD). Current therapeutic strategies in PD are based on slowing down the disease progression. However, they are not successful and potential clinical agents should be based on early biomarkers for PD and mitochondrial dysfunction is one of the most promising targets to be studied. The aim of this special issue is to provide new insights into molecular and cellular pathways related to mitochondria that influence the pathogenesis of PD to further develop new therapeutic approaches.

We invite investigators to contribute original research articles, review articles, and clinical studies that will help in the elucidation of the role of mitochondria in PD, to delay the onset and/or reduce the progression of this disease.

Potential topics include, but are not limited to:

- Mitochondrial dysfunction in the pathogenesis of PD
- Mitochondrial oxidative stress and antioxidant therapy in PD
- Mitochondrial biogenesis, dynamics (fission/fusion), and motility in PD
- Mitophagy
- Relationship between mitochondria and ER in PD
- Preclinical studies in animal models of PD
- Mitochondrial therapies for PD

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/pd/mkop/.

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