

Special Issue on **Oxidative Stress and Inflammation on Nondopaminergic Damage in Parkinson's Disease**

CALL FOR PAPERS

Parkinson's disease is a neurodegenerative disorder characterized by a slow and progressive loss of dopaminergic neurons in the substantia nigra pars compacta resulting in decreased striatal dopamine levels. Clinically, the dopaminergic degeneration is tightly linked with classic irreversible and incapacitating motor symptoms development, such as resting tremor, rigidity, bradykinesia, and postural instability. It is known that the dopaminergic damage involves the production of reactive oxygen species (ROS) through dopamine metabolism that produces superoxide anions, hydroxyl radicals, and hydrogen peroxide. Additionally, dopamine autoxidation produces dopamine-quinone that contributes for the oxidative stress in the cell.

Reactive species are also involved in the formation of Lewy bodies, α -synuclein, and ubiquitin aggregates which are pathological markers for Parkinson's disease. Contrary to the well-defined motor symptoms, the nonmotor symptoms associated with Parkinson's disease include olfactory deficits, constipation, sleep behavior disorders, cognitive impairment, and mood disturbances. These symptoms are currently still poorly understood and often not considered in diagnostic and therapeutic protocols. Moreover, there is considerable evidence that the nonmotor preclinical phase can begin more than 20 years before the motor impairments appearance that suggests the involvement of nondopaminergic damage. Among this evidence, recent studies have demonstrated the role of raphe nuclei (serotonergic neurons), locus coeruleus (noradrenergic neurons), nucleus basalis of Meynert (cholinergic neurons), prefrontal cortex, hippocampus, and amygdala as well as alterations in GABAergic and glutamatergic systems. However, it is unclear yet what or how important the contribution of oxidative stress/redox regulation is on the nondopaminergic cell damage observed in this disorder.

Thus, in this special issue, we invite investigators to contribute original research articles and comprehensive reviews aiming to evaluate the oxidative mechanisms causing the nondopaminergic damage in Parkinson's disease, as well as putative behavioral impairments. We are particularly interested in articles covering the identification of new relevant targets and/or pharmacological approaches.

Potential topics include but are not limited to the following:

- ▶ Human, animal, or in vitro studies including the oxidative stress mechanisms behind the nondopaminergic damage in Parkinson's disease
- ▶ The contribution of oxidative stress, inflammation, and aging on nondopaminergic damage in Parkinson's disease
- ▶ Contribution of redox deregulation in other neurotransmission systems in the development of dopaminergic lesions
- ▶ Behavioral evidence associated with oxidative stress in nondopaminergic cell damage in Parkinson's disease
- ▶ Recent insights into the relationship between redox regulation/oxidative stress and nonmotor symptoms of Parkinson's disease
- ▶ Characterizations of the role of antioxidants as a therapeutic approach for nondopaminergic damage in Parkinson's disease

Authors can submit their manuscripts through the Manuscript Tracking System at <https://mts.hindawi.com/submit/journals/omcl/cdpdd/>.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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