Hindawi

Parkinson's Disease

Special Issue on

Characterization of New Markers in Early Stage Experimental Parkinsonism

CALL FOR PAPERS

This special issue on Parkinson's disease will focus on the early events characterizing the preclinical phase of the disease pathology. The attention is directed to the role of multiple precocious factors responsible for the progressive disruption of dopaminergic signalling within the nucleus striatum and substantia nigra.

Neurotoxicity produced by reactive oxygen species released by an impaired mitochondrial activity and accumulation of toxic oligomeric proteins (i.e., alpha-synuclein) disrupting synaptic compartments by targeting ion channels and receptor function are potential targets of investigation and intervention for finding new possible mechanisms underlying early stages of Parkinson's disease.

We strongly encourage research focusing on the identification of early alterations underlying the initial dysfunction of the nigrostriatal system in experimental models of Parkinson's disease. Indeed, we believe that these studies could set the basis for the identification of early functional and structural markers of the disease and for setting up new therapeutic strategies that might parallel the conventional, clinical approaches in Parkinson's disease treatment.

Authors are invited to submit their research and perspective in the form of original articles or critical reviews of the literature, particularly focusing on early changes occurring in experimental models of Parkinson's disease by single or multiple approaches of investigations.

Potential topics include, but are not limited to:

- Characterization of the molecular, morphological, electrophysiological, or behavioural effects of striatal inflammation occurring at neuronal or glial level in the early phases of Parkinson's disease
- Identification of mechanism underlying the mitochondrial dysfunction produced by genetic alterations or neurotoxic agents in early Parkinsonism
- Assessment of the effects of misfolded proteins aggregation (i.e., alpha-synuclein, amyloid beta) on synaptic transmission of specific striatal neuronal populations
- Analysis of the effects related to disrupted neuronal ion channels/receptors at either pre- or postsynaptic sites within the striatal neurotransmitter signalling systems

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

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