

# CALL FOR PAPERS

Mutations in leucine-rich repeat kinase 2 (LRRK2) are the most common known cause of inherited forms of Parkinson's disease (PD) and genetic variations in LRRK2 locus are associated with an increased risk of developing sporadic PD. LRRK2 is a very attractive therapeutic target and inhibition of its kinase activity is currently one of the prevailing disease-modifying therapeutic strategies for PD. Even though more than 10 years of research have increased our knowledge of LRRK2 function and dysfunction dramatically, many questions remain unsolved. A thorough understanding of the physiological and pathological function of LRRK2 would massively increase our insight into PD and further pave the way for improved PD diagnosis and drug development.

We invite researchers to contribute original research papers as well as state-of-the-art review papers for the upcoming special issue which will cover all aspects of LRRK2 in PD.

Potential topics include but are not limited to the following:

- ▶ Anything related to LRRK2 that may directly or indirectly increase our understanding of Parkinson's disease, for example:
  - ▶ Biomarkers
  - ▶ Pathology
  - ▶ Animal models
  - ▶ Molecular and cellular (dys)function
  - ▶ Therapeutic strategies
  - ▶ Genetics
  - ▶ Neuroinflammation
  - ▶ Structure
  - ▶ High throughput screenings (e.g., lipidomics, proteomics, and RNAseq)
  - ▶ Relation with other PD-related genes

Authors can submit their manuscripts through the Manuscript Tracking System at <http://mts.hindawi.com/submit/journals/pd/lrrk/>.

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Friday, 24 March 2017

### First Round of Reviews

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