Topical Collection

LRRK2-Dependent Neurodegeneration in Parkinson's Disease

Message from the Collection Editors

Mutations in *LRRK2* have been recognized as the most common genetic cause of familial Parkinson's disease, and *LRRK2* itself is considered a risk factor in idiopathic Parkinson's disease. LRRK2 is a large multidomain protein with a GTPase and kinase catalytic core surrounded by protein–protein interaction domains. LRRK2 regulates several cellular functions, including vesicle trafficking, cytoskeletal dynamics, neurotransmitter release, synaptic plasticity, mitochondrial function, autophagy, and immune response. All of these functions are dysregulated in Parkinson's disease, suggesting LRRK2 may play a direct or indirect role. Indeed, preclinical studies have revealed that pathogenic *LRRK2* mutations, notably the p.G2019S substitution at the kinase domain, favor the degeneration of nigrostriatal dopaminergic neurons and formation of alpha-synuclein inclusions, which are neuropathological hallmarks of the disease. The enhancement of kinase activity proved to be instrumental for LRRK2-mediated neurodegeneration, leading to the development of LRRK2 kinase inhibitors as possible disease-modifying agents in Parkinson's disease.

Collection Editors

Dr. Michele Morari

Department of Neuroscience and Rehabilitation, Section of Pharmacology, University of Ferrara, Via Fossato di Mortara 17-19, 44122 Ferrara, Italy

Dr. Mattia Volta

Institute for Biomedicine, Eurac Research-Affiliated Institute of the University of Lübeck, 39100 Bolzano, Italy

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Cells
Editorial Office
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland
Tel: +41 61 683 77 34
cells@mdpi.com

mdpi.com/journal/cells

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Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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Dr. Alexander E. Kalyuzhny
Dental Basic Sciences, University of Minnesota, 308 Harvard St. SE, Minneapolis, MN 55455, USA

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