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LRRK2-Dependent Neurodegeneration 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

Message from the Collection Editors

Mutations in LRRK2 have been recognized as the most common genetic cause of familial Parkinson's disease, and LRRK2itself 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.













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