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# LRRK2-Dependent Neurodegeneration in Parkinson's Disease

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## 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.

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