Over the last few years, several genes for monogenic forms of Parkinson's disease (PD) have been identified. For example, point mutations as well as duplications and triplications have been identified in the gene for α-synuclein (SNCA) in rare families, and common variants in this gene are associated with the risk to develop sporadic PD. Mutations in the gene for LRRK2 are a much more common cause of autosomal-dominant PD, while mutations in several genes have been linked to recessive early-onset variants of PD: parkin, DJ-1, PINK1. These genes implicate various cellular subsystems in PD pathogenesis, such as the proteasomal protein degradation pathways, protection against oxidative stress and mitochondrial function.

Evidence is emerging that low penetrance variants in at least some of these but also several other genes also play a role in the etiology of the common sporadic form of PD. In addition, rare variants with moderate effect size in some other genes, such as the Gaucher's disease associated gene for glucocerebrosidase A (GBA), also significantly influence the disease risk in a subset of patients.

Thus, an increasingly complex network of genes, all contributing to disease risk and progression, is emerging. These findings provide the “genetic entry points” to identify molecular targets and readouts necessary to design rational disease-modifying treatments.