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Gene Technology in Parkinson's

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About the Study

Parkinson's disease (PD) is a neurodegenerative disease that primarily affects the elderly, with a median onset age of 60 years. The precise cause of this pathological change is unknown at this time. Genetic factors, environmental factors, ageing, and oxidative stress may all play a role in the degeneration of dopaminergic neurons in Parkinson's disease. The condition has significant autonomic, cognitive, behavioural, sensory, and sleep components, despite being best characterised as a movement disorder. The formation of intracellular inclusions known as Lewy bodies comprising α-synuclein and the loss of dopamine neurons, most often in the substantia nigra, are hallmarks of Parkinson's disease. The genetic susceptibility factors for Parkinson's disease have been detected, including nine genes related to heritable, monogenic forms of the disease. Over 41 genetic susceptibility loci have been linked with late-onset PD. A few genes have been characterised as potentially causal within these risk loci, but it is still unknown which genes are responsible for PD risk in the majority of cases. It is currently uncertain on a broader scale but on current scenario. There are currently 178 genes known to be linked to Parkinson's disease. The presumption is that genes linked to the same or similar diseases tend to congregate in the same molecular network neighborhood. As a result, determining the distance between candidate genes and known disease genes in the protein-protein interaction (PPI) network is an important step. 28 genes, including those encoding alpha-synuclein (SNCA), leucine-rich repeat kinase 2 (LRRK2), and microtubule-associated protein tau (MAPT), have been related and/or associated with PD in complex forms of parkinsonism, providing novel insight into disease aetiology. In families with multiple-incident parkinsonism, traditional linkage analysis has proved to be an effective method for identifying disease-associated genes and mutations. Although the molecular relationship between alpha-synuclein (SNCA), leucine-rich repeat kinase 2 (LRRK2), and microtubule-associated protein tau (MAPT) has yet to be studied, they deserve special attention in this framework of genomic exploration.

The SNCA gene in humans encodes a protein known as alpha-synuclein. It is contained in large quantities in the brain, but in lesser amounts in the heart, muscle, and other tissues. Alpha-synuclein is primarily found at the tips of neurons in specialised structures called presynaptic terminals in the brain. Alpha-synuclein interacts with phospholipids and proteins within these structures. Chemical messengers called neurotransmitters are released from synaptic vesicles by presynaptic terminals. Neurotransmitter release relays signals between neurons. Alpha-synuclein regulates DNA repair, including double-strand break repair. When alpha-synuclein is depleted in human cells, the number of DNA double-strand breaks (DSBs) produced after bleomycin exposure increases, and the ability to repair these DSBs decreases.

LRRK2, also known as dardarin and PARK8 (from its early association with Parkinson's disease), is a kinase enzyme that is encoded by the LRRK2 gene in humans. LRRK2 belongs to the family of leucine-rich repeat kinases. This gene's variants have been linked to an increased risk of Parkinson's disease. In vivo and in cultured neurons, expression of LRRK2 mutants linked to autosomal dominant Parkinson's disease induces dendritic tree shortening and simplification.

The tau proteins (or proteins, after the Greek letter that bears that name) are a collection of six highly soluble protein isoforms created by alternate splicing from the MAPT gene (microtubule-associated protein tau). According to the tau hypothesis, excessive or abnormal tau phosphorylation causes normal adult tau to convert into paired-helical-filament (PHF) tau and neurofibrillary tangles (NFTs). a The phosphorylation of NFTs is determined by the stage of the disease. At least 19 amino acids are phosphorylated in Alzheimer's disease; pre-NFT phosphorylation occurs at serine 119, 202, and 409, while intra-NFT phosphorylation occurs at serine 409.

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