Editorial

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Use of Gene Technology in Parkinson's Disease (PD)

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Editorial Note

Parkinson's Disease (PD) is a neurological illness that affects mostly the elderly, with a median onset age of 60 years. At this moment, the exact etiology of this pathological alteration is unknown. In Parkinson's disease, genetic factors, environmental variables, ageing, and oxidative stress may all have a role in the degeneration of dopaminergic neurons. Despite being classified as a movement disorder, the illness involves important autonomic, cognitive, behavioral, sensory, and sleep components. Parkinson's disease is characterized by the creation of intracellular inclusions known as Lewy bodies, which contain synuclein, and the death of dopamine neurons, most often in the substantia nigra. Parkinson's disease susceptibility genes have been discovered, including nine genes linked to heritable, monogenic variants of the illness. Late-onset PD has been associated to over 41 genetic susceptibility loci. Within these risk loci, a few genes have been identified as possibly causative, but it is still uncertain which genes are responsible for PD risk in the majority of patients. On a larger scale, it is currently questionable, but in the current circumstances, it is. There are 178 genes that have been associated to Parkinson's disease so far. Genes associated to the same or comparable disorders are thought to cluster together in the same molecular network neighborhood. As a result, calculating the distance between candidate genes and known illness genes in the Protein-Protein Interaction (PPI) network is crucial. 28 genes have been linked to PD in complicated types of Parkinsonism, including those Encoding Alpha-Synuclein (SNCA), Leucine-Rich Repeat Kinase 2 (LRRK2), and Microtubule Associated Protein Tau (MAPT), offering new insight into disease genesis. Traditional linkage analysis has proven to be an effective tool for finding disease-associated genes and mutations in families with multiple-incident Parkinsonism. Despite the fact that the molecular interaction between Encoding Alpha-Synuclein (SNCA), Leucine-Rich Repeat Kinase 2 (LRRK2), and Microtubule-Associated Protein Tau (MAPT) has yet to be investigated, they need special attention in this context of genomic investigation.

The alpha-synuclein protein is encoded by the SNCA gene in humans. Large levels are found in the brain, although smaller amounts are found in the heart, muscle, and other tissues. Alpha-synuclein is predominantly present in the brain's presynaptic terminals, which are specialized structures that contain the ends of neurons. Within these structures, alpha-synuclein interacts with phospholipids and proteins. Presynaptic terminals release chemical messengers called neurotransmitters from synaptic vesicles. The release of neurotransmitters transfers signals between neurons. DNA repair, particularly double-strand break repair, is regulated by alpha-synuclein. The frequency of DNA Double-Strand Breaks (DSBs) created following bleomycin exposure rises when alpha-synuclein is decreased in human cells, and the capacity to repair these DSBs reduces.

LRRK2, also known as dardarin and PARK8 (because to its early link to Parkinson's disease), is a human kinase enzyme encoded by the LRRK2 gene. LRRK2 is a member of the leucine-rich repeat kinase family. Variants of this gene have been related to a higher risk of Parkinson's disease. LRRK2 mutations associated to autosomal dominant Parkinson's disease cause dendritic tree shortening and simplification in vivo and in cultured neurons.

The tau proteins (or proteins, after the Greek letter that bears that name) are a group of six highly soluble protein isoforms derived from the Microtubule-Associated Protein Tau (MAPT) gene by alternative splicing. Excessive or aberrant tau phosphorylation, according to the tau hypothesis, causes normal adult tau to transform into Paired Helical-Filament (PHF) tau and Neurofibrillary Tangles (NFTs). The stage of the illness determines how phosphorylated NFTs are. In Alzheimer's disease, at least 19 amino acids are phosphorylated; preNFT phosphorylation takes place at serine 119, 202, and 409, whereas intraNFT phosphorylation takes place at serine 409.