The Movers and The Shakers: You Can't Dance Without Dopamine

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Parkinson's Disease (PD) is a progressive neurodegenerative illness characterized by rigidity, bradykinesia and resting tremor. Pathologically, PD involves the death of dopaminergic neurons in the substantia nigra of the brain. Two forms of PD exist: familial and sporadic. The causes of sporadic PD are still not fully understood, although α-synuclein, a neuronal protein, is found misfolded in neurons. Among familial forms, autosomal dominant PD is a result of mutations in one of two genes: α-synuclein, which accumulates in substantia nigra dopaminergic neurons, and UCH-L1, whose etiology remains unclear. Autosomal recessive PD involves mutation in parkin, a ubiquitin ligase involved in protein degradation. Until recently, the molecular mechanisms by which α-synuclein, UCH-L1, and parkin were involved in the pathogenesis of PD remained a mystery. Lately, scientists have made significant progress using *in vitro* models, and transgenic fly and mouse models to clarify α-synuclein dependent pathology and symptoms. Scientists have also discovered that mutant parkin leads to α-synuclein aggregation by preventing proper α-synuclein ubiquitination and degradation. Given this new information, and the inadequacies of current drug therapies, scientists are exploring gene therapy, stem cells, and protein therapies as alternative treatments for PD. Scientists remain hopeful that such research will eventually lead to a cure for Parkinson's disease.