



VirginiaTech®

School of Neuroscience

Faculty Candidate

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**“Mitochondrial Quality Control
and its Involvement in the
Pathogenesis of Parkinson’s Disease”**

January 24, 2017
3:30pm – 4:30pm
Kelly Hall 310



Parkinson’s disease (PD) is the most common motor deteriorating neurodegenerative disease causing selective degeneration of dopaminergic neurons. Although a majority of Parkinson's disease cases are sporadic, a handful of monogenic causes have been identified. Genetic and biochemical studies reveal that the products of two genes that are mutated in forms of autosomal recessive PD, PINK1 and Parkin work together in the same pathway to govern mitochondrial quality control. The kinase PINK1 accumulates on the outer membrane of damaged mitochondria, activates Parkin's E3 ubiquitin ligase activity, and recruits Parkin to the dysfunctional mitochondrion. Parkin ubiquitinates outer mitochondrial membrane proteins triggering a selective form of autophagy to degrade the damaged mitochondria. The function of PINK1 and Parkin bolsters previous evidence that mitochondrial damage is involved in the etiology of PD. Understanding the function of genes mutated in hereditary forms of PD yields insights into disease etiology and reveals new pathways in cell biology.

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