The PINK1 gene is mutated in autosomal recessive Parkinson’s disease/Parkinson’s disease with dementia (PD/PDD). PINK1 encodes a mitochondrially targeted serine-threonine kinase, and has been implicated in regulating mitochondrial function, dynamics and degradation via mitophagy. Neurons are able to undergo PINK1-independent mitophagy both in vitro and in vivo, and our studies indicate that upregulation of autophagy plays a neuroprotective role in PINK1-deficient systems. Recently, we have begun defining additional mechanism(s) by which PINK1 loss of function may contribute to neurodegenerative disease. Using neuronal cell lines, primary neurons and patient-derived cells, we have discovered new protein-protein interactions for PINK1 that regulate its stability and ability to support neuronal morphogenesis and dendritic branching. Inhibiting PINK1 degradation increases its bioavailability and protects against toxin-mediated neuronal injury.