## The role and regulation of PINK1/Parkin mitophagy in maintaining mitochondrial health

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Functional mitochondria are critically important for the maintenance of cellular integrity and survival. Two gene products mutated in familial parkinsonism, PINK1 and Parkin, function together to degrade dysfunctional mitochondria through a selective form of autophagy termed mitophagy. PINK1 accumulates on the surface of damaged mitochondria where it simultaneously recruits and activates Parkin's E3 ubiquitin ligase activity. This forms the basis of multiple signaling events that culminate in engulfment of damaged mitochondria within an autophagosome, and degradation by the lysosome. Autophagosomes are double membrane structures whose formation is driven by a core set of conserved proteins termed autophagy-related (Atg) proteins. Atg proteins govern autophagosome initiation from isolation membranes, and then expand these membranes to encapsulate damaged mitochondria destined for lysosomal degradation. Our research aims to understand how autophagosomes form around mitochondria during PINK1/Parkin mitophagy, and how Atg proteins drive the various stages of autophagosome biogenesis.