

Molecular mechanism of Parkin/PINK1-mediated mitochondrial degradation via autophagy pathway

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The quality of mitochondria, essential organelles that produce ATP and regulate many metabolic reactions, should be strictly monitored. The loss of mitochondrial quality control system leads to many types of neurodegenerative disorders such as Parkinson's disease. The two gene products mutated in the familial Parkinson's disease, Parkin and PINK1 have been identified as essential proteins for the clearance of the damaged mitochondria through autophagy machinery termed mitophagy.

Recent significant progress has been made in understanding how the mitochondrial kinase PINK1 and the E3 ligase Parkin work together to identify the damaged mitochondria. Furthermore, the coordinated crosstalk of ubiquitin phosphorylation and autophagy has been identified for effective elimination of the damaged mitochondria.

Here, I discuss the molecular mechanisms that we have identified such as Parkin recruitment to the damaged mitochondria, and precise autophagosomal formation to encapsulate the damaged mitochondria and beyond.