

New insight into Parkinson's disease

April 19 2010

New research provides crucial insight into the pathogenic mechanisms of Parkinson's disease (PD), a prevalent neurodegenerative disorder. The study appears in the April 19 issue of the *Journal of Cell Biology*.

The identification of inherited mutations in genes such as Parkin and PINK1 (PTEN-induced putative kinase 1) has revealed key factors in the development of familial forms of the disease. Parkin adds ubiquitin molecules to other proteins to trigger their degradation, while PINK1 regulates mitochondrial quality control. But how these two [genes](#) work together remains a mystery.

Now, Keiji Tanaka and colleagues show that PINK1 is rapidly and continuously degraded under steady-state conditions when mitochondria are healthy, and that a loss in mitochondrial membrane potential stabilizes PINK1's accumulation. Furthermore, PINK1 recruits Parkin from the cytoplasm to mitochondria with low membrane potential to initiate the disposal of damaged [mitochondria](#).

Interestingly, the ubiquitin ligase activity of Parkin is repressed in the cytoplasm under steady-state conditions; however, PINK1-dependent mitochondrial localization liberates the latent enzymatic activity of Parkin. Some pathogenic mutations of PINK1 and Parkin interfere with the aforementioned events, suggesting they play a role in causing the disease.

More information: Matsuda, N., et al. 2010. J. Cell Biol.
[doi:10.1083/jcb.200910140](https://doi.org/10.1083/jcb.200910140)

Provided by Rockefeller University

Citation: New insight into Parkinson's disease (2010, April 19) retrieved 18 April 2024 from <https://phys.org/news/2010-04-insight-parkinson-disease.html>

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