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Mitochondrial dynamics and Mitophagy in Parkinson's Disease: a fly point of view

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Abstract

Mitochondria are double membrane-bounded organelles residing in the cytoplasm of almost all eukaryotic cells, which convert energy from the disposal of organic substrates into an electrochemical gradient that is in turn converted into ATP. However, the ion gradient that is generated through the oxidation of nutrients, may lead to the production of reactive oxygen species (ROS), which can generate free radicals, damaging cells and contributing to disease. Originally described as static structures, to date they are considered extremely plastic and dynamic organelles. In this respect, mitochondrial dynamics is crucial to prevent potential damage that is generated by ROS. For instance, mitochondria elongate to dilute oxidized proteins into the mitochondrial network, and they fragment to allow selective elimination of dysfunctional mitochondria via mitophagy. Accordingly, mitochondrial dynamics perturbation may compromise the selective elimination of damaged proteins and dysfunctional organelles lead the development of different diseases including and to neurodegenerative diseases.

In recent years the fruit fly *D. melanogaster* has proved to be a valuable model system to evaluate the consequences of mitochondria quality control dysfunction *in vivo*, particularly with respect to PINK1/Parkin dependent dysregulation of mitophagy in the onset of Parkinson's Disease (PD). The current challenge is to be able to use fly based genetic strategies to gain further insights into molecular mechanisms underling disease in order to develop new therapeutic strategies.

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