

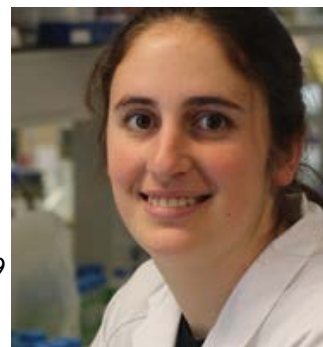
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Current Position

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Keywords

Parkinson's disease – PINK1 – phosphorylation – mitochondria

Science

We are interested in the function of the mitochondrial kinase PINK1, as mutations in the PINK1 gene cause early-onset recessive Parkinson's disease (PD). This neurodegenerative disorder is the most common movement disorder worldwide, but is unfortunately still incurable. The widely reported mitochondrial phenotypes resulting from loss of PINK1 function overlap with what is observed in sporadic PD cases, underscoring the importance of mitochondrial dysfunction in the development of PD.

PINK1 can be (auto)phosphorylated on different residues and while it is rapidly processed in healthy mitochondria, it accumulates on the outer mitochondrial membrane under depolarizing conditions. The functional consequences of this complex regulation remain poorly understood.

We use PINK1 with phosphomimetic mutations to explore how autophosphorylation regulates PINK1's activity and its different functions at the mitochondria. As such, we aim to gain a better understanding of the physiological roles of PINK1 and its substrates and how they are dysregulated in the context of PINK1-related PD.

Recent Fellowships

IWT doctoral fellowship 2010-2014