





## 1 JUNE

at 11.30 a.m. Room A103, Povo 1

## MITOCHONDRIAL HEALTH MAINTENANCE IN NEURONS

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Neurons with their elaborate and extended morphology must employ homeostatic mechanisms that allow neuronal mitochondria to exist far away from the cell body while still retaining a functional proteome. This process, called "Mitostasis", is most likely a finely tuned concert of mitochondrial transport, local protein synthesis and local degradation by proteasomal and autophagic mechanisms. Modulation of these processes may prove beneficial in the treatment of neurodegenerative diseases. However, the processes that allow local translation of mRNAs encoding for mitochondrial proteins are only partially understood. Using the transcript of PTEN-induced kinase 1 (PINK1) as a model substrate, we have discovered that this RNA associates with mitochondria specifically in neurons and uses mitochondria as a means of transport into axons and dendrites. This is a neuron specific mechanism driven by selective expression of an mRNA anchoring complex at the outer mitochondrial membrane. Regulation of this complex by modulation of intracellular signaling pathways impacts local PINK1 function in the removal of damaged organelles (mitophagy) and hence mitostasis. This connects signaling pathways known to be dysregulated in e.g. Alzheimer's disease with the fundamental regulation of mitochondria protein biogenesis and mitophagy in neurons.

## **Contacts**

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