MITOCHONDRIAL TRAFFICKING IN ALZHEIMER’S DISEASE AND HYPOXIC PRECONDITIONING: UNVEILING THE ROLE OF THE HYPOXIA UP-REGULATED MITOCHONDRIAL MOVEMENT REGULATOR

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Title of project/programme

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The project/programme is most relevant to:

Alzheimer's disease & other dementias

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Research Abstract

Axonal transport of mitochondria is mandatory for neuronal function. Among the core components that control mitochondrial transport are Miro and Milton, two proteins that tether mitochondria to kinesin motor proteins. Under hypoxia, the hypoxia up-regulated mitochondrial movement regulator (HUMMR) interacts with Miro-Milton complex, favoring anterograde movement of mitochondria. While moderate hypoxia triggers a pro-survival response (a phenomenon known as hypoxic preconditioning (HP)), severe hypoxia potentiates Alzheimer’s disease (AD), which is also characterized by defective mitochondrial trafficking. Data from our lab show that HP prevents AD-related pathological features including mitochondrial dysfunction. In light of this evidence, we aim 1) to decipher HUMMR role on defective mitochondrial trafficking in AD and 2) to uncover the potential protective effect of HP on HUMMR-mediated mitochondrial trafficking. Using in vitro and in vivo/ex vivo models, we expect to clarify the role of HUMMR in neurodegeneration and neuroprotection and obtain new information to help counteract AD.

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