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Optogenetic investigation of brain network dysfunction in Alzheimer's disease

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The dominant hypothesis in the Alzheimer's field is that protein deposition of amyloid-beta and phosphorylated tau are the cause of this brain disease. This has led to the development of many therapeutic strategies aimed at disrupting amyloid-beta and phosphorylated tau depositions, albeit with limited success, suggesting a lack of fundamental understanding of the molecular etiology of Alzheimer's disease and other related dementia. In this regard, we have found that phosphorylated tau plays an important part in synaptic function by promoting synaptic depression. In addition, by using revolutionary methods like optogenetics, we also found that brain network alterations happen before any sign of protein deposition. Here, we suggest that phosphorylated tau protein at the synaptic terminal is aiming to restore the network balance that is lost at early stages of the disease. In conclusion, our data suggest that studying the basic mechanism underlying memory formation and function will help to elucidate the complexity of brain diseases. In consequence, new tools like optogenetics and computational modelling may offer new therapeutic approaches aimed at restoring normal brain circuit functioning with superior prognosis.

Biography

Mondragon-Rodriguez Siddhartha graduated in Chemical Engineering from Michoacan University of Saint Nicolas of Hidalgo (UMICH) in 1996 - 2002. Master Degree (MsC) in Molecular Biomedicine from National School of Medicine from the National Polytechnic Institute of Mexico City. PhD from Center of Research and Advanced Studies of the National Politechnical Institute in August 2005 - August 2009. PosdoctoralFellow from Universite de Montreal. Faculte de Medecine, Departament de physiologie., Montreal, Qc, Canada. Posdoctoral Position in McGill University.

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