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MIPROTRAN: Transfer of misfolded protein as a pathogenetic mechanism in neurodegenerative disease

Project Description

Alzheimer's and Parkinson's diseases (AD and PD) are neurodegenerative disease that start late in life and where the symptoms gradually worsen. It is still largely unknown what causes these diseases. They are characterized by deposits of two proteins (called aggregated amyloid-ß protein and a-synuclein) in brain cells. These proteins take on an abnormal shape and become "misfolded". Our recent studies suggest that the changes in protein folding in the brain in AD and PD spread from one cell to another, through a novel and potentially very important mechanism. We base this idea on findings we obtained in animal models of AD and observations we made in two PD patients we grafted with fetal dopamine neurons. Our consortium will take a joint effort to investigate whether and how misfolded proteins move between cells in different cell- and animal models of AD, PD and a related disorder called multiple system atrophy (MSA). We will examine strategies to inhibit the transfer of the proteins between cells and study the cellular defense mechanisms against aggregation of misfolded proteins. Our ultimate goal is to lay the foundations for neuroprotective therapies in AD, PD and MSA, based on inhibiting transfer of misfolded proteins.

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