

STAD

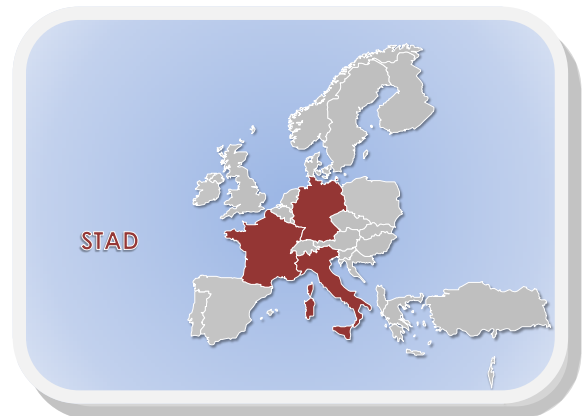
Synapse-to-nucleus communication in Alzheimer's disease

Understanding how fast events at synapses are converted into long-lasting changes of neuronal activity is a very important question in neuroscience. Several recent studies demonstrated that synapses and nuclei are connected by bidirectional communication routes that enable the efficient transfer of information and regulate the long-term structural changes of neuronal function. Moreover, several studies suggest that the disturbance of these communication routes is a common principle in many neurodegenerative diseases.

Our primary hypothesis is that alterations in synapse-to-nucleus transport represent a main event associated with synaptic dysfunction in Alzheimer's disease, which can be exacerbated by dysmetabolism. In particular, STAD will evaluate the properties of three synapse-to-nucleus messengers (Jacob, RNF10 and ICD) by testing whether interfering with their nuclear import can be beneficial or detrimental with respect to the progression of Alzheimer's disease.

This research question will be performed through the development of innovative experimental models, in which synaptic failure, amyloid load and dysmetabolism may reveal the complexity of the human pathology. Overall, STAD will provide a characterization of the role played by the different synapse-to-nucleus pathways in Alzheimer's disease and will generate novel animal models linking Alzheimer's disease and dysmetabolism, thus disclosing a picture of the complex interplay of pathways underlying Alzheimer's disease pathogenesis.

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