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Science

Alzheimer disease is characterized by two types of lesions in the brain: neurofibrillary tangles containing hyperphosphorylated tau proteins and extracellular deposits of amyloid beta ($A\beta$) forming senile plaques. Both the amyloidogenic processing of APP (Amyloid Precursor Protein) which produces $A\beta$ peptides and the non amyloidogenic pathway release the Amyloid Intracellular Domain (AICD). Because of its analogy with NICD (Notch Intracellular Domain) a well studied transcription factor, AICD has been thought to act on transcription to up-regulate the expression of several genes including those encoding the chemokine CXCL5, and the aquaporine AQP1. However, recent results showed that the increased expression of these genes does not result from transcriptional regulation but from epigenetic regulation. Epigenetic is defined as inheritable changes of DNA and chromatin conformation without altering the sequence of DNA. Epigenetic regulations affect both histones and DNA by post-transcriptional modifications like methylation and acetylation. The basis of this project is the understanding of the mechanisms implicated in the epigenetic regulation of the expression of CXCL5 and AQP1 genes.

We will focus on the interaction of AICD with the promoters of CXCL5 and AQP1 genes, and with the epigenetic machinery. We will also analyze the state of DNA methylation and the methylation/acetylation of histones in cellular models. We will extend our observations in normal and pathologic brains.