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Science

Amyloid Precursor Protein (APP) is a type I transmembrane protein, which processing is important for the pathology of Alzheimer's Disease (AD). In fact, APP is precursor to Amyloid Beta (Aβ) peptide that is the main component of senile plaques, one of the well-known neuropathological hallmarks that are found in the brain of AD patients.

Pathogenic processing of APP in AD is believed to be the reflection of a physiological pathway that goes wrong in the course of the disease. However, the function of APP and its processing under physiological conditions remains elusive. APP Knockout (KO) mice do not show any gross abnormality. As APP is a member of a conserved gene family that includes APP, APP-Like Protein 1(APLP1) and APLP2, the lack of any obvious phenotype in APP KO mice could be explained by compensation by other members of APP gene family. To tackle this problem knockout animals for APP combined with APLP1 and/or APLP2 have been generated. Results from genetic knockout studies indicate that there is functional redundancy between the members of APP family protein. Lethality of double knockout (dKO) APP/APLP2, APLP2/APLP1 or triple knockout (tKO) APP/APLPs animals around birth also shows that these family of protein has an important role late in development.

In this study we aim to gain insight into the function(s) of APP by studying APP tKO neurons both in vivo and in vitro. To this end, we use genetic knockout embryonic stem cells and siRNA strategies to investigate the function of APP gene family.

Selected publications

Bergmans BA, **Shariati SAM**, Habets RLP, Verstreken P, Schoonjans L, Müller U, Dotti CG and De Strooper B. (2010) Neurons generated from APP/APLP1/APLP2 triple knockout embryonic stem cell behave normally *in vitro* and *in vivo*: Lack of evidence for a cell autonomous role of APP in neuronal differentiation. Stem Cells doi: 10.1002/stem.296