β-Amyloid Oligomer-Induced Alzheimer’s Model Shows Synaptic Degradation for Use in Target Validation and Drug Development

A. Allouche, P. Goetghebeur, D. Rimet, N. Fischer, Y. Terroire, P. Housset, S. Colin, V. Koziel, A. Köpke, T. Pillot
SynAging SAS, 2 rue du Doyen Roubault, 54518 Vandoeuvre-les-Nancy, France

Oligomeric forms of β-amyloid peptide (AβO) are widely accepted as the initial cause for neurodegeneration in Alzheimer’s disease (AD).

Translational in vitro and in vivo models are essential to reduce the significant attrition during drug discovery for neurodegenerative diseases.

Here, we report highly reproducible in vitro and in vivo AD models, induced by a single icv injection of SynAging’s proprietary low-number AβO preparation.

AβO induce neuronal cell death in rodent primary neurons, as well as in iPS cell derived human neurons.

In rodent models (mice and rats), a single brain injection of minute amounts of AβO results in dramatic and fast impairment of cognitive functions fully established after one week and stable for months.

AβO-induced synaptic loss is shown.

AβO-induced release of pro-inflammatory cytokines is shown.

Unlike transgenic models, SynAging’s models imitate sporadic AD.

AβO Induce Pro-inflammatory Cytokine Release from Mouse Primary Astrocytes (ELISA measurements)

AβO-Induced Cognitive Decline and Synaptic Loss in Mice

AβO-Induced degeneration on Human iPS Cell-Derived Neurons

AβO induced neurotoxicity in rodent primary neurons

AβO Induced Neurotoxicity in rodent primary neurons

AβO induced decrease of hippocampal synaptic proteins (ELISA)