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In vivo Animal Models

Alzheimer's Disease

5xFAD Transgenic Mouse Model

5xFAD (Familiar Alzheimer Disease) mice bear five mutations, three in the APP695 gene [K670N/M671L (Swedish), I716V (Florida), V717I (London)] as well as two mutations in the presenilin 1 gene [M146L, L286V]. Transgene expression is driven by the neuron-specific Thy-1 promoter.

- Increased Aβ in cortex, hippocampus, plasma, and CSF
- Strong amyloid plaque accumulations
- Increased neurofilament light chain levels (NF-L)
- Increased neuroinflammation levels
- Vascular pathology (CAA)
- Spatial and long-term memory deficits

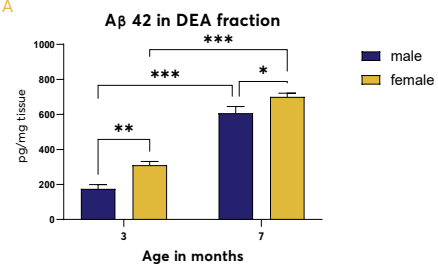
Amyloid-β Levels in Hippocampus

Figure 1

Figure 1:
Aβ42 level in the DEA (A) and FA fraction (B) of the hippocampus of 3- and 7-months old male and femal 5xFAD mice. Aβ42 levels in pg/mg hippocampal tissue. Mean + SEM; n = 8. Two-way ANOVA with Bonferroni 's *post hoc* test. *p<0.05; **p<0.01; ***p<0.001.

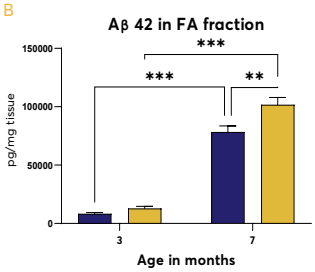
Soluble DEA Fraction

A



Insoluble FA Fraction

B



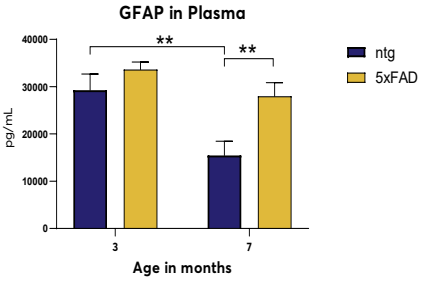
Astrocytosis and Neurodegeneration

Figure 2

Figure 2:
Quantification of GFAP and neurofilament light chain in the plasma of 5xFAD mice. GFAP (A) and NF-L (B) levels in pg/ml in the plasma of 3- and 7-months old 5xFAD mice compared to non-transgenic littermates. Mean + SEM; n = 8. Two-way ANOVA with Bonferroni 's *post hoc* test. **p<0.01; ***p<0.001.

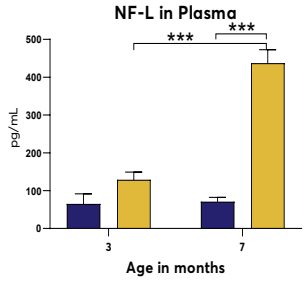
GFAP

A



Neurofilament Light Chain

B



Oakley et al. Intraneuronal β-amyloid aggregates, neurodegeneration, and neuron loss in transgenic mice with five familial Alzheimer's disease mutations: potential factors in amyloid plaque formation. J. Neurosci. 2006 Oct 4;26(40):10129-40.

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Discovery

Important note:
Representative data are shown throughout this document. However, biological variability might cause deviations from shown data.

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