



## APP<sub>SL</sub> Transgenic Mouse Model

The APP<sub>SL</sub> mouse model overexpresses human APP751 with Swedish and London mutations under the control of the neuron-specific murine Thy1 promoter.

- Early onset of brain pathology (3-4 months)
- Progressive learning & memory impairments in the Morris water maze (MWM)
- Progressive increase in amyloid plaque burden and CAA
- Early concomitant microgliosis & astrocytosis
- Increased oxidative stress & altered cholesterol profile

**Figure 1:** Morris water maze. Escape latencies of 6, 9 and 12 months old animals. Mean ± SEM; 6 and 9 months: n = 19 - 21; 12 months: n = 13 - 22; Two-way ANOVA with Bonferroni's post hoc test; \*p<0.05, \*\*p<0.01).

**Figure 2:** Quantification of Aβ levels in APP<sub>SL</sub> mice over age. Amount of Aβ1-38 (A), Aβ1-40 (B) and Aβ1-42 (C) in cortical (CTX) and hippocampal (HC) samples of 6-, 9- and 12-month old APP<sub>SL</sub> mice and 12-month old wild type (WT) littermates measured with MSD immunosorbent assay using the 4G8 antibody. n = 8 - 10 per group. Two-way ANOVA with Bonferroni's post hoc test. \*p<0.05, \*\*p<0.01; \*\*\*p<0.001.

**Figure 3:** Qualitative comparison of plaque pathology of APP<sub>SL</sub> transgenic mice at 6, 9 and 12 month of age compared to non-transgenic littermates. Tissue was labeled with antibody 6E10 (green), collagen IV (red) and DAPI (blue).

Figure 1

### Morris Water Maze

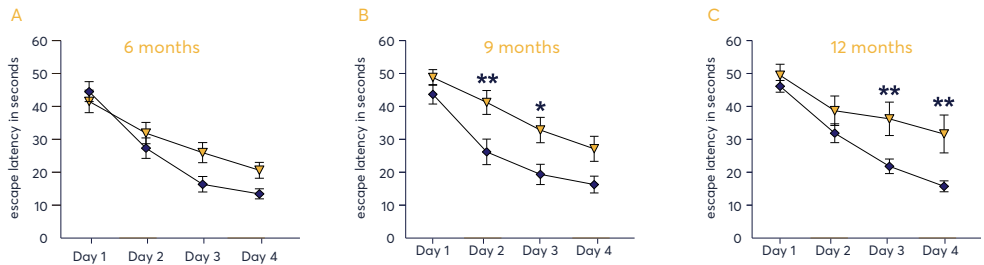


Figure 2

### Aβ Expression

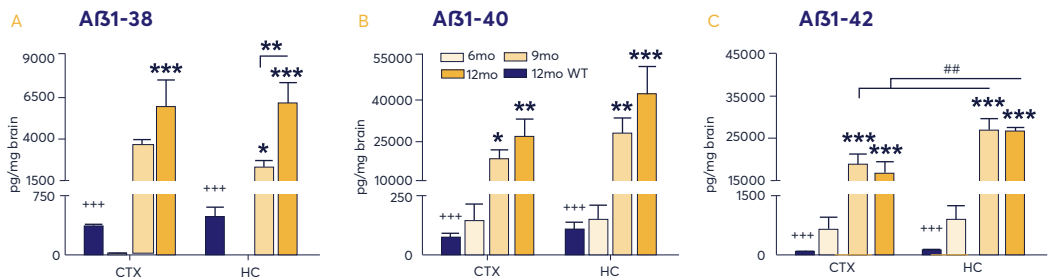


Figure 3

