

APP_{SL} Transgenic Mouse Model

The APP_{SL} 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 & astrogliosis
- Increased oxidative stress & altered cholesterol profile

Morris Water Maze

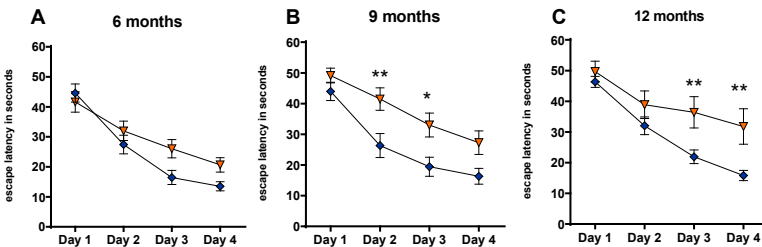


Figure 1. Morris water maze. Escape latencies of 6, 9 and 12 months old animals. Mean \pm SEM; 6 and 9 months: $n = 19 - 21$; 12 months: $n = 13 - 22$; Two-way ANOVA with Bonferroni's *post hoc* test; * $p < 0.05$, ** $p < 0.01$.

A β Expression

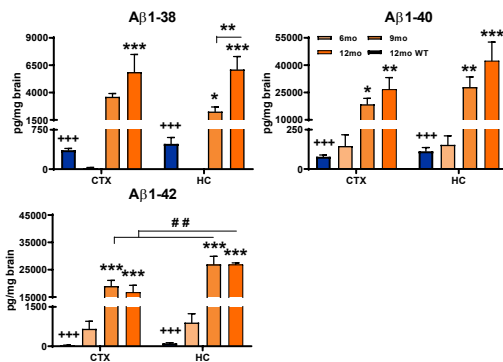


Figure 2: Quantification of A β levels in APP_{SL} mice over age. Amount of A β ₁₋₃₈ (A), A β ₁₋₄₀ (B) and A β ₁₋₄₂ (C) in cortical (CTX) and hippocampal (HC) samples of 6-, 9- and 12-month old APP_{SL} mice and 12-month old wild type (WT) littermates measured with MSD immunosorbent assay using the 4G8 antibody. $n = 8 - 10$ per group. Two-way ANOVA with Bonferroni's *post hoc* test. * $p < 0.05$, ** $p < 0.01$; *** $p < 0.001$.

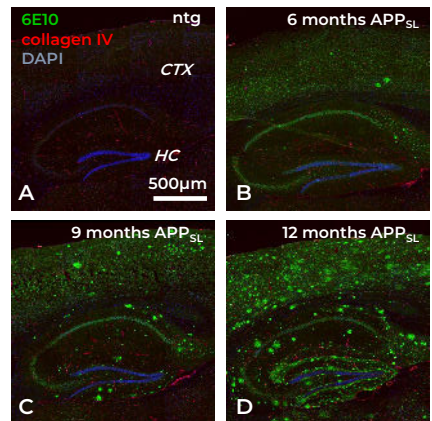


Figure 3: Qualitative comparison of plaque pathology of APP_{SL} transgenic mice at 6, 9 and 12 month of age compared to non-transgenic littermates. Tissue was labeled with antibody 6E10 (green), collagen IV (red) and DAPI (blue).