

Reducing hippocampal extracellular matrix reverses early memory deficits in a mouse model of Alzheimer's disease

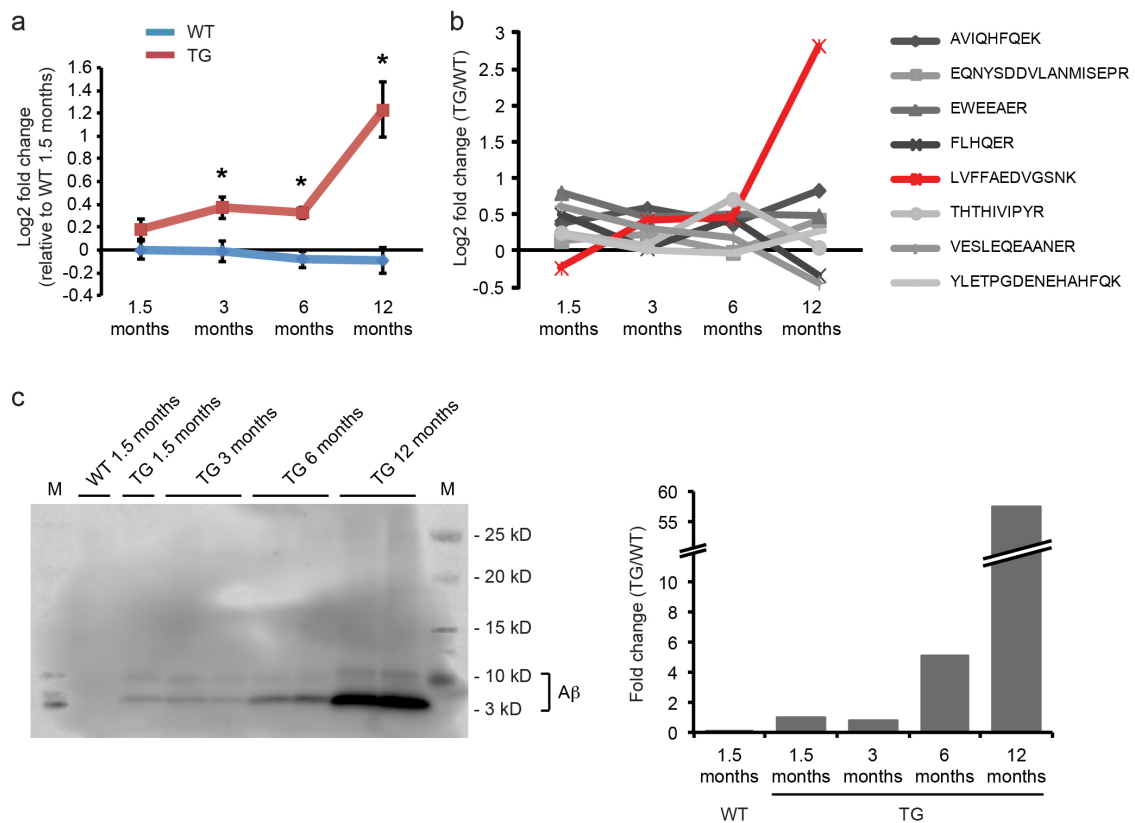
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Suppl. Fig. 2 APP/A β levels at hippocampal synaptic sites are significantly increased in APP/PS1 mice. **a** Proteomics analysis revealed a significant increase in APP levels at hippocampal synaptic sites in APP/PS1 transgenic (TG) mice compared with wildtype (WT) controls at 3, 6 and 12 months of age; $n = 5$ mice per genotype (SAM analysis; mean \pm SEM; *FDR < 10). **b** Increased levels of APP as detected by proteomics analysis are primarily due to an increase in the levels of the A β -specific peptide LVFFAEDVGSNK (indicated in red), in particular at 12 months of age. **c** The increase in synaptic A β levels is further confirmed by immunoblotting using the A β -specific antibody 6E10. An age-dependent accumulation of monomeric and low molecular weight oligomeric A β is observed, starting as early as 1.5 months of age