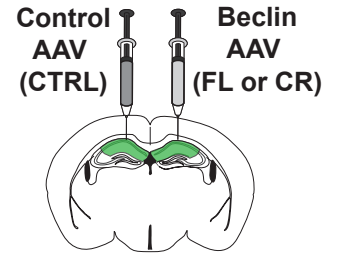
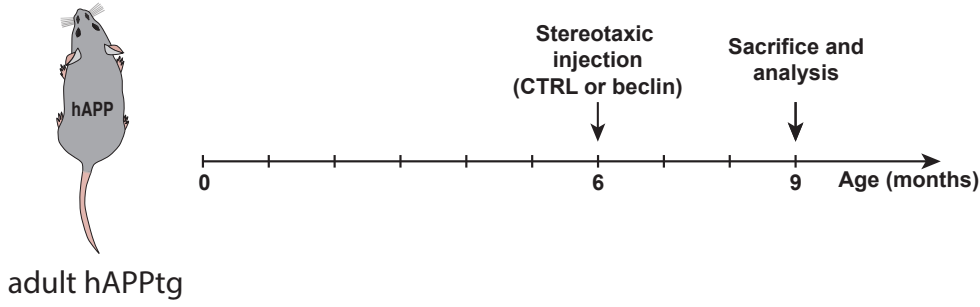
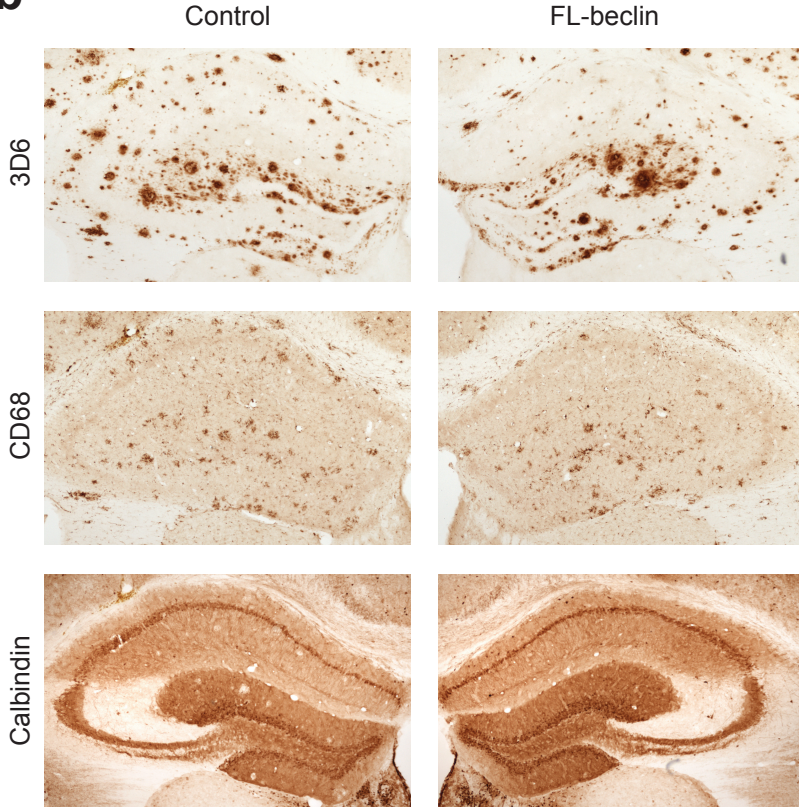


Supplementary Figure S7

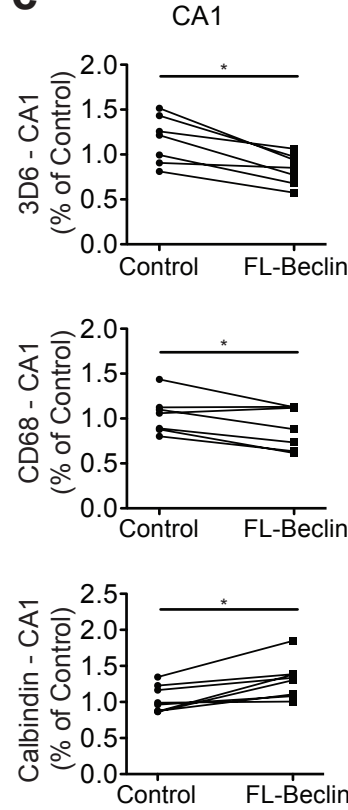
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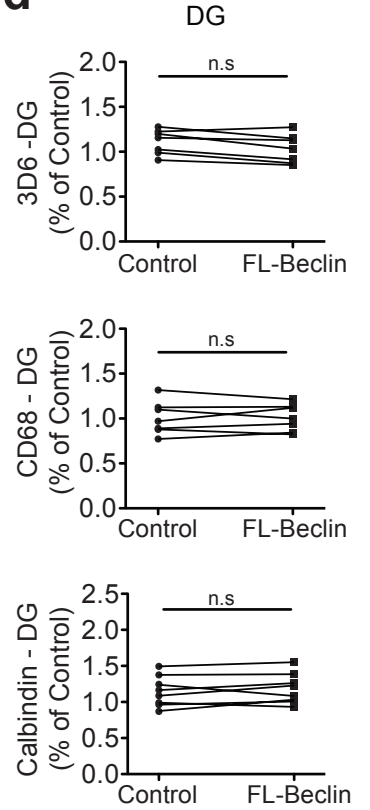
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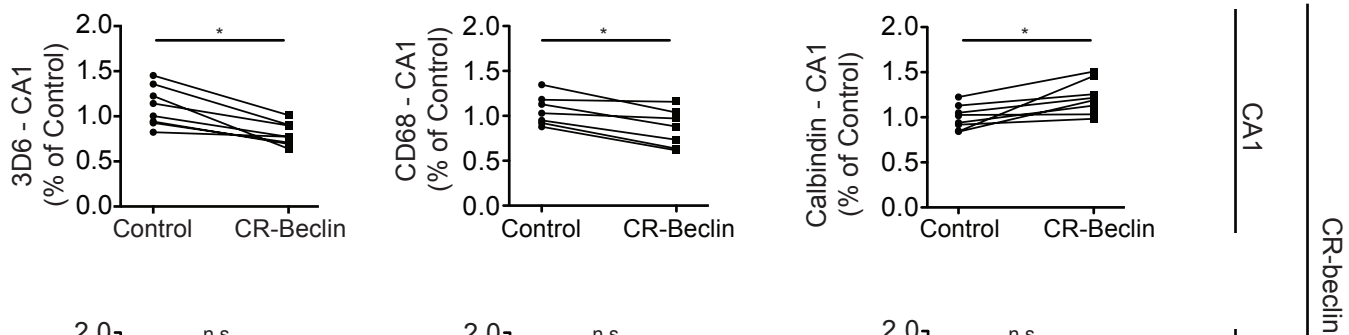
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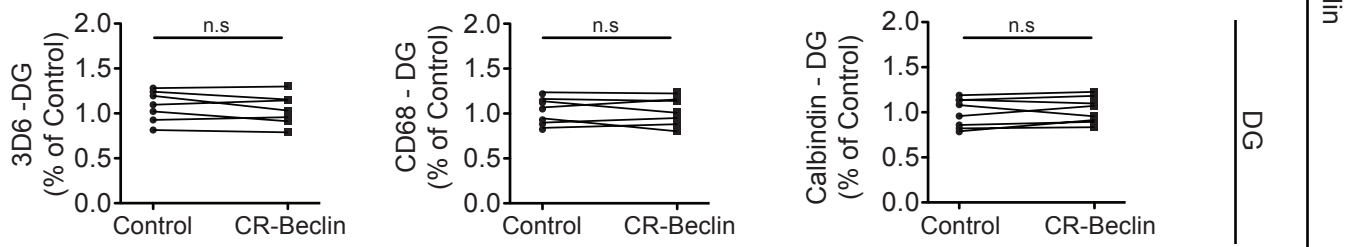
d



e



f



Supplementary Figure S7: Beclin 1 expression reduces Amyloid beta pathology in a hAPP mouse model.

a Schematic of experimental design and time line. 6 months old hAPP-transgenic mice were stereotaxically injected with FL-beclin or CR-beclin into the CA1 and Control AAV into the contralateral hemisphere. Dentate gyrus (DG) was used as a control area not targeted by the AAV injections. Mice were analyzed after 3 months. **b** Amyloid beta (A β) pathology and associated degenerative phenotypes were assessed by immunohistochemical detection of A β (using the 3D6 antibody (top panel), microglial activation marker CD68 (middle panel) and neuronal marker Calbindin (lower panel). Representative hippocampal images from adjacent sections of one animal expressing FL-beclin (right hemisphere) or control (left hemisphere). Scale bar 200 μ m. **c,d** Quantification of 3D6, CD68 and Calbindin immunostaining in the CA1 (**c**) and dentate gyrus area (**d**) of the hippocampus comparing the FL-beclin expressing to the contralateral control hemispheres. Beclin 1 and control AAV were selectively expressed in the CA1 area. **e,f** Quantification of 3D6, CD68 and Calbindin immunostaining in the CA1 (**e**) and dentate gyrus area (**f**) of the hippocampus in CR-beclin and control injected animals (n = 7 mice/group; 4-5 hippocampal sections/brain). Data expressed as mean value for each hemisphere of the same animal; *p < 0.05; compared by paired Student's t-test.