**Spatial reversal learning defect coincides with hypersynchronous telencephalic BOLD functional connectivity in APP$^{NL-F/NL-F}$ knock-in mice**

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## Supplementary Figures

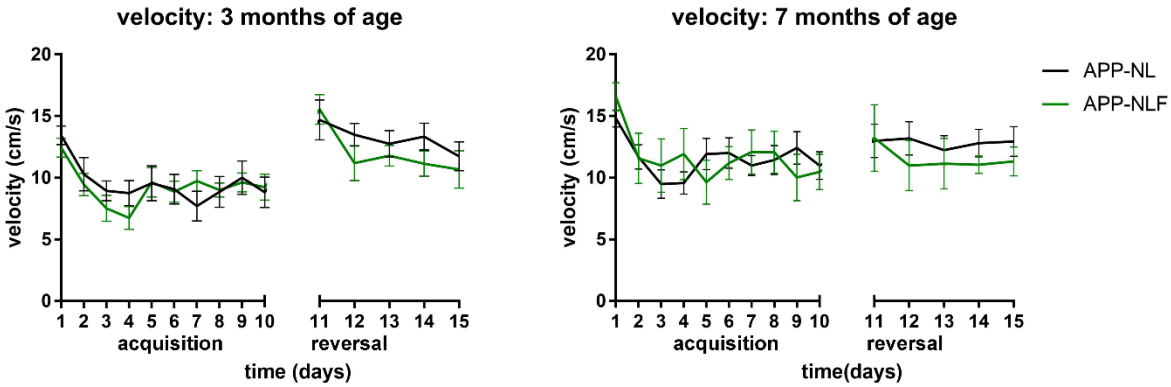


**Figure S1: Velocity during the Morris water maze behaviour task.** Velocity (cm/s) ± standard error shown for the acquisition and reversal phase of the Morris water maze task for 3 and 7 months old $APP^{NL/NL}$ and $APP^{NLF/NLF}$ mice. RM-ANOVA showed no significant differences between groups.

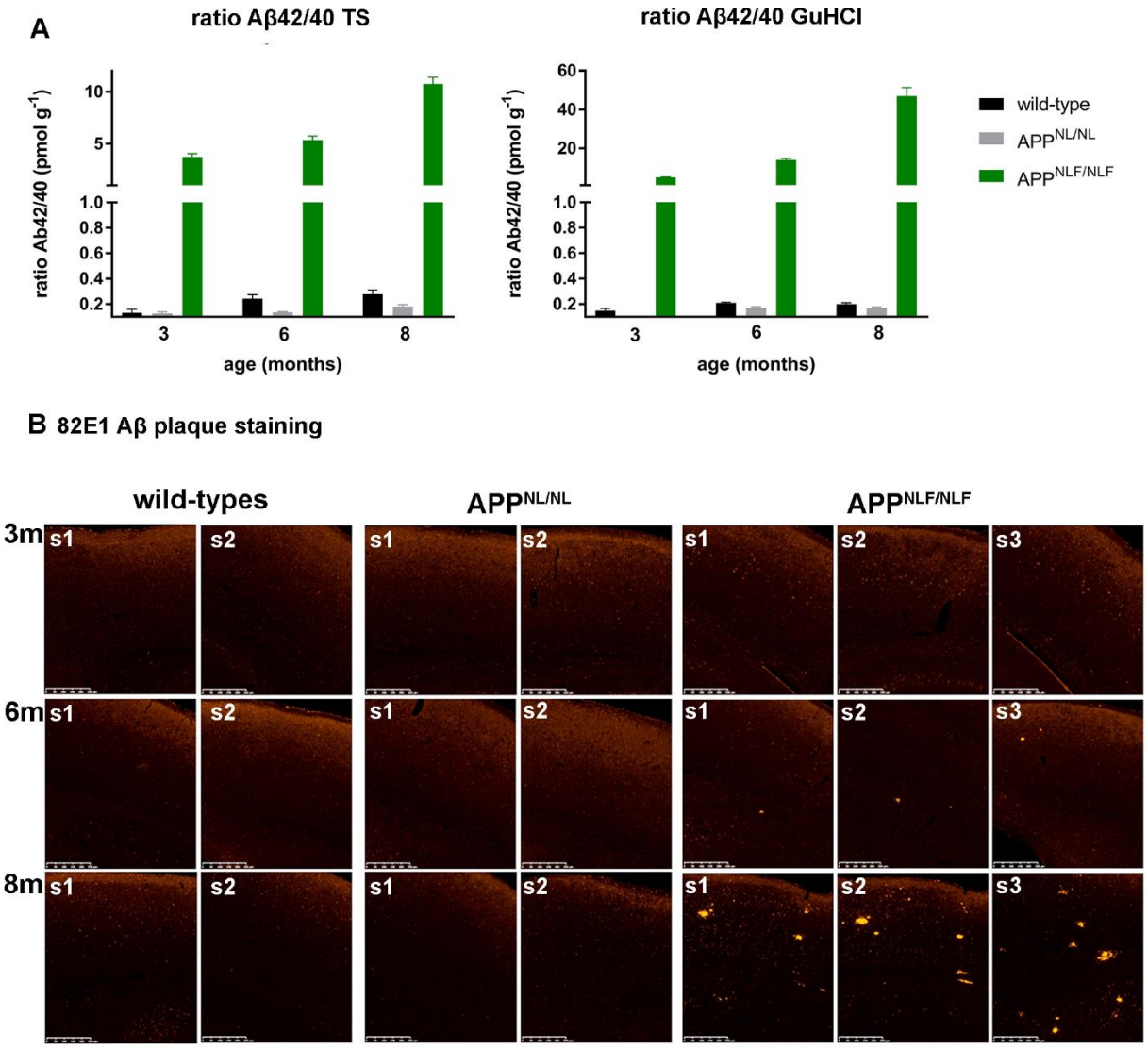


**Figure S2: Aβ pathology. A)** Biochemical quantities of Aβ42/40 ratio in the brains of wild-type, $APP^{NL/NL}$ and $APP^{NLF/NLF}$ mice at 3 (N=3/group), 6 (N=3/group) and 8 (wild-types N=3, $APP^{NL/NL}$ N=3, $APP^{NLF/NLF}$ N=4) months of age. Aβ levels (pmol/g) were measured from Tris-HCl-buffered saline (TS) and guanidine-HCl (GuHCl) fractions and quantified by sandwich ELISA as previously described[1]. Data are shown as ratio Aβ42/40 ± standard error. **B)** Brain sections from 3, 6 and 8 months old wild-type (N=2/age group), $APP^{NL/NL}$ (N=2/age group) and $APP^{NLF/NLF}$ (N=3/age group) were immunostained with 82E1 N-terminal specific Aβ antibody (IBL, Japan) to detect Aβ plaques using a previously described protocol[1]. Scale bars represent 250 µm.

**References:**

1. Saito, T. *et al.* Single App knock-in mouse models of Alzheimer's disease. *Nat Neurosci* **17,** 661–663 (2014).