Supporting Information

Montgomery et al. 10.1073/pnas.0902750106

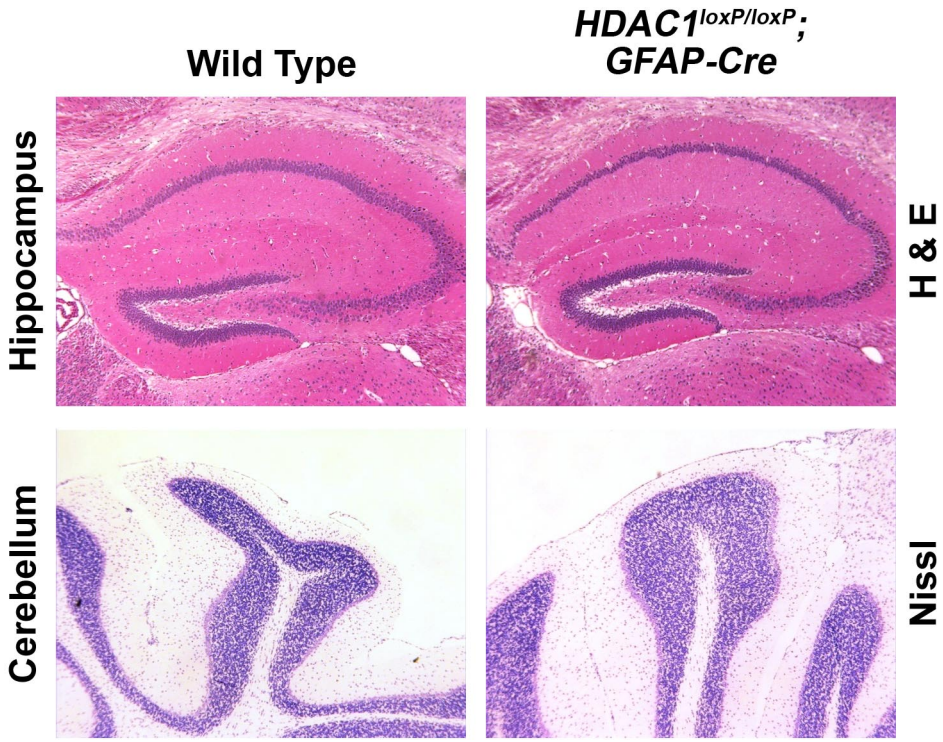

Fig. S1. Hematoxylin and eosin staining of hippocampus (*Upper*) and Nissl staining of cerebellum (*Lower*) from wild-type, *HDAC1*$^{loxP/loxP}$;*GFAP-Cre* mice showing normal hippocampal and cerebellar development in HDAC1 mutant mice.

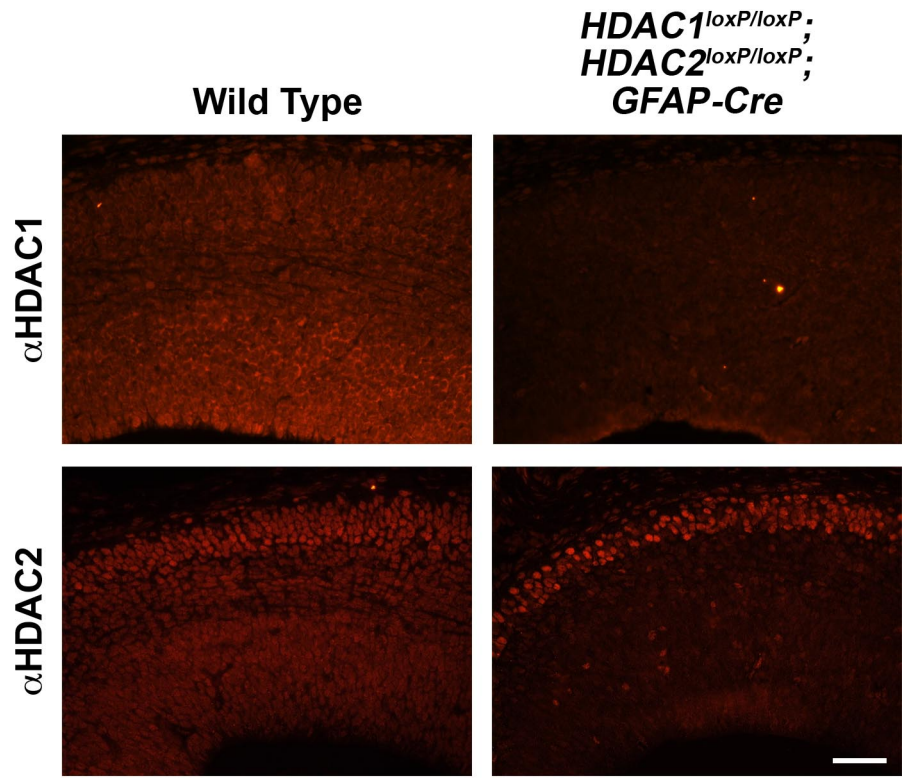

Fig. S2. Immunohistochemistry for HDAC1 (*Upper*) or HDAC2 (*Lower*) on the cortex of wild-type and *HDAC1*$^{loxP/loxP}$, *HDAC2*$^{loxP/loxP}$;*GFAP-Cre* mice to show deletion of HDAC1 and HDAC2 in the ventricular zone and cortical neurons. Staining of HDAC2 persists in the superficial cortical plate neurons. Scale bar, 40 μm.

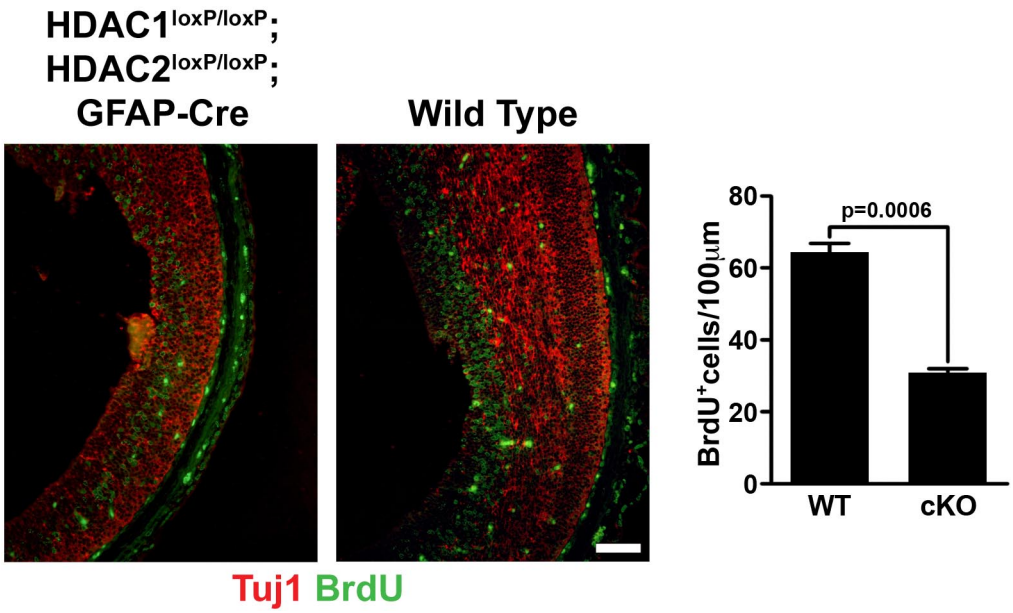

Fig. S3. Immunohistochemistry at E15.5 detecting S-phase neuronal precursors labeled by BrdU (green) and Tuj1 (red) on wild-type and mutant cerebrum cortex. Quantification of BrdU+ cells at E15.5 to assess proliferation. cKO, double conditional knockout. Scale bar, 40 μm.