Figure S2: Similar FE65, FE65L1 and FE65L2 expression patterns in WT mouse brains. In situ hybridization of brain slices with an FE65 antisense probe (A-B), an FE65L1 antisense probe (C-D) and an FE65L2 antisense probe (E-F) in adult WT brains (A, C and E), FE65L1−/− brain (B), FE65−/− brain (D) and FE65−/−;FE65L1−/− brain (F). Our in situ hybridization data concur with the published FE65 expression pattern in adult mouse brain (A) and show that the pattern of FE65 expression does not differ significantly in the FE65L1 null brain (B), indicating that loss of FE65L1 does not produce global or regional upregulation of FE65 expression. In situ hybridization analyses of mouse adult WT brain slices
using antisense probes specific for FE65L1 (C) and FE65L2 (E) revealed lower expression levels of FE65L1 and FE65L2 in the cortex compared to FE65 (FE65>FE65L2>FE65L1), but showed a similar spatial distribution. FE65L1 expression is not elevated in mice deficient for FE65 (D). The apparent increase in FE65 expression in the thalamus of FE65L1 null (D), and for FE65L2 in the olfactory bulb of the FE65<sup>−/−</sup>;FE65L1<sup>−/−</sup> mouse (F) is likely due to a different medial to lateral position of the sagittal sections depicted. Furthermore, Western blot analyses did not confirm increased FE65 protein in the subcortical area of FE65L1<sup>−/−</sup> brains (data not shown) nor was an increase in FE65L2 apparent (see Fig. S1C).