Supplemental Materials

Supplemental Table

Table S1 Clinical characteristics of patients with TLE and controls

Case	Sex/Age (years)	Duration (years)	Preoperative AEDs before surgery	Resection side	Pathological diagnosis
C 1	M/36	0	None	Right	N
C 2	M/29	0	None	Right	N
C 3	F/27	0	None	Left	N
C 4	M/25	0	None	Right	N
C 5	M/40	0	None	Left	N
C 6	F/34	0	None	Left	N
C 7	F/23	0	None	Left	N
E 1	M/36	27	VPA, CBZ, CZP	Left	NL, G
E 2	F/26	7	CBZ, VPA, TPM, OXC	Left	G
E 3	M/29	14	CBZ, PB, LTG, LEV	Right	NL, G
E 4	F/38	18	VPA, PB, CBZ, LTG	Left	G
E 5	M/24	13	CBZ, PHT, VPA, PB	Right	G
E 6	F/28	8	VPA, CBZ, TPM, PB	Left	NL, G
E 7	F/23	6	TPM, CBZ, VPA	Right	G

AEDs, antiepileptic drugs; C, control; CBZ, carbamazepine; CZP, clonazepam; E, epilepsy; F, female; G, gliosis; GBP, gabapentin; LEV, levetiracetam; LTG, lamotrigine; M, male; N, normal; NL, neuron loss; OXC, oxcarbazepine; PB, phenobarbital; PHT, phenytoin; TPM, topiramate; VPA, valproate.

Supplemental Figures

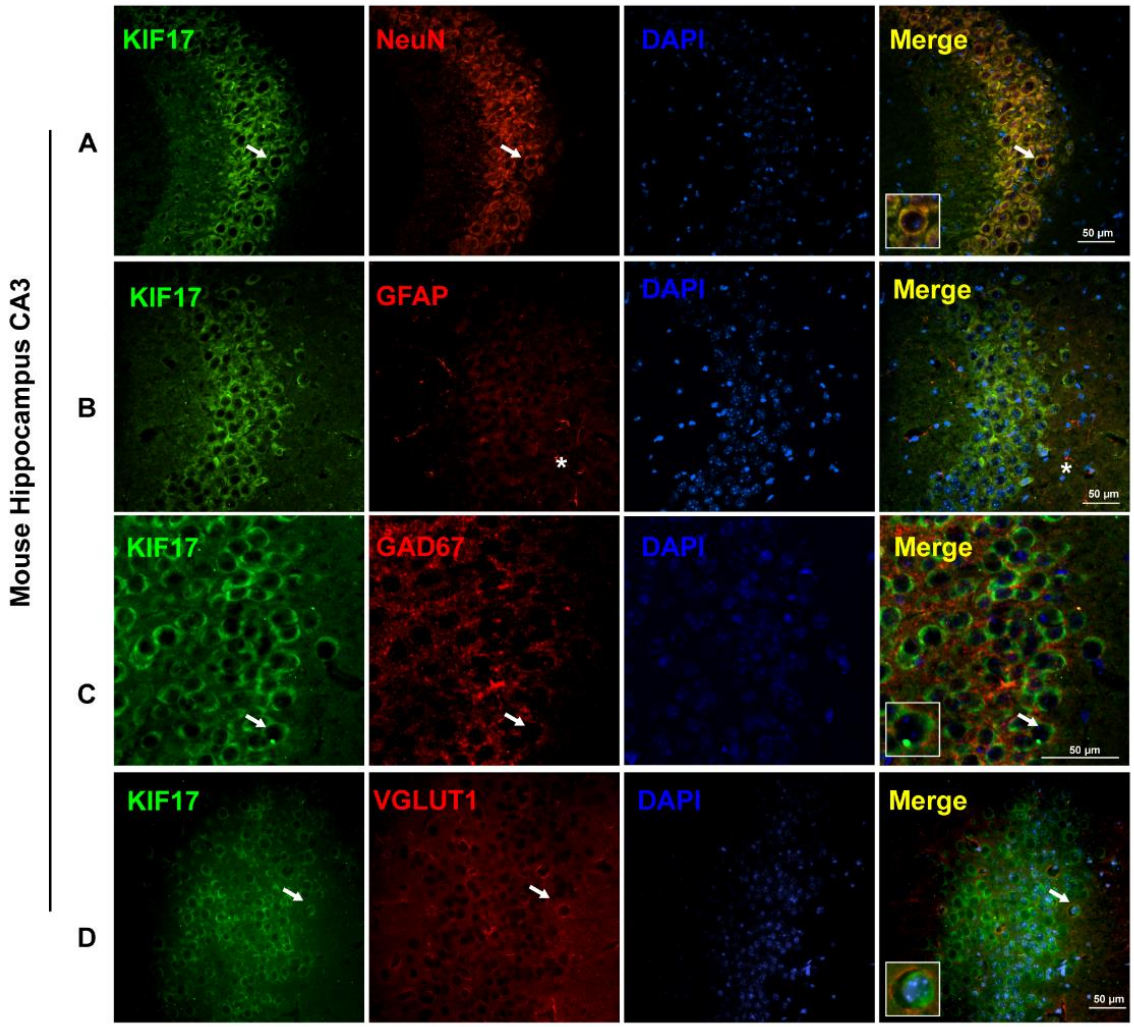

Fig. S1 Localization of KIF17 in control mouse brain. **A, B** In the mouse hippocampus CA3, KIF17 co-localizes with NeuN and rarely with GFAP (*astrocyte). **C, D** KIF17 co-localizes with a GABAergic neuronal marker (GAD67) and a glutamatergic neuronal marker (VGLUT1). Scale bars, 50 μm (arrow indicates positive cell).

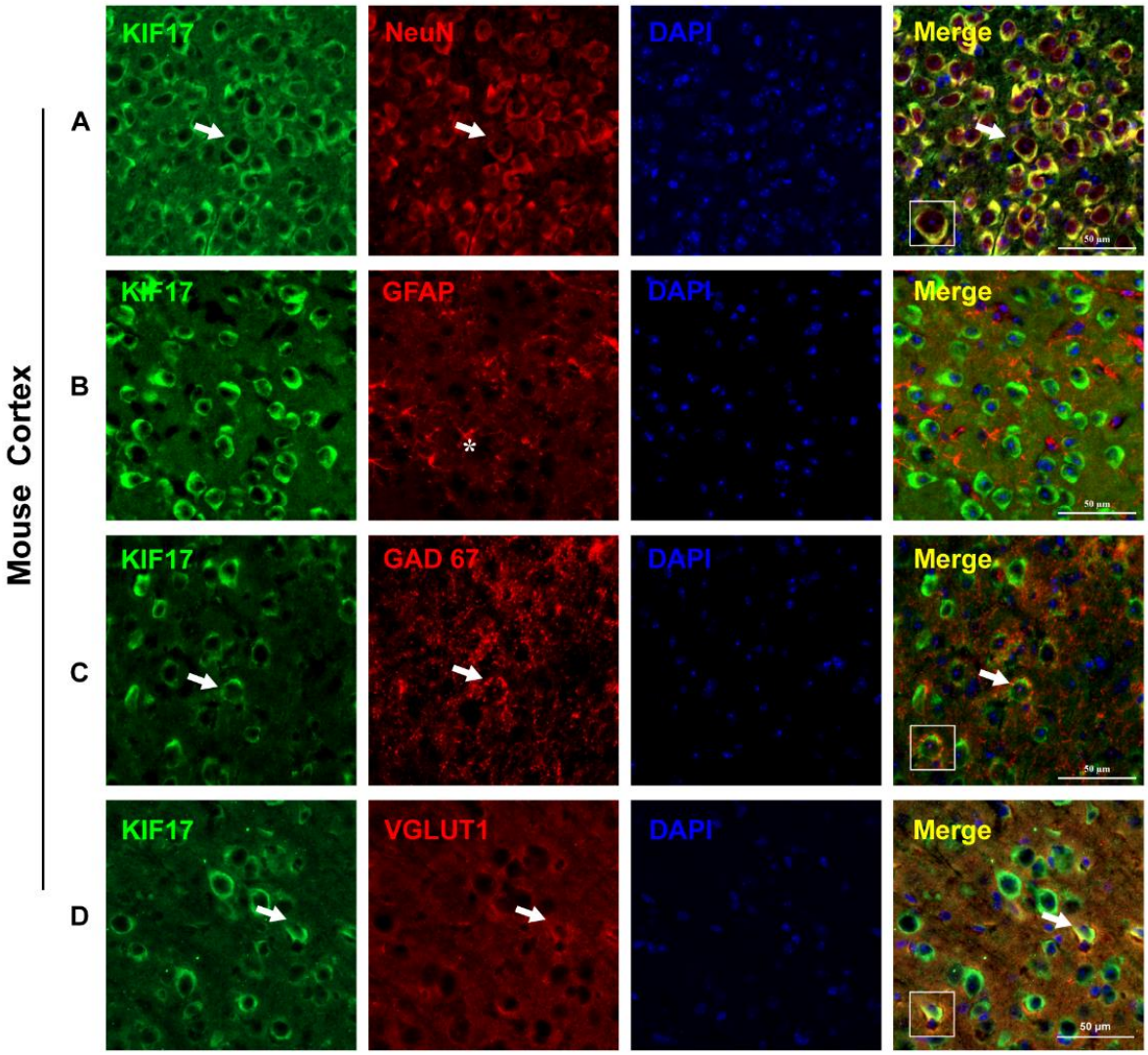

Fig. S2 Localization of KIF17 in epileptic mouse brain. **A, B** In the cortex, KIF17 co-localizes with NeuN and rarely with GFAP (*astrocytes). **C, D** KIF17 co-localizes with a GABAergic neuronal marker (GAD67) and a glutamatergic neuronal marker (VGLUT1). Scale bars, 50 μm (arrow indicates positive cell).

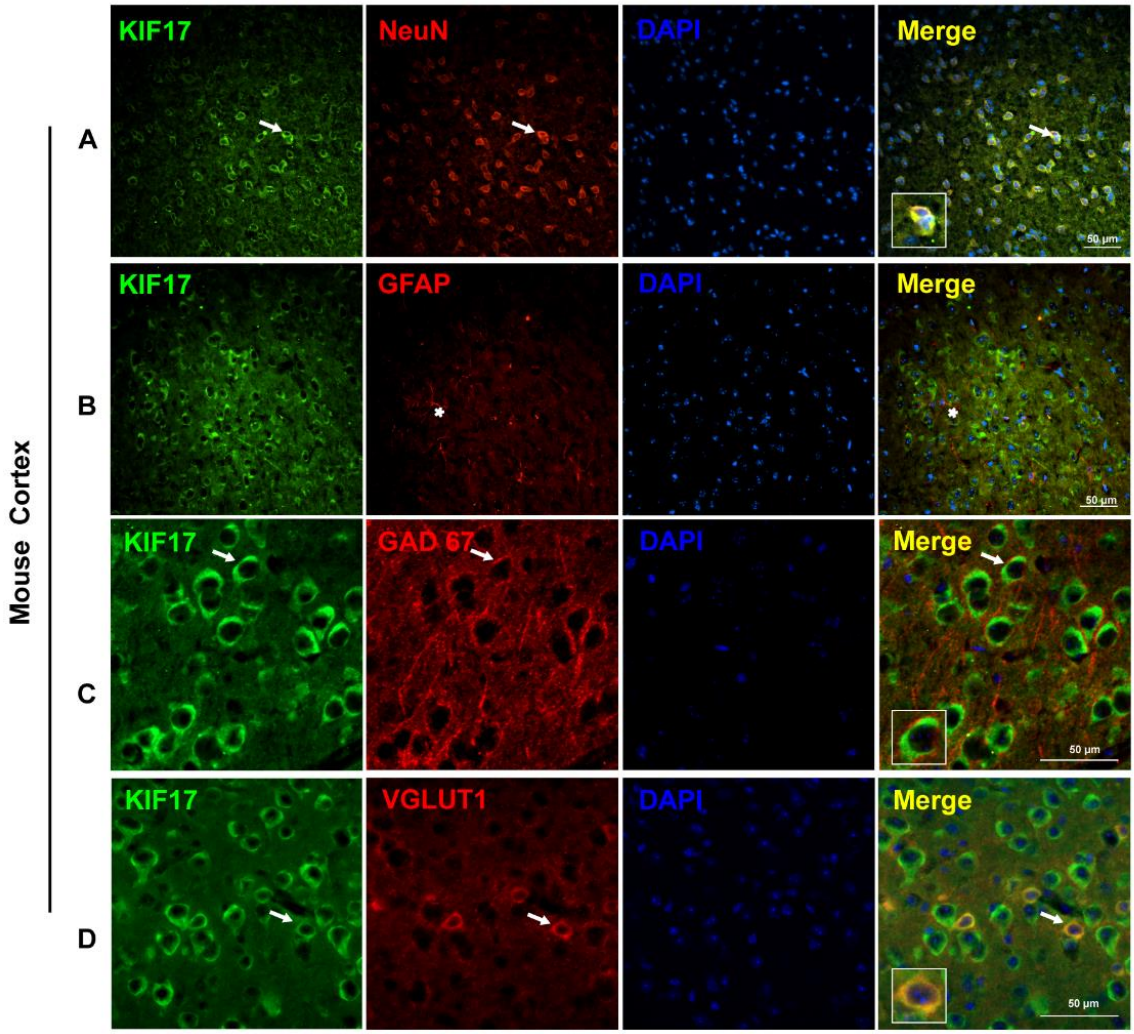

Fig. S3 Localization of KIF17 in control mouse brain. **A, B** In the cortex, KIF17 co-localizes with NeuN and rarely with GFAP (*astrocyte). **C, D** KIF17 co-localizes with a GABAergic neuronal marker (GAD67) and a glutamatergic neuronal marker (VGLUT1). Scale bars, 50 µm (arrow indicates positive cell).

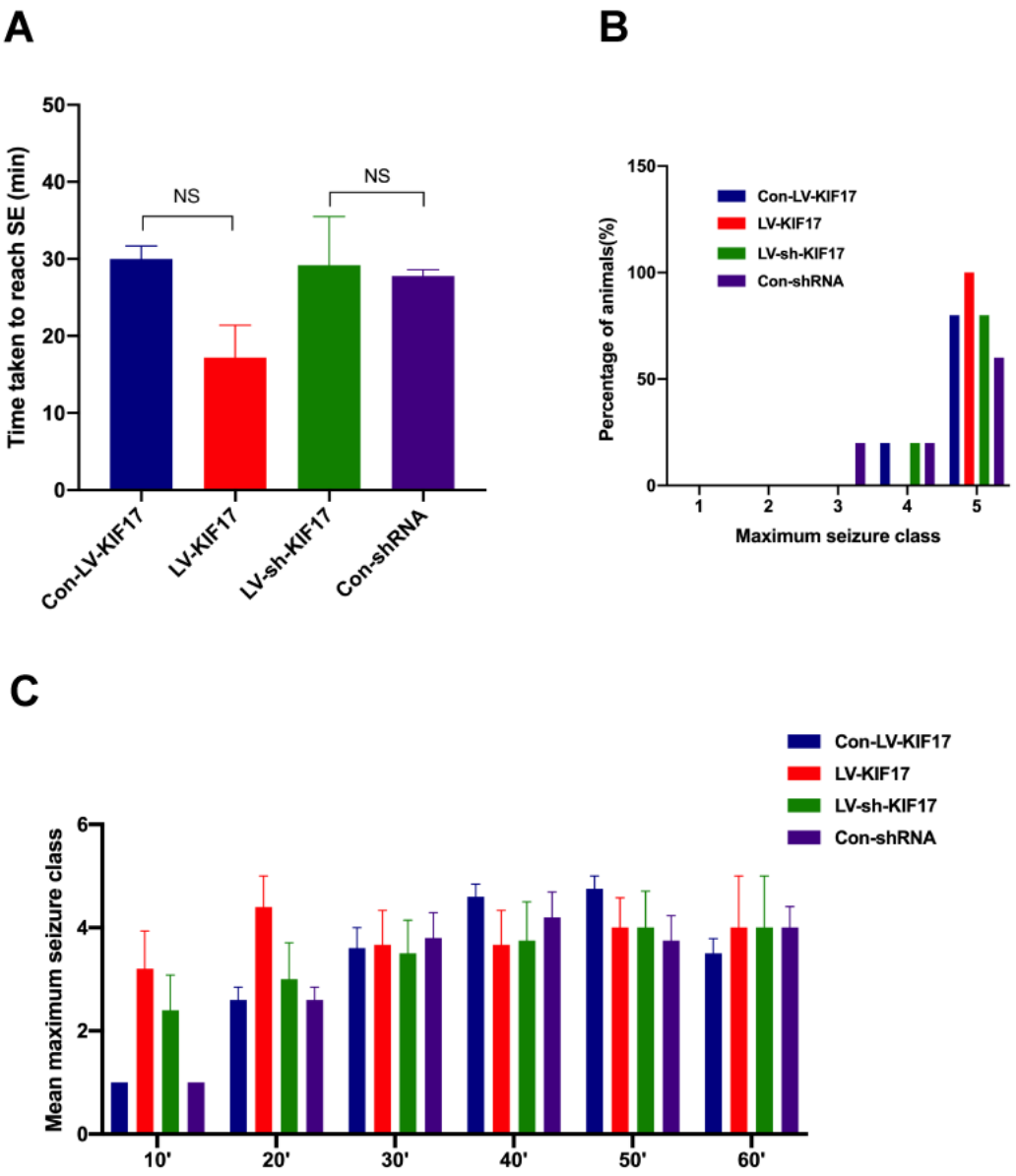

Fig. S4 KIF17 modulates seizure susceptibility. Time taken to reach SE (**A**), percentage of maximum seizure class within 1 h (**B**), and seizure progression (**C**) after KA administration in the four groups (n = 5). Data are presented as the mean ± SEM, NS means P >0.05, one-way ANOVA followed by LSD-t test (**A** and **C**).

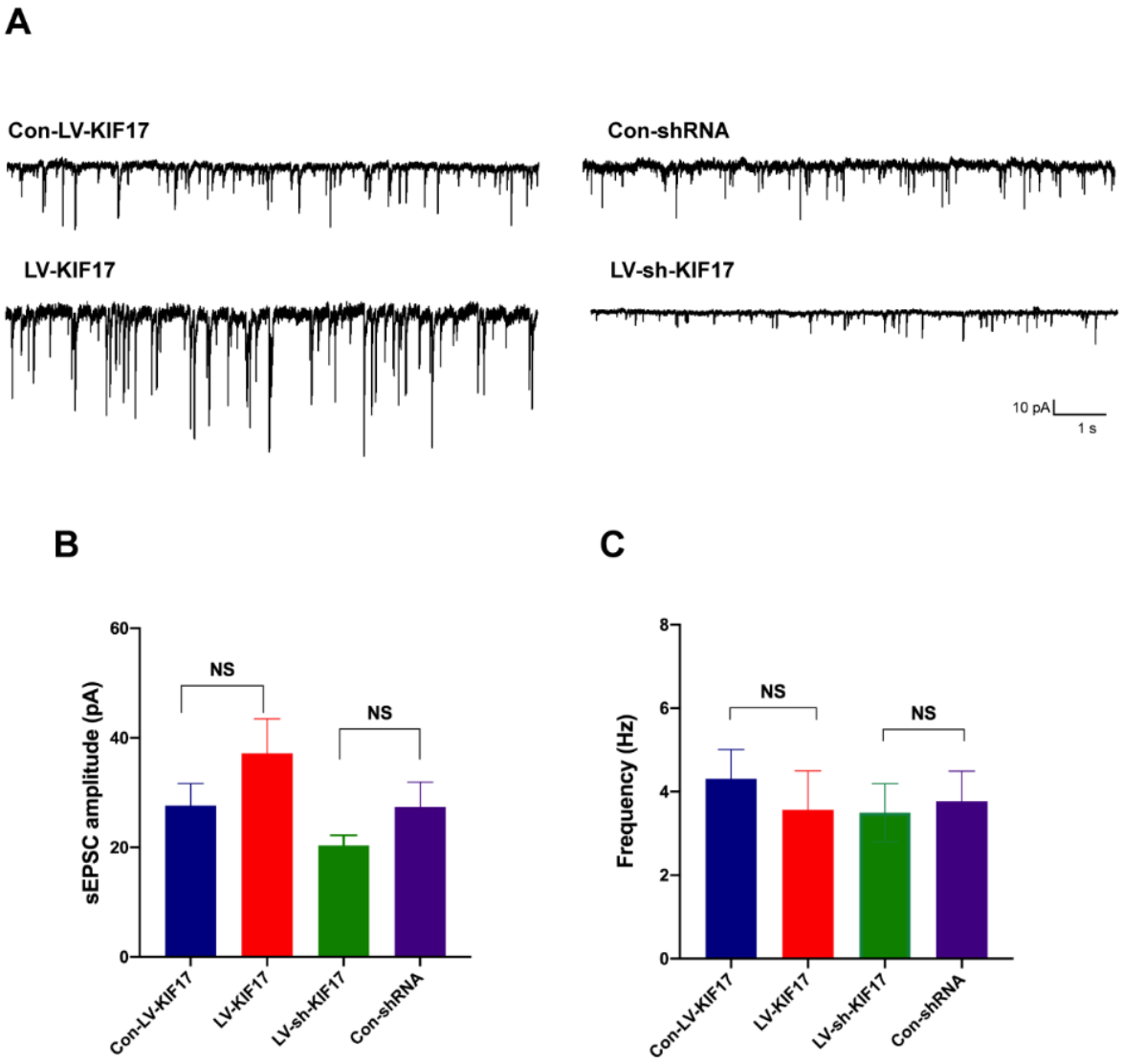

Fig. S5 KIF17 alters the amplitude of sEPSCs. **A–C** Representative traces of sEPSCs (**A**) and amplitude and their frequency (**B** and **C**) (n = 5). Data are presented as the mean ± SEM, NS means P >0.05, one-way ANOVA followed by LSD-t test.

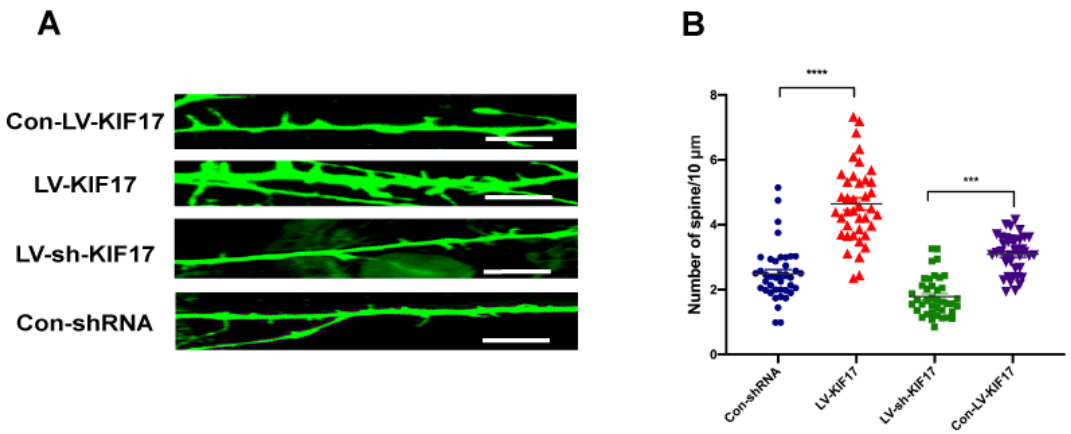

Fig. S6 KIF17 regulates spine density. **A** Representative images of spine density on cultured primary neurons after treatment with Con-LV-KIF17, LV-KIF17, LV-sh-KIF17, or Con-shRNA. Scale bars, 10 μm. **B** Statistics of spine density (n = 50). Data are presented as the mean ± SEM, ***P <0.0005, ****P <0.0001, one-way ANOVA followed by LSD-t test.

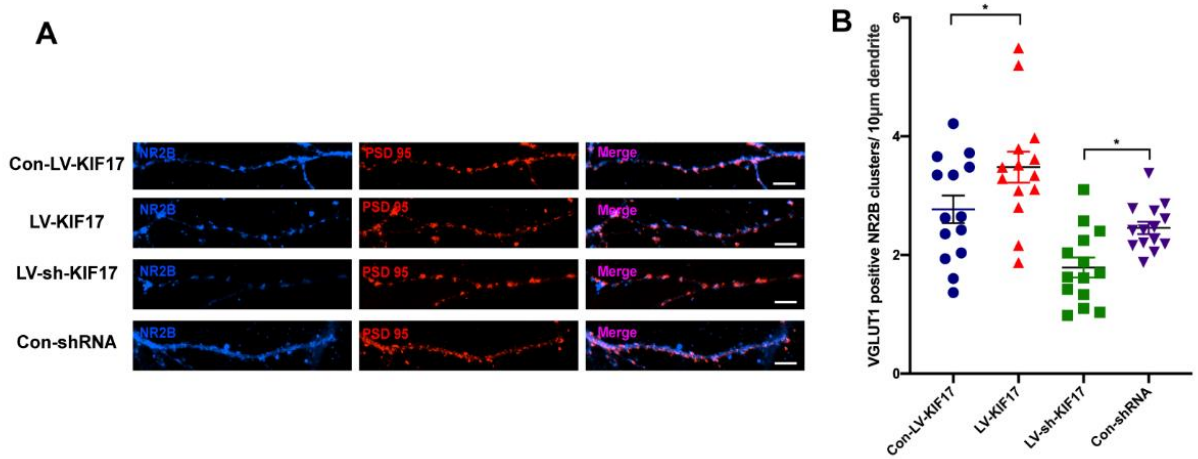

Fig. S7 KIF17 intervention changes the surface expression of NR2B on hippocampal neurons. **A** Representative images showing the co-localization of NR2B and PSD95 in hippocampal neuronal dendrites after KIF17 intervention. Scale bars, 10 μm. **B** Statistics for the PSD95-positive NR2B cluster in each treatment group. Data are presented as the mean ± SEM, n =14, *P <0.05, one-way ANOVA followed by LSD-t test.