Hu et al., http://www.jcb.org/cgi/content/full/jcb.201404092/DC1

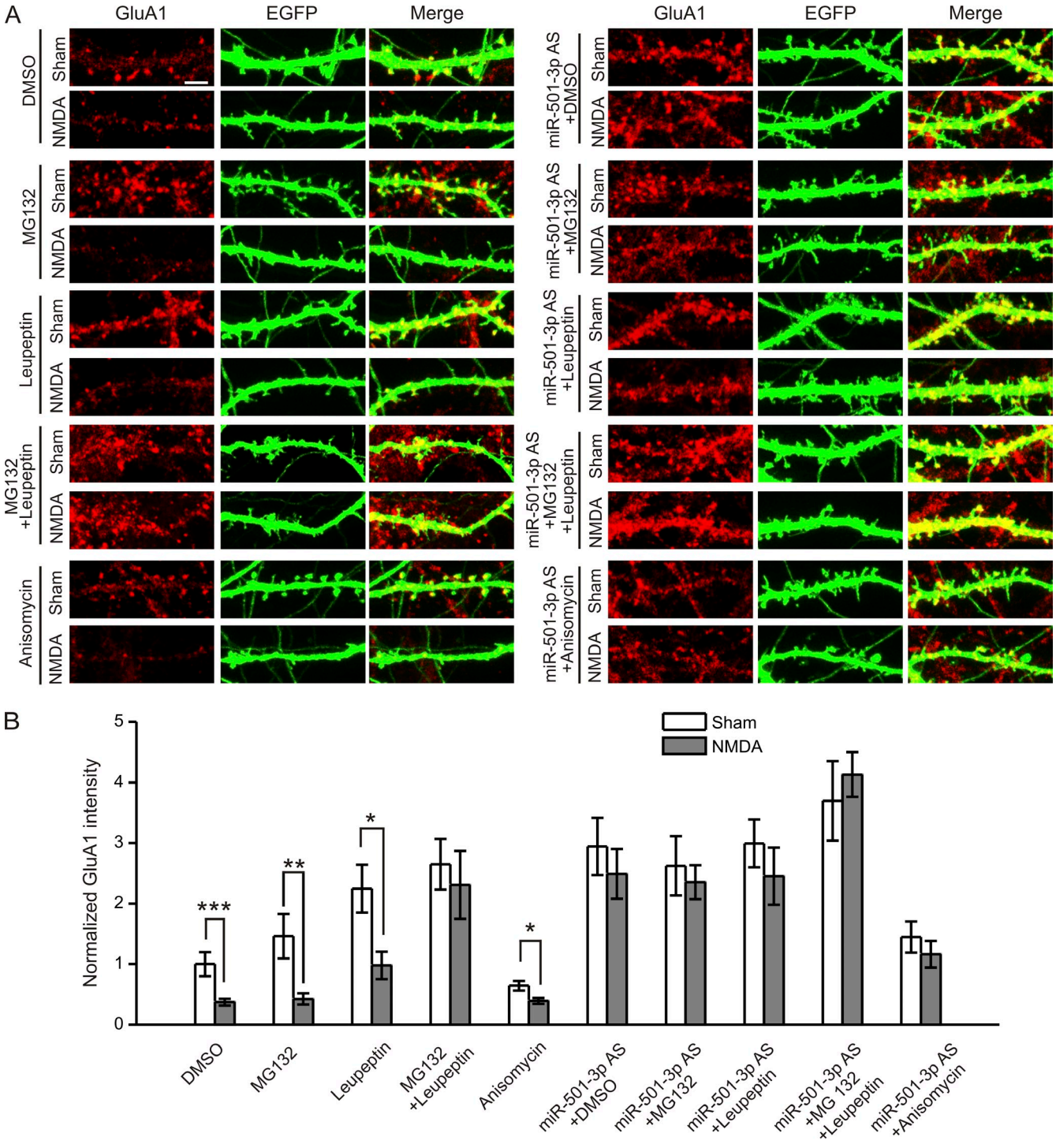

Figure S1. **NMDA-induced GluA1 reduction is caused by miR-501-3p–mediated suppression.** Cultured hippocampal neurons (17 DIV; 3 d after transfection) were treated with NMDA and then stained for GluA1. (A) Representative images of dendrites from transfected neurons. (B) Quantification of A; n = 13–20 neurons for each group. Data are presented as mean ± SEM. Mann-Whitney U test was used for statistical analysis. *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.005$. Bar, 5 µm.

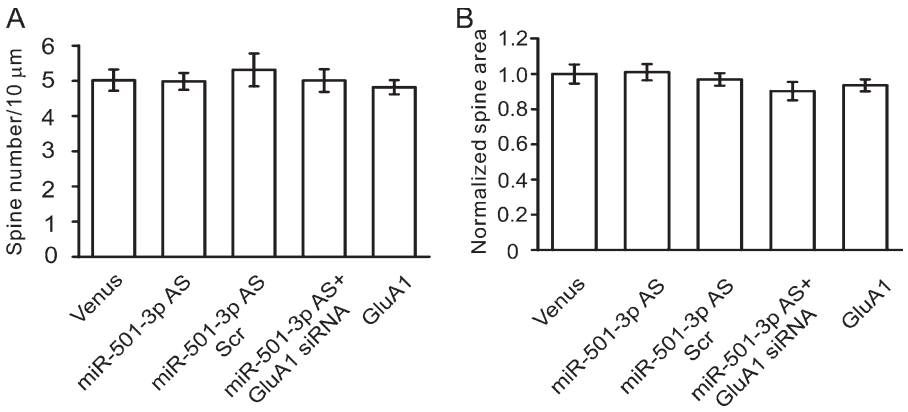

Figure S2. **miR-501-3p antisense oligonucleotides and GluA1 overexpression have no effect on basal spine number and size.** Primary hippocampal neurons were transfected with the designated constructs at 14 DIV and fixed for analysis of dendritic spines at 17 DIV. (A and B) Quantification of dendritic spines in fixed neurons; *n* = 14–16 neurons for each group; data are presented as mean ± SEM; one-way ANOVA was used for statistical analysis.

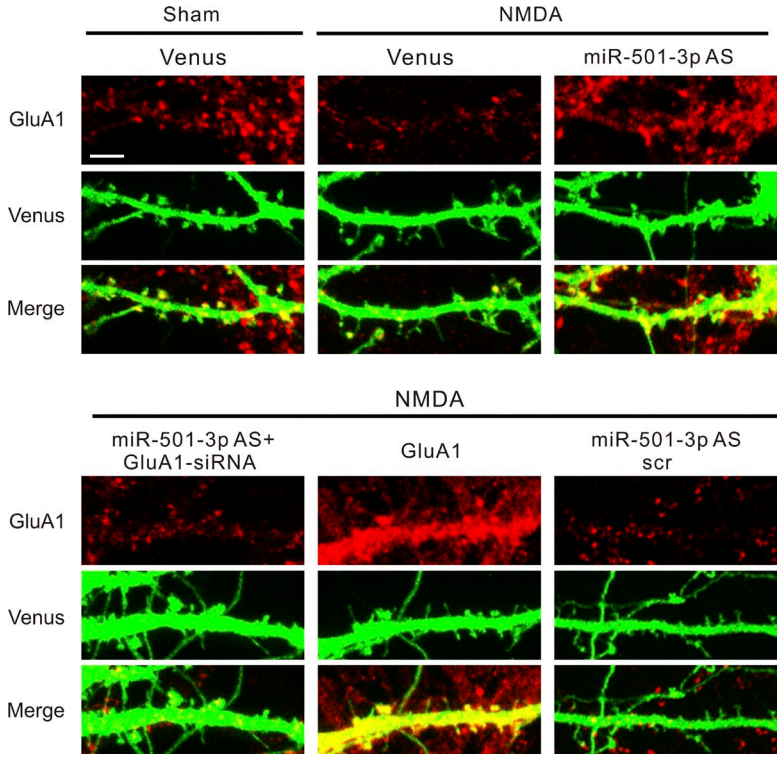

Figure S3. **GluA1 protein expression in neurons used for live imaging experiments.** Primary hippocampal neurons (17 DIV; 3 d after transfection) were treated with NMDA (30 µM for 5 min) and then stained for GluA1 at 90 min after stimulation. Bar, 5 µm.

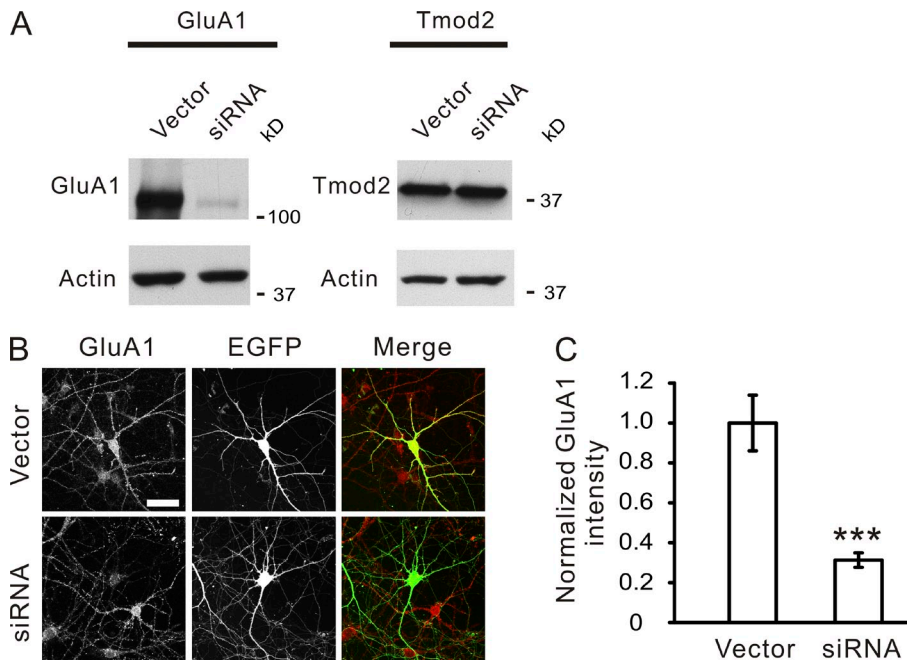

Figure S4. **GluA1 siRNA effectively and specifically knock down GluA1 expression.** Cos7 cells were transfected with constructs expressing GluA1 (A) or Tmod2 (B) along with the empty vector or the GluA1 siRNA construct. Cell lysates were collected at 48 h after transfection for immunoblotting against GluA1 or Tmod2. (C) Cultured hippocampal neurons (14 DIV) were transfected with the EGFP construct along with the empty vector or the GluA1 siRNA construct and stained for GluA1 at 3 d after transfection. (D) Quantification of C; *n* = 16–19 neurons for each group. Data are presented as mean ± SEM. Mann-Whitney *U* test was used for statistical analysis. ***, P < 0.005. Bar, 20 µm.

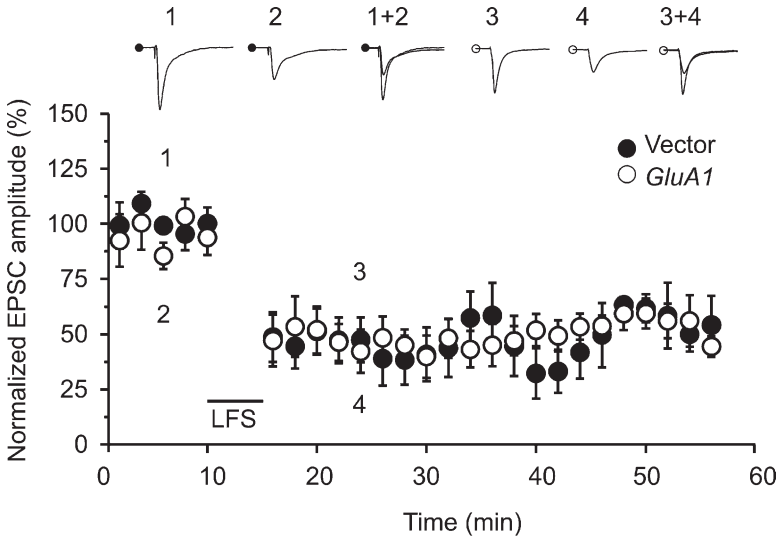

Figure S5. **GluA1 overexpression has no effect on LTD.** Cultured hippocampal slices were transfected with the venus construct alone or along with a construct expressing GluA1. LTD of CA1 neurons was induced by stimulating the Schaffer collateral pathway with low-frequency stimulations (LFS). EPSCs were recorded from transfected CA1 neurons. Data are presented as mean ± SEM.