

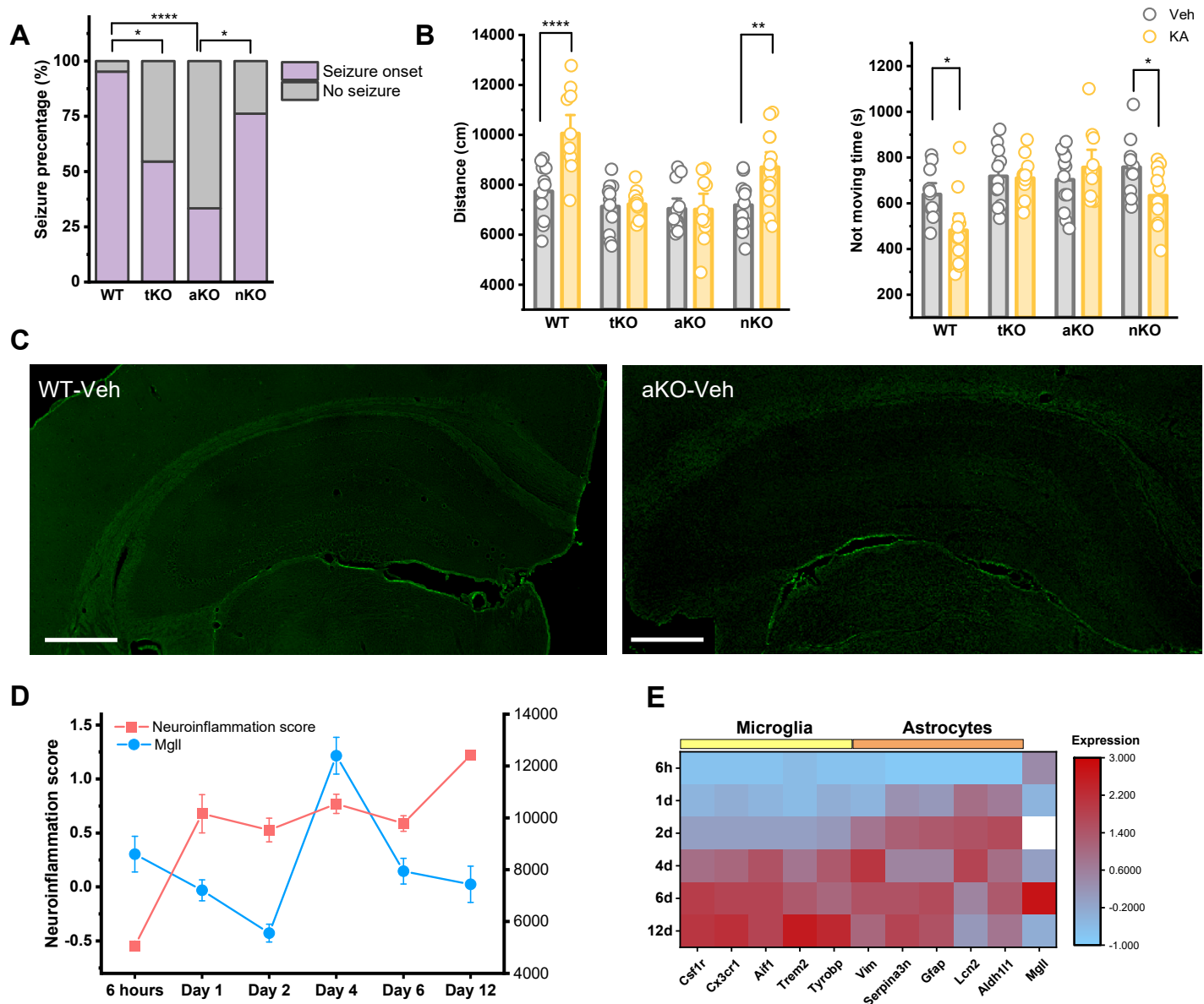


Figure S1. Astrocyte-specific *Mgl1* deletion attenuates kainic acid (KA)-induced seizure susceptibility and hippocampal neuroinflammation (A) Quantification of the percentage of mice developing seizures in WT, TKO, AKO, and NKO mice in one hour (WT: 20 mice, TKO: 22 mice, AKO: 25 mice, NKO: 21 mice). Statistical significance was determined by a Chi-Squared Test with Bonferroni multiple test, * $p < 0.05$, **** $p < 0.001$. (B) Quantification of hyperactive and anxiety-like behaviors in the Open Field test, showing total distance traveled and normalized freezing time of both the baseline and post-seizure injury. Data are means $\pm$ SEM. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.005$, **** $p < 0.001$ (n=11-13, t-test in each genotype). (C) Representative immunofluorescence images of Fluoro-Jade C (FJC) staining in the hippocampus of WT and AKO mice following vehicle treatment. Scale bars: 500 μ m. (D) Temporal profile of the Neuroinflammation score (red) and *Mgl1* expression (blue) over 14 days in CA1. (E) Heatmap illustrating the temporal mRNA expression profiles of *Mgl1*, astrocytic and microglial markers in the hippocampus of mice treated with saline or KA at 6 hours, days 1, 2, 4, 6, and 12 post-injection. The color scale indicates relative expression levels.