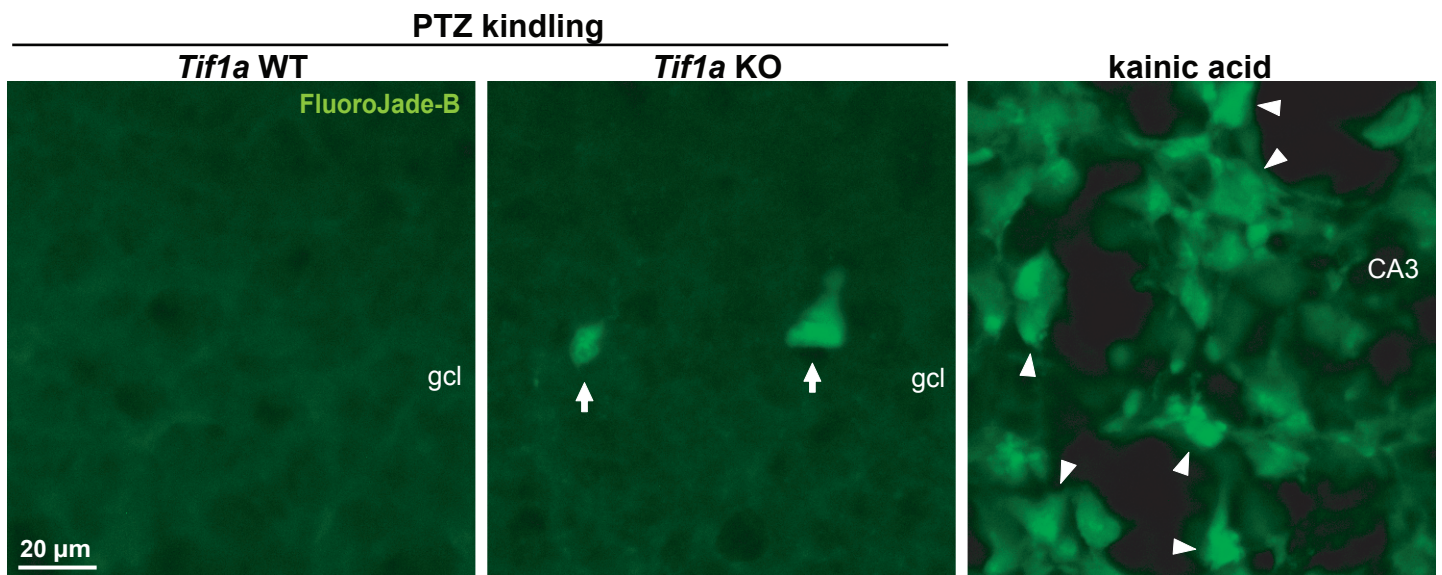


**Figure S1. A,** Mice (C57Bl6 males) were treated with vehicle (control, ctrl) or pilocarpine (300 mg/kg) to induce SE. Representative images of ribosome (NeuroTrace<sup>®</sup> 500/525 Green Fluorescent Nissl)- or DNA (Hoechst 33258) in the dorsal hippocampus of control or Pilo-SE mice (6 weeks post SE). Note apparent reductions in Nissl-staining in the CA and the DG hilar regions of Pilo-SE animals confirming pilocarpine-induced hippocampal neurodegeneration. **B,** Mice (C57Bl6 males) were treated with 10 sub-seizures doses of PTZ (35 mg/kg) or vehicle (control, ctrl) every other day; fully kindled animals that displayed tonic-clonic seizures (at least stage 4) after each of the last 5 injections of PTZ were used for evaluation of ribosome content. Representative images of ribosomal staining (NeuroTrace<sup>®</sup> 500/525 Green Fluorescent Nissl) in the dorsal hippocampus of PTZ-kindled mice are shown; DNA was counterstained with Hoechst 33258. Quantitative analysis of fluorescence intensity revealed no significant changes in DNA-normalized content of ribosomes in the granule cell layer of the DG or stratum pyramidale of the CA1 region of PTZ kindled mice (Fig. 4E-F).



**Figure S2.** FluoroJade-B staining reveals few degenerating neurons in the DG granule cell layer (gcl) of PTZ-kindled *Tif1a* KO mice. Representative images of sections from the study that is described in Fig. 5 are shown. Positive cells are pointed by arrows. Counts of positive cells/section from the DG, CA1, and, CA3 regions are presented in Table 3. Sections from mice that survived 4 days after unilateral intra-amygdalar injection of kainic acid were used as a positive control. As reported in the literature (Brain Research 1213: 140-151. doi:10.1016/j.brainres.2008.03.061), such treatment resulted in appearance of many degenerating (*i.e.* FluoroJade-B-positive) cells in the CA3 region of the ipsilateral hippocampus (arrowheads).

**Supplementary Table S1. Upregulated ribosomal protein mRNAs in the hippocampus of pilocarpine-SE-challenged mice (analysis of the publically available RNASeq dataset GSE72402)<sup>a, b</sup>.**

Time post Pilo-SE	Gene Symbol	Gene Name	Fold Change	Q-value
12h	Rpl8	ribosomal protein L8	3.1	9.63E-15
	Rpl18	ribosomal protein L18	17.6	1.43E-09
	Rplp1	ribosomal protein, large, P1	2.9	1.98E-05
	Rps2	ribosomal protein S2	2.8	0.00021
10 days	Rpl26	ribosomal protein L26	35.6	8.66E-16
	Rpl8	ribosomal protein L8	4.9	8.66E-16
	Rpl18	ribosomal protein L18	27.1	1.39E-15
	Rps3	ribosomal protein S3	2.9	1.60E-15
	Rpl6	ribosomal protein L6	15.1	2.68E-10
	Rpl29	ribosomal protein L29	89.2	1.74E-09
	Rps2	ribosomal protein S2	3.1	2.16E-05
	Rps27l	ribosomal protein S27-like	11	6.01E-05
	Rpl32	ribosomal protein L32	4.7	0.00087
	Rplp2	ribosomal protein, large P2	27.6	0.00149
	Rplp1	ribosomal protein, large, P1	3.3	0.00252
	Rpl39	ribosomal protein L39	10.3	0.00335
6 weeks	Rpl8	ribosomal protein L8	2.2	4.76E-05

<sup>a</sup>Downregulation of many RP transcripts was also observed (Rps15a at 12 h- or 10 day time point; Rpl34, Rps4x, Rps15, Rpl13a, Rps15a, Rpl4, Rpl8 at 6 week time point). However, as such events may have been caused by post SE neurodegeneration, their interpretation is unclear without follow up expression studies at a single cell level.

<sup>b</sup>Hansen, K. F., Sakamoto, K., Pelz, C., Impey, S. and Obrietan, K. (2014) Profiling status epilepticus-induced changes in hippocampal RNA expression using high-throughput RNA sequencing. *Scientific reports*, 4, 6930.