Supplemental Figure Legends

Figure S1. ChR2-GFP expression in PV cells of PV-Cre x Ai32 mice intrahippocampally injected with KA. (A) Brain slice of a chronically epileptic mouse depicts survival of PV cells in a PV-Cre x Ai32 animal treated with IHKA (Inset A': a different single image plane of the outlined area which best visualizes cells seen in panel C). Note the remodeling of the hippocampal formation and loss of a defined laminar structure on the injection side. Scale bar, 500 µm. (B, C) Orthogonal projection of z-stack images showing ChR2-GFP fluorescence (left) and PV immunostaining (middle) co-localization (right) in both the contralateral (B) and ipsilateral (C) hippocampus of PV-Cre x Ai32 mice. Scale bar, 20 µm. (D) Orthogonal projection of z-stack images of the ipsilateral hippocampus from a different chronically epileptic mouse, below the optrode (arrow), shows survival of PV cells in various areas below and above the hippocampal fissure (dashed line). Scale bar, 200 µm.

Figure S2. Post-hoc histological verification of optical fiber locations in chronically epileptic mice with optogenetic seizure intervention

Figure S3. Comparison of object location memory performance based on ChR2 opsin expression status. Opsin negative (n=12) and opsin positive (n=12) animals produced from crossing PV-Cre to Cre-dependent ChR2 reveals no difference between object location discrimination (Opsin negative DI=25.6±5.0, Opsin positive DI=28.3±4.6, p=0.71) or exploration time (Opsin negative, 3.9±0.2 s; Opsin positive, 4.1±0.2 s; p=0.51).

Table S1. Overall animal data for object location memory testing

Table S2. Experimental steps and animal dropout