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Synaptopathy in temporal lobe epilepsy: role of kainate receptor

In both human patients and animal models of TLE, neuronal tissue undergoes major reorganization; some neurons die while others sprout and form novel aberrant connections (Tauck and Nadler, 1985, Represa et al. 1987, Nadler, 2003; Gabriel et al., 2004; Dudek & Sutula, 2007). This phenomenon is best documented in the dentate gyrus where mossy fiber (MF) axons sprout to form aberrant glutamatergic excitatory synapses onto other dentate granule cells (DGCs) leading to the formation of functional recurrent excitatory circuits. This accounts for, in part, the enhanced ability of the hippocampus to generate epileptiform activity. MF sprouting also induces a reorganization of KAR-mediated synaptic transmission, with a shift in the nature of glutamatergic transmission in DGCs (Epsztein et al. 2005, Epsztein et al. 2010, Artinian et al. 2011, Peret et al. 2014; Artinian et al. 2015; Matsuda et al. 2016). Our data reveal that ectopic kainate receptors drastically impact the computational properties of DGCs and play a major role in epileptiform activities.