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Title: Homeostatic reorganization of network dynamics at the border between intact and deafferented cortex

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Cortical trauma causes a dramatic increase in seizure propensity in animal models of posttraumatic epilepsy. In our *in vivo* recordings from behaving cats one to six weeks following partial cortical deafferentation, all experimental animals developed electrographic (in some cases accompanied by behavioral) seizures. The underlying cause of this increased excitability in posttraumatic cortical networks is unclear. Computational models have suggested a role for homeostatic upregulation of intrinsic and synaptic excitability (Houweling et al., Cerebral Cortex 2005). Here, we study changes in spatio-temporal network dynamics after partial deafferentation in a cortical network model composed of pyramidal cells and fast spiking inhibitory interneurons with homeostatic regulation of fast excitatory synaptic transmission. When external input was completely removed from a part of the neuronal population, following an initial period of silence homeostatic plasticity induced an increase in excitatory synaptic transmission causing bursts at low frequency (1-3 Hz). Bursts were initiated in the boundary region between intact and deafferented cortex and propagated through the deafferented population. Furthermore, bursting in the deafferented cells disrupted the asynchronous firing of neighboring neurons with intact inputs. This is in agreement with our recent experimental findings showing that spike and wave discharges during posttraumatic seizures originate near the border between intact and deafferented cortex and then spread into more strongly deafferented regions.
Little is known about the consequences of homeostatic synaptic scaling for spatio-temporal network dynamics. Our findings suggest that homeostatic plasticity can cause reorganization of cortical networks leading to paroxysmal dynamics by alteration of synaptic connectivity in neural populations suffering from severe loss of synaptic inputs.

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