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Program#/Poster#: 165.15/AA9
Title: Homeostatic recovery of a partially deafferented neocortical network as a mechanism for posttraumatic epileptogenesis
Location: San Diego Convention Center: Halls B-H
Presentation Start/End Time: Sunday, Nov 04, 2007, 10:00 AM -11:00 AM
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Trauma is the worldwide most frequently reported etiology of epilepsy. While the exact mechanism of posttraumatic epileptogenesis is debated, our experimental and theoretical work suggests that deafferentation triggers homeostatic upregulation of local recurrent excitation which then supports the occurrence of paroxysmal bursting. In a computational model of a neocortical network with a global homeostatic plasticity rule, we studied the recovery dynamics after spatially inhomogeneous partial deafferentation. The simulated injury corresponded to the removal of the uncorrelated random afferent input to a fraction of neurons (degree of deafferentation between 10% and 100% of all cells). Homeostatic upregulation was modeled by strengthening local excitatory connections.

We found two different types of recovery dynamics as a function of the injury severity. Mild to intermediate trauma (up to 60% deafferentation) resulted in increased activity of pyramidal cells (PYs) with intact input and decreased activity of deafferented PYs. The baseline network firing rate was recovered; however, cells that were silent before deafferentation remained silent throughout the recovery process. In case of more severe trauma (70% to 100% deafferentation), the recovery process was qualitatively different. Specifically, all cells in the network became active, including PYs that were silent before deafferentation. Interestingly, this apparent threshold for network-wide activation reflected also the minimal degree of deafferentation for which we found bursts of action potentials in individual PYs.

In summary, we propose the existence of a threshold for posttraumatic epileptogenesis as a function of the fraction of deafferented cells. Our findings suggest a possibility to develop stimulation protocols which could counteract homeostatic upregulation of excitatory connections in order to prevent epileptogenesis in patients with head trauma.

Disclosures: **F. Frohlich** , None; **M. Bazhenov** , None; **I. Timofeev** , None; **T.J. Sejnowski**, None.

Support: NIDCD, CIHR

[Authors]. [Abstract Title]. Program No. XXX.XX. 2007 Neuroscience Meeting Planner. San Diego, CA: Society for Neuroscience, 2007. Online.

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