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Presentation Abstract

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Title:	Spatial pattern and severity of cortical trauma determine the propensity of post-traumatic epileptogenesis
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Abstract:	Epileptic activity is often documented after a latent period that follows head trauma. This phenomenon has been attributed to the pathological action of homeostatic plasticity following cortical deafferentation that results from trauma. However, the relation between properties of trauma (size or spatial pattern) and the propensity to develop post-traumatic epilepsy (PTE) is unknown. The present study had two objectives: 1) to understand how trauma severity (parameterized as a fraction of deafferented neurons and the degree of deafferentation) affects the propensity of PTE; 2) to understand how spatial patterns of trauma affect the emergence of epileptic like activity. To achieve the objectives, a biophysically realistic computational model of 2D cortical network was developed. The model comprised of 6400 (80x80) pyramidal neurons and interneurons (PY/IN ratio 4:1) organized in a 2D square lattice with locally random synaptic connectivity. Deafferentation was modeled as a reduction in the rate of external input to a preset fraction of neurons, allowing us to control both the severity and the shape of trauma. Following deafferentation, homeostatic plasticity adjusted synaptic strength to bring the network-averaged firing rate to the target value of 5 Hz. Using the model we studied the reorganization of collective activity in response to trauma patterns of different severity and different spatial organization. Epileptic

activity (manifested as highly synchronized network bursts) appeared when the fraction of lesioned neurons exceeded some critical value. For low and medium severity of deafferentation, spatially compact trauma significantly reduced the threshold for PTE as compared to spatially diffuse trauma. By contrast, for nearly complete and severe deafferentation, the diffuse distribution of a small number of trauma-surviving intact neurons significantly alleviated the emergence of PTE. Our study shows that spatial organization of brain trauma can greatly affect the propensity of traumatized network to generate epileptic activity. Based on these observations, we suggest that clinical methods aiming to estimate the likelihood of PTE in conjunction with brain trauma should account for pattern-related aspects.

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