Electrophysiology of the rat barrel cortex following traumatic brain injury

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Abstract: Neuronal injury resulting from direct mechanical impact activates secondary processes of cytotoxicity (excitotoxicity, oxidative stress, and apoptosis), which continue to evolve over hours/days. Following injury, the electrical activity of neuronal populations within relevant brain structures has not been widely studied at the single- or multi-unit level, and the role that the electrical activity may play in the recovery process is only beginning to be addressed. In this study, Long-Evans rats were subjected to traumatic brain injury (TBI) in the barrel cortex through the use of a controlled cortical impactor (CCI) device. With the use of a paired-pulse and 3-pulse whisker deflection paradigm, TBI at the electrophysiological (single/multi-unit and EEG) and histological levels in the barrel cortex of the rat somatosensory system were monitored and evaluated at various time points following recovery.

Post-stimulus time histograms (PSTH) seem to indicate that periods of enhanced excitation and cortical suppression exist following induction of injury. Excitotoxicity and cortical spreading depression (CSD) are often reported following traumatic brain injury and the resulting changes in PSTH amplitudes at multiple time points after TBI induction. The results suggest a possible perturbation in the balance of inhibitory and excitatory neuronal activity in the cortex following injury. These data provide possible insight into the time course of the development of injury and could potentially serve as indicators of outcome. Controlled modulation of inhibitory and excitatory activity at critical time points could lead to more effective interventions for enhancing recovery following TBI.

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