Rescuing deficits in neuronal plasticity after mild TBI

Traumatic brain injury (TBI) continues to be a major socio-economic problem with about 2 million head injuries in the US annually, the majority being mild in severity. To understand better the mechanisms of TBI, we have developed in vitro models using organotypic brain slice cultures that afford precise control over injury biomechanics. With these models, we have previously developed tolerance criteria to determine safe levels of exposure that could be used to engineer better safety systems to prevent TBI. More recently, we have focused on how different mechanical stimuli (injury) may alter neuronal activity and electrophysiological function within hippocampal neuronal networks and explored therapeutic strategies to reverse pathological changes. Our recent findings suggest that after mild TBI, a disruption of dendritic organization may underlie deficits in long-term potentiation, i.e. the cellular correlates of learning and memory. We have identified therapeutic interventions that rescue LTP with therapeutic windows as long as 6 hours after injury. The long-term goal of our research is to reduce the socio-economic costs of TBI by developing novel treatments and by helping others engineer better protection systems.

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