

Plasticity and Axonal Sprouting of Contralateral Cortex after Unilateral Traumatic Brain Injury

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Abstract

According to the Centers for Disease Control and Prevention, an estimated 1.7 million Americans experience Traumatic Brain Injury (TBI) annually and about 52,000 of them die. TBI results in a primary injury of brain tissue. It can also cause a secondary damage as well depending on the severity of the injury, which could lead to different types of dysfunctions such as persistent motor or cognitive deficits. We hypothesize that cortical injury from unilateral TBI will cause plasticity and axon sprouting of the contralateral cortex, which may contribute to functional compensation and recovery. Controlled cortical impact (CCI) is a methods used in our research laboratory to create TBI models in rats and mice. To test our hypothesis, one hemisphere of each mouse brain is moderately injured by the CCI technique to allow us to determine if there is significant axon sprouting in the contralateral cortex. Axon sprouting is expected to occur at certain time period after the injury. To determine the existence and the timing of axon sprouting, two sets of CCI and sham mice were used for histological analysis at two different time points after CCI. The first set contains 4 sham and 6 CCI mice and examined at 6 weeks post-injury; the second set contains 4 sham and 6 CCI mice and examined at 3 weeks post-injury. Immunostaining to growth-associated protein-43 (GAP-43) will be used to detect sprouting axons in the injured cortex. However, the brains are currently in process for the staining. . Further data collection and image analysis will be needed to obtain the results and findings of the research.