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"The effect of moderate TBI on neuronal degradation in the spinal cord"

Traumatic brain injury (TBI) is a major cause of death and disability amongst all age ranges, making it a predominant public health concern. One of the consequences of moderate TBI is enhanced neuronal loss and neurodegeneration. Additionally, alcohol use may exacerbate these negative outcomes and delay or even prevent recovery from a TBI. Moderate TBI is a risk factor for certain neurodegenerative diseases, like Amyotrophic Lateral Sclerosis (ALS). Preliminary data from the Desai lab suggests early changes in the spinal cord may be evident following a single moderate TBI. In this experiment, we hypothesize there will be evidence of motor neuron degeneration in the ventral horn of the spinal cord following a moderate TBI, especially in combination with alcohol use.

Male Wistar rats were randomly assigned to naive, sham, or TBI groups, with half of the sham and TBI rats receiving alcohol. The rats receiving the alcohol were taught to self-administer alcohol over a 4 week period prior to the injury and continued consuming alcohol for 30 minutes 3 times per week following the injury. A craniotomy was performed over the somatomotor cortex, and 3 days later a mild-to-moderate TBI was produced directly onto the intact dura mater using the lateral fluid percussion injury model. Sham animals received craniotomy and matched anesthesia only. Animals were euthanized at either 2 or 12 weeks post-injury. Using immunohistochemistry, we analyzed sections of the lumbar spinal cord to detect the extent of motor neuron degeneration in the ventral horn of the lumbar spinal cord. We used Flouro-JadeC to identify degenerating motor neurons, and NeuN to label mature neurons present.