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"Alcohol and Repeated Mild Traumatic Brain Injury Synergistically Exacerbate Hippocampal Neuroinflammation During Adolescence"

Background: Traumatic brain injury (TBI) is a non-degenerative, non-congenital insult to the brain from an external mechanical force or trauma that affects over 2 million people in the United States each year, leading to $77 billion in healthcare costs. Adolescents participating in athletics are more likely to experience TBIs as well as engage in underage alcohol consumption than non-athletic peers. Both TBI and alcohol produce neuroinflammation, which can lead to neuropathology, especially in the developing adolescent brain. While approximately 90% of TBIs are mild (mTBI), the damage from repeated mild TBI (rmTBI) can accumulate to levels of damage comparable to moderate or severe injury. The primary goal of our study is to analyze the individual and combined effects of alcohol and rmTBI on neuroinflammation during adolescence.

Methods: A mixed sex cohort of adolescent Wistar rats were divided into four groups: Sham+Air, Sham+EtOH, rmTBI+Air, and rmTBI+EtOH. Rats were exposed to 3 days of intermittent alcohol vapor or plain room air, followed by a day of respite. The following day, they received either a mTBI produced by a weight-drop model, or the sham procedure. This cycle was repeated for a total of four episodes. Rats were euthanized 7 days after the final injury, and their brains were collected for immunohistochemistry to assess for neuroinflammation. Here, we measured glial fibrillary acidic protein (GFAP) expression, a marker of astrogliosis, in the CA1, CA2/3, and dentate gyrus regions of the hippocampus.

Results: While GFAP expression in the CA1 showed no significant change, there was a trending effect of injury. In the CA2/3, we observed a significant increase in the rmTBI+Air group, which was further increased in rmTBI+EtOH. Finally, in the dentate gyrus, GFAP expression was significantly increased following either Sham+EtOH and rmTBI+Air, and this effect was further increased in the rmTBI+EtOH group.

Conclusions: Our findings suggest that the combination of rmTBI and alcohol can synergistically promote neuroinflammation within the hippocampus of adolescent rats. Future studies will increase the N and explore sex differences, as well as analyzing additional markers of neuroinflammation.