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**“Examining the Blood-Brain Barrier Following Mild Traumatic Brain Injury”**

**Background:** Between 1.6-3.8 million cases of mild traumatic brain injury (mTBI) are estimated to occur in the United States annually, making it a substantial public health concern. One important component that is damaged during mTBI is the blood-brain barrier (BBB). The BBB is a highly regulated physical and functional interface that separates the peripheral circulation and the central nervous system (CNS). It functions primarily as a selective diffusion barrier at the level of the cerebral microvascular endothelium. In this study, we focus on examining the endothelial glycocalyx of the BBB, and the presence of albumin, an endogenous protein that is typically excluded from the brain by the BBB, in relation to BBB breakdown following mTBI. In order to further validate our Weight-Drop Model of mTBI, we hypothesize it will induce disruption of the BBB.

**Methods:** Male Wistar rats were divided into two cohorts: sham and mTBI. After being anesthetized, rodents received either a single injury with our Weight-Drop model in which a 300-gram weight was dropped through a PVC tube from a height of one meter, or anesthesia only. At 24 hours following this procedure, animals were euthanized, and brain tissue was collected. Immunohistochemistry was performed to assess disruption of the BBB. Tomato lectin was used as our marker for the endothelial glycocalyx. The presence of albumin into the brain was also used to gauge damage. ImageJ was utilized to analyze images of the brain tissue.

**Results:** Analysis of the BBB in mTBI rodents revealed a decrease in tomato lectin labeling in mTBI rodents in both the dorsal cortex and the hippocampus, which corresponds to endothelial glycocalyx shedding in both the dorsal cortex and hippocampus. In addition, we saw a corresponding increase in the amount of albumin allowed into the brain.

**Conclusion:** This preliminary data suggests that the Weight Drop model of diffuse mTBI induces damage to the BBB. Further directions to validate that our model produces damage to the BBB following mTBI include analysis of the tight junction proteins and the endothelial cells. Future studies may also benefit from examining the production of molecules by astrocytes and microglial cells contributing to the dysfunction of the BBB such as TGF-beta, reactive oxygen species (ROS), and MMPs.