

ABSTRACT.

Jourdan, L.K., Broadway, S. Cassen, C., McKinnon, K.N., Ross, A.K., Martin, N.A., Clay, A.M., Carr, R.L. 2023. MODEL FOR THERAPEUTIC DEVELOPMENT IN TRAUMATIC BRAIN INJURY. Project 66. Mississippi State University Shackouls Honors College Summer Undergraduate Research Symposium, August 2.

Traumatic brain injury (TBI) is one of the most frequently occurring injuries with approximately 250,000 injuries and 69,000 deaths annually in the United States. TBI occurs when a forceful blow or whiplash induces damage in the brain. After the injury occurs, the timing of the treatment can be crucial to successful recovery and delayed treatment can have drastic physical, cognitive, and socioeconomic effects. The availability of therapeutics that can be rapidly administered post-injury is also important. The purpose of this research is to develop novel therapeutics to treat TBI that can be administered as a nasal spray formulation thereby allowing rapid administration quickly after diagnosis of a concussion. Thus, a model for TBI needs to be developed that can be utilized to further the research. To achieve this, we utilized a user-friendly platform weight-drop device that can induce TBI in a rat without surgical or pre-injury manipulations. Following anesthesia, 54 adult male rats were administered impact levels of either Sham (no impact), 0.5J, 1.0J, 1.5J, 2.0 J, or 2.5J with 9 rats per group. Post-impact, the rats are given pain medication and anti-sedatives. At 3, 7, and 14 days, 3 rats per impact level were sampled and the hippocampus and cerebral cortex were collected. The use of western blot analysis allowed the quantification of the levels of neuron-specific-enolase (NSE), a marker for neuronal damage, and of glial fibrillary acidic protein (GFAP), a marker for astrocyte activation. Maximum GFAP levels occurred 7 days post-impact. However, maximum NSE levels occurred on day 3 post-impact. Both markers increased as the impact level was raised but reached a plateau at higher impact levels. Based on the experiment, the impact level of 2.0J was decided to be the optimum level that will be used in future experiments to treat TBI.

