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Traumatic brain injury (TBI) is one of the leading causes of death and disability in children. Despite advances in care, morbidity following TBI remains high. Following the initial primary injury, critical secondary injuries develop from multiple mechanisms including hypoxemia, ischemia, hypoglycemia, and excitotoxicity. Treatment of secondary injuries to TBI patients provides a unique opportunity for therapeutic interventions and to overcome the narrow temporal window of efficacy of many therapeutics. Nicotinamide has been shown to provide neuroprotection when administered early after TBI in animal models. The hypothesis of this ongoing study is that NAD treatment before delayed hypoxemia in mice with TBI would reduce axonal injury and neuronal death. Twenty 8-week-old C57/B6 male mice underwent controlled cortical impact (CCI). Twenty-four hours following CCI, the mice were subjected to 60 minutes of hypoxemia (8% FiO2) treatment. Mice were randomly assigned to receive nicotinamide 500 mg/kg IP or an equivalent volume of saline 2 hours before hypoxemia. Mice were sacrificed 24 hour after hypoxemia. The brains of the mice were extracted and sectioned into 50 μm slices to be used for immunohistochemistry. We stained the slices for beta amyloid precursor protein (β-APP), NF200, and 4-HNE. Previous studies have suggested that these biomarkers are highly expressed short term following TBI. We also stained the slices with Fluoro-Jade C. Anterior white matter sections of the corpus callosum were analyzed with ImageJ and the Fluoro-Jade slides were scanned with a confocal. We found little distinguishable change between treatment/vehicle groups for βAPP and NF200 staining. However, our ImageJ protocol has not been correlated with stereology, the current gold standard for quantification. The remaining stains are still in progress of analysis. Furthermore, we began testing a second cohort with longer-term survival to analyze neuronal death.