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***Abstracts will be considered for both poster and platform presentations***

***Neurotrauma (TBI, spinal cord injury, etc.)***

Traumatic brain injury is a major source of acquired disability often resulting in long-term neurological deficits and mental health issues. Within a given year approximately 2.8 million individuals suffer a traumatic brain injury. Following the primary insult, pro-inflammatory responses can drive acute secondary brain injury and long-term neurological dysfunction. Beyond central nervous system dysfunction, there is increasing evidence suggesting profound alterations to the neuroenteric axis following traumatic brain injury. Both clinical and experimental traumatic brain injuries are capable of inducing in gastrointestinal damage and dysfunction in the absence of polytrauma. This includes complications with mucosal ischemia, motility changes, and barrier dysfunction. Despite this being recognized in the clinical population, the potential role of these changes in recovery has widely been ignored, and the mechanism by which gastrointestinal changes occur after traumatic brain injury is poorly understood. Intestinal damage is often underscored or concurrent with changes in the microbial population native to the intestinal tract. The native gut flora is a critical mediator of neuroenteric signaling and is vital for normal development of the brain and immune system and to maintain normal physical and psychological health. Dysbiosis, the disturbance of normal microbial populations, is associated with and can be caused by, and worsen, central nervous system insults, but further characterization of the microbiome after injury is needed. The current research seeks to better understand the changes that occur in the gastrointestinal tract and how microbiome alterations can contribute to secondary injury outcomes in the brain with the goal of identifying novel and non-invasive targets for intervention after traumatic brain injury.