EVALUATION OF NEUROINFLAMMATION AS MODULATOR OF TAU AGGREGATION IN RESPONSE TO REPETITIVE MILD TRAUMATIC BRAIN INJURY

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The pathological misfolding and aggregation of hyper-phosphorylated tau (ptau) protein in neurofibrillary tangles (NFTs) is the main hallmark of a group of neurodegenerative diseases called tauopathies. These include important disorders such as Alzheimer's Disease (AD) and chronic traumatic encephalopathy (CTE). Mounting evidence supports that the aggregation and deposition of misfolded proteins is an early event in the development of AD and CTE. Several studies uphold traumatic brain injury (TBI) as an important risk factor for both disorders, since there is a relationship between TBI severity and frequency, and the vulnerability to develop dementia. Importantly, individuals affected by TBI show elevated levels of ptau in cerebrospinal fluid and NFTs in specific brain areas. Although previous studies on tau mice subjected to TBI show increased ptau burden, the mechanisms underlying this phenomenon have not been explored. Besides, previous research neither recapitulate the effects of repetitive mild TBI (rmTBI), reported to lead to long-lasting neurological consequences. Acute and chronic inflammation are intimately associated to TBI events, but its role on tau aggregation over time remains unknown. Here, we analyze the neuroinflammatory response, at both short- and long-term, in transgenic tau mice after rmTBI. Our data suggest that rmTBI triggers microglia and astroglia activation and that they may be involved in the increase observed on tau pathology in concussed mice. This could indicate that neuroinflammation could have an active role in the increased risk to develop tauopathies after brain concussion.