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"The involvement of inflammatory processes underlying changes in neuronal activity after traumatic brain injury *in vivo* and *in vitro*"

By

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Wednesday, January 18th, 2023
11:00 A.M.
Chen conference room, NJIT, 111 Lock St., Newark; Webex
https://njit.webex.com/njit/j.php?MTID=m11c99131e1038b070cccde7c7c009b84
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ABSTRACT

Despite the prevalence of traumatic brain injuries (TBI) yearly, the outcomes of clinical trials for TBI treatments have been disappointing. To date, effective treatments for acute symptoms and development of chronic TBI-induced neuropathies like epilepsy are lacking. To model the clinical phenomenon that follow these types of traumatic injuries, *in vivo* and *in vitro* systems have been developed. Nonetheless, mechanisms underlying long-term susceptibility to epilepsy and the therapeutic potential of interventions have been difficult to elucidate for different forms of traumatic brain injury, direct impact (blunt injury) or indirect (blast injury). This study examines different forms of traumatic injuries *in vivo* and *in vitro* and their outcomes on neuronal function as well as potential therapeutic interventions to minimize injury-induced changes in neuronal function. We show that neuronal functional changes are capable of being produced in the presence and absence of neuronal loss, but also be due to secondary inflammatory mediators which contribute to alterations in neuronal function. *In vivo*, we show that acutely blocking TLR4 signaling after moderate FPI reduces dentate network excitability and seizure susceptibility. *In vitro*, we show that treatment with an anti-inflammatory cytokine, leukemia inhibitory factor (LIF), acutely after mild biaxial stretch injury reduces neuronal activity buts preserves mitochondrial dynamics. Therefore, we conclude that inflammatory processes contribute to neuronal activity after injury and therapeutic interventions administered early after injury play a role in mitigating injury induced changes in network activity.