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Neuroinflammatory mechanisms of posttraumatic Epilepsy

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Traumatic brain injury (TBI) occurs in as many as 64–74 million people worldwide each year and often results in one or more post-traumatic syndromes, including depression, cognitive, emotional, and behavioural deficits. TBI can also increase seizure susceptibility, as well as increase the incidence of epilepsy, a phenomenon known as post-traumatic epilepsy (PTE). Injury type and severity appear to partially predict PTE susceptibility. However, a complete mechanistic understanding of risk factors and biomarkers for PTE is incomplete. Accumulating evidence supports a significant role for neuroinflammation in the post-traumatic epileptogenic progression. Notably, substantial evidence indicates a role for astrocytes, microglia, chemokines, and cytokines in PTE progression. Microglia and astrocytes are activated and altered after TBI. Cytokines interleukin-1α, TNF and complement C1q are secreted by microglia and can induce the A1 astrocyte phenotype. Astrocytes suffer gap junction uncoupling and present both impaired neurotransmitter clearance and metabolic recycling from synapses. Cytokines interleukin-6, interleukin-1β, transforming growth factor beta, and chemokine CCL2 are secreted in high concentration creating a neuroinflammatory milieu. The cellular alterations and neuroinflammatory factors interactions ultimately contribute to the epileptogenic progression following TBI.

Recent Publications

1. Arisi, GM; Mukherjee, S; Mims, K; Hollingsworth, G; Oneil, K; Shapiro, LA. Neuroinflammatory mechanisms of post-traumatic epilepsy. J Neuroinflammation, 17, p. 193, 2020.
2. Foresti, ML; Arisi, GM: campbell, JJ; mello, LE. Treatment with CCR2 antagonist is neuroprotective but does not alter epileptogenesis in the pilocarpine rat model of epilepsy. Epilepsy & Behavior, 102, p. 106695, 2020.
3. Arisi, G M.; Foresti, M L.; Katki, K; Shapiro, L A. Increased CCL2, CCL3, CCL5, and IL-1β cytokine concentration in piriform cortex, hippocampus, and neocortex after pilocarpine-induced seizures. J Neuroinflammation, 12, p. 129, 2015.

Biography

Gabriel M. Arisi has more than twenty years of neuroscience research practice ranging from histology to molecular biology. A co-discoverer of caramboxin, a neural toxin presents in star fruit. He also contributed for the demonstration of the chemokine CCL2 as a biomarker of neural tissue damage. He has ten years' experience in teaching for medical, biomedical and nurse graduate students. He is proficient in electron and light microscopy and in molecular biology techniques such as immunohistochemistry and multiplex assays.

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