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Role of Brain Inflammation in Heightened Seizure Susceptibility After Early-Life Seizures

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Poster Presentation P75

**ROLE OF BRAIN INFLAMMATION IN HEIGHTENED SEIZURE SUSCEPTIBILITY
AFTER EARLY-LIFE SEIZURES**

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Research suggests a functional role for inflammation as a cause of neuronal hyperexcitability and neuron degeneration. Significant increases in genes that code for classic inflammatory responses have been observed after seizures induced by kainic acid (KA). Activation of microglia (immune cells the CNS) and increases of cytokines and complements are implicated as initiating events for seizure-induced inflammatory responses. Previous work has shown that a single seizure early in life can cause long lasting alterations that lead to a seizure-prone state. This research examines the role neuro-inflammation in the increase of seizure susceptibility. First, increased activation of microglia after early seizures was observed in both juvenile rates and transgenic mice. To more effectively demonstrate the role of inflammation in heightened seizure susceptibility, rats were treated with anti-inflammatory agents after early seizures. From this, it was observed that the anti-inflammatory agents are capable of decreasing both microglia activation and seizure susceptibility implicating a strong role for inflammation in increased seizure susceptibility. However, the use of anti-inflammatory treatments is also associated with risks resulting from immune system modulation.