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Oral Presentation

The Role of Neuroinflammation in Epilepsy: A New Target for Treatment

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Abstract

Despite progress in pharmacological and surgical treatments of epilepsy, little is known about the processes that a healthy brain is rendered epileptic after seizure occurrence. Growing evidence supports the involvement of inflammatory processes, both the adaptive immunity and systemic inflammatory response, in induction of individual seizures as well as in the epileptogenesis. Clinical and experimental investigations indicated that cortical spreading depression (CSD) play a role in epileptogenesis. CSD triggered seizure-like activities in human brain and lead to chronic epilepsy. Poly I:C (Toll-like receptor antagonist) attenuated CSD-induced production of cytokines in the brain and the spleen. In addition, application of poly I:C modulated CSD-induced expression of GABAAα, GABAAβ as well as Hsp70 and GAD65 in the entorhinal cortex. CSD-induced reactive astrocytosis, were paralleled by an increased expression of protein markers indicative of astrocytes and neuroinflammation in ex vivo brain tissues. Cultured astrocytes also showed an enhanced expression of the pro-inflammatory markers in CSD-treated brain. Targeting neuroinflammation with approved and available immunomodulatory treatments may thus represent a strategy to combat or ameliorate CSD-related disorders, such as epilepsy.

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