

(kang, epilepsy)

Brain inflammation plays a role in human epilepsy, but most studies have focused on acquired epilepsies, such as those due to head trauma, viral illness or other insults. Neuroinflammation in genetic epilepsy is poorly understood.

Jing-Qiong (Katty) Kang, MD, PhD, and colleagues previously characterized how mutations in the gene for a GABA-A receptor subunit — part of an ion channel that blunts excitatory signaling — contribute to epileptogenesis in mouse models.

They now report in the journal *Epilepsia* that mice with a particular GABA-A receptor subunit mutation (a model for the genetic epilepsy Dravet syndrome) have increased levels of proinflammatory factors in the brain, but not in the plasma. They found increased neuroinflammation in multiple brain regions and throughout different developmental stages and showed that it was independent of seizure occurrence.

The findings support neuroinflammation as a mechanistic link between genetic and acquired epilepsy and suggest that anti-inflammatory treatments might be beneficial for some forms of genetic epilepsy.

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