Anti-inflammatory effects of the anticonvulsant drug levetiracetam on electrophysiological properties of astroglia are mediated via TGFβ1 regulation

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The involvement of astrocytes as immune-competent players in inflammation and the pathogenesis of epilepsy and seizure-induced brain damage has recently been recognized. In clinical trials and practice, levetiracetam (LEV) has proven to be an effective antiepileptic drug (AED) in various forms of epileptic seizures, when applied as mono- or added therapy. Little is known about the mechanism(s) of action of LEV. Evidence so far suggests a mode of action different from that of classical AEDs. We have shown that LEV restored functional gap junction coupling and basic membrane properties in an astrocytic inflammatory model in vitro.

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