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### Poster Presentation

#### Astrocytes Alterations in Epileptic Disorders

Arezoo Rajabian\*, Mahmoud Hosseini

Neuroscience Research Center, Mashhad University of Medical Sciences, Mashhad, Iran

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#### **Abstract**

**Introduction:** Epilepsy affects about 1% of the population. Approximately 30% of epileptic patients are resistant to current antiepileptic drugs. **Materials and Methods:** This article reviewed the mechanisms underlying epileptic seizures. **Results:** recently, glial cells have been considered as new targets for epilepsy treatment. Glial cells previously were considered as supportive elements for brain function; however, their role is beyond. They deliver neuroactive molecules and adjust synaptic transmission. Increasing  $\text{Na}^+$  channels, decreasing inwardly rectifier  $\text{K}^+$  channels or water channels, reduction of glutamine synthetase, and enhancement of glutamate dehydrogenase are among the characteristics of astrocytes in sclerotic hippocampus, which is commonly seen in the epileptic brain. The release of glutamate from astrocytes enhances neuronal irritability during seizure-like events. Astrocytes are the target of inflammatory molecules and can generate cytokine molecules. Up-regulation of interleukin-1 receptor type 1(IL-1R1) or Toll-like receptors (TLRs) in reactive astrocytes in the human brain has been reported in epilepsy. Cannabinoid (CB) receptors exert an immunomodulatory effect on astrocytes. Astrocytic calcium oscillations can mediate astrogliosis, which is a well-known feature in mesiotemporal lobe epilepsy. Astrogliosis is enough to provoke epileptic seizures. The extracellular and synaptic adenosine (Ado) level is mainly regulated by astrocytes. Seizures occur in animals lacking oligodendrocytic connexin (Cx) Cx32 and astrocytic Cx43. **Conclusion:** the bidirectional flow of molecules between astrocytes and the surrounding microenvironment makes the understanding of ongoing mechanisms more difficult. Each effector in this regard may be considered a therapeutic target for attenuating seizure and epilepsy.

**Keywords:** 1. Gliosis 2. Epilepsy 3. Neuroglia 4. Seizures

**Corresponding Author:** Arezoo Rajabian

**Email:** [rajabianar@gmail.com](mailto:rajabianar@gmail.com)

