Temporal lobe epilepsy etiology

Despite recent advances in our understanding of synaptic transmission associated with epileptogenesis, the molecular mechanisms that control seizure frequency in patients with temporal lobe epilepsy (TLE) remain obscure.

The most common cause of temporal lobe epilepsy is mesial temporal sclerosis.

The expression of some proteins involved in the packaging of vesicular neurotransmitters is altered in TLE. In addition, upregulated expression of annexin family proteins, which are also related to TLE, might play an important role in protection against TLE [1].

Data demonstrate that mTOR signaling is significantly dysregulated in human TLE, offering new targets for pharmacologic interventions. Specifically, clinically available drugs that suppress mTORC1 without compromising mTOR2 signaling, such as rapamycin and its analogs, may represent a new group of antiepileptogenic agents in TLE patients [2].

Water homeostasis has been shown crucial for regulation of neuronal excitability. The control of water movement is achieved through a family of small integral membrane channel proteins called aquaporins (AQPs). Despite the fact that changes in water homeostasis occur in sclerotic hippocampi of people with temporal lobe epilepsy (TLE) , the expression of AQPs in the epileptic brain is not fully characterised [3].

Soluble human epoxide hydrolase 2 is increased in both lateral and medial temporal tissues in temporal lobe epilepsy. Further studies should be conducted as inhibition of this enzyme has resulted in a significant decrease in or stopping of seizures and attenuated neuro-inflammation in experimental epilepsy models in the current literature [4].

Cavernous malformation related temporal lobe epilepsy

Cavernous malformation related temporal lobe epilepsy (CRTLE)

1)
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