

Special Issue

Synaptic Changes in Epilepsy

Message from the Guest Editor

Research on the pathophysiology of epilepsy has focused on persistent intrinsic changes in excitability using various models of experimental epilepsy and also on human tissue from epilepsy surgery. In these studies, the altered transcription of Na⁺, Ca²⁺, or K⁺ channels was described in epileptic tissue and these were referred to as acquired channelopathies. In recent years, more attention has been drawn to neuronal networks affected by disease in tissues such as the hippocampus. It is well established that pathological axon sprouting gives rise to new synapses with uncommon properties in epileptic tissues. However, we now know that also glutamate and GABA receptors may persistently be altered and further transmitter receptors are increasingly being studied. Thus, elucidating epileptic synaptopathy adds significantly to our pathophysiological understanding of seizure initiation and disease progression during epileptogenesis. This Special Issue is dedicated to synaptic changes in epilepsy. Studies on interventions to interfere with epilepsy-associated synaptic changes and thus disease-modifying effects are also welcome.

Guest Editor

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