

**June 3, 2009**

Temporal lobe epilepsy: limbic networks and synaptic mechanisms

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**ABSTRACT OF THE TALK**

Temporal lobe epilepsy is a partial epilepsy disorder in which seizure discharges involve the hippocampus proper, extrahippocampal structures such as the entorhinal and perirhinal cortices, the amygdala, and the temporal neocortex. TLE patients are often unresponsive to antiepileptic drugs and present with a typical pattern of brain damage known as Ammon's horn sclerosis (or mesial temporal sclerosis) that is typically characterized by neuronal loss in hippocampal CA1/CA3 subfields, dentate hilus, layer III of the medial entorhinal cortex, and amygdala.

Similar histopathological changes are seen in laboratory animals by injecting convulsant drugs (e.g., pilocarpine or kainic acid) or by repetitive electrical stimulation of limbic pathways. These experimental procedures induce an initial *status epilepticus* and cause 1 to 4 weeks later a chronic condition of recurrent limbic seizures that are also poorly controlled by antiepileptic drugs. Interestingly, a similar, seizure-free, latent period is seen in TLE patients who can present with an initial insult in early childhood (e.g., birth trauma,

complicated febrile convulsions, brain injury or meningitis) and develop partial seizures in adolescence or early adulthood.

Here, we will discuss the topic of limbic neuronal networks in TLE by: (i) summarizing the changes in synaptic excitability that occur in parahippocampal structures (including the subiculum) of pilocarpine-treated epileptic rats; (ii) reviewing the cell damage that occurs in the mesial temporal lobe of these animals, and particularly the presence of a lesion in the CA3 stratum lacunosum-moleculare that destroys the perforant path and correlates with the presence of spontaneous generalized seizures during the chronic epileptic state; and (iii) discussing how hippocampus-parahippocampal networks interact in the presence of these structural changes.

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