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Modeling of seizure transitions with ion concentration dynamics

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Traditionally it is considered that neuronal synchronization in epilepsy is caused by a chain reaction of synaptic excitation. However, it has been shown that synaptic transmission is not necessary for epileptiform synchronization [1]. In order to investigate the respective roles of synaptic and non-synaptic neuronal coupling in seizure transitions, we developed a computational model of hippocampal network, involving extracellular space, realistic dynamics of Na^+, K^+ and Cl^- ions, the glial uptake and diffusion mechanism. We show that network behavior under synaptic coupling conditions may be quite different from the neurons' activities when specific non-synaptic components are included. In particular, we show that in the extended model, strong discharge of inhibitory interneurons may result in long lasting accumulation of extracellular K^+, which sustains depolarization of principal cells and causes their pathological discharges. This effect is not present in a reduced, purely synaptic network. These results confirm the experimental hypothesis that increase of inhibitory interneurons firing may lead to increased firing in the pyramidal cells through accumulation of extracellular potassium [2]. The model also shows that all potassium clearance mechanisms (glial uptake, sodium-potassium pump, potassium diffusion) are critically important to reproduce the experimental findings. This means that computational modeling of seizure activity without ion dynamics may lead to unrealistic results.

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