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Electrophysiological characterization of homeostatic synapse-driven membrane plasticity in hippocampal CA1 pyramidal cells

In neuronal network, neurons receive information input from synapses and, in response, fire action potential (AP) trains as an output. Chronic changes of global neuronal network activity induce homeostatic plasticity to maintain the functional output of neurons at a stable level. Different forms of homeostatic plasticity modulate synaptic weights or the intrinsic excitability. Here, I focused on the homeostatic synapse-driven membrane plasticity (hSMP) induced by chronic NMDA receptor inactivation or activation in CA1 pyramidal neurons. I used the antagonist and co-agonist to pharmacologically activate or inhibit the NMDAR responses. Then current infection elicited AP firing was recorded as a measurement for neuronal intrinsic excitability. After chronic pharmacological blockade of NMDARs, the excitability of pyramidal neurons was increased. This change in intrinsic excitability was correlated to changes in some membrane properties including a reduced AP threshold, a shortening of AP duration and an increase in input resistance. On the other hand, the chronic activation of NMDARs by a co-agonist caused variable effects on hippocampal slices. In responsive slices, chronic enhancement of NMDARs caused an increase in SK channel mediated mAHP current and a reduction of the input resistance, contributing to the decrease in the intrinsic excitability. These results provide evidence that bidirectional hSMP can be expressed in the excitatory system in hippocampal slices and may contribute to maintain the global output of the network.