Presentation Abstract

Program#/Poster#: 686.07/A68

Presentation Title: Subthreshold conductances regulate local field potentials and theta-frequency spike phase preference of hippocampal model neurons

Location: WCC Hall A-C

Presentation time: Wednesday, Nov 19, 2014, 8:00 AM -12:00 PM

Presenter at Poster: Wed, Nov. 19, 2014, 10:00 AM - 11:00 AM

Topic: ++B.04.f. HCN and non-selective cation channels

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Abstract: Despite the presence of a rich repertoire of somatodendritic subthreshold voltage-gated ion channels, the focus of most studies on local field potentials (LFP) has been confined to the contributions of synaptic and suprathreshold conductances. Can hyperpolarization-activated cyclic-nucleotide gated (HCN) channels and their somatodendritic gradients, given their ability to regulate intrinsic excitability and to introduce an inductive lead in impedance phase, alter LFPs and associated spike phase preference? Here, we computed LFP at different strata of the hippocampal CA1 region using line source approximation of currents from 400 morphologically realistic conductance-based models of pyramidal neurons. These neurons received randomized theta-frequency modulated excitatory inputs at dendrites and perisomatic inhibitory afferents. We inserted HCN channels into all model neurons with an experimentally constrained somatodendritic gradient and computed LFPs and neuronal spike phase preference. We compared these measurements with those obtained in the absence of HCN channels, and observed a significant lead in the LFP phase across different strata of the CA1 region, without significant changes in LFP amplitudes. Further, the presence of HCN channels also resulted in a lag in the spike phase preference and increased spike phase coherence. As HCN channels
alter both excitability and impedance phase, we assessed the relative contribution of these two components by replacing HCN channels with fast-activating counterparts and found a significant contribution of impedance phase to LFP and spike phases. How does plasticity in the synaptic receptors and HCN channels alter LFPs and spike phase? In answering this, we found that an increase in HCN conductance resulted in a progressive increase in LFP phase lead, spike phase lag and spike phase coherence. An increase in either excitatory or inhibitory synaptic conductances resulted in an increase in LFP amplitude, but did not significantly alter spike phase coherence. However, whereas an increase in excitatory conductances introduced a lag in the LFP phase and a lead in the spike phase, an increase in inhibitory conductances led to the opposite. Finally, on changing the phase difference between the theta-modulation of inhibitory and excitatory input afferents, we found that LFP and spike phases reliably reflected this difference. Our results suggest that subthreshold conductances can intricately regulate the LFPs and neuronal spike phase preference through changes in excitability and impedance phase, and identify specific roles for these conductances in phase coding and in the dynamics of cell assemblies.

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