

Poster presentation

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The role of dendritic plasticity in noise induced synchrony

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There are several neurological diseases associated with pathological disturbances in ion channel fluctuations of the dendrite. An example of this is the acquired channelopathy of A-type K⁺ channels that develops in the pyramidal neurons of the CA1 region of the hippocampus in the pilocarpine model of temporal lobe epilepsy [1]. As changes to the expression level of ion channels in the dendritic membrane take place, the excitability of the neuron increases, leading to a greater susceptibility to seizure activity.

In light of this, it is instructive to ask how dendritic plasticity alters the synchronization properties in neuronal populations. It has previously been shown that both active and passive dendritic contributions play a significant role in the phase response of a somatic oscillator, impacting synchronization properties of a neuron on a network level [2]. The aim of this work is to study the influence of both active and passive changes of dendritic conductances on the phase resetting curve (PRC) of an oscillator in the presence of noisy synaptic input, and hence to examine the effect of dendritic plasticity on the synchronization properties and reliability of a repetitively firing neuron.

Given the structure of the somatic phase resetting curve and the dendritic properties of interest, a recent paper lays the groundwork of this study by developing a closed-form approximation for dendritic contributions to the somatic PRC that is expressed entirely in terms of properties of the postsynaptic neuron [3]. Here the system is modeled as a

quasi-linearized dendritic cable coupled to a somatic oscillator. We proceed with our analysis by applying a phase reduction to our model and treating synaptic input as white noise as in [4] and [5]. We use this framework to explore the effects of alterations in the dendritic excitability on the synchronization properties and reliability of a neuron in the presence of noise. A comparison is then made between this analysis, which incorporates the linear and nonlinear contributions of the cable but is indiscriminant in its synaptic filtering, and a second analysis in which a filtered version of the noise is injected directly into the soma, excluding direct dendritic considerations.

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