Abstract View

PROPAGATION IN THALAMOCORTICAL DENDRITES.

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Dendritic propagation of low threshold Ca2+ potentials (LTCPs) and fast Na+ action potentials (APs) were investigated using in vivo and in vitro computational models of 3-D reconstructed neurons from the somatosensory and visual thalamus. Hot-spot, monotonic, bimodal, proximal, uniform channel densities were considered. For each, localized burst inhibition of various intensities elicited a range of LTCP amplitudes. In all cases, LTCPs were uniform and synchronous events: it was not possible to elicit an LTCP that was either localized or spatiotemporally structured on a relevant scale - even when individual trees were differentially modulated. We suggest that this "whole-cell" nature of LTCPs provides an intracellular synchronization signal. In trials using uniform somato-dendritic Na+/K+ channel densities, APs peaked uniformly and nearly synchronously throughout the arbor (initiating tens of usec earlier in some dendrites that had received inhibition). However, the dendritic K+ channels rapidly repolarized their membrane, which truncated calcium gating and hindered further Na+ spiking. In trials using a soma-only Na+/K+ distribution, APs passively backpropagated into the arbor at a rate of ~250um/ms, depolarizing all branches by at least 35mV with a spike shaped timecourse. The absence of dendritic Na+/K+ channels allowed the arbor to continue on an LTCP timecourse, which left the arbor sufficiently depolarized to generate additional Na+ spikes in a burst. We conclude that 1) dendritic Na+/K+ channels are not necessary to produce strong depolarizing signals throughout the dendritic arbor, 2) their absence in the dendrites would enhance bursting, and 3) only moderate dendritic densities could provide virtually instantaneous AP feedback to synaptic mechanisms.
Supported by: HHMI

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