Activity-dependent control of neuronal output by local and global dendritic spike attenuation.
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Neurons possess elaborate dendritic arbors which receive and integrate excitatory synaptic signals. Individual dendritic subbranches exhibit local membrane potential supralinearities, termed dendritic spikes, which control transfer of local synaptic input to the soma. Here, we show that dendritic spikes in CA1 pyramidal cells are strongly regulated by specific types of prior input. While input in the linear range is without effect, supralinear input inhibits subsequent spikes, causing them to attenuate and ultimately fail due to dendritic Na+ channel inactivation. This mechanism acts locally within the boundaries of the input branch. If an input is sufficiently strong to trigger axonal action potentials, their back-propagation into the dendritic tree causes a wide-spread, global reduction in dendritic excitability which is prominent after firing patterns occurring in-vivo. Together, these mechanisms control the capability of individual dendritic branches to trigger somatic action potential output. They are invoked at frequencies encountered during learning, and impose limits on the storage and retrieval rates of information encoded as branch excitability.