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A model for correlation detection based on Ca^{2+} concentration in spines

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Understanding the mechanisms of correlation detection between pre- and postsynaptic activity at a synapse is crucial for the theory of Hebbian learning and development [1,2] of cortical networks. The calcium concentration in spines was experimentally shown to be a correlation sensitive signal confined to the spine: A supralinear influx of calcium into spines occurs when presynaptic stimulation precedes a backpropagating action potential within a short time window. The magnitude of the influx depends on the relative timing t_{post}-t_{pre} [3]. There is strong evidence that NMDA (N-methyl d-aspartate) receptors are responsible for the supralinear effect [3]. Previous simulation studies relate the occurrence of spike time dependent plasticity to this calcium signal [4,5]. However, these simulations mainly focus on pairs and triplets of pre- and postsynaptic spikes, rather than on irregular activity. Here, we investigate the properties of a biologically motivated model for correlation detection based on the calcium influx through NMDA receptors under realistic conditions of irregular pre- and postsynaptic spike trains with weak correlation. We demonstrate that a simple thresholding mechanism acts as a sensitive correlation detector robustly operating at physiological firing rates. We identify the regime (rate, correlation coefficient, detection time) in which this mechanism can assess the correlation between pre- and postsynaptic activity. Furthermore, we show that correlation controlled synaptic pruning acts as a mechanism of homeostasis, and that cooperation between synapses leads to a connectivity structure reflecting the spatial correlations in the input. The detector model allows for a computationally effective implementation usable in large-scale network simulations. On the single synapse level most of the results are confirmed by an analytical model.

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