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Location-specific activity of signaling molecules underlying STDP in a model CA1 pyramidal neuron

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Spike-timing-dependent plasticity (STDP) is a form of bidirectional change in synaptic strength that depends on the temporal order and temporal difference of the pre- and postsynaptic activity [1]. The synapse undergoes long-term potentiation (LTP) if the presynaptic spike precedes the postsynaptic spike, and exhibits long-term depression (LTD) if the temporal order is reversed. Recent physiological observations suggest that the form of plasticity at a synapse depends not only on the timing of the pre- and postsynaptic activity but also on the location of the synapse on the dendritic tree [2]. We proposed a biophysical model of STDP predicting that learning rules are location-dependent [3]. Numerous modeling studies investigate molecular mechanisms of synaptic plasticity (e.g. [4], [5]). However, the influence of the dendritic location of the synapse on the plasticity mechanisms has not been addressed in detailed models of STDP.

It is known that calcium-activated CaMKII and calcineurin cause phosphorylation or dephosphorylation of AMPA-type glutamate receptors, and these changes are thought to underlie LTP and LTD. In this study, we model the trigger of the second messenger cascades, the calcium signal, by pairing the AMPA and NMDA receptor activation with a backpropagating action potential at a spine close to the soma and by pairing the AMPA and NMDA receptor activation with a dendritic spike at a spine in distal dendritic regions. We employ a detailed compartmental model of CA1 cell [6] and adjust the calcium handling mechanism following [7]. The resulting calcium signals are used in a bistable biochemical model of the CaMKII autophosphorylation and dephosphorylation system [5]. In this model, transition from a weakly phosphorylated state to a highly phosphorylated state corresponds to LTP, and transition into the opposite direction leads to LTD.

We show that CaMKII is highly phosphorylated for the multiple pre-post spike pairing protocol and it is weakly phosphorylated if the temporal order is reversed in a proximal spine. These results are consistent with the rules for LTP/LTD induction observed experimentally. However, CaMKII stays highly phosphorylated for the pre-post and post-pre protocols in a distal spine and implies that synapses tend to avoid transitions to LTD neglecting the temporal order of the pre- and local postsynaptic events in distal dendritic regions. The results imply that synapse location is one of the critical factors for plasticity rules at a synapse.

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