Canadian Neuroscience Meeting, CAN 2014

Calcium-dependent calcium decay explains STDP in a dynamic model of hippocampal synapses

Dominic Standage[1], Thomas Trappenberg[2] and Gunnar Blohm[1]

[1]Queen's University, Canada; [2]Dalhousie University, Canada

It is widely accepted that the direction and magnitude of synaptic plasticity depends on post-synaptic calcium flux, where high levels of calcium lead to long-term potentiation and moderate levels lead to long-term depression. At synapses onto neurons in region CA1 of the hippocampus (and many other synapses), NMDA receptors provide the relevant source of calcium. In this regard, post-synaptic calcium captures the coincidence of pre- and postsynaptic activity, due to the blockage of these receptors at low voltage. Previous studies show that under spike timing dependent plasticity (STDP) protocols, potentiation at CA1 synapses requires post-synaptic bursting and an interpairing frequency in the range of the hippocampal theta rhythm. We hypothesize that these requirements reflect the saturation of the mechanisms of calcium extrusion from the postsynaptic spine. We test this hypothesis with a minimal model of NMDA receptor-dependent plasticity, simulating slow extrusion with a calcium-dependent calcium time constant. In simulations of STDP experiments, the model accounts for latency-dependent depression with either post synaptic bursting or theta-frequency pairing (or neither) and accounts for latencydependent potentiation when both of these requirements are met. The model makes testable predictions for STDP experiments and our simple implementation is tractable at the network level, demonstrating associative learning in a biophysical network model with realistic synaptic dynamics.