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A model of activity-dependent changes in dendritic spine density and spine structure

Recent evidence indicates that the geometry of a dendritic spine influences the dynamics of calcium in the spine and is regulated during synaptic plasticity. For instance, a moderate rise in calcium can cause elongation, while a very large increase in calcium causes fast shrinkage and the eventual collapse of a spine. This expansion and shrinkage depends on the frequency of the synaptic input to a spine. Here, we extend previous modeling studies due to Verzi *et al.* (J. Neurophys., 2005) by combining models for activity-dependent spine density and spine stem resistence with one for calcium-mediated spine-stem restructuring. The spine density model is based on the standard dimensionless cable equation, which is used to model the changes in trans-membrane potential in a passive dendrite. Additional equations represent the activity dependent changes in spine density along the dendrite, the current balance equation for the spine head, the calcium concentration in the spine head, and the spine stem resistance. For this continuum model, Hodgkin–Huxley type kinetics represent the changes in trans-membrane potential in the spine head. We are using computational studies to investigate the changes in spine density and structure for a variety of synaptic inputs of different frequencies. In particular, we are using the model to investigate the mechanisms underlying changes in spine density and morphology and the role of spine plasticity in long-term depression (LTD) and long-term potentiation (LTP), which are thought to contribute to learning and memory.