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

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Recent evidence indicates that the morphology and density of dendritic spines are regulated during synaptic plasticity. See for instance a review by [1]. High-frequency stimuli that induce long-term potentiation (LTP) have been associated with increases in the number and size of spines. In contrast, low-frequency stimuli that induce long-term depression (LTD) are associated with decreases in the number and size of spines. Decreases in spine density also occur due to excitotoxicity associated with very high levels of activity such as during seizures.

In this work, we extend previous modeling studies [2] by combining a model for activity-dependent spine density with one for calcium-mediated spine stem restructuring. The model is based on the standard dimensionless cable equation for the changes in membrane potential in a passive dendrite. An additional equation characterizes the activity-dependent changes in spine density along the dendrite. For this continuum model, a typical Hodgkin-Huxley type current balance equation represents the change in membrane potential in an isopotential compartment representing a spine head. Both the cable equation and the current balance equation rely on the spine stem current to represent current flow between the spines and the dendrite. The model also includes equations for activity-dependent changes in the calcium concentration in spines as well as changes in spine stem resistance that depend on the level of calcium in an individual spine. The calcium-mediated changes in spine density and spine stem resistance are based on a conceptual model proposed by Segal et al. [1] where low calcium concentrations lead to spine shrinkage and pruning, an increase in calcium concentration leads to spine elongation and formation of new spines, and significantly higher values cause spine shrinkage and pruning.

We use computational studies to investigate the changes in spine density and structure for differing synaptic inputs and demonstrate the effects of these changes on the input-output properties of the dendritic branch. Moderate amounts of high-frequency synaptic activation to dendritic spines cause an increase in spine stem resistance, which is correlated with spine stem elongation. In addition, the spine density increases both inside and outside the input region. The model is formulated so that this LTP-inducing stimulus eventually leads to structural stability. In contrast, a prolonged low-frequency stimulation paradigm that would typically induce LTD results in a decrease in stem resistance (correlated with spine shortening) and an eventual decrease in spine density.

References

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