

Poster presentation

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Modeling structural plasticity in dendrites with multiple spine types

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Introduction

Dendritic spines can change in number and shape in response to various physiological, behavioral or pathological states. This activity-dependent structural plasticity exists over a vast range of time scales, from minutes to days or weeks [1,2]. Although spines may have a continuum of shapes, evidence suggests that spines may cluster in defined groups by their shapes [3-6]. Further evidence suggests that spine morphology correlates with distance from the cell body [3,4]; proximal to the soma, where the dendrite has the largest diameter, spines have short necks and appear stubby, whereas distally, where the dendrite is thinner, spines have thinner and longer stems. At intermediate distances, a variety of spine types are seen including the intermediate mushroom-shaped spines.

We formulate a stage-structured population model for activity-dependent spines of three types: stubby (type-I), mushroom (type-II), and thin (type-III). Each spine type is characterized electrically by a spine stem resistance and chemically by a parameter that controls the level of calcium accumulation in the spine head. Transitions between spine types are driven by intraspine calcium levels that depend on local electrical activity. A continuum formulation based on the cable equation [7] represents a dendritic branch and includes the stage transitions between stubby, mushroom, and thin dendritic spines. The model allows for a study of the interaction between the many activity-dependent (active or passive) spines and for an investigation of the impact of their individual and collective dynamics on the output properties of the dendrite.

Simulations are run for both passive and active spines to investigate how dendritic diameter and synaptic input frequency influence spine morphology along the dendrite. In the passive case, the model is reduced to a system of ordinary differential equations because evidence shows spatially uniform repetitive synaptic input to passive spines, within an input region, drive spine restructuring within that region, with little or no effect on dendritic output and structural change outside the input region. This is in stark contrast to excitable spines where synaptic activity can drive restructuring and dendritic output far from the input region. Finally, we discuss how this model can be extended to study the formation and loss of spines as well as how to handle different input frequencies.

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