

POSTER PRESENTATION

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A spatiotemporal model of spine calcium dynamics in the hippocampus

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Ca²⁺-signalling in dendritic spines is required for NMDA receptor-dependent synaptic plasticity at glutamatergic synapses in the hippocampus [1]. However, it is not clear whether plasticity induction is dependent solely on the global signal, i.e., the spine volume-averaged Ca²⁺ signal; or whether plasticity induction is also sensitive to Ca²⁺-channel nanodomain signaling [2]. A working hypothesis of this work is that temporal and spatial variations in postsynaptic intracellular [Ca²⁺]-fields may be significant factors governing the signaling cascades that lead to either long-term synaptic potentiation or depression. Direct measurement of [Ca²⁺] distributions in dendritic spines is experimentally difficult but we can investigate this hypothesis using mathematical models of Ca²⁺ diffusion.

We have developed a spatio-temporal model of Ca²⁺ diffusion in three dimensions. We then study our model using finite element methods. The model allows predictions of intracellular [Ca²⁺]-field responses to combinations of pre- and post-synaptic spikes with nanometre and millisecond spatio-temporal resolution. Our results so far indicate that Ca²⁺ signalling is highly spatially non-uniform and that Ca²⁺ signal differences between induction protocols is dependent on location within the spine. This has implications for the ultimate biological role of the Ca²⁺ signal given that the relevant receptors in the spine are organised inhomogeneously [3].

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