BMC Neuroscience

Poster presentation

An efficient Ca^{2+} based plasticity rule with combined Ca^{2+} sources

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from Sixteenth Annual Computational Neuroscience Meeting: CNS*2007
Toronto, Canada. 7–12 July 2007

Published: 6 July 2007

BMC Neuroscience 2007, **8**(Suppl 2):P93 doi:10.1186/1471-2202-8-S2-P93

A number of research groups have proposed generative, Ca^{2+} based plasticity models in recent years. Such rules are based on the premise that moderate, above-basal levels of post-synaptic Ca^{2+} lead to long term depression (LTD) and that high levels lead to long term potentiation (LTP). We present such a rule and discuss its assumptions and implications.

Our rule has similarities with two models in [1] in that Ca^{2+} may enter the post-synaptic density (PSD) through voltage gated channels $Ca^{2+}(V)$ and NMDA receptor (NMDAR) mediated channels $Ca^{2+}(V, NMDA)$. Unlike Model 1 in their study and the model of the Shouval group [2], our model achieves spike time dependent LTD without the requirement that back-propagating action potentials (BAP's) have a long tail. Thus, we do not assume this tail is sufficient to expel Mg^{2+} from glutamate-bound NMDAR's. In our model, LTP and LTD processes are compounded while Ca^{2+} exceeds LTP and LTD thresholds respectively. We do not use a specific function of peak Ca^{2+} or the time-integral of pre- and post-synaptic interactions.

The simple formulation of our model makes fewer assumptions about the underlying biology of NMDAR-dependent plasticity than the models in [1] and [2], but our simulations of spike-time dependent plasticity (STDP) experiments show similar output to theirs. For *post-before-pre* spike pairings, depression is graded because the respective time courses of Ca^{2+} and NMDAR-activation are sufficiently long to interact with one another. $Ca^{2+}(V)$ is spatially non-specific because it is driven by the BAP, but NMDAR's provide an indicator of pre-synaptic plasticity that interacts with this Ca^{2+} source. We use NMDAR's in this role for convenience, as other molecules could serve this purpose. This mechanism is similar to Model 2 in [1] where the two Ca^{2+} sources are separate. Here, the Ca^{2+} sources are combined to exceed the LTP threshold, resulting in the much-debated LTD window at long-latency *pre-before-post* pairings.

Our model points to several mechanisms for experimental study. For instance, spatially non-specific $Ca^{2+}(V)$ must integrate with $Ca^{2+}(V, NMDA)$ in the PSD very quickly to produce LTP. Alternatives to rapid integration at the PSD include the possibility that plasticity-inducing processes determine the relative levels of Ca^{2+} inside and outside the PSD, that $Ca^{2+}(V, NMDA)$ exceeds $Ca^{2+}(V)$ by some margin, or that Ca^{2+}-dependent release from internal stores plays a role in this regard.

References

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