Poster presentation

An efficient Ca²⁺ based plasticity rule with combined Ca²⁺ sources

Dominic Standage* and Thomas Trappenberg

Address: Faculty of Computer Science, Dalhousie University, Halifax, Canada

Email: Dominic Standage* - standage@cs.dal.ca

* Corresponding author

A number of research groups have proposed generative, Ca²⁺ based plasticity models in recent years. Such rules are based on the premise that moderate, above-basal levels of post-synaptic Ca²⁺ 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 Ca²⁺ 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 Ca²⁺ exceeds LTP and LTD thresholds respectively. We do not use a specific function of peak Ca²⁺ 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 Ca²⁺ 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 Ca²⁺ 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 Ca²⁺ sources are separate. Here, the Ca²⁺ 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 Ca²⁺ inside and outside the PSD, that $Ca^{2+}(V, NMDA)$ exceeds $Ca^{2+}(V)$ by some margin, or that Ca²⁺-dependent release from internal stores plays a role in this regard.

References

1. Karmarkar U, Buonomano D: **A model of spike-timing dependent plasticity: one or two coincidence detectors?** *J Neurophysiol* 2002, **88**:507-513.
2. Shouval H, Bear M, Cooper L: **A unified model of NMDA receptor-dependent bidirectional synaptic plasticity.** *PNAS* 2002, **99**:10831-10836.