Plasticity and Homeostasis

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In its most naive form Hebbian plasticity leads to diverging synaptic weights. Solutions to this such as normalization models, Bienenstock-Cooper-Munro (BCM) theory, and hard bounds have been proposed to deal with this problem. However, their biological relevance is not always clear, yet, many aspects of synaptic plasticity models depend strongly on the solution chosen. We argue that simple weight-dependent plasticity, in which potentiation is relatively weaker for stronger synapses, is an effective way to deal with the weight divergence, and is consistent with a number of biological observations. We show how these 'soft-bound' plasticity rules maximize synaptic information storage capacity in a single neuron recognition memory task.

It has been observed that the synaptic spine volume increases (decreases) with increases (decreases) in synaptic strength. We show how this effect leads to plasticity that depends on the strength of the synapse. In particular, stronger synapses will have larger spines and therefore experience lower Ca transients than small synapses. Thus we hypothesize that this is the biophysical mechanism behind the weight synaptic plasticity. Moreover, the model predicts that large synapses will have meta-stable states that are relatively immune to depression.

Homeostasis has often been introduced alongside synaptic plasticity models to ensure stability of neuronal networks and induce competition between synapses. We introduce a framework for linear homeostatic controllers. We show that stability of a single neuron does not guarantee stability of a network of neurons. In particular, we find that slow oscillations can develop that defeat the purpose of homeostasis. Moreover we find that adding more filters in the feedback loop can also have de-stabilizing effects on the network level. These results constrain biological and engineered homeostatic controllers.