

Abstract View

HOMEOSTATIC METAPLASTICITY ACCOUNTS FOR SYNAPTIC SCALING.

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Recently, a new property of synaptic plasticity, named synaptic scaling, has been described (Turrigiano et al., 1998). Synaptic scaling is an activity-dependent physiological mechanism for preserving cortical homeostasis. Its underlying biophysical basis, however, is not yet elucidated.

Homeostatic metaplasticity had previously been proposed as a means of stabilizing the inherently unstable Hebbian plasticity. For example, the BCM theory of synaptic plasticity assumes a sliding modification threshold that stabilizes learning while producing selective receptive fields (Bienenstock et al., 1982). Here, we suggest a biophysical formulation of metaplasticity as the underlying mechanism for synaptic scaling. We have recently developed a unified theory of synaptic plasticity that can account for various induction paradigms (Shouval et al., 2002). In this model, AMPA receptor plasticity is induced by calcium transients through NMDA receptors. Thus changing the properties of NMDA receptors can alter the form of synaptic plasticity.

We propose a form of metaplasticity that is based on cell-wide activity-dependent regulation of NMDA receptor conductance. Simulations of this combined plasticity-metaplasticity system show that, as the average input rate increases, synaptic weights decay, and the resulting distribution of synaptic weights is unimodal. In addition, the output-firing rate is roughly maintained, even in the absence of hard constraints on individual or the average synaptic weights. Therefore, metaplasticity and synaptic plasticity can account for the experimental observations of synaptic scaling and maintain cortical homeostasis. Additional mechanisms that explicitly enforce scaling are not required.

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