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An adaptive spike-timing-dependent plasticity rule

Jesper Tegnér[a,b,*], Ádám Kepecs[c]

[a] *Stockholm Bioinformatic Center, SCFAB, S-106 91 Stockholm, Sweden*
[b] *Department of Numerical Analysis and Computer Science, Royal Institute of Technology, S-100 44 Stockholm, Sweden*
[c] *Volen Center for Complex Systems, Brandeis University, Waltham, MA 02454, USA*

Abstract

We examine the conditions under which spike-timing-dependent plasticity (STDP) normalizes post-synaptic firing rates. Our simulations show that the rate normalization property of STDP is fragile and small changes in the LTD/LTP ratio or pre-synaptic input rates can lead to high firing rates. We propose an adaptive scheme to dynamically control the LTP/LTD ratio. The biophysics of synapses lead us to suggest a control mechanism using action potential-induced calcium influx, a known mediator of synaptic plasticity. This adaptive STDP rule is shown to stabilize the post-synaptic firing rates under a variety of perturbations. © 2002 Published by Elsevier Science B.V.

Keywords: Learning; Homeostatic mechanism; Hebbian; Spike correlations

1. Introduction

Spike-timing-dependent plasticity (STDP) has been proposed to solve two fundamental issues of learning in neural networks [9]. First, how synaptic competition is achieved and second, how neuronal firing rates are stabilized in the presence of a large number of excitatory recurrent collaterals. Song and Abbott have shown the conditions under which SDTP leads to synaptic competition [9]. Gerstner et al. argued that STDP will normalize firing rates [4]. Recent work by van Rossum et al. [13] has demonstrated that an STDP type of rule does not necessarily guarantee competition between synaptic afferents. It has been clear, however, that the competition and normalization properties

* Corresponding author. Stockholm Bioinformatic Center, SCFAB, S-106 91 Stockholm, Sweden.
E-mail address: jespert@nada.kth.se (J. Tegnér).

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are strongly dependent on the ratio between LTD and LTP in the STDP learning rule [9]. Here, we further examine the sensitivity of the normalization property on the learning ratio. We propose an adaptive rule which dynamically regulates the learning ratio.

2. Methods

We use a model of STDP developed by Song et al. [9]. The learning kernel $L(\tau) = A_{\mathrm{LTP}} \exp(\tau/\tau_{\mathrm{LTP}})$ if $\tau < 0$ and $-A_{\mathrm{LTD}} \exp(-\tau/\tau_{\mathrm{LTD}})$ if $\tau > 0$ (Fig. 1A). Following [9] and use $\tau_{\mathrm{LTP}} = \tau_{\mathrm{LTD}} = 20$ ms for the time window of learning. The learning ratio is $\alpha = A_{\mathrm{LTD}}/A_{\mathrm{LTP}} = 1.05$, which reflects the net dominance of depression during learning. Synaptic weights changes according to

$$\frac{\mathrm{d}w_i}{\mathrm{d}t} = \int L(\tau) s_{\mathrm{pre}}(t+\tau) s_{\mathrm{post}}(\tau)\,\mathrm{d}\tau, \quad w_i \varepsilon [0, w_{\max}], \tag{1}$$

where $s(t)$ denotes a delta function representing a spike at time t. Correlations between input rates were generated as in [9] by adding a common bias rate in a graded manner across synapses. If Fig. 2, an adaptive scheme is used to change α dynamically, according to the equations discussed in the text.

3. Results

3.1. The learning ratio controls neuronal gain

We use computer simulations to examine the normalization property of the STDP rule (Fig. 1A). As shown in Fig. 1B, the input–output relationship of our simulated neuron is approximately normalized after learning. We refer to the slope of the resulting input–output function as the effective neuronal gain. In the presence of input correlations neuronal gain is not normalized (Fig. 1C). Increasing the strength of input correlations increases the slope of the input–output curve leading to larger effective neuronal gain. Next, we examined how neuronal gain depends on the depression/potentiation ratio in the STDP rule. We define $\alpha = \mathrm{LTD}/\mathrm{LTP}$. In Fig. 1B and C we used $\alpha = 1.05$ [9]. By changing α, the neuronal gain call be controlled. Fig. 1D shows that the high-gain- and high-rate mode induced by strong input correlations is reduced to a lower-gain and lower-rate mode by increasing the α ratio.

3.2. Biophysical basis for an adaptive STDP learning rule

These observations suggest that if synaptic input is correlated it would be useful to dynamically regulate the depression/potentiation ratio. Guided by the known biophysics of synaptic plasticity we propose that Ca^{2+} dynamics in post-synaptic spines can control the neuronal plasticity. When a neuron fires, high-voltage activated calcium channels

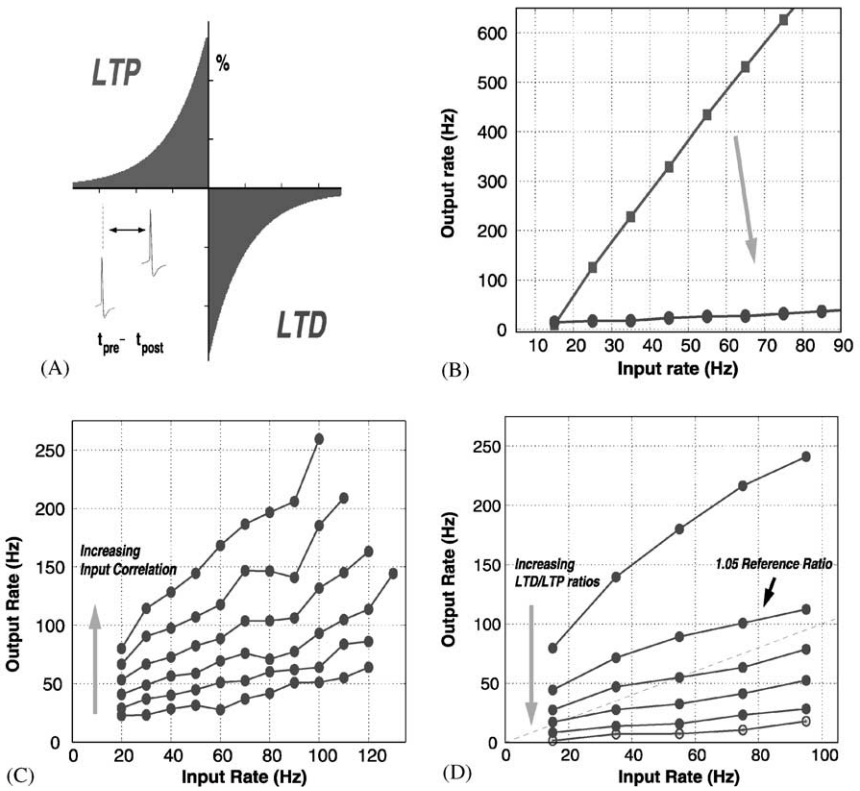

Fig. 1. (A) The STDP learning kernel. (B) STDP controls neuronal gain. The slope of the dependence of the post-synaptic output rate on the pre-synaptic input rate is referred to as effective neuronal gain. The initial firing rate is shown by the top curve while the lower line displays the final post-synaptic firing rate. The gain is reduced provided that the depression/potentiation ratio (1.05 here) is large enough. Uncorrelated input. (C) Increasing input correlations increases neuronal gain. When the synaptic input is strongly correlated the post-synaptic neuron operates in a high-gain mode characterized by a larger slope and larger baseline rate. Input correlations were uniformly distributed between 0 and a maximal value. The maximal correlation in the direction of the arrow: 0.0; 0.2; 0.3; 0.4; 0.5; 0.6; 0.7. α ratio is 1.05. Note that for further increases in the pre-synaptic rates, post-synaptic firing can increase above 1000 Hz. Data not shown here as it probably represents a non-physiological regime. (D) The depression/potentiation ratio sets the neuronal gain. The α ratios are in the direction of arrow: 1.024; 1.05; 1.076; 1.1025; 1.155; 1.2075. Maximal input correlation is 0.5.

open. This process allows calcium levels to track changes in the firing rate on a rapid time scale. When the resting Ca^{2+} is elevated in a spine, several changes occur. First, the calcium level required for synaptic depression is easier to reach. Second, increased resting calcium levels inhibit NMDA channels and thus calcium influx due to synaptic input. Both of these effects in turn increase the probability of LTD induction. Because of buffering, changes in the resting calcium levels are transient, thus, the time-scale of changes in our adaptive rule is very fast. This chain of events can be captured by

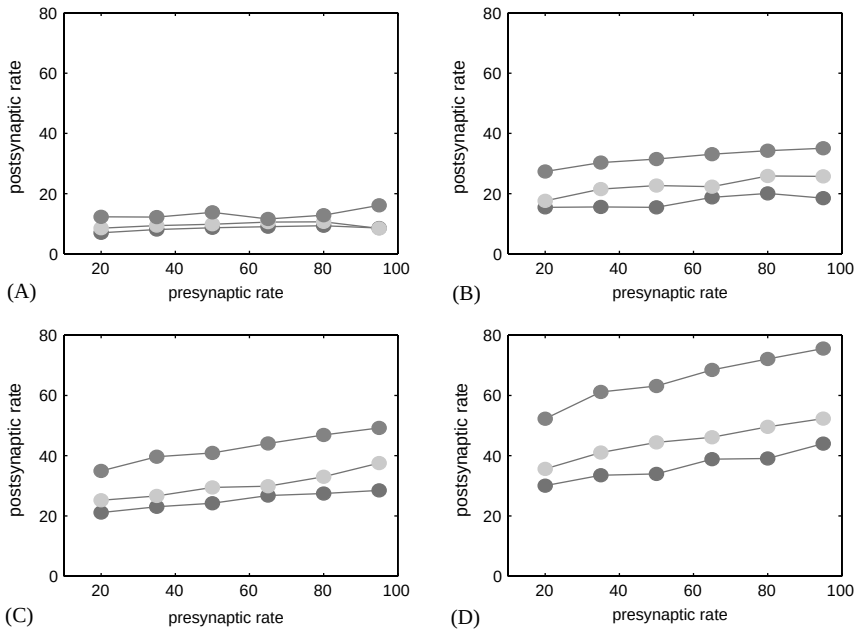

Fig. 2. Correlation and rate sensitive regimes with an adaptive STDP learning rule. (A) The post-synaptic rate (y-axis) is essentially unchanged when the pre-synaptic rate is increased. Increased synaptic input correlations (dots) have a small effect on the post-synaptic firing rate ($\gamma = 1.25$). (B) Reducing the parameter ($\gamma = 0.75$) controlling the strength of the calcium on the post-synaptic firing rate slightly increases the dependence of the post-synaptic firing rate on the amount of synaptic input correlations. (C) and (D) show how the neuronal gain (the slope) increases further when γ is reduced (0.5 and 0.25, respectively), $p = q = 1, \tau_{Ca} = 50$ ms, $\tau_{Ca} = 500$ ms.

the following first-order kinetic system:

$$\frac{\mathrm{d}[\mathrm{Ca}]}{\mathrm{d}t} = -[\mathrm{Ca}]/\tau_{\mathrm{Ca}} + \gamma \left[\sum_i \delta(t - t_i)\right]^p, \tag{2}$$

$$\tau_\beta \frac{\mathrm{d}\beta}{\mathrm{d}t} = -\beta + [\mathrm{Ca}]^q. \tag{3}$$

The parameter p determines how the calcium concentration scales with the post-synaptic firing rate (delta spikes δ above) and q controls the learning sensitivity. γ controls the rise of steady-state calcium with increasing post-synaptic rates (r_{post}). The time constants τ_{Ca} and τ_β determine the calcium dynamics and the time course of the adaptive rule, respectively. Note that this formulation controls the asymmetry of the learning rule, $\beta = \alpha - 1$.

We coupled this adaptive scheme to the STDP rule used previously. Our simulations demonstrate that the adaptive STDP rule controls the effective neuronal gain. As shown in Fig. 2 the postsynaptic firing rate after learning depends on both the rate as well as the correlation of the input. Fig. 2A shows perfect normalization and thus the

post-synaptic response does not depend on either the pre-synaptic rate or the strength of input correlations. This happens when γ is high, which could be interpreted as a large calcium channel conductance. Decreasing the conductance of the calcium channel, γ, results in less than perfect rate normalization. Fig. 2B shows an intermediate regime, where post-synaptic response is only weakly dependent on input rates. Increasing input correlations, however, does increase response.

4. Discussion

We show that the normalization property of the STDP rule is highly sensitive to the LTD/LTP ratio. Changing either the neuronal dynamics (not shown) or the input correlations (Fig. 1) can drive the neuron out of the balanced firing regime [2]. We propose that an adaptive control of the LTD/LTP ratio can stabilize the STDP rule. Interestingly, the known biochemical and biophysical processes in synaptic spines provide the physiological basis for the adaptive control scheme. Calcium levels can track the firing rate [5] and calcium is a crucial mediator of synaptic plasticity [7]. Recently, experimental observations show that NMDA-receptors are inhibited by calcium [8]. Taken together, these findings lead us to propose a scheme where calcium levels track post-synaptic firing, and in turn inhibit the NMDA conductance. Lowering calcium influx through NMDA channels shifts the balance between LTP and LTD. We found that this scheme could control the effective neuronal gain after learning and increase the robustness of the learning rule. Depending on the maximal calcium conductance, neurons can be differentially sensitive to input correlation and/or input rates (Fig. 2). Further mathematical analysis of the adaptive STDP rule is presented elsewhere [10]. The adaptive scheme introduces new parameters into the learning equations. These parameters determine the learning mode (correlation/rate sensitive) of the neuron and are robust against small mistuning. Therefore, meta-plasticity controlling these second order learning parameters could enable neurons to operate in differentially rate and correlation sensitive regimes.

Our adaptive scheme is akin to a number of known homeostatic mechanisms and models. It has been suggested that intrinsic conductances and synaptic strengths are under the control of homeostatic mechanisms [3,6,11,12]. Our work is a natural extension of learning algorithms by incorporating homeostatic principles [1]. While we also use calcium as the mediator of homeostatic control, the biophysics of spines lead us to use a very fast (millisecond) time-scale in contrast to the hours or days observed for intrinsic plasticity and synaptic scaling.

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