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Correlation based learning from spike timing dependent plasticity

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Abstract

We explore a synaptic plasticity model where potentiation and depression are induced by precisely timed pairs of synaptic events and postsynaptic spikes. We include the observation that strong synapses undergo relatively less potentiation than weak synapses, whereas depression is independent of synaptic strength. After random stimulation the synaptic weights reach a stable equilibrium distribution. Competition can be introduced separately by a mechanism that scales synaptic strengths as a function of postsynaptic activity. The plasticity rules select inputs which have a strong correlation with other inputs. © 2001 Published by Elsevier Science B.V.

Keywords: Hebbian plasticity; Synaptic competition, Activity dependent scaling, Temporal learning

Changes in the synaptic connections between neurons are widely believed to contribute to memory storage, and the activity-dependent development of neuronal networks. These changes are thought to occur through correlation-based, or Hebbian, plasticity. Recently, plasticity known as spike-timing dependent plasticity (STDP) has been observed [1,2,8]. STDP synapses are modified as follows: If a synaptic event precedes the postsynaptic spike, the synapse is potentiated. If it follows the postsynaptic spike, the synapse is depressed. STDP rules have been implemented in several modeling studies [3,6,7]. However, as commonly implemented, these learning rules are unstable and require hard bounds on the synaptic weights.

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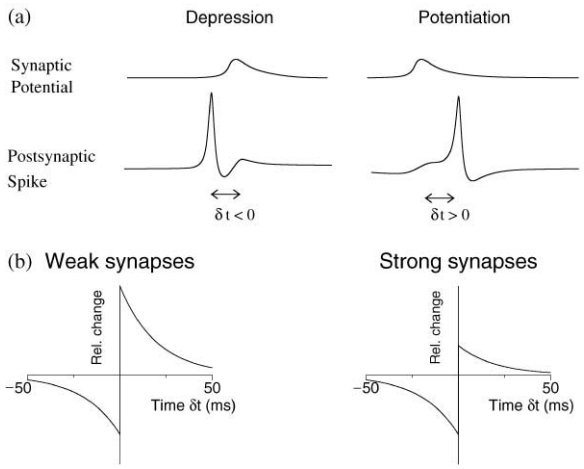

Fig. 1. Spike timing dependent plasticity. (a) Synapses are potentiated if the synaptic event precedes the postsynaptic spike. Synapses are depressed if the synaptic event follows the postsynaptic spike. (b) The amount of change falls off exponentially as the time difference between pre and postsynaptic spike increases ($\tau = 20$ ms). In addition, the amount of potentiation decreases for stronger synapses, whereas depression is independent of synaptic size.

Here we present an intrinsically stable STDP learning rule. It incorporates the experimental observation that potentiation is weaker for strong synapses [2,4,5]. This learning rule generates a stable, unimodal, positively-skewed distribution of synaptic weights that closely resembles the distribution of quantal amplitudes measured from central neurons [13]. The weights depend on their correlation with other inputs, so that learning occurs through cooperation between inputs. Finally, competition is almost absent, and can be introduced independently by implementing activity-dependent synaptic scaling [9,13]. This scaling does not change the shape or the stability of the weight distribution.

1. The plasticity rules

Synapses can be potentiated and depressed by pairing a synaptic event with a postsynaptic spike. If the synaptic event occurs before the postsynaptic spike, the synapse will be potentiated; if the postsynaptic spike precedes the synaptic event the synapse will be depressed [2,8,15]. The amount of conductance change decreases approximately exponentially with the time difference between the synaptic event and the postsynaptic spike, δt. Such a synaptic modification window is illustrated in Fig. 1. The amount of change also depends on the initial synaptic size [2,4,5]. The relative amount of depression is independent of synaptic weight, whereas the relative amount of potentiation decreases for stronger synapses, see Fig. 1b. We assume that the amount of potentiation is inversely proportional to the weight, see also [7].

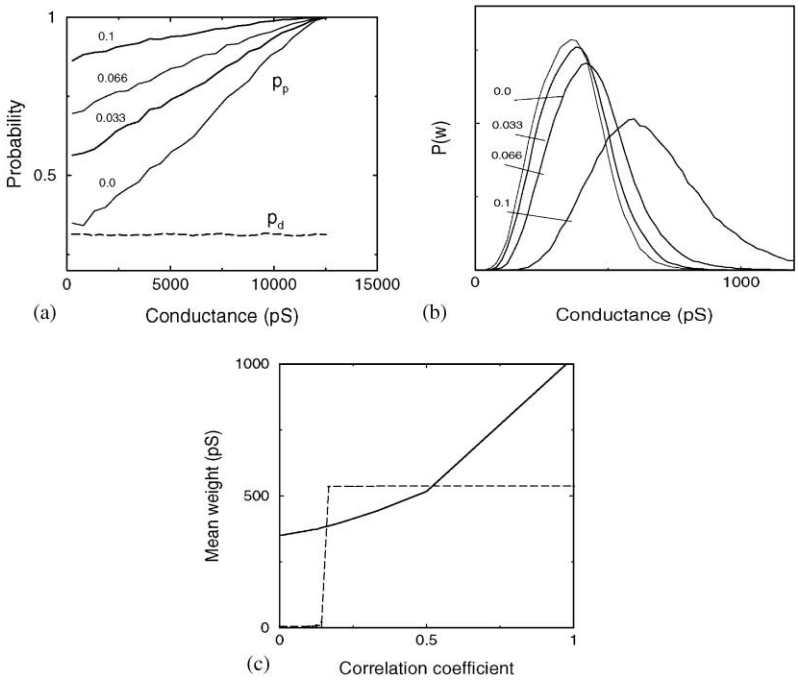

Fig. 2. Simulation of the effect of correlation in the inputs on the synaptic weights. The inputs consisted of four groups of 25 synapses having different amounts of correlation within the group (correlation coefficients: 0, 0.033, 0.066, 0.1). (a) The probability for inducing potentiation, p_p and depression p_d vs. the weight. The probability for inducing potentiation is increased when correlations between inputs are present, whereas the probability for inducing depression is unaltered. The labels indicate the correlation coefficient. (b) The weight distributions of the different groups. The different amounts of correlation lead to the coexistence of multiple weight distributions. The weights of the more strongly correlated groups are larger. (c) The mean conductance of the different groups as a function of the correlation. Also shown: model without weight-dependent LTP (dashed line).

Denoting potentiation as $w \to w + w_p$ and depression as $w \to w + w_d$, the plasticity rules are

$$w_p = c_p e^{-\delta t/\tau_{STDP}}, \quad w_d = -c_d w e^{\delta t/\tau_{STDP}}, \tag{1}$$

where c_d is the average amount of relative depression after one pairing, $c_d = 0.003$; and c_p is the average amount of potentiation after one pairing, $c_p = 1$ pS. Note the difference in weight dependence. This weight dependence has drastic consequences for the model. The rules yield a stable, unimodal weight distribution (for example see Fig. 2b). Competition between synapses means that the potentiation of one synapse, slightly depresses all others. This model shows little competition. In contrast, in models without weight dependence an artificial limit on the maximal weight has to be imposed and, interestingly, strong competition between the inputs occurs [12].

2. Activity-dependent scaling

Our model yields stable Hebbian learning without competition. Nevertheless, competition is useful for developmental processes such as ocular dominance column plasticity, and output rate normalization is useful when the input rate or the number of inputs undergoes large changes. Therefore we include activity-dependent scaling (ADS) of synaptic weights in the model. Activity-dependent scaling is a mechanism that adjusts the synaptic weights to regulate the postsynaptic activity. The postsynaptic activity is measured with a slow-varying sensor, $a(t)$. It increases with every postsynaptic spike, and decays exponentially between spikes

$$\tau\frac{\mathrm{d}a(t)}{\mathrm{d}t} = -a(t) + \sum_i \delta(t - t_i),$$

where t_i is the spike time. Activity-dependent scaling scales the weights to prevent too low or too high activity levels. The scaling is thought to be multiplicative and independent of presynaptic activity [13]. We update the weights every time-step according to

$$\frac{\mathrm{d}w(t)}{\mathrm{d}t} = \beta w(t)[a_{\mathrm{goal}} - a(t)] + \gamma w(t)\int_0^t \mathrm{d}t'[a_{\mathrm{goal}} - a(t')]. \tag{2}$$

The second term allows for a slow yet strong scaling. As it accumulates the error, it dominates in the long run. As a result the steady state activity level will eventually become equal to its goal value. The scaling shifts the point where potentiation and depression are balanced, thus adjusting the mean weight while preserving the shape of the weight distribution, consistent with experimental observations [13].

3. The weight distribution

Given the plasticity rules and the ADS we can calculated how the weight distribution evolves under random stimulation (Poisson inputs) using a Fokker–Planck approach [11,14]. The weights are equilibrated when potentiation, depression and activity dependent scaling are equally strong. We concentrate on the average equilibrium weight. There are two effects: First, *how much* does the synapse change with potentiation/depression? This is given by Eq. (1). Secondly, *how often* is the synapse potentiated/depressed? The probability that a presynaptic spike leads to depression is simply a constant p_{d}, proportional to the postsynaptic firing rate ρ_{out}: $p_{\mathrm{d}} \propto \rho_{\mathrm{out}}$. Potentiation requires a presynaptic event before the postsynaptic spike. This is more complicated, as the presynaptic event can help to cause a postsynaptic spike. One finds approximately [14]

$$p_{\mathrm{p}}(w) = p_{\mathrm{d}}(1 + w/W_{\mathrm{tot}}) \quad (\text{but } \leqslant 1).$$

Thus, the probability for inducing a spike increases with the weight of the synapse w, and decreases in the presence of other synaptic inputs, W_{tot}. Finally, activity

dependent scaling changes the weight. We assume that an equilibrium state has been reached such that the activity dependent scaling is a constant. We consider the case that all synapses are identical and have the same synaptic rate, ρ_{in}.

The average weight is the weight which experiences no net strengthening or weakening, it obeys

$$0 = \text{LTD} + \text{LTP} + \text{ADS} \tag{3}$$

$$0 = -\rho_{\text{in}} p_{\text{d}} c_{\text{d}} w + \rho_{\text{in}} p_{\text{p}} c_{\text{p}} + \gamma w \int_0^t \mathrm{d}t' [a_{\text{goal}} - a(t')], \tag{4}$$

$$w = w_0 \frac{1}{1 - \gamma' - w_0 / W_{\text{tot}}}, \tag{5}$$

where $w_0 = c_{\text{p}}/c_{\text{d}}$ and $\gamma' = \gamma \int_0^t \mathrm{d}t' [a_{\text{goal}} - a(t')]/(\rho_{\text{in}} p_{\text{d}} c_{\text{d}})$.

4. Correlated input potentiates synapses

Next, we analyze the effect of correlations in the inputs. Correlation is implemented by randomly distributing N Poisson trains among the inputs. Every time-step the Poisson trains are redistributed. For every synaptic event there is a chance $1/N$, that it is shared by another synapse. This yields a cross-correlation coefficient between trains $C(\Delta t) = 1/N\delta(\Delta t)$. The cooperation between inputs will increase the probability that a synaptic event causes a spike. The presence of the correlations increases the probability for potentiation to $p_{\text{p}}(\boldsymbol{w}) = p_{\text{d}}(1 + C \cdot \boldsymbol{w}/W_{\text{tot}})$, where C is the matrix of correlation coefficients.

We diagonalize the correlation matrix. We denote its eigenvalues as λ_i and it's eigenvectors as $\boldsymbol{e}_i$, with components e_i^k. The weight vector can be written as a sum of the eigenvectors

$$\boldsymbol{w} = w_0 \sum_i \frac{\sum_k e_i^k}{1 - \gamma' - \frac{w_0}{W_{\text{tot}}} \lambda_i} \boldsymbol{e}_i.$$

This equation predicts the effect of the correlations on the weights as seen in simulations, Fig. 2c. The learning reminds us of Oja's model [10]. In that model the weight vector becomes equal to the eigenvector with the largest eigenvalue, the principal eigenvector, $\boldsymbol{e}_1$. Thus Oja's model acts as a principal component analyzer. Also here, the principal eigenvector will be enhanced. However, let us point out the differences: (1) The structure of the correlations is different: here the correlations are coincident synaptic inputs, instead of correlation in the synaptic rates. (2) Apart from the eigenvalue, also the sum of the components of the eigenvector enters in the equation. If this sum is zero, the eigenvector does not contribute. (3) In Oja's model both $\boldsymbol{e}_1$ and $-\boldsymbol{e}_1$ are valid solutions; here the sum in the numerator breaks such symmetries.

In contrast, in models without weight dependent potentiation the synapses have essentially zero or maximal weight. When correlations in the input are introduced,

inputs with correlation above a certain threshold obtain maximal weight, below that the weights are zero, see Fig. 2c.

5. Discussion

We have introduced a plasticity model of spike timing dependent plasticity. In contrast to most other models it includes the observation that strong synapses undergo relatively less potentiation than small ones, whereas depression does not depend on synaptic strength. This yields a stable, realistic distribution of synaptic weights. The competition is the result of activity dependent scaling. The competition and learning therefore can occur on different time-scales, and with different strengths. This contrasts with other models of learning, where competition is directly tied to the plasticity to normalize the learning.

Our model selects inputs which have strong correlations. The learning is similar, but not identical to Oja's model, as in general it selects inputs which have strong correlations. The precise consequences of the learning rules in a network setting are being investigated.

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