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A variety of competitive properties arising from STDP incorporating metaplastic regulation

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Abstract Spike-timing-dependent plasticity (STDP) induces competition among inputs, which is required for the construction of functional neuronal circuits, while maintaining the basic features of Hebbian plasticity. Here, we theoretically examine the competitive function of STDP incorporating a metaplastic activity-dependent feedback (ADFB) mechanism, wherein higher postsynaptic activity suppresses LTP, in cases where a neuron receives two groups of correlated inputs. We demonstrate that there are four distinct types of competitive properties depending on the relative input frequency between the different groups and the correlation time among the inputs within the same group. (1) Competition with a bi-stable synaptic weight distribution (for identical frequencies and brief correlation). (2) No competition (for identical frequencies and prolonged correlation). (3) Competition preferring strong input activity (for different frequencies and brief correlation).(4) Competition preferring weak input activity (for different frequencies and prolonged correlation). This may suggest that ADFB regulation can modulate the Hebbian competition properties associated with STDP to increase its ability to reflect input firing properties.

Key words Plasticity · STDP · Synaptic competition · Metaplasticity · Neocortex

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1 Introduction

The development of functional neuronal connections depends on the competitive interaction between inputs.[1] In the presence of competition, the strengthening of some inputs causes the weakening of the others, thereby producing the input selectivity of neurons while maintaining the level of postsynaptic activity. A conventional view is that the consequence of competition is determined by the relative strength of input activities, such that the synapses that are frequently activated are strengthened, while those that are less frequently activated are suppressed. This type of activity-dependent competition is consistent with the Hebbian rule of plasticity, because the frequently activated inputs will tend to produce strong postsynaptic discharge and therefore can be potentiated. However, recent observations suggest that some forms of neocortical plasticity cannot be explained by Hebbian competitive mechanisms; for example, in the adult rat, the active use of single whiskers for exploring a new environment produces a contraction of the cortical representation of the inputs from the frequently used whiskers.[2] The plasticity mechanism governing activity-dependent competition may be altered depending on the characteristics of inputs arising from sensory stimuli.[3]

Hebbian-based competition has been suggested to emerge automatically through spike-timing-dependent plasticity (STDP).[4] In STDP, the synapses that are activated slightly before and after the postsynaptic event are potentiated and depressed, respectively. Therefore, a group of temporally correlated inputs, which tends to arrive just before postsynaptic spikes and can frequently contribute to evoking them, are selectively potentiated. However, our recent study[5] has revealed that when STDP is accompanied by metaplastic activity-dependent feedback (ADFB) modulation, wherein LTP is suppressed by the feedback of postsynaptic activity, the correlated inputs can be either potentiated or depressed depending on whether the correlation time is shorter or longer than a threshold, respectively. This finding suggests that the ADFB mechanism may serve to increase

the ability of STDP to encode the firing statistics of inputs, so that the resulting synaptic behavior can exhibit either Hebbian or anti-Hebbian properties according to the correlation structure of the input spikes.[5]

In this study, in order to further examine the impact of such switching in the plasticity mechanism on activity-dependent competition, we examined the dynamics of a synaptic population emerging through STDP incorporated with the ADFB mechanism. We constructed a conductance-based pyramidal neuron model that receives inputs from two groups of plastic synapses, which are correlated within each group, and investigated the distribution of synaptic efficacies obtained by STDP. The results show that, depending on both the correlation time for the inputs within the same group and the relative activation frequency between the inputs of different groups, STDP can exhibit four types of competitive dynamics under ADFB modulation.

2 Methods

2.1 Neuron model

We used a two-compartment neuron model consisting of a soma and a dendrite.[5] Both of the compartments contained voltage-dependent sodium and potassium currents. A voltage-gated Ca^{2+} current and a Ca^{2+}-dependent potassium current were included in the dendritic compartment to reproduce the firing rate adaptation exhibited in pyramidal neurons.

The neuron receives random inputs, generated by Poisson processes, from 4000 excitatory and 800 inhibitory synapses.[5] The excitatory inputs consist of AMPA- and NMDA-mediated currents, while the inhibitory inputs are mediated by GABA. To examine correlation-based competition, we divided the excitatory synapses into two equally sized groups (2000 synapses each). We introduced independent correlations of equal magnitude to both of them by the method given by Song and Abbott.[6] The firing rate of the inputs within the same group has a correlation function that decays exponentially with a time constant τ_c (i.e., correlation time). The inhibitory synapses are activated by uncorrelated homogeneous Poisson processes. The mean firing rates for both the excitatory and inhibitory inputs are set to 3 Hz, unless otherwise stated. Considering a very low success rate (around 10%) of synaptic transmission in central synapses,[7] this input rate may approximately correspond to a 30-Hz firing frequency, which is within the physiological range of the sensory-evoked response of cortical neurons.

2.2 Synaptic weight modification

The synaptic modification by STDP acts on all the excitatory (AMPA) synapses in the model. We define $\Delta t = t_{post} - t_{pre}$ to be the time lag between the pre- and postsynaptic action potentials. The weight change Δw induced by STDP is described as follows:

$$\Delta w = \begin{cases} A_+ \exp(-\Delta t/\tau_+), & \text{for } \Delta t > 0, \\ -A_- \exp(\Delta t/\tau_-), & \text{for } \Delta t \le 0. \end{cases} \quad (1)$$

Here, A_+ (see below) and A_- (= 0.004) represent the magnitude of LTP and LTD, respectively.[5] $\tau_+ = \tau_- = 20$ ms are the parameters that set the length of the temporal window of STDP. When a pre- or postsynaptic event occurs, the synaptic weights w are modified stepwise according to the additive rule of STDP. The weight changes caused by all the spike pairs are summed linearly. The upper bound of synaptic weights (w_{max}) is imposed to stabilize the learning dynamics.

Experimental findings suggest that LTP and LTD in STDP may depend on different signaling pathways: the activation of NMDA receptors (NMDARs) for LTP and that of other signaling receptors (e.g., metabotropic glutamate receptors (mGluRs)) for LTD.[8] This may suggest that when higher postsynaptic activity facilitates Ca^{2+} entry through the voltage-gated Ca^{2+} channels, the Ca^{2+}-dependent desensitization of NMDARs will suppress LTP without affecting LTD. Additionally, functional NMDARs consist of obligatory NR1 subunits and modulatory NR2 subunits. The fact that the Ca^{2+}-dependent desensitization occurs in NR2A- but not NR2B-containing NMDARs[9] implies that the expression pattern of NR2 subunits may regulate the level of Ca^{2+}-dependent desensitization. Therefore, to examine the effects of the subunit- and activity-dependent desensitization of NMDARs, we introduced the ADFB modulation of the magnitude of LTP proposed by our previous studies.[5,10]

$$A_+(t) = A_+^0 - k_{max}\rho f_{post}(t) \quad (2)$$

Here, $A_+^0 = 0.008$ and $k_{max} = 0.068$ ms[5]. $f_{post}(t)$ denote the postsynaptic firing rate at time t. The parameter ρ is used to represent the expression pattern of distinct NMDAR subunits: $\rho = 0$ corresponds to the state where NMDARs comprise NR1 and NR2B subunits, as in the case of neonatal neurons, whereas $\rho = 1$ denotes the state where the NMDARs contain many NR2A subunits, as in the case of mature neurons.[10] We set the value of ρ to 1 throughout this study. The postsynaptic frequency was estimated by the equation $f_{post}(t) = \int_0^\infty \lambda \exp(-\lambda\tau) S_{post}(t - \tau) d\tau$ by using the postsynaptic spike train $S_{post}(t) = \Sigma_{t_{post}} \delta(t - t_{post})$.

3 Results

In order to examine how the competition induced by STDP depends on input firing properties, we examined the synaptic distribution in the equilibrium state by changing the correlation time τ_c, when the neuron receives two groups of correlated inputs (Fig. 1A and D). The figures show that when the correlation time is sufficiently short, the synaptic weights segregate into the two input groups, with the one winning the competition suppressing the other. However, when the correlation time becomes prolonged ($\tau_c > 20$ ms), the synaptic weights of both groups converge to the same average strength, implying the absence of competitive interaction between the input groups. This result is clarified by

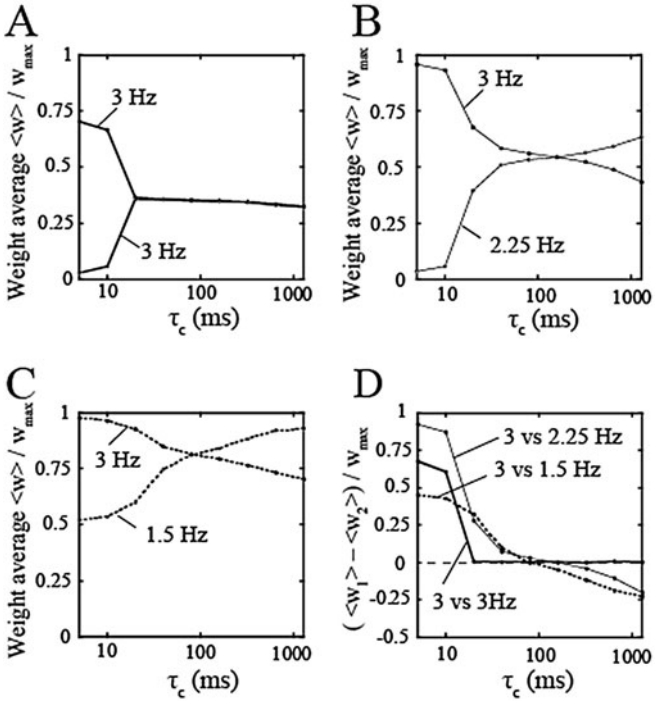

Fig. 1. **A–C** The weight averages of the two input groups as a function of τ_c in the equilibrium state. The mean input frequencies for the two groups are the same (3 Hz) in **A**, whereas that of one group is decreased by either 25% in **B** or by 50% in **C**. The input frequency for each group of synapses is shown in the labels. **D** The difference in average weight between the two groups in the equilibrium state is plotted as function of τ_c for the three input cases corresponding to **A–C** by using the same line style

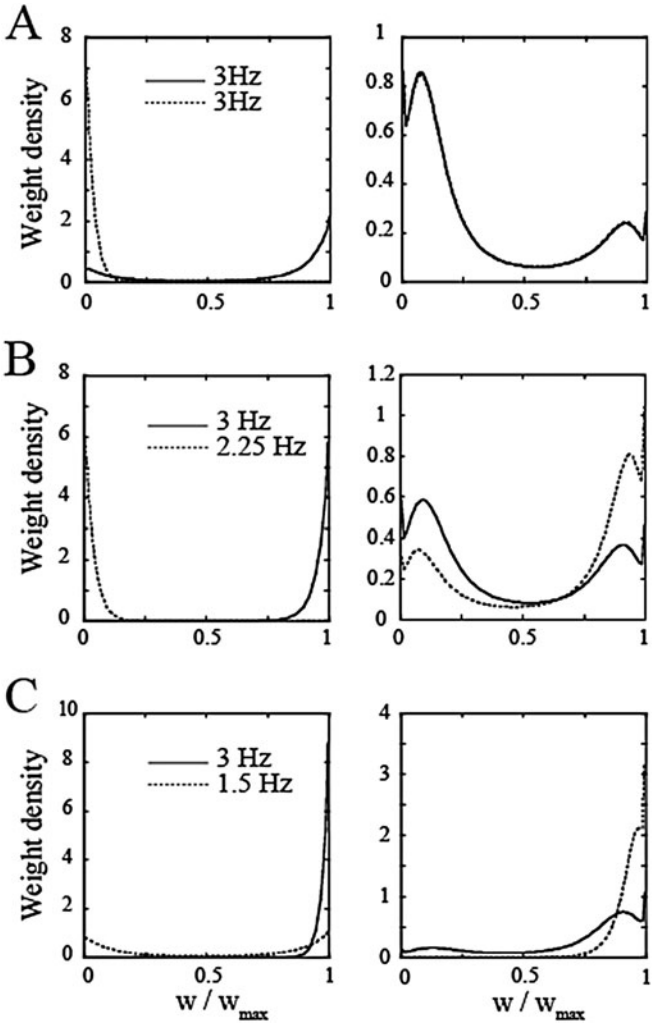

Fig. 2. The steady-state weight distributions of the two synaptic groups are shown by the solid and dashed lines. *Left* and *right* columns show the cases for τ_c = 5 ms and τ_c = 1280 ms, respectively. The mean input frequencies for the two groups are the same in **A**, whereas they are different in **B** and **C**. The input rate for each group is shown in the legends. Since the weight distribution fluctuates even at equilibrium, we have taken the temporal average of the weights over a sufficiently long period

examining the steady-state weight distributions (Fig. 2A). The figure shows that for smaller τ_c, the final weight distribution is bimodal and the two groups converge to distinct distributions[6] (Fig. 2A, left). In contrast, for larger τ_c, the weight distributions of the two groups have an identical characteristic form that contains local peaks at positions slightly apart from the boundaries (Fig. 2A, right). The competitive state obtained for smaller τ_c is bi-stable because the inputs are symmetric under exchange of the two groups, and therefore, which group becomes dominant at a certain time is determined by earlier input activities.[10] The results here appear to be consistent with previous results[5] showing that STDP exhibits Hebbian and anti-Hebbian properties for smaller and larger τ_c values, respectively. For smaller τ_c, the Hebbian property of STDP can drive input correlation-based competition.[6] In the presence of prolonged input correlation, if either group of synapses is potentiated more than the other group, then the activity of the potentiated input group will tend to control the timing of postsynaptic spiking. However, it would be difficult for the input group to be continuously potentiated due to the anti-Hebbian mechanism, where a group of inputs having prolonged correlation tends to be strongly weakened by the occurrence of many post-pre-timing spike pairs.[4,5]

Furthermore, when the mean input frequency for one group was decreased, the dominant group in the equilibrium state was reversed with changes in the correlation time (Fig. 1B–D): the group activated by higher frequency inputs suppresses the other group at smaller τ_c, whereas the group activated by lower frequency inputs becomes dominant at larger τ_c. These changes are accompanied by a significant modification in the proportion of synapses within each group that accumulates near either the upper or lower boundary (Fig. 2B and C). These results imply that STDP functions to strengthen frequently and less-frequently activated inputs in the case of brief and prolonged correlation times, respectively, which may also be consistent with the finding of a switch from a Hebbian to an anti-Hebbian plasticity regime through an increased correlation time.[5]

4 Conclusion

In this study, we have examined how an STDP model incorporating the ADFB mechanism regulates competition between two groups of correlated inputs. The results have demonstrated that four distinct types of competitive properties emerge from STDP, depending on the relative difference in the frequencies with which the two groups generate inputs, and the correlation time of the input activity within each group. (1) Competition producing a bi-stable synaptic pattern (for the same input frequencies and small correlation time). (2) No competition (for the same input frequencies and large correlation time). (3) Competition with a bias toward stronger input activity (for different input frequencies and small correlation time). (4) Competition with a bias toward weaker input activity (for different input frequencies and large correlation time). Cases (1) and (3) can be expected from Hebbian plasticity, whereas (2) and (4) may result from anti-Hebbian plasticity, as mentioned above. Strong Hebbian learning has a nondemocratic aspect, since it will permit only a small number of frequently activated inputs to acquire control of many postsynaptic neurons. Therefore, in biological systems, it would be useful to regulate the strength of Hebbian effects. The present findings suggest that ADFB regulation enables the modification of the Hebbian properties associated with STDP, which may be useful for efficient central processing.

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