The Fifteenth International Symposium on Artificial Life and Robotics 2010 (AROB 15th '10),
B-Con Plaza, Beppu,Oita, Japan, February 4-6, 2010

A variety of competitive properties arises from STDP incorporating metaplastic regulation

S. Kubota[1], J. Rubin[2], T. Kitajima[3], and T. Nakamura[1]

[1]*Department of Biomedical Information Engineering, Yamagata University*
4-3-16 Jonan, Yonezawa, Yamagata, 992-8510, Japan
[2]*Department of Mathematics, University of Pittsburgh*
301 Thackeray Hall, Pittsburgh, PA 15260, USA
[3]*Department of Bio-System Engineering, Yamagata University*
4-3-16 Jonan, Yonezawa, Yamagata, 992-8510, Japan
(Tel : 81-238-26-3356; Fax : 81-238-26-3299)
(kubota@yz.yamagata-u.ac.jp)

Abstract: Spike-timing-dependent plasticity (STDP) induces competition among inputs, required for the construction of functional circuits, while maintaining the basic features of Hebbian plasticity. Here, we examine the competitive functi ons of STDP incorporating a metaplastic activity-dependent feedback (ADFB) mechanism, wherein higher postsynaptic activity suppresses LTP, in cases where a model 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 bistable synaptic weight distribution (for identical frequency and brief correlation), (2) no competition (for identical frequency and prolonged correlation), (3) competition preferring strong input activity (for different frequencies and brief correlation), and (4) competition preferring weak input activity (for different frequencies and prolonged correlation). Our results suggest that ADFB regulation enables the modification of the Hebbian competition properties associated with STDP to increase its abilities to reflect the firing properties of input neurons.

Keywords**:** Plasticity, STDP, Synaptic competition, Neocortex.

I. 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. Such activity-dependent competition is consistent with 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 new environment produces the 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 the sensory stimuli [3].

The 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 is potentiated and depressed, respectively. Therefore, a group of temporally correlated inputs, which tends to arrive just before the 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 such that the resulting synaptic behavior can exhibit either Hebbian or anti-Hebbian property according to the correlation structure of input spikes [5].

In the present study, to further examine the impact of such switching in the plasticity mechanism on the activity-dependent competition, we examine the dynamics of synaptic population emerging through STDP incorpo-

rated with the ADFB mechanism. We construct a conductance-based pyramidal neuron model that receives inputs from two groups of plastic synapses, which are correlated within each group, and investigate 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 different groups, STDP can exhibit four types of competitive dynamics under ADFB modulation.

II. METHODS

1. Neuron model

We used a two-compartment neuron model consisting of a soma and a dendrite [5]. Both the compartments contain voltage-dependent sodium and potassium currents. A voltage-gated Ca^{2+} current and a Ca^{2+}-dependent potassium current are included in the dendritic compartment to reproduce 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 are comprised of AMPA- and NMDA-mediated currents, while the inhibitory inputs are mediated by GABA. To examine the 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 (correlation time). The inhibitory synapses are activated by uncorrelated homogeneous Poisson processes. The mean firing rate for both the excitatory and inhibitory inputs are set to 3 Hz, unless otherwise stated. Considering a very low success rate (around 10%) of the synaptic transmission in central synapses [7], this input rate may approximately correspond to 30 Hz of firing frequency, which is within the physiological range of the sensory-evoked response of cortical neurons.

2. Synaptic weight modification

The synaptic weight modification by STDP acts on all the excitatory (AMPA) synapses. 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:

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

Here, A_+ (see below) and A_- (>0) represent the magnitude of LTP and LTD, respectively [5]. $\tau_+ = \tau_- = 20$ ms are the parameters to decide 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 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 Ca^{2+}-dependent desensitization occurs in NR2A- but not NR2B-containing NMDARs [9] implies that the expression pattern of NR2 subunits may regulate Ca^{2+}-dependent desensitization. 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 ref. [5]:

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

Here, A_+^0 and $k_{\max}$ are positive parameters. $f_{post}(t)$ denotes the postsynaptic firing rate at time *t*. The parameter ρ is used to represent the expression pattern of distinct NMDAR subunits: ρ = 0 corresponds to the state where NMDARs are comprised of NR1 and NR2B subunits, whereas ρ = 1 denotes the state where the NMDARs contain many NR2A subunits. Therefore, in Eq. 2, the feedback effect is strengthened by the increased value of ρ, which corresponds to the enhanced expression of NR1/NR2A NMDARs exhibiting activity-dependent desensitization. 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 $S_{post}(t) = \sum_{t_{post}} \delta(t - t_{post})$.

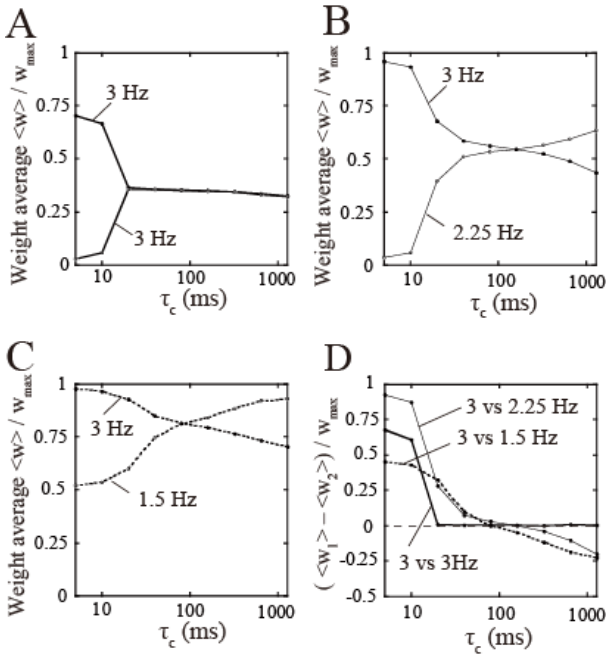

Fig.1. (A–C) The weight averages of the two input groups as function of τ_c at the equilibrium state. The mean input frequency of the two groups is the same (3Hz) in (A), whereas that of either one group is decreased by 25% in (B) or by 50% in (C). The input frequency for each group of synapses is shown in the labels. (D) The difference in the weight averages of two groups at 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.

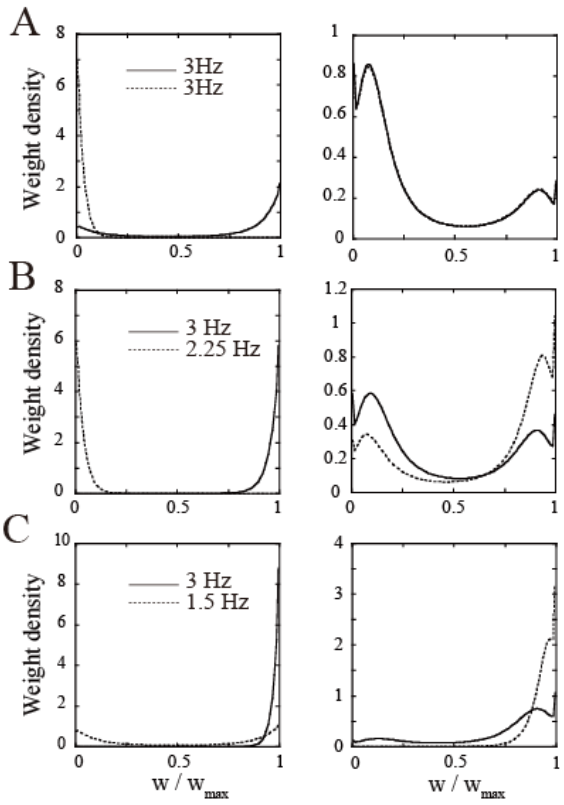

Fig.2. The steady-state weight distributions of the two synaptic groups are shown by the solid and dashed lines. Left and right columns show the cases for τ_c = 5 ms and τ_c = 1280 ms, respectively. The mean input frequencies for the two groups are the same in (A), whereas they are different in (B) and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

III. RESULTS

To examine how the competition by STDP depends on the input correlation properties, we examined the synaptic distribution at the equilibrium state by changing the correlation time τ_c, when the neuron receives two groups of correlated inputs (Figs. 1A and 1D). 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 (τ_c > 20 ms), the synaptic weights of both the groups converge to the same average strength, implying the absence of competitive interaction. This result is clarified by the steady-state weight distribution (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 the identical characteristic form that contains local peaks at the positions slightly apart from the boundaries (Fig. 2A, right). The competitive state obtained for smaller τ_c is bistable because the inputs are symmetric under exchange of the two groups, and therefore, which group becomes dominant at the present time can be determined by the past input activities [10]. The results here appear to be consistent with the previous result [5] that STDP exhibits Hebbian or anti-Hebbian property for smaller or larger τ_c values, respectively. For smaller τ_c, the Hebbian property of STDP can drive correlation-based competition as in the previous study [6]. In the presence of prolonged correlation, if either one group is potentiated more than the

other, the potentiated group will tend to control the timing of the postsynaptic spiking. However, the activation of inputs with longer correlation time produces many post-pre timing spike pairs that cause LTD [4]. Thus, it would be difficult for the correlated group to be continuously potentiated in the presence of anti-Hebbian mechanism, in which a group of inputs having prolonged correlation tends to be strongly weakened [5].

Furthermore, when the mean input frequency for one group was decreased, the dominant group at the equilibrium state was reversed with changes in the correlation time (Figs. 1B–1D): 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 the significant modification in the proportion of synapses within each group that accumulates near either the upper or lower boundary (Figs. 2B and 2C). These results imply that STDP functions to strengthen frequently and less-frequently activated inputs in the case of brief and prolonged correlation time, respectively, which may also be in line with the prediction by the switching from the Hebbian to anti-Hebbian plasticity through the increased correlation time [5].

IV. CONCLUSION

In this study, we have examined how an STDP model incorporated with 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 bistable synaptic pattern (for the same input frequencies and small correlation time); (2) no competition (for the same input frequencies and large correlation time); (3) the competition with a bias towards stronger input activity (for different input frequencies and small correlation time); and (4) the competition with a bias towards 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. A strong Hebbian learning has a non-democratic 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 required for efficient central processing.

ACKNOWLEDGMENTS

This study is partially supported by Grant-in-Aid for Scientific Research (KAKENHI(19700281), Young Scientists (B)) from the Japanese government. S.K. is partially supported by the Program to Accelerate the Internationalization of University Education from the Japanese government and the International Research Training Program from Yamagata University.

REFERENCES

[1] Miller KD (1996), Synaptic economics: competition and cooperation in synaptic plasticity. Neuron 17: 371-374
[2] Polley DB, Chen-Bee CH, Frostig RD (1999), Two directions of plasticity in the sensory-deprived adult cortex. Neuron 24:623-627
[3] Feldman DE, Brecht M (2005), Map plasticity in somatosensory cortex. Science 310: 810-815
[4] Song S, Miller KD, Abbott LF (2000), Competitive Hebbian learning through spike-timing-dependent synaptic plasticity. Nature Neuroscience 3:919-926
[5] Kubota S, Rubin, J, Kitajima T (2009), Modulation of LTP/LTD balance in STDP by an activity-dependent feedback mechanism. Neural Networks 22:527-535
[6] Song S, Abbott LF (2001), Cortical development and remapping through spike timing-dependent plasticity. Neuron 32:339-350
[7] Hessler NA, Shrike AM, Mallnow R (1993), The probability of transmitter release at a mammalian central synapse. Nature 366:569-572
[8] Bender VA, Bender KJ, Brasier DJ et al. (2006), Two coincidence detectors for spike-timing-dependent plasticity in somatosensory cortex. Journal of Neuroscience 26:4166-4177
[9] Krupp JJ, Vissel B, Heinemann SF et al. (1996), Calcium-dependent inactivation of recombinant N-methyl-D-aspartate receptors is NR2 subunit specific. Molecular Pharmacology 50:1680-1688
[10] Kubota S, Kitajima T (submitted), Possible role of cooperative action of NMDA receptor and GABA function in developmental plasticity.