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PAPER

A Model for Ocular Dominance Plasticity Controlled by Feedforward and Feedback Inhibition

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SUMMARY The maturation of inhibitory transmission through γ-aminobutyric acid (GABA) is required to induce ocular dominance (OD) plasticity in the visual cortex. However, only circuits that are mediated by specific $GABA_A$ receptors can selectively elicit OD plasticity, implying a role of local circuits involved in GABA inhibition in this process. In this study, in order to theoretically examine the effects of such local pathways associated with cortical inhibition on the induction of OD plasticity, we compared synaptic modification dynamics regulated by feedforward inhibition and those regulated by feedback inhibition. Feedforward inhibition facilitated competitive interactions between different groups of inputs conveying correlated activities, which were required for the emergence of experience-dependent plasticity. Conversely, feedback inhibition suppressed competitive interactions and prevented synapses from reflecting past sensory experience. Our results suggest that the balance between feedforward and feedback inhibition regulates the timing and level of cortical plasticity by modulating competition among synapses. This result suggests an importance of activity-dependent competition in experience-dependent OD plasticity, which is in line with the results of previous experiments.

key words: *STDP, GABA, synaptic competition, visual cortex*

1. Introduction

The closure of one eye during a critical period in early life can shift the dominant neuronal response to visual stimuli towards the open eye [1]. Experimental evidence has suggested that ocular dominance (OD) plasticity may be triggered by the maturation of cortical inhibition, which is regulated by γ-aminobutyric acid (GABA) [2]–[8] ([7], [8] for review). When the development of GABA inhibition is suppressed due to the targeted deletion of an isoform of the GABA synthetic enzyme, glutamic acid decarboxylase (GAD65), the onset of OD plasticity is delayed until inhibition is pharmacologically restored [2]. Likewise, OD plasticity can be prematurely triggered by enhancing GABAergic function through the pharmacological treatment [3] or the overexpression of brain-derived neurotropic factor (BDNF), which regulates the maturation of inhibition [4], [5]. These observations strongly suggest the existence of a threshold inhibition level that is required for the induction of visual cortical plasticity.

However, not all GABA functions are involved in controlling OD plasticity. It has been shown that using a knockin mutation to α subunits that render $GABA_A$ receptors ($GABA_A$Rs) insensitive to benzodiazepines, only α1-containing $GABA_A$Rs are involved in visual cortical plasticity [6]. Since $GABA_A$Rs containing α1 subunits are mainly located at somatic synapses that receive inputs from parvalbumin (PV)-positive cells [9], it has been widely suggested that local pathways mediated by PV interneurons are highly involved in OD plasticity [6]–[8], [10]. This idea also agrees with the finding that the maturation of perisomatic inhibition by PV cells coincides with a critical period [11], [12].

The PV cells are also known to play a key role in providing feedforward inhibition in the cortical circuits [13]. A recent study on thalamocortical inputs [14] shows that the axons from the thalamus make stronger and more frequent excitatory connections onto fast-spiking (FS) interneurons (i.e., putative PV cells [15]). In addition, a study on functional excitatory connections onto layer 2/3 interneurons in the visual cortex suggests that FS cells receive strong feedforward excitatory connections from layer 4 [16]. Further, the same study shows that non-FS adapting interneurons (also known as accomodating interneurons) receive dominant lateral input from layer 2/3, implying that the feedback inhibition within the same cortical layer is mainly mediated by a class of interneurons other than PV cells. Taken together, the activities of PV cells, which may be highly required for OD plasticity [6]–[8], [10], are associated with feedforward inhibition, whereas the activities of a different type of adapting interneurons may be mainly involved in feedback inhibition.

Therefore, to examine a role of such local pathways associated with inhibition in OD plasticity, we performed simulation of a simple model, in which a single neuron receives the left- and right-eye inputs conveying correlated activities. We compared the effects of feedforward and feedback inhibition on the synaptic dynamics with spike-timing-dependent plasticity (STDP) [17], [18]. We first simulated a situation in which the activities of two visual inputs are temporally modulated, as in the experiments of monocular deprivation (MD) and reverse lid suture [19]. In this experimental paradigm, the originally deprived eye is reopened after the end of the initial MD period, while the non-deprived eye is sutured closed. We found that experience-dependent synaptic modifications take place in the presence of competitive interaction between the correlated input groups [20], [21], which is given by feedforward inhibition, but not by feedback inhibition. The effects of changing the strength of feedforward and feedback inhibition on the level of activity-

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dependent competition was also examined. It was found that the balance between them can gradually control the level of competition. Our results suggest that the interactions between the local circuits associated with feedforward and feedback inhibition may be involved in regulating experience-dependent cortical plasticity by modulating the competition among synapses.

2. Methods

We constructed a simplified model of a visual cortical cell which includes feedforward and feedback inhibition by modifying a previous model of STDP [20], [22] (Fig. 1). The postsynaptic cell, described as a leaky integrate-and-fire (LIF) neuron (caption in Fig. 1), receives random inputs from 1000 excitatory and 200 inhibitory synapses. Excitatory inputs are mediated by AMPA-type glutamate receptors, while inhibitory inputs are of GABA type. To simulate sensory inputs from two eyes to a visual cortical cell, excitatory inputs were assumed to consist of two equally sized groups [22].

Excitatory and inhibitory synaptic currents are described according to a conductance-based model [20]. Conductances of excitatory and inhibitory synapses (g_{exc} and g_{inh}, respectively) obey the following equations:

$$g_{exc}(t) = \bar{g}_{exc} w e^{-t/\tau_{exc}}, \tag{1}$$

$$g_{inh}(t) = \bar{g}_{inh}(e/\tau_{inh}) t e^{-t/\tau_{inh}}, \tag{2}$$

where $\bar{g}_{exc} = 0.015$, $\bar{g}_{inh} = 0.005$, $\tau_{exc} = 5$ ms, and $\tau_{inh} = 10$ ms (conductances were measured in units of leak conductances for all cases) [22]. w represents the weight of each excitatory synapse, and is temporally modified by STDP.

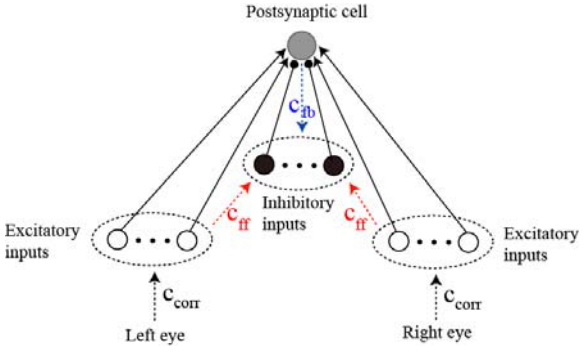

Fig. 1 Simulation model. A LIF neuron receives two groups of excitatory inputs and a group of inhibitory inputs. Excitatory connections to the postsynaptic cell are modified by STDP. Inhibitory inputs are activated by spikes through the feedforward (red) and feedback (blue) connections, eliciting the activation of inhibitory inputs following the excitatory input activation and postsynaptic spiking, respectively. The membrane potential V of the LIF neuron obeys $\tau_m(dV/dt) = I + g_{leak}(E_{leak} - V)$ with $\tau_m = 20$ ms, $E_{leak} = -74$ mV, and $g_{leak} = 1$ [20]. When the membrane voltage reaches -54 mV, the neuron fires and the voltage is reset to -60 mV after the refractory period of 1 ms. The parameter values of c_{ff} and c_{fb} (Eq. (4)) determine the strength of feedforward and feedback inhibition, respectively. c_{corr} (Eq. (3)) decides the correlation of input activities within the same group.

Each group of excitatory synapses is assumed to receive correlated inputs resulting from the activities of retinal ganglion cells in each eye. Furthermore, we introduce the feedforward and feedback excitatory connections that activate inhibitory synapses, as shown with red and blue lines in Fig. 1. A high computational cost is required to model the circuits involved in feedforward and feedback interaction between multiple neurons. However, there is evidence that under sufficiently strong noisy conditions like the *in vivo* state, postsynaptic firing probability is approximately proportional to the summation of postsynaptic potentials (PSPs) [23], [24]. Therefore, we assume that the activation times of all presynaptic inputs are described by simple non-stationary Poisson processes, the rate of which depends on PSPs [25].

Thus, the activation frequency of the two groups of excitatory AMPA synapses ($r_1^{exc}(t)$ and $r_2^{exc}(t)$) and that of inhibitory GABA synapses ($r^{inh}(t)$) are described as follows:

$$r_I^{exc}(t) = c_{corr} \sum_f \varepsilon(t - t_I^f) + r_{uncorr}^{exc} \quad (I = 1, 2), \tag{3}$$

$$r^{inh}(t) = \frac{c_{ff}}{n_{exc}} \sum_i \sum_f \varepsilon(t - t_{exc,i}^f) + c_{fb} \sum_f \varepsilon(t - t_{post}^f) + r_{uncorr}^{inh}. \tag{4}$$

Here, $\varepsilon(t)$ is a function that describes the time course of PSPs, and $\varepsilon(t) = (t/\tau_e^2)e^{-t/\tau_e}$ with $\tau_e = 20$ ms for $t \geq 0$ and $\varepsilon(t) = 0$ otherwise. In Eq. (3), t_I^f denotes the arrival time of the fth spike to the Ith group of excitatory synapses from retinal ganglion cells, and t_I^f is determined using a homogeneous Poisson process with a frequency of $r_{inp} = 5$ Hz. The spike arrival times for the two groups are independent, and therefore, the activities of the different groups are uncorrelated to each other. The parameter c_{corr} determines the strength of correlation among the inputs within each group, and r_{uncorr}^{exc} is a component of the activation rate corresponding to the uncorrelated spontaneous firing. In Eq. (4), $t_{exc,i}^f$ and t_{post}^f denote the fth firing time of the ith excitatory synapse and postsynaptic cell, respectively. Thus, the changes in c_{ff} and c_{fb} regulate the strength of feedforward and feedback inhibition, respectively, by modifying the strength of excitatory connections activating inhibitory inputs (Fig. 1). n_{exc} (= 500) is the number of excitatory synapses for each group, and r_{uncorr}^{inh} represents the activation rate of uncorrelated firing of inhibitory synapses.

The activation of feedforward and feedback inhibition increases the total inhibitory currents received by a postsynaptic cell. Because the effects of total inhibition level on STDP-induced competition were investigated in detail in a previous study [21], this study mainly addresses the influence of feedforward and feedback inhibition without changing the total input activities as follows. First, the uncorrelated component of the activation rate of excitatory synapses is decreased by increasing c_{corr} such that $r_{uncorr}^{exc} = 12 - c_{corr} r_{inp}$ (in Hz), unless otherwise stated. In this case, because $\int_{-\infty}^{\infty} \varepsilon(t)dt = 1$ holds, $r_I^{exc}(t)$ in Eq. (3) is maintained at 12 Hz, independent of c_{corr}. Second, the spontaneous

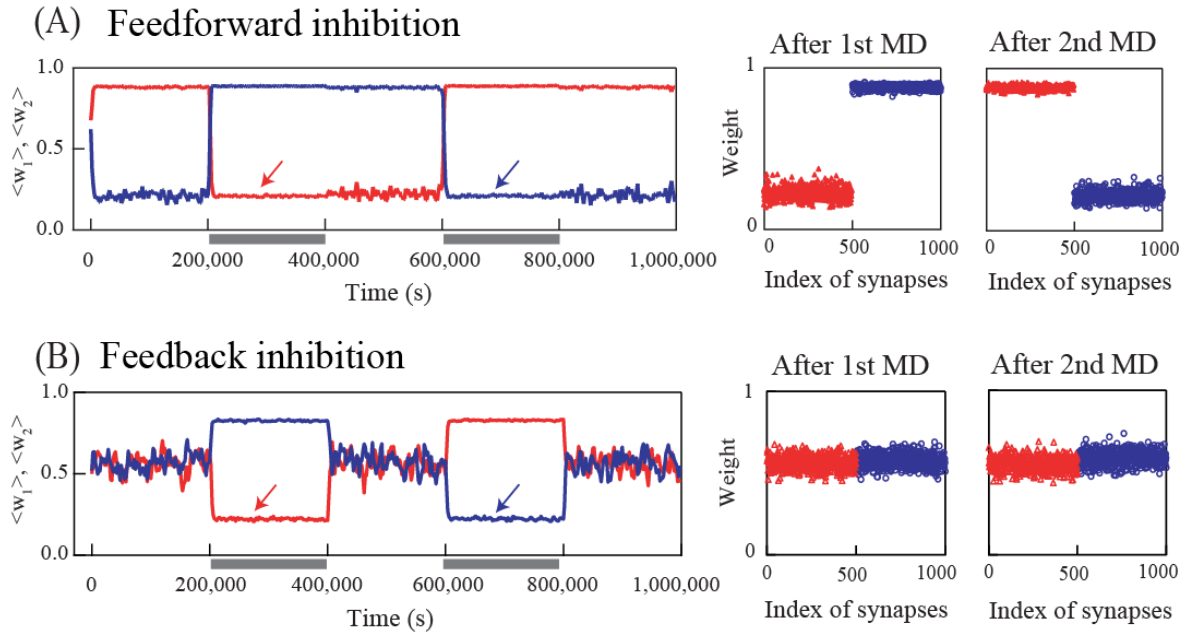

Fig. 2 Synaptic weight dynamics in response to MD and reverse suture in the presence of feedforward inhibition (A) or feedback inhibition (B). Left column: the time course of average weights for the two input groups. One input group (red line) receives deprivation for 200,000 s < t < 400,000 s (gray bar), while the other group (blue line) receives it for 600,000 s < t < 800,000 s (gray bar). The group receiving deprivation is denoted by the colored arrow. Center and right columns: the weight distributions of the two groups after the first MD (averaged over 450,000 s to 550,000 s; center column) and after the second MD (averaged over 900,000 s to 1,000,000 s; right column). The strength of feedforward and feedback inhibition (c_{ff} and c_{fb}, respectively) are set to be $c_{ff} = 1$, $c_{fb} = 0$ (A) or $c_{ff} = 0$, $c_{fb} = 0.085$ (B). These parameter values are selected such that the firing rate obtained in the absence of MD (40 Hz) is the same for (A) and (B).

rate of inhibitory synapses is modified such that $r^{inh}_{uncorr} = 12(1 - c_{ff})$, maintaining the rate of inhibitory synapses at 12 Hz in the absence of feedback inhibition. Additionally, in the presence of feedback inhibition, we examined cases in which the influence of changing the inhibitory synapse activity is virtually removed (Fig. 4B; see Results).

STDP is assumed to act on all of the excitatory synapses that converge on a postsynaptic cell (Fig. 1). With the time lag Δt between pre- and postsynaptic spikes, the change in the synaptic efficacy by STDP is :

$$\Delta w = \begin{cases} A_+ e^{-\Delta t/\tau_+}, & \text{for } \Delta t > 0, \\ -A_- e^{\Delta t/\tau_-}, & \text{for } \Delta t < 0, \\ 0, & \text{for } \Delta t = 0. \end{cases} \quad (5)$$

Here, $A_+ = 0.005$ and $A_- = A_+/0.98$ denote the size of long-term potentiation (LTP) and long-term depression (LTD), respectively. The value of A_- is assumed to be slightly larger than that of A_+ in order to avoid unstable synaptic dynamics [22]. $\tau_+ = \tau_- = 20$ ms determines the temporal width of the STDP curve. We assume that the amounts of STDP from all spike pairs are linearly summed, and the upper and lower bounds (1 and 0, respectively) limit the magnitude of synaptic weights.

3. Results

To explore the functional role of local pathways associated with GABA inhibition, we simulated synaptic modification dynamics in an experiment with MD, which was followed by reverse suture of the opposite eye (Fig. 2) [19]. The OD shift caused by MD has been shown to rely on the difference in the temporal correlations of retinal afferent activities rather than on the difference in the input activity levels between the two eyes [26], [27]. Therefore, we incorporated the effects of MD by decreasing the strength of input correlation (c_{corr}) for the group corresponding to the deprived eye.

We found two types of qualitatively different synaptic dynamics, as shown in Figs. 2A and 2B. Figure 2A (left) shows the time course of the average weights of two groups when only the feedforward inhibition is introduced ($c_{ff} = 1$, $c_{fb} = 0$). Here, the excitatory synapses of both groups are initially activated with the same input correlation ($c_{corr} = 0.6$). For the first period of MD (200,000 s < t < 400,000 s; gray bar), the input correlation of one group (denoted by a red arrow) is removed by decreasing the value of c_{corr} to 0. The input correlation of this group is recovered after the end of the first MD period. After that, the input correlation of the opposite group is removed for 600,000 s < t < 800,000 s (gray bar; denoted by a blue arrow), which corresponds to the reverse suture (second MD). Both groups maintain input correlation after t > 800,000 s. The result of Fig. 2A (left) shows that one input group becomes dominant over the other group, even in the absence of MD. This fact suggests

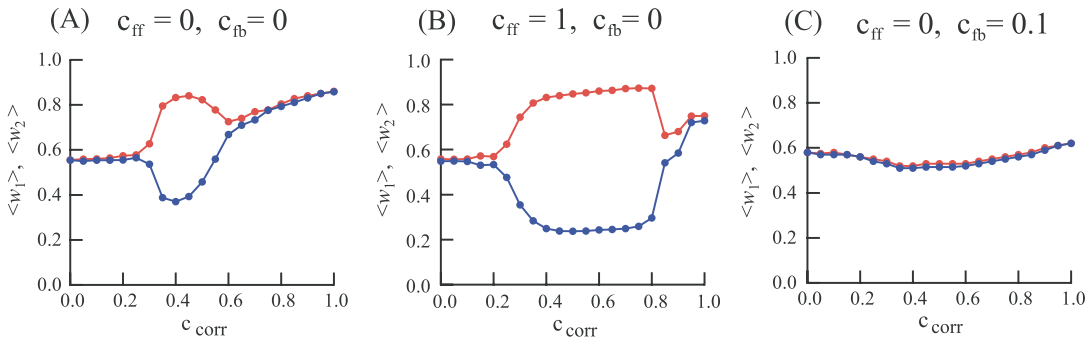

Fig. 3 The average weights of the two groups at the equilibrium of STDP are plotted as a function of the strength of the input correlation c_{corr} within the same group. The strength of the local connections associated with feedforward and feedback inhibition is set to be $c_{ff} = 0$, $c_{fb} = 0$ for (A), $c_{ff} = 1$, $c_{fb} = 0$ for (B), or $c_{ff} = 0$, $c_{fb} = 0.1$ for (C). We ensured that STDP reached equilibrium by checking that the weight average did not change for sufficiently long period.

an involvement of competitive interactions between the correlated input groups, which are elicited by STDP function [21]. During each period of MD, the group whose input correlation has been removed is strongly weakened and therefore the non-deprived group becomes dominant, consistent with experimental observations (e.g., [1]). In addition, the synaptic pattern produced by MD is maintained even after the end of MD period. Thus, the weight distribution after each period of MD (Fig. 2A, right two figures) clearly reflects which input group has received deprivation during a recent period of MD.

On the other hand, when the feedback inhibition is introduced (Fig. 2B; $c_{ff} = 0$, $c_{fb} = 0.085$), both groups rapidly converge to a state where the average weights fluctuate at nearly the same level, suggesting a lack of competition between the input groups. The value of c_{fb} used in Fig. 2B is decided such that the postsynaptic firing rate obtained is nearly the same as that of Fig. 2A (40 Hz). This also implies that the total amount of inhibitory currents is identical for the two cases. Importantly, in contrast to the case with feedforward inhibition (Fig. 2A), the weight distributions of the two groups, following each MD period, do not significantly differ from each other (Fig. 2B, right two figures). Therefore, the synaptic weight pattern cannot reflect which group has received input deprivation.

The comparison between Figs. 2A and 2B indicates that past sensory experience can be embedded into synaptic patterns only in the presence of competitive interactions, which are provided by feedforward inhibition, but not by feedback inhibition. We also found that with neither feedforward nor feedback inhibition ($c_{ff} = c_{fb} = 0$), the weight distributions of the two groups converge to be nearly the same in the absence of MD. Therefore, in this case, the synaptic pattern was not able to retain information about the sensory experience of MD, as in Fig. 2B.

In order to clarify how the competitive function is regulated by local circuits associated with feedforward and feedback inhibition, we plotted the average weights of the two groups at the equilibrium of STDP by changing the level of input correlation (c_{corr}) for three different values of c_{ff} and c_{fb} (Fig. 3). With neither feedforward nor feedback inhibition (Fig. 3A; $c_{ff} = c_{fb} = 0$), the occurrence of competition was restricted to a small range of c_{corr} ($0.3 < c_{corr} < 0.6$). However, by introducing feedforward inhibition (Fig. 3B; $c_{ff} = 1$, $c_{fb} = 0$), the competitive range of c_{corr} was prolonged ($0.2 < c_{corr} < 0.8$), suggesting that feedforward inhibition promoted the emergence of competition. In contrast, a low level of feedback inhibition prevented the induction of competition for all values of c_{corr} (Fig. 3C, $c_{ff} = 0$, $c_{fb} = 0.1$). These results indicate that feedforward inhibition promotes competitive interaction, whereas feedback inhibition strongly suppresses it.

To quantify the strength of competitive interaction, we calculated the ratio of the average weights between the two groups at the equilibrium state (Fig. 4). Higher level of feedforward inhibition was found to not only prolong the competitive range of c_{corr}, but also significantly increase the level of competition when it occurred (Fig. 4A). Conversely, weaker feedback inhibition ($c_{fb} = 0.02$; Fig. 4B, red line) considerably decreased the level of competition, which disappeared at $c_{fb} = 0.1$ (Fig. 4B, blue line). To virtually remove the effects of feedback inhibition on the total input activity, we examined a case in which feedback inhibition was absent ($c_{fb} = 0$), but the mean frequency of inhibitory synapses was maintained to be the same as that for $c_{fb} = 0.1$ by adjusting r^{inh}_{uncorr} (Fig. 4B, black dashed line). In this case, a certain level of competition remained for a large range of c_{corr} ($0.2 < c_{corr} < 0.6$). This result implies that the suppressive effect of feedback inhibition on the competition cannot be simply explained by the alteration in total input activities, although the detailed mechanism for producing this effect remains to be elucidated. As shown in Fig. 4C, when feedforward and feedback inhibition coexisted, the increase in feedforward inhibition and the decrease in feedback inhibition gradually increased the level of between-group competition. This result suggests that the balance between the local circuits involved in feedforward and feedback inhibition can effectively regulate the level of activity-dependent competition.

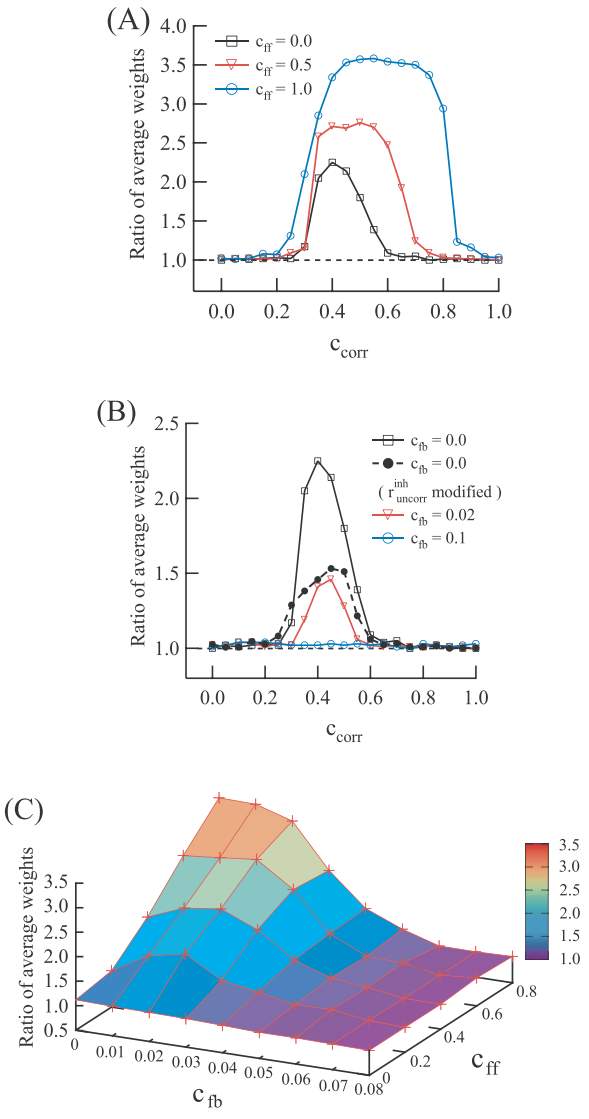

Fig. 4 The change in the level of between-group competition regulated by the local pathways associated with feedforward and feedback inhibition. (A and B) The ratio between the average weights of the two groups at the equilibrium of STDP is plotted as a function of the level of input correlation c_{corr}. The effects of changing the strength of feedforward and feedback inhibition are shown in (A) and (B), respectively [c_{ff} = 0.0 (black), 0.5 (red), or 1.0 (blue), and c_{fb} = 0.0 in (A). c_{fb} = 0.0 (black solid), 0.02 (red), or 0.1 (blue), and c_{ff} = 0.0 in (B)]. The black dashed line in (B) shows a case in which feedback inhibition is absent, but the mean activation rate of inhibitory inputs are kept at that obtained with c_{ff} = 0.0 and c_{fb} = 0.1 by adjusting the value of r^{inh}_{uncorr} (Eq. (4)). (C) The ratio of the average weights between the two groups as a function of c_{ff} and c_{fb} (c_{corr} = 0.6).

4. Discussion

Several experiments have shown that an increased level of cortical GABA activities is critical for inducing OD plasticity [2]–[8]. However, the notion that the level of inhibition determines plasticity is not consistent with the observation that only GABA functions mediated by α1-containing $GABA_A$ receptors drive plasticity [6]. Because this observation suggests that local circuits associated with GABA inhibition may play a role in regulating plasticity, we explored whether the pathways associated with feedforward and feedback inhibition are selectively involved in cortical plasticity.

We showed that the activation of feedforward inhibition promotes competition between input groups (Fig. 4A), whereas the activation of feedback inhibition prevents competition and counterbalances the effect of feedforward inhibition (Figs. 4B and 4C). In addition, we demonstrated that in the presence of competitive interaction given by feedforward inhibition, which group becomes dominant depends on past sensory experience, providing the basis of experience-dependent plasticity (Fig. 2A). The synaptic function that reflects past experience of inputs disappeared when the competitive interaction is lost in the presence of feedback inhibition (Fig. 2B).

When the two groups of synapses are segregated into strong and weak ones by the competitive interaction, there exist two stable synaptic patterns, such that one group is dominant or the other group is dominant. Since bistable dynamics can produce a "memory" of past experience of input stimuli [28], synaptic pattern can retain information about earlier sensory experiences, as in Fig. 2A. However, in the absence of competition, the weight distributions of both groups are identical and the synaptic dynamics are monostable. Thus, the information about earlier sensory experiences cannot be retained in the synaptic pattern, as in Fig. 2B. Therefore, our model predicts that the competitive mechanism may be required for producing bistable synaptic dynamics, which provides the synaptic circuits with the ability of reflecting sensory experience of MD. This idea is in line with the observations that the activity-dependent competition between the inputs from two eyes is involved in OD plasticity [1], [29]–[31].

Recent studies have also proposed a role of GABA in controlling visual cortical plasticity [27], [32]. In a study by Kuhlman et al. [27], it has been theoretically shown that GABA inhibition preferentially reduces synaptic efficacy of less coherent inputs, producing an OD shift toward more coherent inputs. Additionally, the experiments of the same study have revealed that stronger GABA inhibition ensures that coherent inputs can control postsynaptic spiking. Another role of GABA was proposed by Kanold and Shatz [32], who have studied the function of subplate neurons in regulating OD plasticity through GABA maturation. They found that higher levels of inhibition generate a shift in OD toward the non-deprived eye during MD, while lower levels of inhibition prevent MD from producing an OD shift. These studies suggest that a function of GABA is to regulate which of two inputs become dominant during the period of MD [27], [32]. In contrast, our model showed that the local circuits associated with GABA inhibition can control whether the results of MD are embedded in synaptic patterns after the end of MD. It is important to distinguish between the following two functions of GABA inhibition: how the direct effects of MD (i.e., which input becomes stronger) are controlled, which has been considered by previous stud-

ies [27], [32], and how the consequences of MD are stabilized in synaptic weights, which is considered in the present study. The GABA functions proposed by previous studies do not contradict our model. Thus, their hypotheses can coexist with ours.

It has been proposed that local connections mediated by PV interneurons are particularly important for triggering visual cortical plasticity, as mentioned above. An experiment on the visual cortical circuits suggests that the PV cells and a type of adapting interneurons are mainly involved in feedforward and feedback inhibition, respectively [16]. Thus, PV cell-mediated feedforward inhibition might play a key role in visual cortical plasticity, which agrees with the prediction of our model. Additionally, it is conceivable that some portion of PV cells might receive both feedforward and feedback inhibition [16], [33]. In this case, it can be expected from our model that a relative balance between the feedforward and feedback inhibition may regulate OD plasticity through modulating the level of activity-dependent competition (Fig. 4C).

A main limitation of our model is that it does not take into account lateral connectivity within cortical circuits that may affect sensory responses and plasticity [7], [34]. Experimental findings have suggested that the pharmacological blockade of intracortical horizontal connections may disrupt the receptive fields of visual cortical cells and OD plasticity [34]. Future work should focus on constructing a model of interconnected cortical neurons and investigating how lateral connections, in addition to feedforward and feedback inhibition, regulate visual cortical plasticity.

5. Conclusion

We proposed a model to explain the role of local circuits associated with feedforward and feedback inhibition in OD plasticity, motivated by the observation that only specific localized circuits are involved in this type of plasticity [6]. We showed that stronger feedforward inhibition can enhance OD plasticity, whereas feedback inhibition tends to suppress it. Our results appear to be consistent with the fact that PV interneurons, which are mainly associated with the feedforward inhibition in the cortical circuits, may play a critical role in driving OD plasticity [7], [13].

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