Delay Compensation Through Facilitating Synapses and STDP: A Neural Basis for Orientation Flash-Lag Effect

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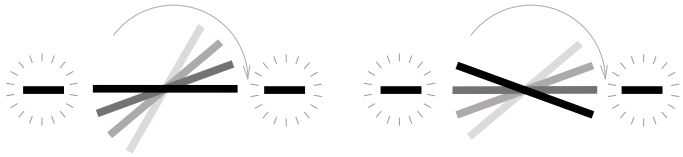
***Abstract*— In orientation flash-lag effect (FLE), a continuously rotating bar in the center is perceived to be misaligned toward the direction of rotation when compared to a briefly flashed pair of flanking bars that are actually aligned. The implication of this simple visual illusion is quite profound: The effect may be due to motion extrapolation, undoing the effects of neural conduction delay. Previously, we showed that facilitating synapses may be a neural basis of such a delay compensation mechanism in other forms of FLE such as luminance FLE. However, the approach based on a single neuron cannot be applied to orientation FLE since firing rate in a single neuron cannot represent the full range of orientations. Here, we extend our model to multiple neurons, and show that facilitating synapses, together with adaptation through Spike-Timing-Dependent Plasticity (STDP), can serve as a neural basis for delay compensation giving rise to orientation FLE.**

I. Introduction

Flash-Lag Effect (FLE) occurs in visual perception when the state of a changing stimulus such as luminance [1], position in a moving object [2], or orientation in a rotating object [3] is misperceived. For example, Fig. 1 shows the orientation FLE. In this version of FLE, the rotating bar in the center is perceived to be tilted at the time of flash of the flanking bars when in fact it is horizontally aligned with the flanking bars. This kind of illusion is common, as it is found in several other modalities, including motor performance [4], auditory perception [5]; and in various visual modalities such as color, pattern entropy, and luminance [1].

One explanation for the FLE is based on the idea that it occurs due to an extrapolation process in the nervous system, which helps compensate for neural conduction delay [3], [6]. (See Sec. IV for alternative hypotheses.) The reasoning is that significant delay exists in neural systems, and if uncompensated, the organism will always live in the past, thus being unable to interact in real-time with the dynamically changing environment (Fig. 2). FLE occurs because the moving (or changing) object gets extrapolated based on the past trajectory information, while the abruptly flashed object does not, and hence the misalignment. As a result, the current internal state can be aligned with the external state. At time = n in Fig. 2, the internal state would look like Fig. 1b if extrapolation was carried out.



(a) Physical (b) Perceived

Fig. 1. **Orientation Flash-Lag Effect.** Grayscale indicates bar orientation over time (black = present). The two short flanking bars are flashed when the rotating bar is horizontal (a), however, the rotating bar is perceived as misaligned (b).

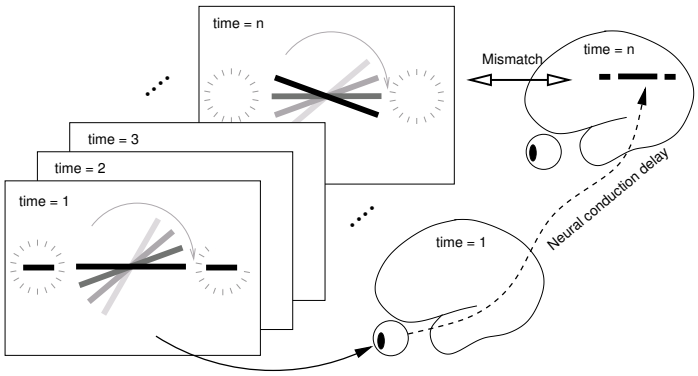


Fig. 2. **Neural Delay Causing Mismatch in Internal and External State.** The rotating bar (black bar in the middle) is horizontal when the flanking bars are flashed (time = 1). The same input is received at the retina (bottom right). The input takes time ($n-1$) to reach higher visual areas (top right). If the delay is not taken into consideration, the perceived orientation of the rotating bar will be misaligned with the environmental state at time = n, since the bar continues to rotate during the ($n-1$) time period.

Our main research question is, what can be the neural basis of such an extrapolation process? In previous work, we showed that facilitating synapses [7], [8], a form of dynamic synapse, can function as an extrapolator to undo the effect of neural delay, thus giving rise to FLE [9]. In that work, we modeled a single neuron, and tested our idea in luminance FLE [1]. However, the same approach cannot be applied to other forms of FLE such as orientation FLE, since unlike luminance, the

full range of orientation cannot be represented by a single neuron. Changing firing rate may be able to represent dark to bright, but orientation-tuned cells in the visual cortex are narrowly tuned, thus one neuron can only represent a narrow range of orientation (see [10] for a review). To address this issue, we extended our previous *facilitation model* to include multiple neurons, and allowed facilitation to go across neurons. As it turns out, facilitating synapses are not enough to give rise to orientation FLE. We found that mechanisms such as Spike-Timing-Dependent Plasticity (STDP) [11], [12] is necessary to set up the connection strengths, so that cross-neuron facilitation can carry out extrapolation in the direction of change.

In the following, we will first provide details of our model (Sec. II), and present the experiments and their results (Sec. III). Finally, we will discuss issues arising from our work (Sec. IV), and conclude with some outlook into the future (Sec. V).

II. Model Description

A network of leaky integrate-and-fire neurons [13] was constructed to test facilitating dynamics and STDP as the neural basis of extrapolation. In order to represent the full range of orientation, we modeled twelve orientation-sensitive neurons at a 15°. Fig. 3 shows the arrangement of the neurons, and their lateral excitatory connectivity (connections were only between immediate neighbors). Each neuron fires according to a distribution centered around its preferred orientation, maximally firing when the preferred orientation was present in the input, and gradually less as the input veers away (Fig. 4, "No FLE"). Note that during orientation FLE, the distribution shifts toward the direction of rotation (Fig. 4, "FLE").

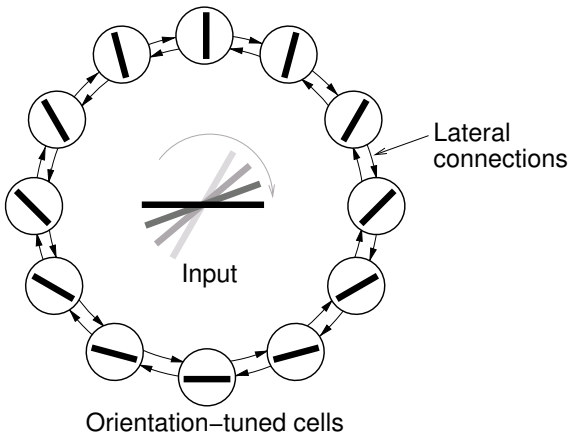


Fig. 3. **Bilaterally Connected Orientation-Tuned Cells.** Twelve orientation-tuned neurons are shown, with excitatory lateral connections between immediate neighbors. The connectivity shown here is a simplification of similar arrangements near orientation pinwheels in the primary visual cortex [14], [10].

Each neuron has a facilitating dynamics, and the lateral connections are trained using STDP. Note that initially, all the neurons have same synaptic weights on their bilateral connections (i.e. rotationally symmetric). This symmetry is broken by synaptic adaptation induced by repetitive activation

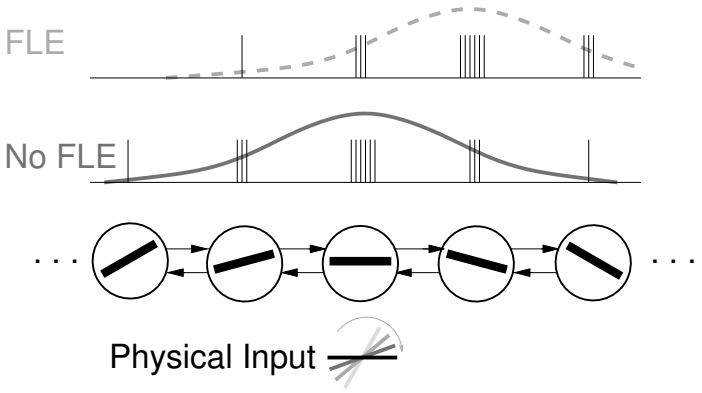


Fig. 4. **Response of Orientation Cells with and without FLE.** The spike responses (vertical bars) and firing-rate distributions (curves) are shown, with (top; dashed) and without (bottom; solid) Flash-Lag Effect. The x-axis represents the spatial span across the connected orientation-tuned cells (Fig. 3). Without FLE (solid), the firing-rate distribution is centered around the middle (cf. Fig. 1*a*). With FLE (dashed), the distribution is shifted toward the direction of input rotation (cf. Fig. 1*b*).

(i.e. rotating visual stimulus). The two sections below present these mechanisms in detail.

A. Facilitating Dynamics

The description in this section closely follows Lim and Choe [9]. Dynamic synapses generate a short-term change in synaptic strength which shows activity-dependent decrease (depression) or increase (facilitation) in synaptic transmission. The process occurs within several hundred milliseconds from the onset of the activity (for reviews see [15], [16], [17]). Especially, facilitating synapses cause a gradual increase in postsynaptic response through increasing synaptic efficacy with successive presynaptic spikes. (Synaptic efficacy is the fraction of neural transmitter released when presynaptic action potentials arrive at the axon terminal.) Unlike depressing synapses, the rate of neural transmitter release is not constant but is a dynamic variable in facilitating synapses.

According to the dynamic synapse model by Markram and colleagues, based on neurophysiological data [8], [7], synaptic efficacy U evolves over time as described in the following differential equation:

$$\frac{dU}{dt} = -\frac{U}{\tau_{\mathrm{f}}} + C(1-U)\delta(t-t_{\mathrm{s}}), \tag{1}$$

where τ_{f} is the time constant for the decay of U; C a constant determining the increase in U when a successive action potential (AP) arrives at time t_{s} at the synaptic terminal; and $\delta(\cdot)$ the Dirac delta function. This equation is already suitable enough to replicate the extrapolating dynamics when the activation level is increasing. However, it is not capable of handling cases where the activation level is decreasing. Ideally, extrapolation should work for both increasing and decreasing trends.

Here we modify the equation by redefining C as a dynamic variable which is varied in proportion to the change of input

firing rate:

$$C = Sgn\left(I(n-1) - I(n)\right)\left(\frac{I(n-1)}{I(n)}\right)r, \qquad (2)$$

where $Sgn(\cdot)$ is the sign function, $I(n)$ is the interspike interval between the n-th spike and the $(n-1)$-th spike which reflects whether a spike train consists of high-frequency APs or low-frequency APs. The first term in Eq. 2 determines the sign of C: "$+$" for increase or "$-$" for decrease in firing rate. The second term represents the ratio of the change in frequency, and r is a gain parameter. As the input firing rate increases, C becomes positive and increases proportional to the rate of change in frequency. On the contrary, as the firing rate decreases, $I(n)$ becomes larger which results in a negative C and thus leads to the decrease in the rate of change in the synaptic efficacy U.

With this, we can now fully describe our membrane potential model (cf. [8], [7], [18]). The time course of postsynaptic current $P(t)$ at time t triggered by incoming spikes is defined as follows:

$$P(t) = Ee^{-\frac{t}{\tau_{\rm p}}}, \qquad (3)$$

$$E = wAU, \qquad (4)$$

where E is the excitatory postsynaptic potential (EPSP) amplitude; $\tau_{\rm p}$ the time constant of decay in $P(t)$; w weight or scaling factor of A; A a constant for maximum postsynaptic response amplitude; and U the synaptic efficacy as defined above. Finally, the membrane potential $V_m(t)$ at time t evolves as follows:

$$V_m(t) = V_m(t-1)e^{-\frac{t}{\tau_{\rm m}}} + P(t)(1 - e^{-\frac{t}{\tau_{\rm m}}}). \qquad (5)$$

The membrane potential is determined by the membrane current $P(t)$ at that moment and the previous membrane potential $V_m(t-1)$, both of which are regulated by a membrane time constant $\tau_{\rm m}$ ($= 300$ ms). The last part of the spiking neuron model is the spike generation mechanism. Once V_m exceeds the spike threshold θ ($= -54$ mV), a spike is generated, and V_m is reset to -70 mV followed by an absolute refractory period of $\tau_{\rm refrac}$ ($= 4$ ms) during which spikes cannot be generated.

To allow for cross-neuronal interaction, we extended the single neuron model above to include synaptic input from neighboring neurons, as shown in Fig. 3. Each cell in Fig. 3 can receive spikes from other neurons to the left and to the right as well as input spikes delivered from peripheral neurons. Through the sequence of spikes, facilitating dynamics propagate to neighboring neurons and increase the postsynaptic neurons' responses. At the same time, the connection weights w are also updated through STDP (see Sec.II-B for details). As a result of STDP, that facilitating activity propagates only to the neurons located in the direction of rotation.

B. Spike-Timing-Dependent Plasticity (STDP)

The lateral connections in our model (Fig. 3) are initially directionally symmetric, meaning that there is no preferential activation in the clockwise or the counter-clockwise direction.

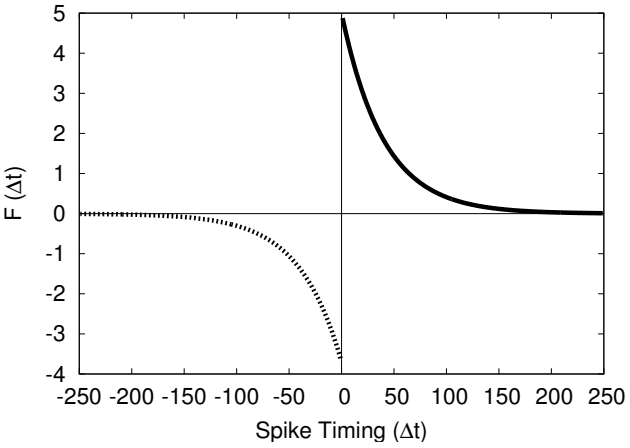


Fig. 5. **Illustration of STDP.** The illustration shows the dynamic increase or decrease in the amount of synaptic modification F based on spike timing Δt (postsynaptic spike time $-$ presynaptic spike time) as shown in Eq. 6.

However, for extrapolation to happen in a particular direction, there needs to be directionality.

Such a learning process that gives rise to directionality can be found in Spike-Timing-Dependent Plasticity (STDP). STDP changes the synaptic weight between two neurons when they fire together within a small time interval [11], [12]. Fig. 5 shows the basic principle underlying STDP. When the presynaptic neuron fires first (firing time $t_{\rm pre}$), and then the postsynaptic spike (firing time $t_{\rm post}$), then the difference $\Delta t = t_{\rm post} - t_{\rm pre} > 0$, since $t_{\rm post} > t_{\rm pre}$. On the other hand, if the postsynaptic neuron fires first, $\Delta t < 0$. The synapse will be strengthened if $\Delta t > 0$, and weakened if $\Delta t < 0$.

To include STDP in our model, we used the following synaptic modification function [12]:

$$F(\Delta t) = \begin{cases} A_+ e^{-\frac{\Delta t}{\tau_+}} & \text{if } \Delta t > 0 \\ -A_- e^{\frac{\Delta t}{\tau_-}} & \text{if } \Delta t < 0 \end{cases}, \qquad (6)$$

where A_+ and A_- are constants determining the maximum range of modification, and τ_+ and τ_- the time constants of exponential decay. For our simulations, we used the following values: $A_+ = 5.0$, $A_- = 3.7$, $\tau_+ = 80$, and $\tau_- = 105$.

The actual weight adaptation for connection from neuron j to neuron i, with firing time t_j and t_i, respectively, was carried out as follows:

$$w_{i,j} \leftarrow w_{i,j} + \alpha F(\Delta t); \qquad (7)$$

where α is the learning rate ($= 0.02$) and $\Delta t = t_i - t_j$. In case $w_{i,j}$ reached zero, it was not decreased any further.

Fig. 6 in the next section shows the evolution of the connection weights in the direction and in the opposite direction of input rotation. When a particular target (postsynaptic) neuron has orientation preference that is in the rotating direction relative to the source (presynaptic) neuron, the source neuron fires first and then the target neuron, so STDP strengthens the connection. STDP weakens the connection in the opposite case (i.e. the postsynaptic neuron fires first). A critical factor

in this experiment is that the input rotation should not be too fast nor too slow, so that neighboring neurons can fire *within* the adaptation window (the width of positive or negative range in Fig. 5, which is found to be around ± 80 ms in the experimental literature [19]). Also note that the parameters τ_+ and τ_- in Eq. 6 affect the width of positive and negative adaptation windows, respectively. Interestingly, this timing roughly corresponds with the timescale of orientation FLE. For example, as the orientation of the stimulus sweeps by at 100 ms per neuron (i.e. 25 RPM), the neurons generate several spikes while the input is at their preferred orientation. With this, the bilaterally connected presynaptic and postsynaptic neurons fire together within a short time interval (about 100 ms) and Δt will fall within the STDP range. Thus, the synaptic weights between the neurons can be changed.

III. Experiments and Results

To test, in a multi-neuron environment, the contribution of facilitating dynamics and STDP in delay compensation, we used a network of neurons arranged as in Fig. 3. The experiments were carried out in view of orientation FLE, illustrated in Fig. 4.

We conducted three separate experiments: orientation perception with (1) STDP only, (2) facilitating synapses only, and (3) both STDP and facilitating synapses. The results are reported in the following sections. In all experiments, the input was rotated in the clockwise direction, at a speed of 25 RPM, and the input firing rate was set to 10 spikes/100 ms.

A. Orientation perception with STDP

First, we tested our model with only STDP, without facilitating synapses. The input bar was rotated at the speed of 25 RPM, while the connection weights were allowed to adapt according to Eq. 7. For those connections pointing toward the direction of input rotation, the weight increased (solid curve) since presynaptic spike preceded postsynaptic spike within the small time interval (Fig. 5). On the other hand, for the connections pointing in the opposite direction, the weight decreased (dotted curve). Fig. 6 shows these results.

To calculate the firing rate at any given moment, we used a fixed sliding window of width 100 ms. We measured the firing rate when the input was oriented to optimally stimulate neuron 2. Fig. 7 shows the results. Initially, when the weights have not adapted much, the firing-rate distribution is peaked at neuron 2 and is symmetric. After the weights have reached a stable state, the distribution becomes asymmetric, with a shift toward the right, the direction of input rotation. However, the location of the peak did not change (neuron 2). The results can be interpreted as no orientation FLE occurring in this experiment.

B. Orientation perception with facilitating synapses

In the next experiment, we used only facilitating synapses, without STDP. All weights were initialized to 1.0, and remained fixed throughout the experiment. Since the weights did not have any directionality, we expected no orientation FLE to occur, which turned out to be the case.

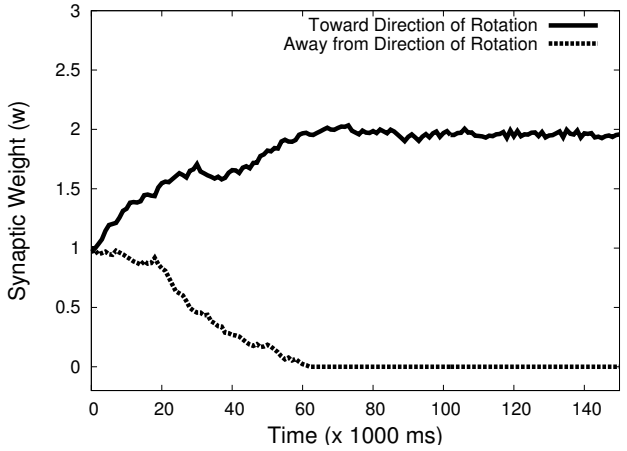


Fig. 6. **Adaptation of Synaptic Weight using STDP.** The synaptic weights w in the conenctions from neuron 2 to two immediate neighbors are shown. The weight to the neuron in the direction of input rotation increases (solid curve), while that in the opposite direction decreases (dotted curve).

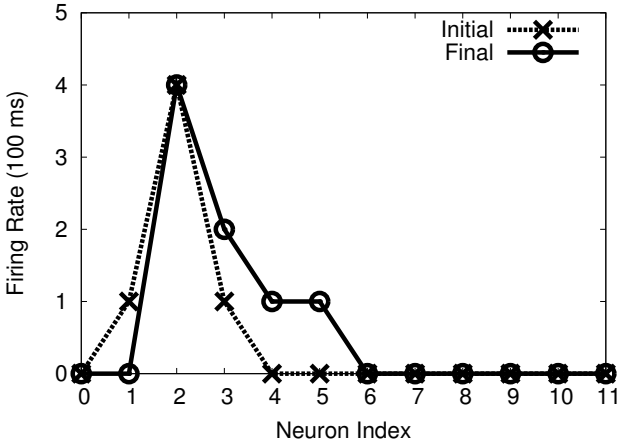


Fig. 7. **Firing Rate Distribution of STDP-only Experiment.** The dotted line with 'x' represents the initial firing rates of neurons. The solid line with 'o' denotes the changed firing rate after the synaptic weights reached a stable state through STDP. This is a snapshot of firing rates (i.e. number of spikes for 100 ms) when the actual input orientation was neuron 2's preferred stimulus. With STDP, the firing rates of the neurons changed due to the synaptic weight modification (skewed toward rotation direction). However, the maximally firing neuron (neuron 2) did not change (i.e., no FLE).

The only changing quantity in this experiment was the synaptic efficacy in the facilitating synapses, following Eq. 1. Fig. 8 shows the evolution of the synaptic efficacy U over time, for a single neuron. The U value increases while the input bar is optimally stimulating the neuron, and decays as soon as the input bar rotates out of the optimal range.

As expected, the firing rate distribution did not change from its symmetric peaked distribution centered around the optimally tuned neuron, for the given input orientation (Fig. 9). The results suggest that, again, orientation FLE did not occur, and facilitating synapses alone are not enough to account for cross-neuron facilitation (or extrapolation across neurons).

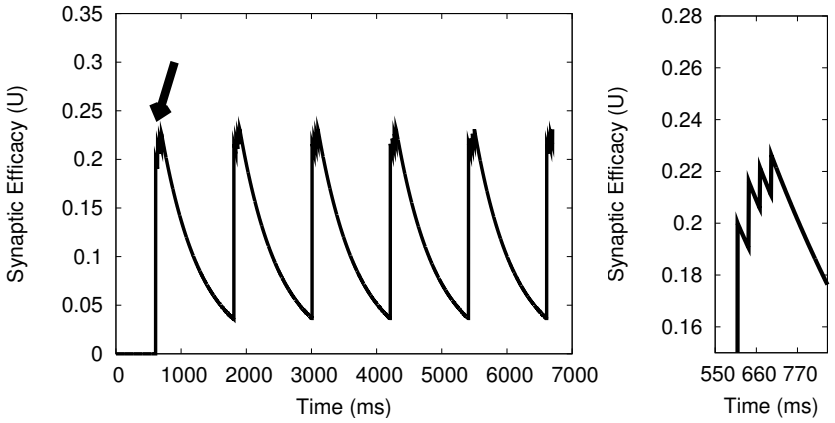


Fig. 8. **Adaptation of Synaptic Efficacy Through Facilitating Synapses.** The synaptic efficacy in a single neuron is shown. During the period of stimulation (when the rotating input is stimulating the neuron) synaptic efficacy increases. Shown to the right is a close-up of the part on the left marked with an arrow.

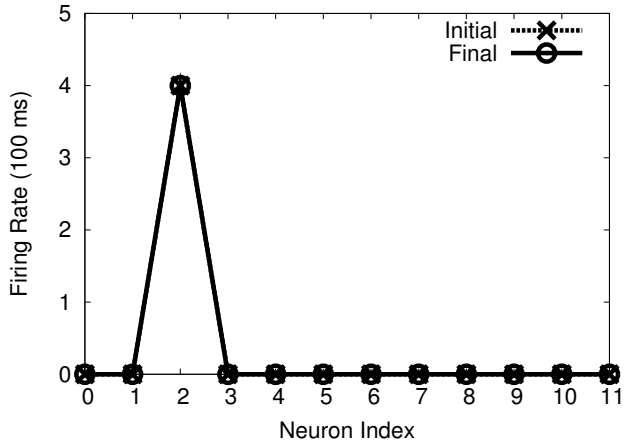


Fig. 9. **Firing Rate Distribution of Facilitating-synapse-only Experiment.** The distributions of firing rates of initial state (dotted line with 'x') and final state (solid line with 'o') are shown. Because the synaptic efficacy U is rapidly changed (increase and decrease) by a small amount (see Fig. 8, right), facilitating synapse alone did not affect the firing rate of the neighboring neurons. Thus, the maximally firing neuron (neuron 2) did not change (i.e., no FLE).

C. Orientation perception with STDP + facilitating synapses

The final experiment combined STDP and facilitating synapses. The experimental condition was the same as in Sec. III-A and III-B for the STDP and the facilitating synapse parameters, respectively.

In this experiment two factors contributed to synaptic transmission between two neurons: the very short-term increase in synaptic efficacy due to facilitating dynamics, and the longer-term increase in synaptic strength due to STDP. Fig. 10 shows the combined effect of these two factors over time. The gradually increasing long-term trend in STDP weight is accented with rapidly changing facilitating dynamics.

STDP gives the model directionality, and facilitating dynamics provides extra influence in the direction of input rotation, thus, allowing the model to shift its firing rate distribution significantly. Fig. 11 (thick arrow) shows that the peak in the firing rate distribution shifts toward the direction of input rotation (that is, to the right) by two steps (neuron 4), when the actual input at that moment optimally stimulates neuron 2. Thus, we can conclude that orientation FLE occurred in this case.

An important result to be noted is that when the input firing rate was low (e.g. 5 spikes/100 ms), the shift of the firing rate distribution was not observed (i.e. no FLE; the data are not shown here). The reason is that to make facilitating dynamics to be activated, the neuron should receive high frequency input spikes [16]. This result can be a potential answer to a puzzling phenomenon, the Flash-Lead effect: When the luminance of the rotating bar is sufficiently low (relative to the flashed objects), the rotating bar appears behind the flashed object (opposite of FLE; [20]). Even our results could not explain the Flash-Lead effect exactly (i.e. shift of firing rate distribution toward the opposite direction), it was shown that the sensitivity of facilitating activity to the spike frequency may play a role in modulating the degree of misalignment.

Rotation termination or rotation reversal can be further tested with our model. However, in this paper, we assumed that a separate network (as shown in Fig. 3) is responsible for the other directional input which is independently adapted by FAM and STDP to give rise FLE in the opposite direction. For example, similar directionally selective rotational arrangements can be found near orientation pinwheels in the visual cortex [10]. As for rotation termination, it has been shown that motion stopping signal conveyed by inhibitory synaptic transmission can suppresses facilitated postsynaptic membrane potential to reduce the flash-lag effect [21].

D. Summary of Results

The results showed that STDP or facilitating synapses alone cannot serve as a robust mechanism for delay compensation, as shown in their lack of ability to give rise to orientation FLE in a multi-neuron setting. Only when both are combined, delay compensation can work properly, thus rise to orientation FLE.

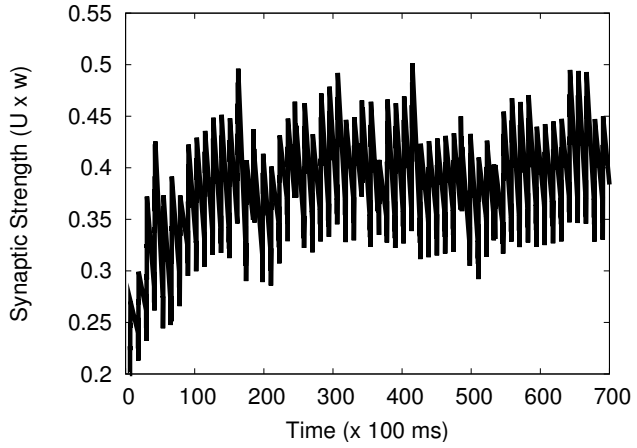


Fig. 10. **Adaptation of Synaptic Strength ($U \cdot w$ in Eq. 4) Through STDP plus Facilitating Synapses (Along Direction of Rotation).** Synaptic strength ($U \cdot w$) dynamically increases because of the gradually increasing synaptic weight w and the fast changing synaptic efficacy U.

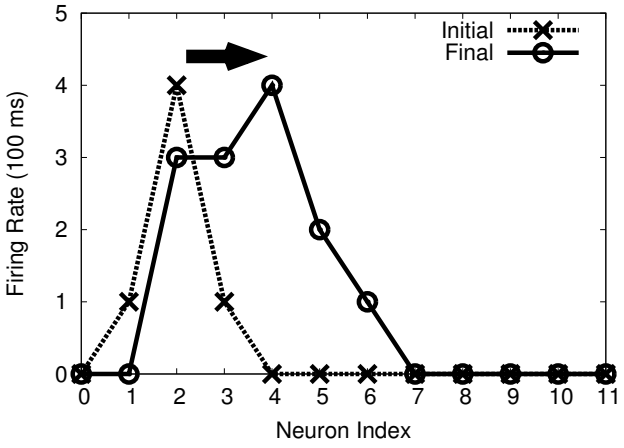


Fig. 11. **Firing Rate Distribution of STDP plus Facilitating-synapse Experiment.** The distribution of firing rates significantly shifted toward the rotating direction (as marked with an arrow). The position of maximally firing neuron changed from neuron 2 to neuron 4 (dotted line with 'x' for initial state; solid line with 'o' for final state). After training with STDP plus facilitating synapse, with the input at neuron 2's preferred orientation, neuron 4 showed maximum firing rate instead of neuron 2. This result indicates that the perception of the rotating bar shifted, just like in orientation FLE.

IV. Discussion

The main contribution of this paper is to draw attention to the relationship between STDP, facilitating synapses, and delay compensation. Through our model and experiments, we showed the link between the (1) psychophysical phenomenon of orientation FLE, (2) neurophysiological mechanisms of STDP and facilitating synapses, (3) and theoretical problems associated with neural delays and extrapolative compensation.

STDP and facilitating synapses are well-known phenomena in neurophysiology, and there are several theories on the possible role of these mechanisms. Existing theories on the role of STDP are varied. For example, STDP is supposed to help reduce spike train variability [22]. It was also shown that STDP may be involved in topology-preserving mapping [23]. Others suggested STDP as the neural mechanism for cortical orientation tuning [24]. The perspective closest to ours is that of [25]: They argued that STDP may implement a predictive function, where what will next occur in an input sequence is predicted. (See also [26] for STDP's anticipatory dynamic function in CA1. General discussion of the role of prediction in brain function can be found at [27] and [28].) However, they did not relate this predictive ability to the idea of delay compensation. From our perspective, these predictive mechanisms are actually predicting the present, not predicting the future. Also, our work further combines facilitating synapses with STDP, to give the predictive mechanism a faster and more dynamic timescale, which turned out to be necessary to account for perceptual phenomena such as orientation FLE.

Facilitating synapses (and dynamic synapses in general) have been studied in the context of memory or temporal information processing. For example, they were discussed in relation to sensitization and habituation [29], [30], or temporal information processing [7], [8], [16], [31], [32]. Again, our view provides a new insight on the possible role of facilitating synapses—that of extrapolatory delay compensation. It is interesting to note that there is a view that memory is explicitly linked to predictive functions [27], thus all the views above may be complementary.

It would be worth to verify whether the variable term C defined in Eq. 2 is plausible in terms of neurophysiology. (Note that we modeled the synaptic efficacy increase factor C as a variable instead of constant.) It is known that facilitation is driven by elevated calcium levels in presynaptic terminals. Recently, a neurophysiological mechanism (e.g., involving calcium chelator BAPTA) has been found, which may be responsible for the variation in C [33].

Turning our attention to psychophysics, Flash-Lag Effect is actually a much more complicated perceptual phenomenon than we described in the introduction. For example, FLE does not occur near motion-reversal points [34], or when the luminance of flashed object is sufficiently high [20]. There are several alternative hypotheses regarding the cause of FLE. Differential latency model suggests that FLE occurs because the visual system responds with shorter latency to moving stimuli than to flashed stimuli [34], [35]. However, recent results suggest that differential latency model may have limitations [5], [36]. Another hypothesis is postdiction, which suggests that information received in the future is incorporated into the estimation of a past perceptual event [37], [38]. Postdiction helps explain anomalies near motion reversal or motion termination where extrapolation model cannot provide an accurate account. Despite these limitations, motion extrapolation model has desirable properties [39], [40], [41], [42], and the limitations can be overcome, computationally and neurophysiologically [9].

Finally, there is an important implication of our research on detecting and treating neurological disorders such as autism and dyslexia. For example, autistic children have problem de-

tecting coherent motion [43], and have problem in processing moderately rapid motion [44], [45]. People with dyslexia also have difficulty in processing rapidly changing stimulus [46]. These results suggest that people with autism or dyslexia may have problem in motion processing, and one reason for the malfunction may be related to delay compensation mechanisms. If this is the case, these deficits can be tested with various forms of FLE. Luminance FLE would be better than other forms of FLE because it includes the change of object property only and not direct motion. With these, we can discriminate more clearly whether the autistic/dyslexic people have deficit in delay compensation or just in motion perception itself. Our prediction is that people with autism or dyslexia, with malfunction in delay compensation, will not be able to see the illusion in FLE.

For treatment of these disorders, first, the neurophysiological process that underlies the disorder needs to be identified. Our model suggests that the control mechanism of synaptic efficacy in facilitating synapses may be one major substrate of autism/dyslexia. (For a review and a comprehensive neural model of autism, see [47].) The extrapolation mechanism can be disrupted at least in two ways: (1) a normal extrapolation mechanism is not able to catch up with rapid growth, which results in an abnormal increase in axonal conduction delay; and (2) a malfunction in the extrapolation mechanism itself. One anatomical indication that case (1) above may be the case is that, in autism, the brain grows rapidly [48], [49]: In such a case, the delay caused by a sudden growth in spatial dimension may be more than the delay compensation mechanisms can cope with, thus leading to failure. As for case (2), to our knowledge, there has been no systematic experimental investigation under this perspective. Further physiological tests can assess whether case (2) can indeed be the case.

V. Conclusions

It this paper, we argued that delay compensation mechanisms at the neural level can account for orientation FLE. Going beyond a single neuron in our previous work, we showed that facilitating synapses, if combined with STDP, can allow extrapolation based on cross-neuronal facilitation. Through extrapolating the past to the present, an organism with internal delay can successfully cope with the dynamic environment in real-time, rather than living in the past. Our framework, with the specific neural basis, holds potential for the early detection (and possible treatment) of dysfunctions such as autism and dyslexia, where those affected commonly show error in processing fast-changing stimulus.

Acknowledgment

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