Extrapolative Delay Compensation Through Facilitating Synapses and Its Relation to the Flash-lag Effect

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***Abstract*— Neural conduction delay is a serious issue for organisms that need to act in real time. Various forms of Flash-lag effect (FLE) suggest that the nervous system may perform extrapolation to compensate for delay. For example, in motion FLE, the position of a moving object is perceived to be ahead of a brief flash when they are actually co-localized. However, the precise mechanism for extrapolation at a single-neuron level has not been fully investigated. Our hypothesis is that facilitating synapses, with their dynamic sensitivity to the rate of change in the input, can serve as a neural basis for extrapolation. In order to test this hypothesis, we constructed and tested models of facilitating dynamics. First, we derived a spiking neuron model of facilitating dynamics at a single-neuron level, and tested it in the luminance FLE domain. Second, the spiking neuron model was extended to include multiple neurons and spike-timing-dependent plasticity (STDP), and was tested with orientation FLE. The results showed a strong relationship between delay compensation, FLE, and facilitating synapses/STDP. The results are expected to shed new light on real-time and predictive processing in the brain, at the single neuron level.**

Keywords: delay compensation, dynamic synapses, flash-lag effect, spike timing dependent plasticity (STDP), computational neuroscience

I. Introduction

Flash-lag effect (FLE) refers to the phenomenon in visual (and other forms of) perception where the state of a changing stimulus such as luminance [1], position of a moving object [2], or orientation of a rotating bar [3] is perceived ahead of its current state. Fig. 1–2 illustrate examples of the flash-lag effect. The various perceptual effects relating to FLE are quite extensive, and detailed reviews can be found in [4], [5], [6].

There are several competing theories regarding the underlying mechanism of FLE: extrapolation for delay compensation [3], [7], [8] (see Fig. 1*c*), differential latency [9], [10], [11], [12], temporal averaging [13], [14], and postdiction [15], [16], to list just a few. It seems that not one theory can account for the complete gamut of FLE phenomena, with each theory having its own strengths and weaknesses, and thus the debate is still on-going (see e.g., [4] vs. [5], and [17]). We are inclined toward the extrapolation hypothesis because this account links FLE to behavioral performance (such as interceptive behavior) directly linked to survival, and known limitations can be overcome through mechanisms like backward masking [4] (see Sec. IV for details). For example, our previous computational evolution simulations have shown the effectiveness of extrapolative neural dynamics in delayed control tasks [18], [19].

Our main concern in this paper is about the possible neural mechanisms of extrapolation, in relation to FLE. The precise neural mechanism responsible for such an extrapolatory effect, especially at the single-neuron level, has not been fully investigated. The main hypothesis of this paper is that facilitating synapses [20], [21] help perform extrapolation in order to compensate for neural conduction delay, and FLE is a resulting phenomenon. There are existing previous works on the neural basis of extrapolation. However, they were all based on some form of population code, i.e., the mechanisms were not at a single neuron level. For example, Berry et al. [22] and Fu et al. [23] showed that biphasic temporal responses subsequently read out from a population code can account for perceptual effects similar to the FLE; and Baldo and Caticha [6] demonstrated how the patterns of activation on a feedforward network of leaky integrate-and-fire neurons can predict a wide range of FLE phenomena. In contrast, our focus is on single-neuron mechanisms of extrapolation (and FLE), although the explanatory range of our model is limited compared to the more complex network-level models, e.g., [6]. One thing we wish to mention at this point is that our earlier work on delayed control tasks showed that extrapolation at a single-neuron level can be more resilient than network-level dynamics [18], [19].

In the following, first, we will derive a spike-based single-neuron model for facilitating activity, followed by test results in luminance FLE (Sec. II). The single-neuron-level facilitation model will be extended to cross-neuronal facilitation to account for flash-lag effects that involve a changing stimulus not representable by a single neuron. We will provide details of our model and how cross-neuronal facilitation can be implemented by combining facilitating synapses and Spike-Timing Dependent Plasticity (STDP), and present computational results for orientation FLE (Sec. III). Finally, we will

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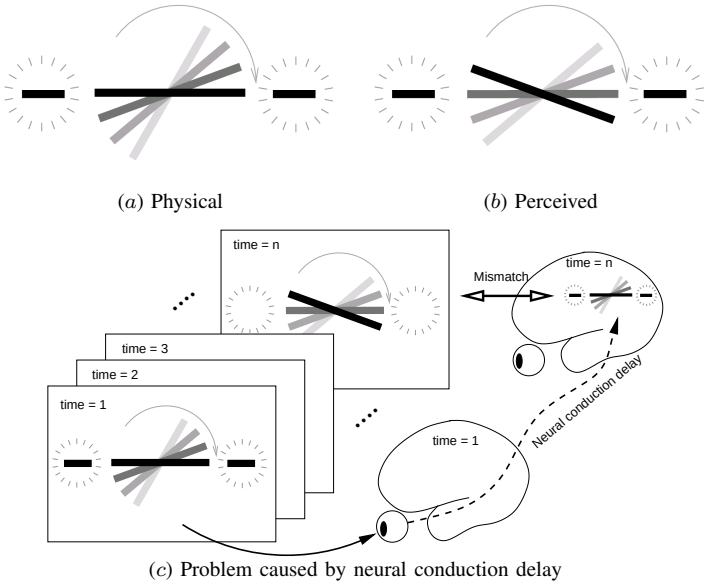


(a) Physical (b) Perceived

(c) Problem caused by neural conduction delay

Fig. 1. **Orientation Flash Lag Effect.** (a) Physical condition: Two flanking bars are flashed when the long rotating bar in the middle is aligned horizontally. The gray scale indicates bar orientation over time (dark = present, light = further into the past). (b) Perceived condition: The rotating bar is perceived to be tilted in the direction of motion. (c) Various events during the perception of the FLE stimulus is shown. At time $t = 1$, the physical stimulus is received at the periphery (retina). By time $t = n$, the horizontally aligned stimulus is received in the central visual area, with a delay. By that time, the environment's state has changed (flashing bars are gone, and the long bar has rotated further). Without FLE, the external state and the internal perception would mismatch (as indicated).

address potential issues in the discussion section (Sec. IV) before concluding (Sec. V).

II. Single-neuron Facilitation Model

In this section, we will describe a spike-based single-neuron model of facilitation grounded in known neurophysiological mechanisms. The main mechanism we will adopt is that of facilitating synapses [20], [21]. In facilitating synapses, the excitatory postsynaptic potential (EPSP) amplitude is dynamically increased when a high-frequency barrage of presynaptic spikes arrive. Such an increased level of activation may implement an extrapolatory function.

Below, we will extend previous models of facilitating synapses by Markram et al. [20], [24], [25] to derive a spike-based model of facilitation. The model will be tested with luminance FLE which is simple enough to be modeled at a single-neuron level.

A. Model description

One potential cellular process that can implement extrapolation in spiking neurons is the synaptic dynamics found in facilitating synapses. These synapses generate short-term plasticity showing activity-dependent decrease (depression) or increase (facilitation) in synaptic transmission occurring within several hundred milliseconds from the onset of activity (for reviews see [26], [27]). Especially, facilitating synapses cause augmentation of postsynaptic response by increasing synaptic efficacy (probability of neurotransmitter release) with successive presynaptic spikes.

According to the dynamic synapse model by Markram et al. [20], [24], [25] synaptic efficacy U of facilitating synapses evolves over time as follows:

$$\frac{dU}{dt} = -\frac{U}{\tau_f} + C(1-U)\delta(t-t_s), \tag{1}$$

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 arrives at the synaptic terminal at time t_s; and $\delta(\cdot)$ the Dirac delta function. This equation is already suitable enough to model the facilitating 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 directions (see Sec. II-B and Fig. 2).

To address this issue, the equation can be altered to make C a dynamic variable which is varied in proportion to the change in input firing rate:

$$C = \mathrm{Sign}\left(I(n-1) - I(n)\right)\left(\frac{I(n-1)}{I(n)}\right) r, \tag{2}$$

where $\mathrm{Sign}(\cdot)$ is the sign function, and $I(n)$ is the interspike interval between the n-th and the $(n-1)$-th spike which reflects whether a spike train consists of high-frequency or low-frequency action potentials. 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 firing frequency, and the third term r is a gain parameter. As the input firing rate increases, C becomes positive and it increases proportional to the rate of change in firing frequency. On the contrary, as the firing rate decreases, $I(n)$ becomes larger which results in a negative C and thus leads to a decrease in the synaptic efficacy U.

With this, we can now fully describe an updated membrane potential model (cf. [20], [24], [28]). The time course of postsynaptic current $P(t)$ at time t triggered by incoming spikes is defined as follows:

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

$$E = wAU, \tag{4}$$

where E is the excitatory postsynaptic potential (EPSP) amplitude; τ_p the time constant of decay in $P(t)$; A a constant for maximum postsynaptic response amplitude; w the weight or scaling factor of A (note that w is constant in a single neuron model); and U the synaptic efficacy as defined above.

Finally, according to a standard leaky integrate-and-fire neuron model [29], the membrane potential $V_m(t)$ at time t can then be calculated as follows:

$$V_m(t) = V_m(t-1)e^{-\frac{t}{\tau_m}} + P(t)(1-e^{-\frac{t}{\tau_m}}). \tag{5}$$

The membrane potential is determined by the membrane current $P(t)$ at time t and the previous membrane potential $V_m(t-1)$, both of which are regulated by a membrane time constant τ_m. The last part of the spiking neuron model is the spike generation mechanism. Once V_m exceeds the spike threshold θ, a spike is generated, and the membrane voltage returned to V_rest (~ -70 mV) after an absolute refractory period of τ_refrac during which spikes cannot be generated.

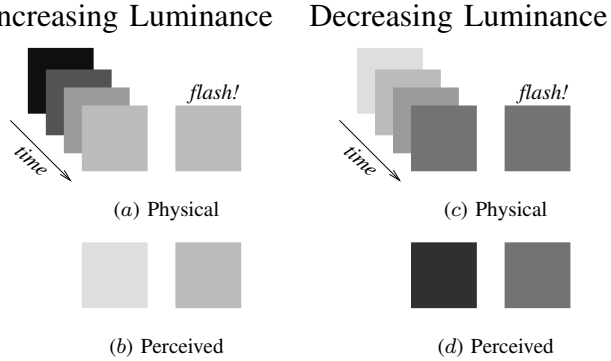


Fig. 2. **Luminance flash-lag effect.** Flash-lag effect is also observed in a spatially stationary time-varying stimulus. A stationary visual patch becomes brighter over time (a, left), and at a random time a patch with equal luminance (a, right) is flashed next to the changing patch. (a) Physically, the two objects have the same brightness. (b) However, the time-varying patch appears brighter than the flashed patch. (c and d) When the patch becomes darker, it is perceived darker than the flashed one.

After the refractory period, membrane potential $V_m(t)$ rises again by facilitated postsynaptic current $P(t)$ and generates subsequent spikes in the postsynaptic neuron.

B. Experiments and results: Luminance FLE

In luminance FLE, a stationary object continuously becoming brighter appears brighter than a neighboring flashed object of equal luminance (and analogously, darker for an object becoming darker) [1] (Fig. 2). It has been shown that neurons in the primary visual cortex respond in a manner correlated with perceived brightness rather than responding strictly to the light level in the receptive field [30], [31]. This finding shows that what we perceive is not the same as the physical stimulus. Such perceptual phenomena in luminance FLE, expressing extrapolation (brighter than bright and darker than dark), can be modeled at a single-neuron level using facilitating synapses.

Sensory signals such as photons hitting the retina are converted into spikes (or action potentials) through sensory transduction. These spikes cause a chain reaction through the sensory pathway to reach the primary sensory area (the primary visual cortex, in case of vision). Let us focus on the last part of the journey of these spikes, where the thalamocortical input spike train releases neurotransmitters from the presynaptic neurons to a postsynaptic neuron through facilitating synapses. Further simplifying this, let us assume that there is only one synapse. With this setup, we can model the extrapolatory phenomenon described above, by varying the spike firing rate in the presynaptic neuron (i.e., the brighter the more action potentials, the darker the less action potentials).

We tested two types of input: (1) increasing firing rate (visual stimulus becoming brighter) and (2) decreasing firing rate (visual stimulus becoming darker). The parameters used for the simulation below were as follows: initial value for synaptic efficacy $U = 0.3$; U-recovery time constant $\tau_f = 220$ ms; postsynaptic potential time constant $\tau_p = 30$ ms; membrane current time constant $\tau_m = 250$ ms; spike threshold $\theta = 175$ mV; $V_{\text{rest}} = 0$ mV; duration of absolute refractory period $\tau_{\text{refrac}} = 5$ ms; maximum postsynaptic response amplitude $A = 300$; and C-gain $r = 0.35$.

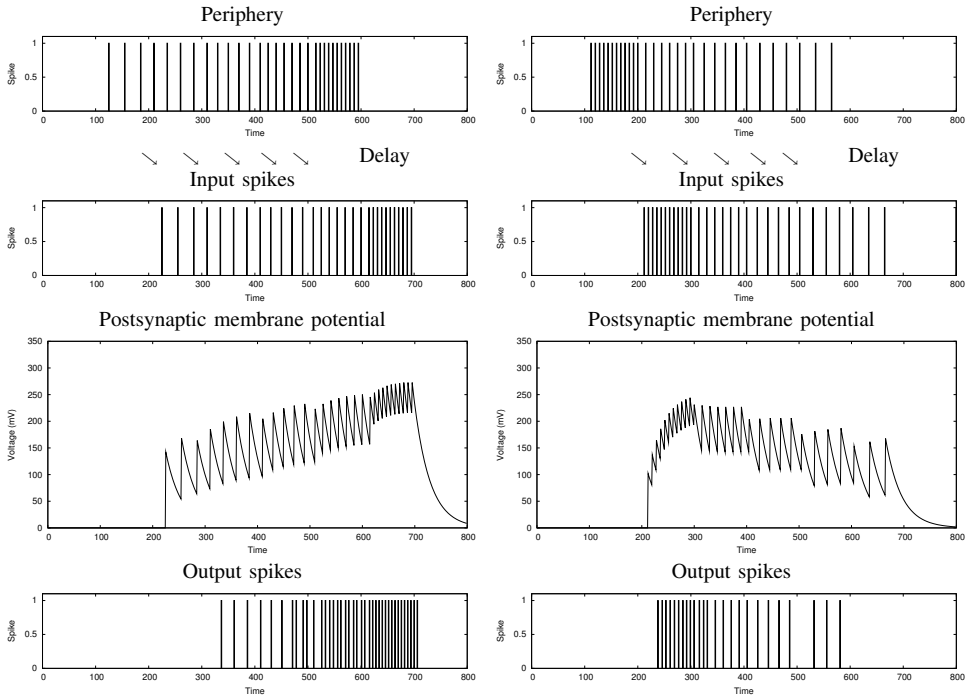
The results are shown in Fig. 3. The facilitating synapse model generated extrapolatory neural activity for both increasing and decreasing firing rate conditions. Dynamic change in the synaptic efficacy U caused the postsynaptic neuron to generate more spikes than the input when the input firing rate was increasing (Fig. 3a). On the other hand, the postsynaptic neuron generated less spikes than what it received when the input firing rate was decreasing (Fig. 3b).

This kind of behavior is quite reasonable if we consider conduction delay. Suppose the spikes in the presynaptic neuron (the second row in Fig. 3a) originated earlier (about 100 ms) in peripheral sensors (the top row). Here is an example sequence of events. (1) Peripheral spiking at 400 ms would be replicated at 500 ms in the presynaptic neuron in the second row, due to the 100 ms delay. (2) The postsynaptic neuron (bottom row) receiving input from the presynaptic neuron (second row) at 500 ms fires based on information from 400 ms in the periphery. (3) However, the postsynaptic neuron's firing rate at 500 ms (bottom row) is the same as that of the presynaptic neuron's firing rate at 600 ms (second row) which represents a peripheral event at 500 ms. This means that the postsynaptic neuron, at time 500 ms, is exactly firing at the same frequency as the peripheral neuron at time 500 ms (refer to (1) above), precisely reflecting the present environmental state. Note that the presynaptic (second row) and the postsynaptic (bottom row) neuron in Fig. 3a are both located in the central nervous system. The changes of firing rates from the three neurons (peripheral, presynaptic, and postsynaptic neuron) are plotted in Fig. 4a. Around 500 ms to 600 ms, the number of spikes in the postsynaptic neuron becomes the same as that of the peripheral neuron.

In case of decreasing firing rate (Fig. 3b), all the experimental conditions and notations were the same as the increasing case except that the input spike frequency was decreasing. According to Eq. 1 and Eq. 2, the constant C for determining the increase in synaptic efficacy becomes negative which leads to a gradual decrease in membrane potential of the postsynaptic neuron. The third row in Fig. 3 shows the decreasing membrane potential, and the bottom row the output spikes generating fewer spikes than that of the input spikes. As shown in the increasing case, the postsynaptic neuron's firing rates are closely matched with those of the peripheral neuron in spite of neural delay. (See Fig. 4b for the precise firing rate from the three neurons.)

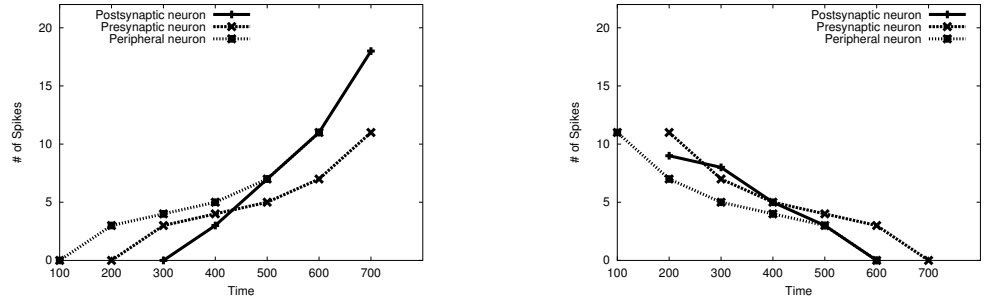
In contrast with the previous models by Markram et al. [20], [24], [25], the modified equation (Eq. 1 and Eq. 2) was able to generate extrapolated neural activity under decreasing, as well as increasing, firing rate conditions. These experiments suggest that facilitating synapses can implement a general extrapolatory neural mechanism.

In sum, the time-varying stationary stimuli (brighter or darker in luminance) were extrapolated to be perceived close to the present luminance, while that of the abrupt stimulus (i.e., flashed patch) was the same as the luminance 100 ms in the past (no time for facilitation to build up). Such a difference in the perceived luminance of the two stimuli might cause FLE. With such a mechanism, organisms can keep the inner state synchronized with that of the physical environment in



(a) Increasing firing rate (b) Decreasing firing rate

Fig. 3. **Extrapolation with facilitating synapses under varying firing rate.** (a) Activation in neurons with facilitating synapses are shown, where a sequence of input spikes shows an increasing firing rate. The top row shows the input spike sequence generated at a peripheral neuron, the second row the input spikes arrived at a presynaptic neuron with neural delay (around 100 ms), the third row the membrane potential of the output neuron, and the bottom row the output spike sequence. The output firing frequency from 400 ms to 600 ms (bottom row) are closely matched with those of the presynaptic neuron's firing rate from 500 ms to 700 ms (second row), and in turn those of the peripheral neuron's firing rate from 400 ms to 600 ms (top). (See Fig. 4a for the exact number of spikes.) This shows that, in spite of the delay ($\Delta t = 100$ ms), the output firing rate at a certain moment exactly reflects the input firing rate in the periphery at that instant and thus help the internal state to be in synchrony with the external state. (b) The same experiment as in (a) except that the input firing rate was decreased. Decreasing firing frequency in the input causes facilitating synapses to decrease its synaptic efficacy and make the postsynaptic neuron generate less spikes than the input spikes. The overall effect is analogous to that of (a).



(a) Number of spikes for increasing luminance (b) Number of spikes for decreasing luminance

Fig. 4. **Change of firing rates in luminance FLE.** The change of firing rate over time is shown for (a) increasing luminance and (b) decreasing luminance. The x axis represents time intervals (e.g., 200 denotes from 100 ms to 200 ms) and the y axis the number of spikes generated during that time frame at peripheral (dotted line with '$*$'), presynaptic (dotted line with 'x'), and postsynaptic neuron (solid line with '+'). (a) In the increasing luminance experiment, the postsynaptic neuron needed some boosting time for facilitatory activity (around 100 ms) and started to generate more spikes than the presynaptic neuron after 400 ms, showing the same firing rate as that in the peripheral neuron at 500–600 ms. (b) In the decreasing case, the postsynaptic neuron generates fewer spikes than the presynaptic neuron, showing the same firing rate as the peripheral neuron after 400 ms.

real time.

III. Cross-neuronal Facilitation Model

In the previous section, we showed that facilitating synapses can function as an extrapolator to undo the effect of neural delay, thus giving rise to FLE. However, the same approach cannot be applied to other forms of FLE such as orientation FLE (Fig. 1), since unlike luminance the full range of orientation cannot be represented by a single neuron. Changing the firing rate in a single neuron 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 [32] for a review).

To address this issue, we extended our spike-based facilitation model to include multiple neurons, and allowed facilitation to go across neurons. We found that mechanisms such as Spike-Timing-Dependent Plasticity (STDP) [33], [34] are necessary to set up the connection strengths, so that

cross-neuron facilitation can carry out extrapolation in the direction of change. STDP refers to a synaptic plasticity mechanism where the order of presynaptic and postsynaptic spike determines the sign of the adaptation: either increased or decreased synaptic efficacy. STDP favors the causal direction, as postsynaptic firing after presynaptic firing would increase the synaptic strength.

In this section, we will first provide details of our model and explain how cross-neuronal facilitating dynamics can be implemented by including STDP, and test the model with orientation FLE.

A. Model description

In order to represent the full range of orientation, we modeled twelve orientation-sensitive neurons at a 15$^{\text{o}}$ increment. Fig. 5a shows the arrangement of the neurons, and their lateral excitatory connectivity (connections are only between immediate neighbors). Each neuron fires according to a distribution centered around its preferred orientation, maximally firing when the preferred orientation is present in the input, and gradually less as the input veers away (Fig. 5b, "No FLE"). Note that during orientation FLE, the distribution shifts toward the direction of rotation (Fig. 5b, "FLE"). Each neuron has a facilitating dynamic, and the lateral connections are trained using STDP.

To allow for cross-neuronal interaction, we extended the single neuron model in Sec. II-A to include synaptic input from neighboring neurons, as shown in Fig. 5a. Each cell in Fig. 5a 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 activity propagates to neighboring neurons and increases the postsynaptic neurons' responses. At the same time, the connection weights w are updated through STDP. Note that cross-neuronal facilitating dynamic follows the single facilitation dynamics as described in Sec. II-A. The only difference here is that w in Eq. 4 is a variable, not a constant. As a result of STDP, 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. 5a) are initially directionally symmetric, meaning that there is no preferential activation in the clockwise or the counter-clockwise direction. However, for extrapolation to happen in a particular direction, there needs to be directionality. A learning process that gives rise to such a 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 [33], [34]. When the presynaptic neuron fires first (firing time t_{pre}), and then the postsynaptic spike (firing time t_{post}), then the difference $\Delta t = t_{\text{post}} - t_{\text{pre}} > 0$, since $t_{\text{post}} > t_{\text{pre}}$. 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 synaptic modification function in [34]:

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

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

$$w_{ij} \leftarrow w_{ij} + \alpha F(\Delta t); \tag{7}$$

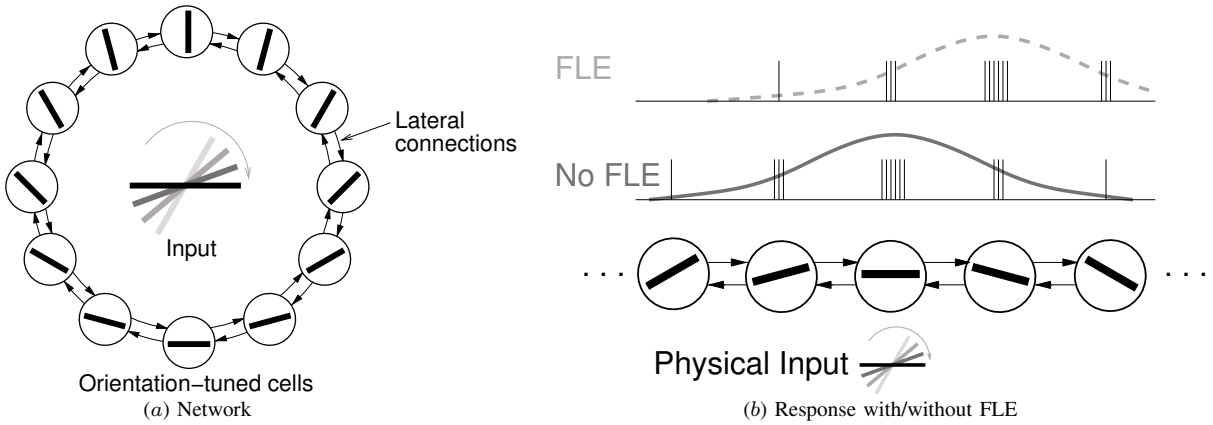
where α is the learning rate ($= 0.02$) and $\Delta t = t_i - t_j$. In case w_{ij} reached zero, it was not decreased any further.

Fig. 6 in the next section shows the evolution of the connection weights toward or against the 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 fires, 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 STDP adaptation window ($\approx \pm 80$ ms [36]). 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 to the timescale of orientation FLE. For example, as the orientation of the stimulus sweeps by at 100 ms per neuron (i.e. 25 rotations per minute (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 80 ms) and Δt will fall within the STDP range. Under this condition, the synaptic weights between the neurons can be adapted.

C. Experiments and results: Orientation FLE

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. 5a. The experiments were carried out in the orientation FLE domain, illustrated in Fig. 1. We conducted three separate experiments: orientation perception with (1) STDP only, (2) facilitating synapses only, and (3) both STDP and facilitating synapses. 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 1 spike/10 ms.

1) STDP only: First, we tested our model with only STDP, without facilitating synapses. The connection weights were allowed to adapt according to Eq. 7. Fig. 6 shows the results. For those connections pointing toward the direction of input rotation, the weight increased (Fig. 6, solid curve) since presynaptic spike preceded postsynaptic spike within the small time interval. On the other hand, for the connections pointing



(a) Network (b) Response with/without FLE

Fig. 5. **Bilaterally connected orientation-tuned cells and FLE.** (a) 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 [35], [32]. (b) 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 in (a). Without FLE (solid), the firing-rate distribution is centered around the middle, coinciding with the physical input shown below (cf. Fig. 1a). With FLE (dashed), the distribution is shifted toward the direction of input rotation (cf. Fig. 1b).

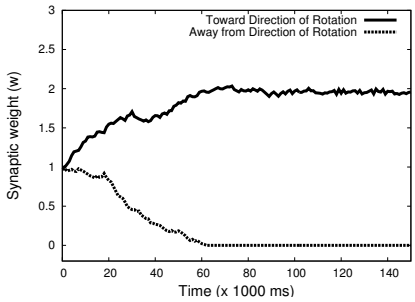


Fig. 6. **Adaptation of synaptic weights using STDP.** The synaptic weights w (y-axis) in the connections from neuron 2 to its two immediate neighbors are shown over time (x-axis). The weight to the neuron in the direction of input rotation increases (solid curve), while that in the opposite direction decreases (dotted curve).

in the opposite direction, the weight decreased (Fig. 6, dotted curve).

To calculate the firing rate at any given moment, we used a fixed sliding window with a width of 100 ms. We measured the firing rate when the input was oriented to optimally stimulate neuron 2. Fig. 7a 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, which is 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.

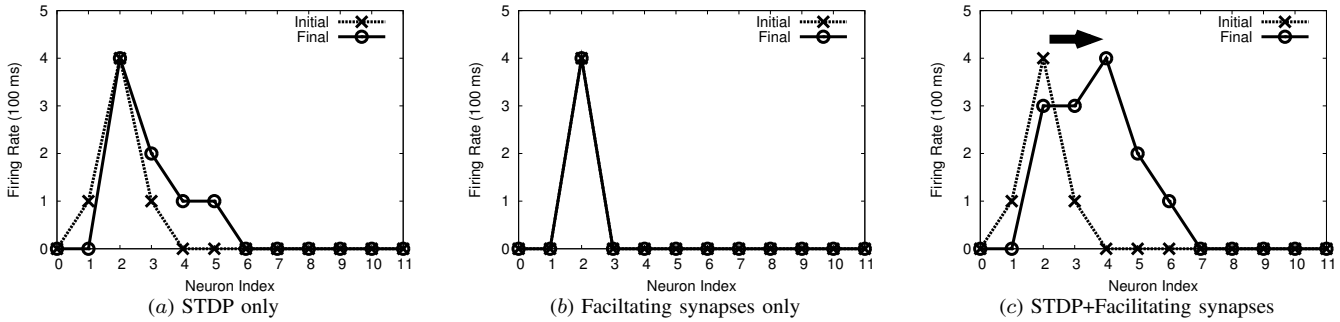
2) Facilitating synapses only: In this experiment, we used only facilitating synapses, without STDP. All weights were initialized to 1.0, and remained fixed throughout the experiment. Since the weights did not have any directionality, we expected that no orientation FLE would occur, which turned out to be the case. The only changing quantity in this experiment was the synaptic efficacy in the facilitating synapses (Eq. 1). As a result, 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. 7b). The results suggest that, again, orientation FLE did not occur, and facilitating synapses alone are not enough to account for cross-neuronal facilitation (or extrapolation across neurons).

3) STDP with facilitating synapses: The final experiment combined STDP and facilitating synapses. Other experimental conditions were the same as in the two experiments above for the STDP and the facilitating synapse parameters. 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. The gradually increasing long-term trend in STDP weight amplified the rapidly changing facilitating dynamics. Fig. 8a–b shows that the adaptation of synaptic efficacy from the initial state (Fig. 8a: no directionality) to the final state (Fig. 8b: with directionality).

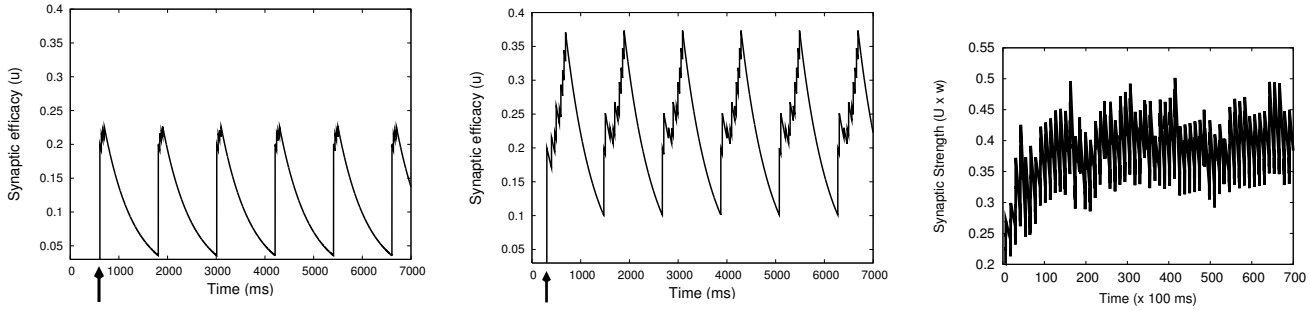
The combined effect of these two factors (STDP and facilitating synapses) over time is shown in Fig. 8c. STDP gives the model directionality, and facilitating dynamics provides extra influence in the direction of input rotation, thus allowing the model to shift its firing rate distribution significantly. Fig. 7c shows that the peak in the firing rate distribution shifts toward the direction of input rotation (to the right) by two steps (neuron 4), when the actual input at that moment optimally stimulates neuron 2. This shift (indicated by the arrow) suggests that orientation FLE occurred.

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 giving rise to orientation FLE. See Table I for a summary of experiments, results, assumptions, and proposed neural bases.



(a) STDP only (b) Faciltating synapses only (c) STDP+Facilitating synapses

Fig. 7. **Firing rate distributions.** Firing rate distributions under different experimental conditions are shown. (a) Results from the STDP-only simulations are shown. The dotted line with '$\times$' represents the initial firing rates of the neurons. The solid line with '$\circ$' 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 during a 100 ms interval) when the actual input orientation was neuron 2's preferred stimulus. With STDP, the firing rates of the neurons were skewed toward the direction of rotation due to the synaptic weight modification. However, the maximally firing neuron (neuron 2) did not change (i.e., no FLE). (b) Results from the facilitation-only experiments are shown (the plotting conventions are the same as in a). Because the synaptic efficacy U is rapidly changed by a small amount, 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) Results from the STDP+facilitation experiments are shown (the plotting conventions are the same as in a). The distribution of firing rates significantly shifted toward the rotating direction (as indicated by an arrow). The position of maximally firing neuron changed from neuron 2 to neuron 4. After training with STDP, and facilitating synapses added, when the neuron 2's preferred orientation was given in the input, 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.



(a) Initial U (b) Final U (c) Evolution of $U \cdot w$

Fig. 8. **Adaptation of synaptic efficacy through STDP + facilitating synapses.** The evolution of synaptic efficacy U in a single neuron is shown. (a) Neuron 2 shows rapid increase of synaptic efficacy during the presence of a preferred input orientation. (b) After training under STDP + facilitation synapses, the dynamics of synaptic efficacy highly increases. Note that synaptic efficacy starts boosting up earlier than in the initial state (compare the parts indicated by arrows). The strengthened connections allowed the neighboring neurons in the direction of rotation to generate predictive spikes. (c) Adaptation of synaptic strength ($U \cdot w$ in Eq. 4) through STDP plus facilitating synapses (along the direction of rotation) is shown. Synaptic strength ($U \cdot w$) dynamically increases because of the gradually increasing synaptic weight w and the fast changing synaptic efficacy U.

TABLE I

SUMMARY OF ASSUMPTIONS AND RESULTS

Experiments	Results	Assumptions	Neural basis
Luminance FLE: increasing luminance	Higher output firing rate than input firing rate for increasing luminance input	Facilitation is needed for single-neuron-level extrapolation	Facilitating synapses [20]
Luminance FLE: decreasing luminance	Lower output firing rate than input firing rate for decreasing luminance input	The factor C in the facilitating synapse equation (Eq 1) is a variable quantity depending on the history of input spikes	Facilitating synapses [20], with modifications (Sec. II-A)
Orientation FLE	Shifted response distribution toward the direction of rotation	Facilitation, as well as STDP, is needed for cross-neuronal extrapolation	Facilitating synapses [20] and STDP [33], [34]

IV. DISCUSSION

A. Contributions

The main contribution of this paper is to relate facilitating synapses, delay compensation (through extrapolation), and FLE; thus linking neurophysiology, computational function, and psychophysics. Unlike previous models of FLE and related phenomena [22], [23], [6], our model provides extrapolation at a single-neuron level. Another contribution of our work is that we have shown STDP alone may not be sufficient for effective extrapolation/delay compensation, and facilitating synapses can again play an important role (cf. Rao and Sejnowski [37] where STDP alone was proposed as a predictive mechanism).

B. Notes on Model Parameters

In our paper, the values of synaptic parameters were tuned, within reasonable range, to derive the desired behavior. All the parameter values for the facilitating synapse model were within the range of empirical data found in neurophysiology [24], [20], [38]. We also referenced STDP related literatures [34], [39], [40] to set the STDP model parameters, again within in known ranges.

C. Limitations

Being an extrapolation model of FLE, our approach suffers from the same potential shortcomings of Nijhawan et al.'s extrapolation theory [4]. As pointed out by Krekelberg and Lappe [5], the extrapolation theory's weakness is in not being able to account for (1) the lack of FLE in motion termination and motion reversal [11], and (2) reversal of FLE in low-luminance condition (low luminance supposedly causes longer latency, thus a stronger FLE is predicted by the extrapolation theory) [10] (also see [14]). Nijhawan proposed a backward-masking solution for issue (1) [4] (also see the next paragraph), however, it is unclear so far how issue (2) can be resolved. One potential explanation could be related to facilitating dynamic's dependence on high-frequency input [27]. Under low luminance, firing rate may become generally lower, and the extrapolation mechanism in facilitating synapses may fail (i.e., no FLE). It would be interesting to find the perceptual (luminance) threshold where FLE disappears, and the firing rate at which facilitating dynamic disappears and find any correlation between the two. One interesting behavior we observed in our model in relation to the above discussion is when the input firing rate was lowered in the orientation FLE experiment (e.g. less than 10 spikes per 100 ms). Note that 1 spike/10 ms was used in the other experiment in Sec. III-C. As the input spike frequency decreases (which can happen when the oriented bar gets fatter or thinner than the optimal thickness [41]) the shift in the firing rate distribution was reduced (i.e., weaker or no FLE). The reason is that to make facilitating dynamics to be activated, the neuron should receive high frequency input spikes [27]. Results show that the adaptation of synaptic efficacy through STDP and facilitating synapses under low firing rate (7 and 5 spikes/100 ms) is significantly reduced compared to the high firing rate condition. With the same connections set up through STDP, suboptimal input could not invoke dynamic change of synaptic efficacy, i.e., it was not enough to shift the firing rate distribution (Fig. 9).

Another complex issue arises when we consider the variability in response latency. For example, the latency between stimulus onset and neural response in various loci in the visual pathway are known to show a range of variability [42]. If this is the case, compensation for such latency may become very difficult, since at every moment an accurate estimation of the latency needs to be in hand, which may be hard to obtain. One counter argument to this is that in most cases, the latency variability is exhibited at the population level, thus the latency may be fixed at a single-neuron level, and in this case it could be compensated. However, it turns out that latency variability exists even at the single-neuron level, for example

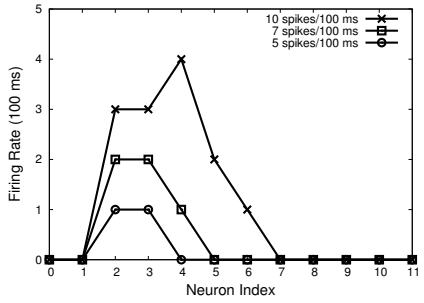


Fig. 9. **Change in the distribution of firing rate depending on the input frequency.** The effects of decreasing the input spike frequency (e.g., fatter input bar) on the response distribution are shown. As the number of input spikes decreases (line with '×' represents 10 spikes/100 ms, □ for 7, and ○ for 5 spikes/100 ms), the shift of firing rate distribution is reduced (i.e., weaker FLE). Also, the overall magnitude of firing rate decreases as the input frequency gets smaller.

in the simple cells in the primary visual cortex [43]. One possibility is that such response latency is mostly due to the variability in the integration time in the membrane potential (see [44]), and not in the axonal conduction delay (which we are trying to compensate). In this case, the relatively fixed axonal conduction delay will dictate the mean of the latency distribution (which can be compensated), and the integration time variability will account for the variance (which may be hard to compensate). More theoretical investigation is needed to unequivocally determine if compensation can be done for such a variable component in the latency.

Finally, our model of FLE introduced in this paper is purely bottom-up. A question here is whether such a model should include top-down influence. In top-down processing, a perceiver's concept and higher level knowledge also influence the visual perception. Eagleman et al.'s approach (postdiction) can be thought of as a first step toward top-down processing [15]. Their main idea is that visual perception is not only feedforward processing: Current visual information can be corrected by future information and the result of integration is what we perceive. It would be interesting to extend our low-level neuronal model to include higher level feedback which can account for such an integration process. For example, backward masking suggested by Nijhawan [4] can come from higher level intervention.

D. Relation to Other Works

The view presented in this paper provides new insights on an alternative role of facilitating synapses—that of extrapolatory delay compensation. Existing theories regarding facilitating synapses included sensitization and habituation [45], [46], or temporal information processing [24], [20], [27], [47], [48]. Synaptic plasticity (including facilitating synapses) are typically related to memory functions, but our results indicate it could be more about prediction. However, note that these views are ultimately compatible: As Hawkins argued, memory may be explicitly linked to predictive functions [49].

There are various existing theories on the role of STDP. For example, STDP is supposed to help reduce spike train variability [50]. It was also shown that STDP may be involved

in topology-preserving mapping [51]. Others suggested STDP as a neural mechanism for cortical orientation tuning [52]. The perspective closest to ours is that of Rao et al. [37]: They argued that STDP may implement a predictive function, where what is to occur next in an input sequence is predicted. (See [49] and [53] for a general discussion of the role of prediction in brain function.) However, they did not relate this predictive ability to the idea of delay compensation. From our perspective, these predictive mechanisms may have started out as prediction of the present, not prediction of 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.

Our dynamic account of extrapolation and the proposed facilitating dynamics is interestingly related to perceptual phenomena due to spatial extrapolation on static stimuli. For example, Changizi presented a theory that angle misperception in static images is due to what he calls 'perceiving the present' [54]. The basic argument is that what we perceive is what the stimulus would look like right now in an 'ecological' context (e.g., as if the perceiver has been moving forward), given the input stimulus from a short while in the past (due to conduction delay). Although the end result looks like there is spatial extrapolation (since you are only looking at a static stimulus), the whole idea depends on a more dynamic argument (movement). Only through a history of movement in the past, such extrapolations could be set up. The work we presented in Sec. III-C with its cross-neuronal facilitation may be closely linked to Changizi's account. Mechanisms like STDP can set up the causal link, and facilitating synapses can carry out the predictive projection of activity through a previously exercised motor-induced perceptual change cycle (see [55] for how the thalamocortical loop can play a role in such a predictive preactivation). It is no coincidence that Changizi's framework is called 'perceiving the present'. Similar arguments can link our dynamic account of extrapolation and other extrapolative phenomena involving discretely presented sequences of stationary stimuli, such as representational momentum [56] (see [57] for a review).

Finally, we would like to compare our model with that of Baldo and Caticha [6] which seems to be the most comprehensive neural model of FLE. Their model consists of three layers (input, hidden, and output) of leaky integrate and fire neurons, with on-center/off-surround receptive fields. Thus, their model includes inhibitory connections, as well as excitatory connections. Also, they have facilities for attentional modulation by forceful activation in the output layer targeted at the locus of attention. Their model is capable of replicating (1) the standard FLE, (2) Frölich effect ([58]; when a moving object appears abruptly, the initial segment of the object's trajectory is not perceived), (3) luminance-ratio-dependence of FLE, (4) speed-dependence of FLE magnitude, (5) flash-initiated cycle, (6) flash termination and flash reversal, (7) absence of Frölich effect when flashed object is shown, removed, and shown again, and (8) attentional modulation of FLE. Our current model can deal with (1), (2), (4), and (5). With the addition of inhibitory influence, effectively a backward masking influence proposed by Nijhawan [4], we expect our model can account for (6). (Note that our model only has excitatory influence.) If we add forced attentional activation as in Baldo and Caticha's experiments, we expect our model can also handle (8). Our model cannot physically represent the stimulus condition of (3), and we are not certain how our model will behave in case (7). In sum, considering that our model does not have additional features like inhibitory connections and forced attentional activation, it fares well compared to Baldo and Caticha's model.

E. Predictions

We expect that our approach can be extended to explain other extrapolatory phenomena such as flash-lag effects in color, pattern entropy, or localization. Moreover, we expect that the extended cross-neuronal facilitation model may be able to account for other visual illusions such as the Fröhlich effect. Note that the facilitating mechanism generated the first output spike train after a certain amount of delay (see Figures 3a and 3b, bottom rows): In addition to the neural transmission delay from peripheral neurons to visual cortex neurons, the postsynaptic neuron needed boosting time to respond to the initial input spike train. This is consistent with the Fröhlich effect [59], in which the moving object may be invisible during the initial period due to the silence of neurons.

Another prediction is that the time-frame for orientation FLE should be within the time-frame of the STDP adaptation window, as suggested in Sec. III. This is because STDP needs to set the directionality for the propagation of facilitating activity, and synaptic change can only happen when the pre- and post-synaptic neurons fire within the STDP window. Also, in relation to the orientation FLE experiments, the results presented in Fig. 9 forms a direct prediction regarding suboptimal orientation stimuli, such as fatter or thinner input.

Our model makes neurophysiological predictions as well. If facilitating synapses indeed function as delay compensators, facilitating synapses should be found more often in places where delay compensation is needed more, for example, at the end of long, slow axons, or where precise real-time information is needed. Also, the term C defined in Eq. 2 could change over time, unlike the original model first proposed by Markram et al. [20] where it is constant. There are some clues that suggest this could be the case: It is known that facilitation is driven by elevated calcium levels in presynaptic terminals. Recent neurophysiological findings such as the involvement of calcium chelator BAPTA, playing a role of Ca^{2+} buffer [60], can be one potential basis of the variance in C.

V. Conclusion

In this paper, we have shown that dynamics found in facilitating synapses can serve as a neural basis of extrapolation. Experiments with our spike-based facilitating model in luminance FLE showed that facilitating postsynaptic activity can generate extrapolated neural activity under both increasing and decreasing firing rate conditions. Going beyond the single neuron model, we showed that facilitating synapses, if

combined with STDP, can allow extrapolation based on cross-neuronal facilitation and may give rise to orientation FLE. In sum, we showed that facilitating synaptic dynamics can serve as a delay compensation mechanism, which can give rise to the various flash-lag effects, and help biological organisms to perceive the present environmental state in real time. The single-neuron-level extrapolative mechanism proposed in this paper have general utility beyond the flash-lag effect, and we expect the framework to be more widely applied in interpreting experimental data and in engineering applications (e.g., as in [61]).

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