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Spike propagation synchronized by temporally asymmetric Hebbian learning

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Abstract. Synchronously spiking neurons have been observed in the cerebral cortex and the hippocampus. In computer models, synchronous spike volleys may be propagated across appropriately connected neuron populations. However, it is unclear how the appropriate synaptic connectivity is set up during development and maintained during adult learning. We performed computer simulations to investigate the influence of temporally asymmetric Hebbian synaptic plasticity on the propagation of spike volleys. In addition to feedforward connections, recurrent connections were included between and within neuron populations and spike transmission delays varied due to axonal, synaptic and dendritic transmission. We found that repeated presentations of input volleys decreased the synaptic conductances of intragroup and feedback connections while synaptic conductances of feedforward connections with short delays became stronger than those of connections with longer delays. These adaptations led to the synchronization of spike volleys as they propagated across neuron populations. The findings suggests that temporally asymmetric Hebbian learning may enhance synchronized spiking within small populations of neurons in cortical and hippocampal areas and familiar stimuli may produce synchronized spike volleys that are rapidly propagated across neural tissue.

1 Introduction

The average firing rate of a neuron is often considered the primary measure of its activity. Recent studies have reported that spike synchronization occurs in cortical neurons with millisecond precision and is modulated by task conditions (Riehle et al. 1997; Prut et al. 1998; but see Oram et al. 1999; Steinmetz et al. 2000; Fries et al. 2001; Salinas and Sejnowski 2001). Since several presynaptic spikes are usually required to produce a postsynaptic spike, synchronous spikes in postsynaptic neurons may be elicited by synchronous spikes in a population of presynaptic neurons (Salinas and Sejnowski 2000). It has been speculated that this sequence is repeated and that neuron populations can propagate synchronous spikes (Abeles 1991). A similar hypothesis was proposed for hippocampal place cells, where spike synchronization within tens of milliseconds was reported for place cells that code for the same location of the animal (Skaggs et al. 1996). Such spike synchronization occurs in pyramidal place cells of CA3 and CA1 regions as well as in other stages of hippocampal processing (O'Keefe and Recce 1993) and seems to be propagated across these processing stages (Skaggs et al. 1996).

Simulated networks consisting of integrate-and-fire neurons propagate synchronized spike volleys across neuron populations without loss of synchrony (Herrmann et al. 1995; Diesmann et al. 1999). However, these networks require exclusive feedforward connections between neuron populations, which seems to be inconsistent with cortical and hippocampal anatomy. Feedback and intragroup connections are likely to lead to recurrent excitation and would thereby disperse synchronous activity. In addition, these simulation studies did not take into account that axonal spike propagation delays between pyramidal neurons vary considerably, which would further disperse spike volleys. What mechanisms could shape neural networks to make propagation of synchronous spike volleys possible?

We investigate here the role of this temporally asymmetric Hebbian learning in sharpening spike synchronization. Temporally asymmetric Hebbian learning is characterized by long-term potentiation (LTP) if a presynaptic spike precedes a postsynaptic spike within a brief time window or by long-term depression (LTD) if the temporal order of the spikes is reversed (Fig. 1a). These mechanisms for synaptic plasticity have been observed in connections between cortical and

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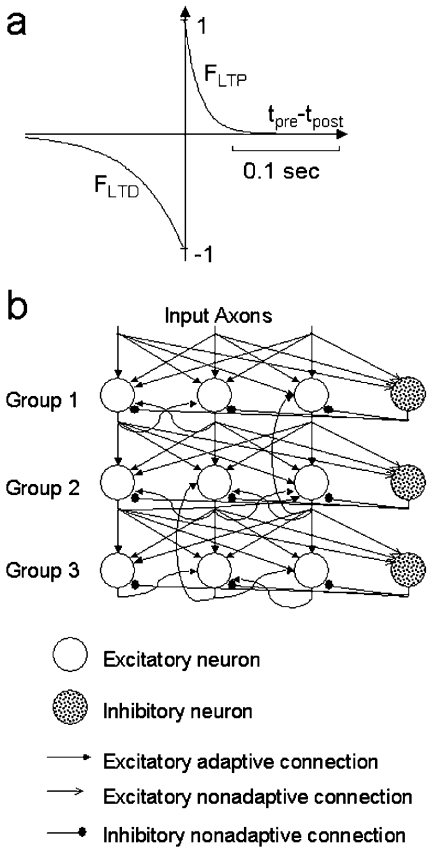

Fig. 1. a Temporally asymmetric Hebbian learning. A synapse is potentiated if the presynaptic spike precedes the postsynaptic spike ($t_{\text{pre}} - t_{\text{post}} > 0$) and depressed if the presynaptic spike follows the postsynaptic spike ($t_{\text{pre}} - t_{\text{post}} < 0$). Modification functions F_{LTP} and F_{LTD} are shown versus the arrival time difference between the presynaptic and the postsynaptic spike. **b** Network architecture. The networks consist of 45 excitatory and 9 inhibitory integrate-and-fire neurons that are subdivided into three groups with 15 excitatory and 3 inhibitory neurons within each group (not all neurons are shown). The axonal propagation delays between groups randomly vary between 4 ms and 14 ms, and the axonal propagation delays within groups are 4 ms. The excitatory projections to excitatory neurons are adapted according to temporally asymmetric Hebbian learning (Sect. 2)

hippocampal pyramidal neurons (Markram et al. 1997; Bi and Poo 1998; Debanne et al. 1998; Feldmann 2000). To investigate how temporally asymmetric Hebbian learning affects synchronous spikes, we have simulated the propagation of spike volleys in a network of integrate-and-fire neurons with feedforward, feedback, and intragroup connections and with randomly varying connection delays (Fig. 1b). We then show that the adaptation of the excitatory conductances produces spike synchronization within small neuron populations.

2 Methods

2.1 Neuron model

The complete network consists of leaky integrate-and-fire neurons with excitatory neurons (AMPA synapses) and inhibitory neurons (GABA-like synapses). The membrane potential, $V(t)$, of each neuron is computed with

$$C\frac{\mathrm{d}V}{\mathrm{d}t} = g_{\text{leak}}(V_{\text{rest}-V}) + I_{\text{AMPA}} - I_{\text{GABA}} + I_{\text{noise}} \ ,$$

with a resting membrane potential $V_{\text{rest}} = -74$ mV, leak conductances $g_{\text{leak}} = 25$ nS for excitatory and 18 nS for inhibitory neurons, membrane time constants $\tau = 20$ ms (excitatory) and 12 ms (inhibitory), and a membrane capacity $C = \tau g_{\text{leak}}$. When the membrane potential $V(t)$ reaches a threshold of -54 mV, a spike is generated and the voltage is reset to -60 mV (Troyer and Miller 1997). The duration of an absolute refractory period is 2 ms. The term I_{noise} represents influences of neurons that are not explicitly simulated and is computed as a shot noise with a mean value of 408 pA with a standard deviation of 60 pA (Mainen and Sejnowski 1995). For each synapse, the time course of the synaptic current is modeled with

$$I_{\text{syn}}(t) = -g(t)(V_{\text{syn}} - V(t)) \sum_k \exp(-(t - t_k)/\tau_{\text{syn}}) \ ,$$

where t_k denotes the arrival time of the kth presynaptic spike. A synaptic reversal potential V_{syn} is set to zero for excitatory and to -75 mV for inhibitory synapses. A time constant τ_{syn} is set to 3 ms for all synapses (Spruston et al. 1995). The variable $g(t)$ represents the maximal conductance of a connection. For the chosen parameters, a conductance of 2 nS for an excitatory connection provides, for one presynaptic spike, a voltage increase of about 1 mV in the postsynaptic neuron. The values of the initial conductances are set similar to measured values and tuned in exploratory simulations. The initial conductances, $g(t)$, for excitatory connections are selected from a Gaussian distribution with a mean of 1.8 nA and a standard deviation of 0.6 times the mean conductance (Markram et al. 1998). The conductances are limited to values below 2.7 times the mean conductance (Markram et al. 1998). For inhibitory connections, the conductances are set to 8 nS (Gupta et al. 2000).

Long-term adaptation of connection conductances according to a temporally asymmetric Hebbian rule appears to occur between excitatory cortical and striatal neurons (Markram et al. 1997; Bi and Poo 1998; Debanne et al. 1998; Feldmann 2000; but see Tao et al. 2000) and is implemented by adapting the connection conductances according to experimental findings in the hippocampus (Selig et al. 1999). As in previous work (Song et al. 2000; Rubin et al. 2000; van Rossum 2000), the amplitude of LTP or LTD depends on the time difference between presynaptic and postsynaptic spike arrivals and is computed using the modification functions

$$F_{\text{LTP}}(t) = \sum_k \exp(-(t - t_k)/\tau_{\text{LTP}})$$

$$F_{\text{LTD}}(t) = -\sum_m \exp(-(t - t_m)/\tau_{\text{LTD}})$$

where t_k and t_m denote the arrival times of the kth presynaptic and mth postsynaptic spike, respectively ($t - t_k \geq 0, t - t_m \geq 0$). Decay times $\tau_{\mathrm{LTP}} = 20$ ms and $\tau_{\mathrm{LTD}} = 60$ ms are estimated from experimental data measured in the cortex (Feldman 2000) and hippocampus (Bi and Poo 1998; Debanne 1998).

Following Rubin et al. (2000), we assume that a postsynaptic spike induces a change in the conductance that is proportional to the difference between the current conductance and the maximal conductance:

$$\Delta g(t) = \eta((g_{\max} - g(t))F_{\mathrm{LTP}}(t)) \ ,$$

whereas a presynaptic spike induces a change

$$\Delta g(t) = \eta g(t) F_{\mathrm{LTD}}(t) \ .$$

The learning rate, η, is set to a value of 0.18.

2.2 Network connectivity

The complete network model consisted of repeated neuron populations with each group containing 15 excitatory and three inhibitory neurons (Fig. 1b). To guarantee propagation of the first input volley, all excitatory neurons in a group projected to all neurons in the following group. The probability for feedback and intragroup connections between excitatory neurons was 18%. All connections between excitatory neurons were adaptive, whereas the other connections were not adaptive. As the distance between neuron populations is thought to be larger than that within neuron populations, the transmission delays of feedforward and feedback connections were randomly selected from a uniform distribution between 4 and 14 ms, and the connection delays within populations were set to 4 ms. The selected transmission delay range is similar to that of delays between spikes of CA3 neurons and EPSP onsets in CA1 neurons (Debanne et al. 1996). For cortical horizontal axons with transmission velocities in the order of 0.1 mm/ms (Bringuier et al. 1999), transmission delays correspond to axons of up to about 1 mm length. To be consistent in each of the repeated neuron populations, the input axons were given the same delays and adaptation properties as the other feedforward projections.

Inhibitory neurons project to all excitatory neurons within the same population. Since the modeled inhibitory neurons are assumed to project only locally, they did not project outside of their neuron group. Therefore, excitatory responses to an input volley should arrive several milliseconds before inhibitory responses to the same volley as is consistent with experimental findings (Volgushev et al. 1993).

2.3 Newtwork input

The input axons are activated with 20 spike volleys at a frequency of 10 Hz. For each input volley, spike times are randomly selected from a Gaussian distribution with a standard deviation of 10 ms. The maximal dispersion of input spike volleys is then limited by setting spike time variations exceeding 25 ms to this maximal value. One spike is presented to each input axon per volley. In addition to these spike volleys, input axons are activated at random times with an average frequency of one spike per second to mimic background activity.

2.4 Computation of number of spikes per volley and volley dispersion

Two measures are defined to characterize spike volleys: the number of spikes per volley and the volley dispersion. Both measures are computed using a time interval of 100 ms duration that begins with the earliest possible response to an input volley. The earliest possible response is computed with the maximal variation of the input spike volley and the minimal propagation delay time for each successive group. The sum of all spikes in a neuron group during this interval is called the number of spikes per volley. To compute the volley dispersion, the background activity is eliminated by subtracting a 500 Hz baseline from the spike histogram of all neurons in a group. From this net histogram, the volley dispersion is computed as the standard deviation of the spike times.

2.5 Simulations

The values of several model parameters are tuned in exploratory simulations within physiologically plausible limits. The mean value of the initial conductances and the probability for feedback and intragroup connections are set to achieve propagation of the first input volley across all neuron populations. Furthermore, the value of the maximal excitatory conductance is set to stabilize the average number of spikes per volley during learning. The value of the learning rate, η, guarantees small conductance changes per volley and sufficient changes for 20 volley presentations.

The equations are integrated using time steps of 2 ms and the results confirmed with 1 ms time steps. The source code, written in the MATLAB programming language, is available at www.cnl.salk.edu/~suri/Suri_Sejn.

3 Results

To investigate the propagation of spike volleys across neuron populations, the 15 input axons of the simulated network were stimulated with 20 spike volleys presented at a frequency of 10 Hz (Sect. 2). Due to repeated input volley presentations, propagation across neuron populations synchronized spike volleys (Fig. 2a). Since temporally asymmetric Hebbian learning depends on the time difference between presynaptic and postsynaptic spikes, it was investigated whether the connection conductances depend on the arrival times of the presynaptic

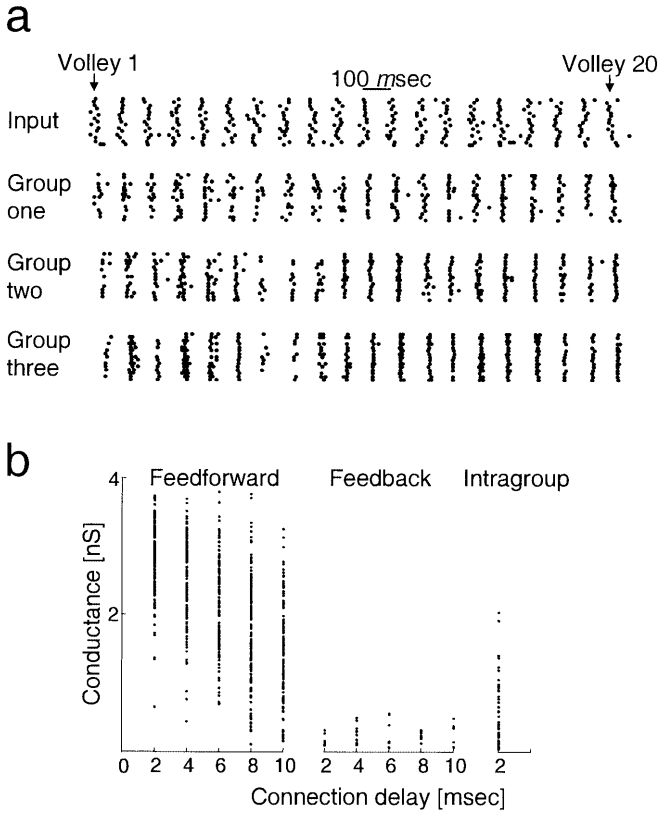

Fig. 2. **a** Propagation across neuron populations synchronized spike volleys due to repeated input volley presentations. The 15 input axons were activated with 20 spike volleys (top line) that were propagated across neuron populations one (line 2), two (line 3), and three (line 4). For each neuron group, spikes of the 15 excitatory neurons are shown in 15 successive lines. Spike volleys became progressively synchronized for successive groups and for successive volley presentations. Since all time constants of the network are much smaller than the intervolley interval, this synchronization appears to be caused by temporally asymmetric Hebbian learning. **b** Connection strength after 20 volley presentations depends on connection types and connection delays. Synaptic conductances of feedforward connections (mean 2.1 nS) became much larger than those of feedback (mean 0.26 nS) and intragroup connections (mean 0.76 nS). Furthermore, conductances of feedforward connections with short delays became larger than those with long delays (correlation $= -0.57, t(673) = 18, p < 0.001$)

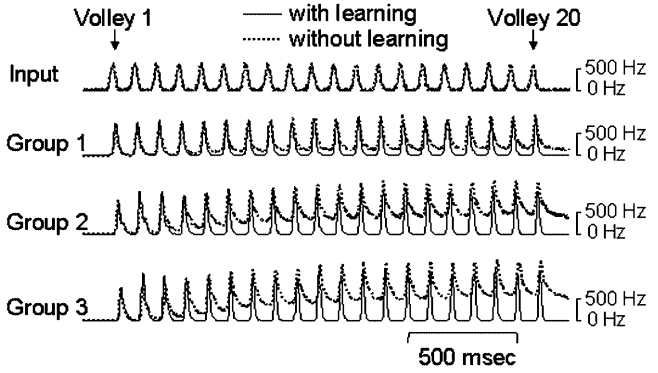

Fig. 3. Average spike frequencies of the input axons and the excitatory neurons within groups with learning (red lines) compared to without learning (blue lines). The simulation shown in Fig. 2a was repeated 500 times for different seeds of the pseudo-random number generators. As in Fig. 2a, propagation across neuron populations progressively synchronized spike volleys due to temporally asymmetric Hebbian learning. Learning led to rapid propagation of these synchronized volleys. Without learning, the average baseline firing rate progressively increased because for 72 out of the 500 simulations recurrent activity led to high and sustained firing rates

spikes within the 20th spike volley. For each synaptic connection, the conductance was computed as a function of the spike arrival time respective to the center of the 20th input spike volley. For all three neuron populations, learning significantly strengthened synapses transmitting early presynaptic spikes as compared to those transmitting late presynaptic spikes (group 1: correlation coefficient $= -0.49$, probability for nonzero correlation coefficient $t(305) = 9.8$, $p < 0.001$; group 2: correlation $= -0.74$, $t(279) = 18$, $p < 0.001$; group 3: correlation $= -0.36$, $t(265) = 6.3$, $p < 0.001$). Without learning, there were no correlations between the arrival times and the conductances (not shown), since the initial conductances were randomly selected (Sect. 2). Spikes transmitted by feedforward connections should usually precede spikes transmitted by feedback and intragroup connections because each connection adds an additional delay. Since learning led to a negative correlation between spike arrival times and connection strengths, conductances of feedforward connections were strengthened while those of intragroup and feedback connections were weakened. For the same reason, feedforward connections with short connection delays became significantly stronger than those with long connection delays (Fig. 2b). The simulation shown in Fig. 2a was repeated 500 times with different seeds of the pseudo-random number generators. Figure 3 depicts the computed average firing rates. As shown in Fig. 2a, propagation across neuron populations progressively synchronized spike volleys due to learning (dispersion $\pm$ standard error in volley 20 was for input 6.8 ± 0.1 ms; group one 4.3 ± 0.1 ms; group two 3.3 ± 0.1 ms; group three 2.7 ± 0.07 ms). This synchronization was not related to a change in the number of spikes per volley (input 16.51 ± 0.05 spikes; group one 14.9 ± 0.1; group two 15.7 ± 0.2 spikes; group three 16.9 ± 0.3 spikes). Without temporally asymmetric Hebbian learning (learning rate $\eta = 0$), firing rates in 72 simulations increased to high and sustained values, which led to a progressively increasing baseline of the average firing rate. Even after removing these 72 simulations with high firing rates as outliers (criterion: more than 75 spikes in the last 100 ms of the simulation), propagation across neuron populations did not synchronize spike volleys (for 500 simulations dispersion $\pm$ standard error in volley 20 was for input 6.8 ± 0.1 ms; group one 6.1 ± 0.2 ms; group two 7.1 ± 0.2 ms; group three 6.2 ± 0.3 ms). The numbers of spikes per volley remained unchanged (input 16.5 ± 0.05 spikes; group one 16.8 ± 0.2 spikes; group two 18.5 ± 0.5 spikes; group three 17.9 ± 0.6 spikes). To quantify synchronization, we defined a criterion for each simulation to determine whether it synchronized input volleys after learning. In group three, we required that volley 20 consisted of at least

10 spikes with a maximum of 3.5 ms dispersion and that the simulation was not an outlier due to high firing rates. According to this criterion, $65 \pm 2\%$ of the simulations synchronized with learning and only $17 \pm 2\%$ synchronized without learning.

To investigate how adaptation in each connection type contributes to propagation of synchronous spike volleys, three variants of the network were studied: a variant without adaptations in intragroup connections, a variant without adaptations in feedback connections, and a variant without adaptations in feedforward connections. For two of the three variants, dispersions after learning were larger than those of the standard network (in volley 20 of group three: no adaptation within groups, dispersion 4.4 ± 0.07 ms, 29.5 ± 0.5 spikes; without adaptation in feedback connections, dispersion 4.2 ± 0.2 ms, 14.4 ± 0.5 spikes; without adaptation in feedforward connections, dispersion 2.3 ± 0.07 ms, 11.2 ± 0.2 spikes). Furthermore, all three model variants synchronized spike volleys significantly less frequently than the standard network ($17.5 \pm 2\%$, $30 \pm 2\%$, and $45.2 \pm 2\%$, respectively), indicating that synchronization was caused by adaptations of all connection types. Since the size of neuron populations may influence propagation of synchronous spikes, the number of neurons in each group was doubled in an additional network variant. Consequently, the number of input axons and the number of spikes per input volley were also doubled. This resulted in a network of 36 neurons per group activated with 30 input spikes per volley. To avoid network instability, the initial values of all synaptic conductances were set to half of their standard values. We found that the dispersions of the 20th volley in the three neuron populations were slightly smaller than those of the standard network and that the number of spikes per volley decreased slightly (input 7.9 ± 0.07 ms, 33.0 ± 0.07 spikes; groups one 4.8 ± 0.08 ms, 25.8 ± 0.2 spikes; group two 3.3 ± 0.08 ms, 24.8 ± 0.4 spikes; group three 2.4 ± 0.05 ms, 25.2 ± 0.6 spikes). Furthermore, synchronization of spike volleys was more frequent than for the standard network ($75 \pm 2\%$), suggesting that temporally asymmetric Hebbian learning also synchronizes spike volleys for larger neuron populations.

4 Discussion

Our simulation results show that temporally asymmetric Hebbian learning leads to progressive synchronization of spike volleys as they are propagated across neuron populations. Weight adaptation strengthens the synapses that are activated by the first spikes in a volley and weakens those that are activated by the latter spikes. Therefore, feedforward connections with short axonal delays are strengthened as compared to feedforward connections with long axonal delays, feedback, and intragroup connections. These adaptations cause the synchronization of spike volleys within several tens of neurons that share equal propagation delays to input volleys. These findings suggest that temporally asymmetric Hebbian learning facilitates rapid propagation of synchronous spike volleys across cortical and hippocampal areas. Since this learning rule synchronizes spike volleys only after repeated volley exposure, synchronization may help to distinguish between familiar and unfamiliar sensory experiences.

These findings are specific for rhythmic input spike patterns. For each neuron, the postsynaptic spike occurrence shifts during learning to the first spikes of the presynaptic volleys, as these first spikes "regularly precede" or "predict" the occurrences of the presynaptic spike volleys. The situation is quite different for nonrhythmic or sequential spatiotemporal input patterns such as those that occur to moving visual stimuli in the visual cortex (Rao and Sejnowski 2000). Under these circumstances, the recurrent connections may take on a dominant role and temporally asymmetric Hebbian plasticity may favor the lateral flow of synchronous activity between cortical columns. The balance between recurrent, feedback and feedforward synchronous flow will depend on the nature of the input patterns as well as the timing of spikes generated in populations of neurons at each level of the hierarchy.

For some values of the model parameters, substantial firing rate changes hamper the emergence of synchronous spikes. For large initial values of excitatory conductances, input volleys trigger sustained firing (Fig. 3). Otherwise, if conductances of excitatory feedforward connections are small, the learning rule prevents any further adaptations of the conductances once a neuron ceases to fire. Although stabilization of firing rates can be achieved with some implementation variants of temporally asymmetric Hebbian learning (Song et al. 2000; van Rossum et al. 2000), the variant chosen here does not have such normalization properties (Rubin et al. 2000).

The current network model simplifies several cortical and hippocampal mechanisms that may hamper the propagation of spike volleys. It is possible that local inhibitory neurons may themselves generate synchronous activity that entrains populations of pyramidal neurons (Tiesinga et al. 2001). Furthermore, the network does not take into account that frequency-dependent depression and facilitation influences connection conductances between pyramidal neurons (Markram 1996; Selig et al. 1999). Although our simulations with depressing excitatory synapses led to similar results as those presented here (not shown), effects of learning were not clearly distinguishable from those of synaptic depression because the time constants of synaptic depression are usually longer than the duration of the chosen intra-trial interval.

For a single neuron, a previous simulation study suggested that temporally asymmetric Hebbian learning enhances the synaptic conductances with matching axonal spike transmission delays (Gerstner et al. 1996). Our results suggest that, in a population of neurons, the connection conductances activated by the first volley spikes become greatly enhanced. Therefore, feedback and intragroup connections become negligibly small during learning. Sustained recurrent activity within neuron populations, which is believed to be the physio-

logical basis of short-term memory, seems to diminish due to temporally asymmetric Hebbian learning (Figs. 2b and 3). Similar to previous simulation results (Levy et al. 2001), our results suggest that sustained short-term memory activity would persist if synchronous spike volleys were propagated in a loop consisting of several neuron populations. In such loops connection conductances become substantially large because their activation predicts spike volley arrivals (Levy et al. 2001). Comparing simulated feedforward connections with cortical bottom-up projections and simulated feedback connections with cortical top-down projections suggests that spikes carried by top-down connections predict spiking of neurons in lower areas by using high-level information about future sensory input. The hypothesis that top-down connections carry predictions or explanations of their target neurons activity was used to simulate extra-classical receptive-field effects (Rao and Ballard 1999) and binocular rivalry (Dayan 1998). The current study suggests that such top-down predictions result from differences in connection delays and temporally asymmetric Hebbian learning.

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