Phase Precession and Recession with STDP and Anti-STDP

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Abstract. We show that standard, Hebbian spike-timing dependent plasticity (STDP) induces the precession of the firing phase of neurons in oscillatory networks, while anti-Hebbian STDP induces phase recession. In networks that are subject to oscillatory inhibition, the intensity of excitatory input relative to the inhibitory one determines whether the phase can precess due to STDP or whether the phase is fixed. This phenomenon can give a very simple explanation to the experimentally-observed hippocampal phase precession. Modulation of STDP can lead, through precession and recession, to the synchronization of the firing of a trained neuron to a target phase.

1 Introduction

Spike-timing dependent plasticity (STDP) is the dependence of synaptic changes on the relative timing of pre- and postsynaptic action potentials, a phenomenon that has been experimentally observed in biological neural systems [1,2,3]. The type of STDP that has been mostly studied is characterized by the potentiation of a synapse when the postsynaptic spike follows the presynaptic spike within a time window of a few tens of milliseconds, and the depression of the synapse when the order of the spikes is reversed. This type of STDP is sometimes called Hebbian, because it is consistent with the original postulate of Hebb that predicted the strengthening of a synapse when the presynaptic neuron causes the postsynaptic neuron to fire. Experiments have also found synapses with anti-Hebbian STDP (also called anti-STDP), where the sign of the changes is reversed, in comparison to Hebbian STDP [4,5,6,7].

Many studies have investigated the computational properties of Hebbian STDP, and have shown its function in neural homeostasis, unsupervised and supervised learning [8,9,10,11,12,13,14,15,16,17,18]. Anti-Hebbian STDP is, at a first glance, not as interesting as the Hebbian mechanism, as it leads, by itself, to an overall depression of the synapses towards zero efficacy [19]. We have recently

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shown that modulating STDP with a reward signal (i.e., having both Hebbian and anti-Hebbian STDP) leads to reinforcement learning [20,21]. None of these studies have specifically investigated the consequences of STDP in oscillatory networks.

Here we study through computer simulations the effects of Hebbian and anti-Hebbian STDP in networks of neurons that fire periodically with a common period. This has biological relevance because there is such rhythmical activity in the brain, for example the hippocampal theta rhythm [22,23]. We first describe our model (Section 2) and then demonstrate some general effects induced by STDP in oscillatory networks (Section 3). Afterwards we study the interplay between these effects and oscillatory inhibition (Section 4) and how the effects can be used to teach a neuron to fire at a given phase (Section 5).

2 Methods

We study an integrate-and-fire neuron driven by N_e excitatory and N_i inhibitory input neurons. The excitatory synapses are plastic, while the inhibitory ones are static. This setup is similar to the one in [10]. We model the network's rhythmic activity by considering that input neurons fire periodically with a common period $T = 125$ ms (corresponding to the 8 Hz theta hippocampal rhythm). In the brain, neurons sometimes skip cycles, while still firing at a constant phase, but we ignore this possibility here, for the sake of simplicity, and consider that each of the input neurons fires once per period, at a predetermined phase ϕ_k. These phases are generated randomly at the beginning of the experiments. The phases of excitatory neurons are generated uniformly between 0 and 2π. In experiments where we use inhibitory neurons, the total inhibition is considered to be modulated by the global oscillation, as in other models of the hippocampal theta rhythm [26,27], and thus their phases are generated with a probability density $p(\phi_k) = [\cos(\phi_k) + 1]/(2\pi)$ (see also Fig. 2e-g).

The dynamics of the postsynaptic integrate-and-fire neuron is given by the following equation:

$$\tau_m \frac{\mathrm{d}V(t)}{\mathrm{d}t} = -(V - V_0) + \sum_{k=1}^{N_e+N_i} g_k(t)\,[E_k - V(t)]\,, \tag{1}$$

where V is the membrane potential, V_0 =-70 mV is the resting potential, τ_m =20 ms is the decay time constant, g_k are synaptic conductances and E_k are reversal potentials. When the membrane potential reaches a threshold of -54 mV, the neuron fires and V is reset to -60 mV. We consider E_k=0 mV for excitatory synapses and E_k=-70 mV for inhibitory ones (parameters from [10,24]).

Each presynaptic spike determines an instantaneous rise in the synaptic conductance, which decays then exponentially. Thus, the dynamics of the synaptic conductances is given by

$$\frac{\mathrm{d}g_k(t)}{\mathrm{d}t} = -\frac{g_k(t)}{\tau_g} + g_k^s(t)\,\Phi_k(t), \tag{2}$$

where $\tau_g = 5$ ms, g_k^s are the peak synaptic conductances, and $\Phi_k(t)$ represents the firing train of input neuron k as a sum of Dirac functions:

$$\Phi_k(t) = \sum_{n=0}^{\infty} \delta\left(t - (n\,T + \phi_k)\right). \tag{3}$$

For inhibitory synapses, g_k^s is constant and is generated randomly at the beginning of the experiment, with an uniform distribution, between 0 and g_{max}^s. For excitatory synapses, g_k^s is also initialized randomly between 0 and g_{max}^s, but varies in time due to STDP. As in previous studies [10,25], we use an exponential dependence of plasticity on the relative spike timings, we consider that the effect of different spike pairs is additive, and we limit the range of possible synaptic strengths with hard bounds, between 0 and g_{max}^s. To model Hebbian as well as anti-Hebbian STDP, we consider that plasticity is modulated by a variable $r(t)$ that can be positive as well as negative. Hence, the dynamics of the excitatory synaptic conductances is given by

$$\frac{\mathrm{d}g_k^0(t)}{\mathrm{d}t} = r(t)\ \left[\Phi_0(t)\ A_+ \sum_{\mathcal{F}_k^t} \exp\left(-\frac{t - t_k^f}{\tau_+}\right) + \Phi_k(t)\ A_- \sum_{\mathcal{F}_0^t} \exp\left(-\frac{t - t_0^f}{\tau_-}\right)\right], \tag{4}$$

with the additional hard bounds. We noted with $\mathcal{F}_k^t$ the set of firing times t_k^f previous to t of input neuron k, and $\mathcal{F}_0^t$ is the analogue for the postsynaptic neuron. $\Phi_0(t)$ is the spike train of the postsynaptic neuron; $A_\pm$ are constant parameters that determine the magnitude of synaptic changes, $A_+ = 0.005\ g_{max}^s$, $A_- = -A_+$; $\tau_\pm$ are the decay time constants of the exponential STDP windows, $\tau_+ = \tau_- = 20$ ms.

Following [10], we use a set of variables P_k^+ that track the influence of presynaptic spikes and P_0^- that tracks the influence of postsynaptic spikes on the synapses. These variables simplify the simulation and may also have biochemical counterparts in biological neurons. We then have:

$$\frac{\mathrm{d}P_k^+}{\mathrm{d}t} = -\frac{P_k^+}{\tau_+} + A_+\ \Phi_k(t) \tag{5}$$

$$\frac{\mathrm{d}P_0^-}{\mathrm{d}t} = -\frac{P_0^-}{\tau_-} + A_-\ \Phi_0(t) \tag{6}$$

$$\frac{\mathrm{d}g_k^0(t)}{\mathrm{d}t} = r(t)\ \left[\Phi_0(t)\ P_k^+ + \Phi_k(t)\ P_0^-\right] \tag{7}$$

In some of the experiments, we use a homeostatic mechanism [28] that scales up or down the synapses in order to keep the postsynaptic firing rate constant, at one spike per oscillation period T. We estimate the postsynaptic firing rate ν by using a leaky accumulator (equivalent to an integration with an exponential kernel),

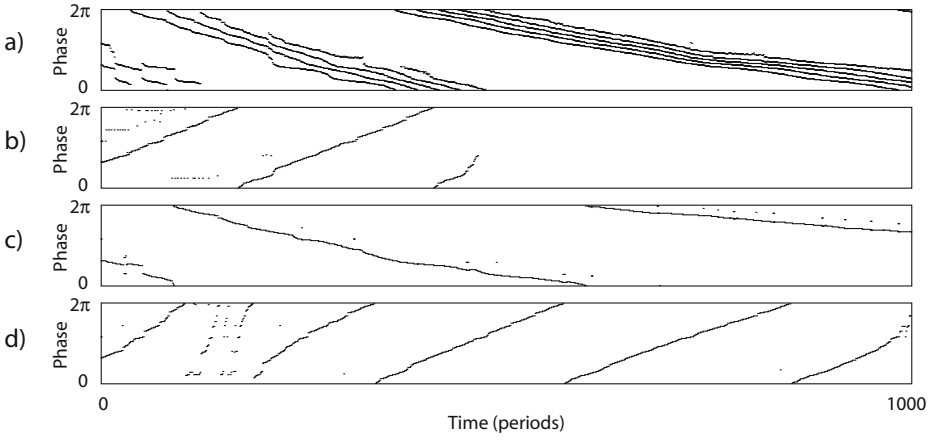

Fig. 1. The evolution in time of the phase of postsynaptic spikes relative to the input oscillation. The graphs illustrate the first 1000 periods of the experiments. All experiments start with identical conditions. a), c): Hebbian STDP. b), d): Anti-Hebbian STDP. a), b): Without homeostasis. c), d): With homeostasis.

$$\frac{\mathrm{d}\nu(t)}{\mathrm{d}t} = -\frac{\nu(t)}{\tau_\nu} + \frac{1}{\tau_\nu}\,\Phi_0(t), \tag{8}$$

with $\tau_\nu = 500$ ms. We then scale all excitatory synapses according to

$$\frac{\mathrm{d}g_k^0}{\mathrm{d}t} = \alpha\; g_k^0\; \left[\frac{1}{T} - \nu(t)\right], \tag{9}$$

with $\alpha = 0.04$. This mechanism is applied additionally to the plasticity mechanisms already mentioned.

The network is simulated with a timestep of 0.5 ms.

3 Precession and Recession

We first consider a setup with $N_e = 1000$ excitatory input neurons and no inhibitory input. We use $g^s_{max} = 0.014$ and no homeostasis. If we set $r(t) = 1$, i.e. Hebbian STDP, and let the network run, we observe that the phase of the postsynaptic spikes relative to the input oscillation precesses, i.e. has a tendency to occur earlier in the cycle (Fig. 1a). This is consistent with previous observations that STDP tends to reduce the latency of postsynaptic firing in response to the same stimulus (input) [10,29] and that STDP allows the postsynaptic neuron to predict its input [12]. These properties of STDP also make inputs that fire before the postsynaptic neuron to become more and more effective in causing the postsynaptic neuron to fire, and eventually increase the total excitation that this neuron receives. This means that the neuron may start to fire more spikes per period, a phenomenon that can be seen in Fig. 1a.

If we set $r(t) = -1$, i.e. we have anti-Hebbian STDP, we observe the opposite, namely that the phase of the postsynaptic spikes recesses (has a tendency to occur later in the cycle), and that the excitation that the neuron receives diminishes, eventually leading the neuron to stop firing (Fig. 1b). This is consistent with the previous observation that anti-Hebbian STDP leads to a global weakening of the synapses [19].

However, if we also introduce the previously mentioned homeostatic mechanism that keeps the postsynaptic neuron firing once per period, we observe that the precession/recession corresponding to Hebbian/anti-Hebbian STDP becomes a stable behavior of the neuron (Fig. 1c,d).

4 Precession Control Through Oscillatory Inhibition

We now add to the previously described setup $N_i = 1000$ inhibitory input neurons, that provide an oscillatory inhibitory input current to the postsynaptic neuron (each inhibitory neuron fires once per cycle, and the number of neurons that fire at a particular phase oscillates as a function of phase). We use $r(t) = 1$, $g^s_{max} = 0.015$ and homeostasis. The phase precession is not disturbed by the oscillatory inhibition (Fig. 2a). Precession is stopped, however, by a much stronger inhibition, for example if we reduce the number of excitatory inputs from 1000 to 500 (Fig. 2b), as the neuron can fire only at phases where excitation overcomes inhibition.

This means that by modulating the ratio of excitation versus oscillatory inhibition, in conjunction with STDP, we may switch from precession to a state of constant phase firing. This is illustrated in Fig. 2c-d, where, after the firing phase stabilizes because oscillating inhibition dominates excitation, we increase the excitation received by the output neuron, by adding extra excitatory inputs. Until $t_1 = 1200\,T$, the postsynaptic neuron is driven by 500 excitatory neurons. From t_1 to $t_2 = 1500\,T$, we constantly add new excitatory inputs to the postsynaptic neuron until their number reaches 1000 at t_2. With greater excitation, the phase starts to precess. From t_2 to $t_3 = 1800\,T$, we gradually remove the newly added excitatory inputs; excitation decreases and then phase stabilizes again to a value close to the one previous to the increase in excitation.

This very simple model is thus capable to explain the basic features of hippocampal phase precession. It has been observed that when a rat moves through the receptive field of a place cell, the firing rate of the neuron correlates with the position in the place field, and the firing phase of the neuron precesses as the animal traverses the place field. The initial phase at which the neuron starts firing when the animal enters the place field is constant for every traversal of the field [30]. The simple model presented here is consistent with these observations: as the excitation of the place cell increases because the animal enters into its receptive field, its firing phase precesses simply because of STDP and because excitation overcomes the phase locking by the oscillatory inhibition.

Among the many computational models that tried to explain phase precession, only two others used STDP. The first one used STDP to explain the skewness

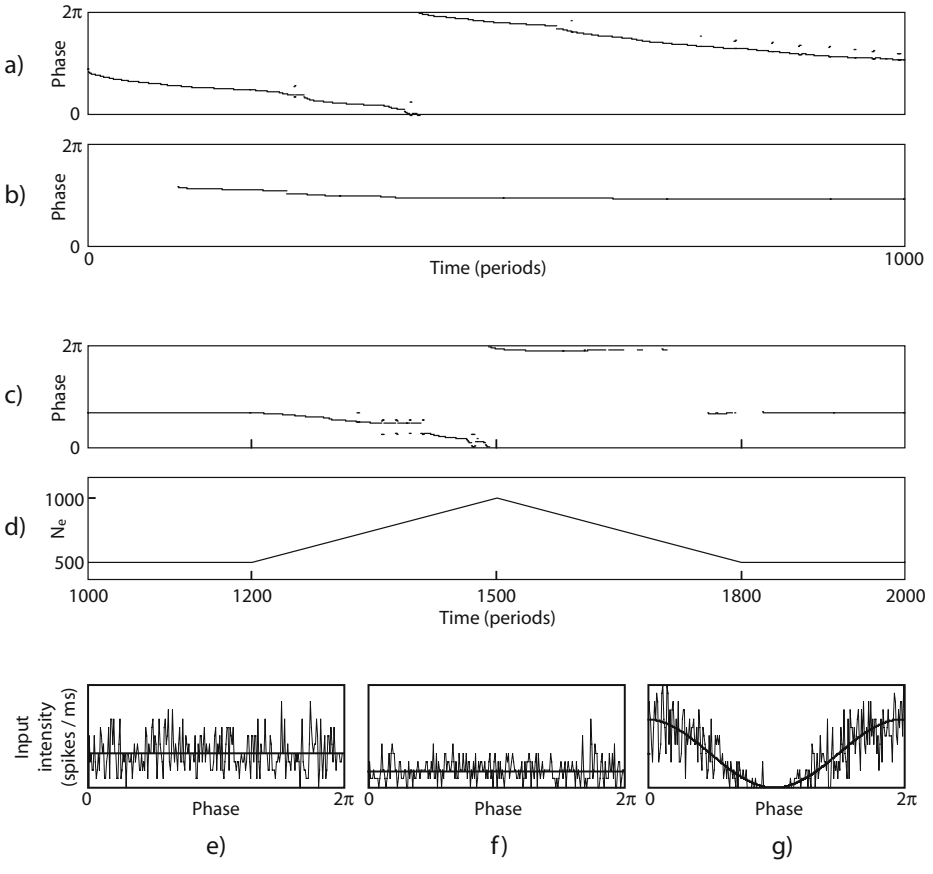

Fig. 2. a)-c) Effect of oscillatory inhibition on the dynamics of the phase of postsynaptic spikes relative to the input rhythm. All experiments start with identical conditions. a) 1000 excitatory inputs, 1000 inhibitory inputs. b) 500 excitatory inputs, 1000 inhibitory inputs. c) 500–1000 excitatory inputs, 1000 inhibitory inputs. d) The evolution in time of the number of the excitatory inputs for the experiment presented in c). e)-f) Intensity of the total excitatory and, respectively, inhibitory inputs (number of spikes per time unit) as a function of phase. The smooth line represents the average number of input spikes per timestep corresponding to the probability with which they were generated, the rugged line represents the actual histogram of the input spikes as a function of phase, corresponding to the experiments illustrated here. e) Excitatory input intensity for the experiment presented in a). f) Excitatory input intensity for the experiment presented in b). g) Inhibitory input intensity for all experiments. In the experiment presented in c), the input intensity varies between the one presented in f) and the one presented in e).

of the place fields, which, at its turn, explained phase precession, through interaction with the inhibitory oscillation [31]. The second one takes from STDP only the idea of temporally asymmetric interactions between neurons, as it uses

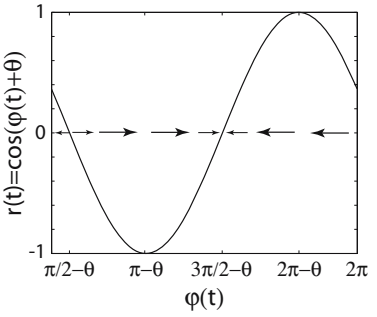

Fig. 3. The phase ϕ_0 precesses if $r(t) > 0$ and recesses if $r(t) < 0$, as indicated by the arrows. If $r(t) = \cos(\varphi(t) + \theta)$, the phase converges to $\phi_0 = 3\pi/2 - \theta$ because this is a stable point for the dynamics of ϕ_0; $\phi_0 = \pi/2 - \theta$ is an unstable equilibrium point

neurons with continuous activations instead of spiking neurons [32]. The model presented here is much simpler than these previous models, yet it captures the essential features of hippocampal phase precession.

5 Controlling the Firing Phase by Modulating STDP

Since the firing phase can be manipulated by STDP and anti-STDP, it is straightforward to devise a mechanism for moving it to a target phase, by modulating STDP. Modulation of STDP by a global reward signal proved to be a robust reinforcement learning mechanism for generic spiking neural networks and could be implemented in the brain by a neuromodulator [20,21]. Here we may use a similar modulation, but with a form that depends on the target phase at which we want the postsynaptic neuron to fire, instead of an external reward.

For example, if the variable $r(t)$ that modulates STDP oscillates as a function of the input oscillation phase, with the same period, and the STDP temporal constants $\tau_\pm$ are smaller than the oscillation period, the output neuron will always decrease its phase if the phase is in certain intervals, and increase it in others. If an oscillatory $r(t)$ is a continuous function of input oscillation phase $\varphi = 2\pi\ t/T$, and has both positive and negative values, the phase of the postsynaptic neuron will have at least two points of equilibrium ($r = 0$), among which one will be stable and one unstable. For example, if $r(t) = \cos(\varphi(t) + \theta)$, the equilibrium point will be $\phi_0 = 3\pi/2 - \theta$ (see Fig. 3). The firing phase of a postsynaptic neuron with synapses featuring STDP modulated by an $r(t)$ of this form will thus move to the phase of stable equilibrium. This means that we can train a neuron or a population of (independent) neurons to fire at a particular phase by using STDP in conjunction to an appropriate form of $r(t)$. The efficacy of this approach is illustrated in Fig. 4. The neuron learns the target firing phase within 200 periods (25 s). The same signal $r(t)$ may train an arbitrary number of neurons to fire at the same phase, thus synchronizing them.

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