

# Spike-driven Synaptic Plasticity for Learning Correlated Patterns of Asynchronous Activity

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**Abstract.** Long term synaptic changes induced by neural spike activity are believed to underlie learning and memory. Spike-driven long term synaptic plasticity has been investigated in simplified situations in which the patterns of asynchronous activity to be encoded were statistically independent. An extra regulatory mechanism is required to extend the learning capability to more complex and natural stimuli. This mechanism is provided by the effects of the action potentials that are believed to be responsible for spike-timing dependent plasticity. These effects, when combined with the dependence of synaptic plasticity on the post-synaptic depolarization, produce the learning rule needed for storing correlated patterns of asynchronous neuronal activity.

## 1 Introduction

It is widely believed that synaptic plasticity is the phenomenon underlying learning and memory. In the last years many models of spike-driven plastic synapses have been proposed (see e.g. [1, 2]). Most of these theoretical works were devoted to modeling experimental results on single pairs of synaptically connected neurons [2], to studying regulatory mechanisms based on spike-timing dependent plasticity [3], or to relating the long term modifications to the neuronal activity to be encoded [1]. Only very recently a few investigators started to study the formation of memory and the collective dynamical behavior of networks of integrate-and-fire neurons connected by spike-driven plastic synapses [4, 5]. Here we make a further step in the direction of understanding the computational relevance of spike-based synaptic dynamics by extending the model introduced in [1]. The Hebbian paradigm there implemented was good enough to embed in the synaptic matrix an extensive number of random, uncorrelated patterns of asynchronous activity. However going beyond these simplistic patterns requires a regulatory mechanism that ensures a balanced redistribution of the memory resources among the different stimuli to be stored. This is usually achieved by introducing a mechanism that guarantees a global normalization of the synaptic weights (see e.g. [6]). One possible local mechanism is studied in [7, 8] where the internal state of the synapse is changed according to a Hebbian paradigm only if it is necessary, i.e. if the pattern of activity to be encoded is not yet correctly memorized. For instance, when a stimulus to be learnt imposes a pattern of elevated activity to a subset of neurons, the synapses connecting these

neurons are potentiated only if the activity of the post-synaptic (output) neuron is below some threshold value. Otherwise the synapse is left unchanged because the pattern of activity imposed by the external stimulus is already stable and the memory resources can be exploited in a more efficient way by other stimuli. Learning prescriptions based on this principle permit to memorize complex correlated patterns [7], and to classify real data, like handwritten digits [8], provided that learning is slow, i.e. that every pattern has to be presented several times. These non-monotonic learning rules are achieved here by introducing in the synaptic dynamics of [1], which depends solely on the sub-threshold depolarization of the post-synaptic neuron, an extra dependence on the effects of the post-synaptic action potential. In particular an extra depression occurs when the pre-synaptic spike follows the post-synaptic action potential within some time interval. This combination of depolarization and spike-timing dependence makes the model more realistic and captures the observations of recent experiments on long term synaptic modifications [9].

## 2 The synaptic dynamics

The synaptic dynamics can be fully described in terms of a single internal variable  $X$  which represents the state of the synapse and determines the synaptic efficacy. In the absence of any pre or post-synaptic spike the synapse is made bistable by a recall force that drives the internal state to one of the two stable states: to  $X = 1$ , that corresponds to the upper bound, if  $X$  is above some threshold  $\theta_X$ , or to the lower bound  $X = 0$  if  $X < \theta_X$ . Above  $\theta_X$  the synapse is potentiated and synaptic efficacy on the post-synaptic neuron is elevated, whilst below  $\theta_X$  the synapse is depressed (low efficacy). These dynamics can be expressed as follows:

$$\frac{dX}{dt} = -\alpha\Theta(-X + \theta_X) + \beta\Theta(X - \theta_X)$$

where  $\alpha$  and  $\beta$  represent the two refresh currents. Pre- and post-synaptic spikes trigger temporary modifications of the internal variable. They depend on the depolarization  $V$  of the post-synaptic neuron [1] and on the time that passed since the last occurrence of a pre or a post-synaptic spike [2]. When the depolarization is high ( $V > V_H$ ) every pre-synaptic spike pushes  $X$  upwards, weakly if the last post-synaptic spike has been emitted within the previous time interval  $T_-$ , strongly otherwise. If the depolarization is low ( $V < V_L$ ), the pre-synaptic spikes induce a depression which is either weak, if there is no recent post-synaptic spike (within a time interval  $T_-$ ), or strong otherwise. Post-synaptic spikes preceding pre-synaptic action potentials provoke small upwards jumps. The table below summarizes the effects of the spikes and the corresponding probability of occurrence when the pre- and post-synaptic neurons fire asynchronously, with a Poisson distribution, at a mean frequency  $\nu_{pre}$  and  $\nu_{post}$  respectively.  $Q_a$  ( $Q_b$ ) is the probability that  $V > V_H$  ( $V < V_L$ ) upon the arrival of a pre-synaptic spike. The modifications induced by the spikes leave a long term memory trace only if  $X$  crosses the threshold  $\theta_X$  during the stimulation. Otherwise the state prior to the stimulation is restored by the refresh currents.

Effects of a pre-synaptic spike that arrives at time $t$		
Condition	Effect	Prob. of occurrence
$V > V_H$ and no spike in $[t - T_-, t]$	$X \rightarrow X + a$	$\nu_{pre} Q_a e^{-\nu_{post} T_-}$
$V > V_H$ and $k$ spikes in $[t - T_-, t]$	$X \rightarrow X + a - ka'$	$\nu_{pre} Q_a e^{-\nu_{post} T_-} \frac{(\nu_{post} T_-)^k}{k!}$
$V < V_L$ and no spike in $[t - T_-, t]$	$X \rightarrow X - b$	$\nu_{pre} Q_b e^{-\nu_{post} T_-}$
$V < V_L$ and $k$ spikes in $[t - T_-, t]$	$X \rightarrow X - b - kb'$	$\nu_{pre} Q_b e^{-\nu_{post} T_-} \frac{(\nu_{post} T_-)^k}{k!}$
Effects of a post-synaptic spike that arrives at time $t$		
$k$ spikes in $[t - T_+, t]$	$X \rightarrow X + ka'$	$\nu_{post} e^{-\nu_{pre} T_+} \frac{(\nu_{pre} T_+)^k}{k!}$

## 2.1 Mean firing rates vs distribution of the depolarization

The probabilities of occurrence of the temporary modifications  $a, b, a', b'$  control the direction in which the synapse is modified by the activity of the pre- and post-synaptic neurons.  $Q_a$  and  $Q_b$  depend on the statistics of the depolarization of the post-synaptic neuron under stimulation and can be calculated analytically when the neuron is embedded in a large network of interacting neurons and is subject to the heavy bombardment of synaptic inputs. Such an in-vivo like situation is emulated by injecting into an integrate-and-fire neuron a gaussian current characterized by its mean  $\mu$  and its variance  $\sigma^2$ . The stationary distribution  $p(V)$  can be computed as in [11, 1] and is given by:

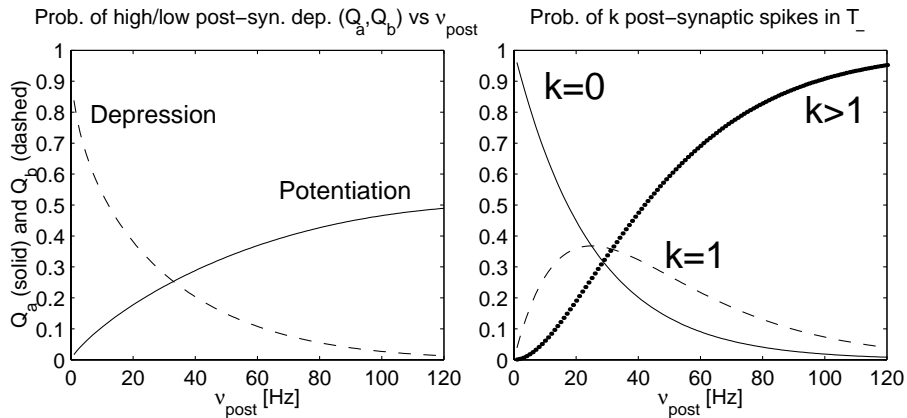
$$p(V) = \frac{\nu}{\mu} \left[ \Theta(V - H) \left( 1 - e^{-\frac{2\mu}{\sigma^2}(\theta - V)} \right) + \Theta(H - V) \left( e^{-\frac{2\mu}{\sigma^2}H} - e^{-\frac{2\mu}{\sigma^2}\theta} \right) e^{\frac{2\mu}{\sigma^2}V} \right]$$

where  $H$  is the reset potential,  $\theta$  is the threshold for emitting a spike and  $\nu$  is the mean firing frequency:

$$\nu = \left[ \tau_r + \frac{\sigma^2}{2\mu^2} \left( e^{-\frac{2\mu\theta}{\sigma^2}} - e^{-\frac{2\mu H}{\sigma^2}} \right) + \frac{\theta - H}{\mu} \right]^{-1}$$

$\tau_r$  is the absolute refractory period.  $Q_a$  and  $Q_b$  are given by the integral of  $p(V)$  in the interval  $[V_H, \theta]$  and  $[0, V_L]$  respectively. It is straightforward to compute these integrals analytically.  $\mu$  and  $\sigma$  characterize the synaptic input and depend on the network interactions. We assume that  $\nu_{post}$  is changed by increasing or decreasing the average spike frequency of a subpopulation of pre-synaptic neurons [1]. If the recurrent feedback of the post-synaptic neurons does not affect much the network activity, then the parameters of the input current move along a linear trajectory in the  $(\mu, \sigma^2)$  space. We chose  $\mu$  as an independent parameter, and  $\sigma^2 = J\mu + K$ . In a network of excitatory and inhibitory neurons, in which in a spontaneous activity state the recurrent input is as large as the external input, we have that  $J = J_E$  (the average coupling between excitatory neurons) and  $K = \nu_0^I N_I J_I (J_I + J_E)$ , where  $\nu_0^I$  is the spontaneous activity of the  $N_I$  inhibitory neurons that are projecting to the post-synaptic cell (mean coupling  $J_I$ ).  $Q_a$  and  $Q_b$  are plotted in Fig. 1. As the external stimulus increases  $\nu_{post}$ , the distribution of  $V$  changes in such a way that  $Q_b$  decreases and  $Q_a$  increases. This makes it rather likely that when the two neurons are simultaneously activated most of the

numerous temporary modifications of the synapse are upwards and the synapse tends to potentiate. On the contrary, when the pre-synaptic neuron is active and the post-synaptic neuron has low spontaneous activity, most of the jumps are downwards and the synapse is eventually depressed.



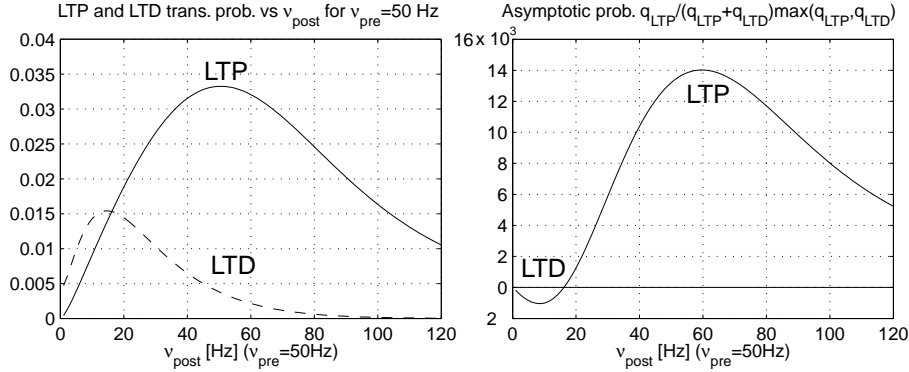
**Fig. 1.** Left: Probability of occurrence of upwards ( $Q_a$ ) and downwards ( $Q_b$ ) jumps upon the arrival of a pre-synaptic spike for different post-synaptic activities. Right: Probability that  $k$  post-synaptic spikes are within a time window  $T_-$ . It determines the depressing and regulatory effects of the action potential on the synaptic dynamics.

When  $\nu_{pre}$  is low, the few jumps are usually not enough to drive the internal state across the threshold. It can be proven that this scheme of modifications (a Hebbian paradigm) maximizes the signal of the stimuli that can be extracted by the network dynamics from the synaptic matrix [12]. Hence  $Q_a$  and  $Q_b$  contain most of the relevant information to encode the patterns of activities imposed by the stimulus. The extra jumps ( $a', b'$ ) due to spike-timing depend essentially on the product of the mean frequencies of the pre and the post-synaptic neuron. The ratio between the number of potentiating events and the number of depressing events is roughly constant when the pre- and the post-synaptic frequencies are multiplied by the same factor. This activity dependence does not contribute much to the signal, but has an important regulatory role. Indeed, if  $T_- \gg T_+$ , as the frequency of the post-synaptic neuron increases the fraction of cases in which the direction of the temporary modification is controlled by the depolarization  $V$  decreases and depression becomes predominant, especially if one considers the possibility of multiple spikes within the time interval  $T_-$ .

### 3 The non-monotonic learning rule

The next step is to compute the probability that a stimulus induces a stable modification in the synaptic couplings, i.e. that during the stimulation the synapse makes a transition from one stable state to another stable state. The transition probabilities control the learning process. Low probabilities correspond to a slow learning scenario in which the stimuli need to be presented a large number of times to be learnt. This is the price to be paid if one wants an optimal redistribu-

tion of the memory synaptic resources among the stimuli to be learnt [12, 7, 8]. Indeed, high transition probabilities allow fast acquisition of information about the stimuli, but limit dramatically the storage capacity of the network because past stimuli are forgotten very quickly [12]. For uncorrelated patterns of activities the optimal storage capacity is achieved when the coding level is low (a small fraction of neurons is activated by each stimulus) and the LTD probability is small enough, compared to the LTP probability, to preserve the balance between potentiations and depressions [12]. For correlated patterns a control mechanism is needed to stop learning in case the stimulus is already correctly encoded in the synaptic matrix [7, 8].



**Fig. 2.** Left: LTP and LTD transition probabilities ( $q_{LTP}, q_{LTD}$ ) as a function of post-synaptic frequency for  $\nu_{pre} = 50$  Hz. Right: The normalized asymptotic probability which combines  $q_{LTP}$  and  $q_{LTD}$  to summarize the behavior of the synapse for a wide range of post-synaptic activities.

### 3.1 Solving the long term synaptic dynamics

The transition probabilities can be approximately calculated by using the density approach introduced in [1]. Since the pre and the post-synaptic neuron fire asynchronously, the Takács equations that govern the density function  $p(X, t)$  of the synaptic internal variable can be easily generalized to incorporate the extra jumps  $a'$  and  $b'$  of the new model. For example, for  $X < \theta_X$  we have:

$$\frac{\partial p(X, t)}{\partial t} = \alpha \frac{\partial p(X, t)}{\partial^{(+)} X} + \nu_{pre} [A(X, t) - p(X, t)] + \nu_{post} Q'_a [p(X - a', t) - p(X, t)]$$

where the term with  $\alpha$  accounts for the refresh current and  $A$  contains the temporary changes induced by the spikes:

$$A(X, t) = Q_a P_0(t) \left[ \sum_{k=0}^{\infty} \delta(X - a + kb') Q'_b(k) \right] + Q_a \left[ \sum_{k=0}^{\infty} p(X - a + kb') Q'_b(k) \right] + Q_b \left[ \sum_{k=0}^{\infty} p(X + b + kb', t) Q'_b(k) \right]$$

where  $Q'_b(k) = e^{-\nu_{post}T_-}(\nu_{post}T_-)^k/k!$ . We assume that  $p(x, t) = 0$  for  $x \notin [0, 1]$ . The equation for the discrete probability  $P_0 = \text{prob}(X = 0)$  is modified in a similar way. The equations are approximated because the temporal auto-correlations of the post-synaptic  $V$  are not considered (see [1] for more details).

## 4 Conclusions

The numerical solution of the density equations provides the LTP and LTD transition probabilities  $q_{LTP}$  and  $q_{LTD}$  for any pair of  $\nu_{pre}, \nu_{post}$  (Fig. 2). The parameters of the synaptic dynamics can be easily tuned to achieve: 1) LTP and LTD trans. probabilities that maximize the signal embedded in the synaptic matrix by each pattern of activity (LTP for high  $\nu_{post}$  and LTD for low  $\nu_{post}$ ); 2) a ratio between LTP and LTD that ensures the balance of total number of potentiations and depressions; 3) the preservation of memory on very long time scales (the transitions in case of spontaneous activity of both pre- and post-synaptic neurons are negligible); 4) a regulatory mechanism that blocks LTP when  $\nu_{post}$  is too high, and stops LTD when  $\nu_{post}$  is too low. This is the mechanism required to store correlated patterns [7, 8]. It also resembles the BCM rule [10] and it is essentially based on the extra effect of the action potential that signals when the activity of the post-synaptic neuron is too elevated.

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