

# Toward STDP-based population action in large networks of spiking neurons

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**Abstract.** We present simulation results that clarify the role of Spike-Timing Dependent Plasticity (STDP) in brain processing as a putative mechanism to transfer spatio-temporal regularities, as observed in sensory signals, toward action, expressed as a global increase of the target population activity, followed by a reset. The repetition of this activation-reset mechanism gives rise to a series of synchronous waves of activity when the same stimulus is repeated over and over. Our simulation results are obtained in recurrent networks of conductance-based neurons under realistic coupling constraints.

## 1 Introduction

Synchronous activity patterns are found in natural and artificial recurrent neural networks when (i) recurrent excitatory and inhibitory influences balance each other and (ii) when the membrane potential of neurons undergo strong variations (i.e. when the neurons strongly amplify their input). The parametric conditions under which synchrony can be obtained have been analyzed, from the most simple binary units [1], to integrate and fire [2] or more elaborate conductance-based models [3, 4]. The functional role of this synchronous activity is not fully clarified yet. Spontaneous brain activity is characterized by a high degree of irregularity [5]. Some synchrony can however be observed directly or indirectly when the brain is involved in a task [6, 7]. Synchronous firing is commonly observed in the first steps of olfactory recognition [8]. It is generally admitted that synchrony can help to transmit a structured signal from population to population [9, 10] or to help distant populations to “work together” [7].

The final brain processing activity may finely rely on the interplay between asynchrony and synchrony. Following Freeman [8], we consider that a transition to synchrony may be observed when the brain faces an event of particular interest. In order to allow such responses to rely on experience, we moreover suppose that some plasticity process may allow to enhance synchrony in a network of units spontaneously displaying irregular activity.

The Spike-Timing Dependent Plasticity (STDP) is a serious candidate for implementing such synaptic plasticity effects. The rule originates from biological evidences [11] showing that an excitatory synapse is potentiated if the EPSP (Excitatory Post-Synaptic Potential) is shortly followed by the emission of an action potential at the soma, and depressed in the opposite case (when the AP is followed by the EPSP). An important effect of STDP is that it reduces

the latency of a neuron's response to a given input [12, 13]; the behaviour of STDP has also been studied on larger, recurrent networks, but the results are more ambiguous: while it has been shown that in some conditions it can lead to the emergence of neuronal groups [14, 15], these results seem to depend on the precise implementation, since a slightly different rule on a different neuron model gives different results: there is no structure development, strong synapses remaining unstable [16].

We propose here to clarify the role of Spike-Timing Dependent Plasticity (STDP) in recurrent networks of conductance-based neurons<sup>1</sup> under realistic coupling constraints.

Several implementations of the STDP update mechanism have been proposed in the literature. Our implementation is all-to-all [18] and additive: the weight update depends on every previous synaptic event (the influence of the less recent events fading with time) and doesn't take into account the current weight of the synapse. Concretely:

$$w'_{ij}(t) = w_{ij}(t - dt) + \frac{\alpha}{N} [S_i(t)\varepsilon_j(t - d_{ij}) - \varepsilon_i(t)S_j(t - d_{ij})]$$

where the trace  $\varepsilon_i(t) = \sum_{s < t, S_i(s)=1} \exp\left(-\frac{t-s}{\tau_m}\right)$  can be considered as a short-

term estimation of the firing rate of the  $i^{\text{th}}$  neuron and  $\alpha$  is a learning parameter ( $\alpha = 1$  in simulations). This update scheme divides in two terms. The first term corresponds to the synaptic potentiation: a significant weight increment takes

<sup>1</sup>We consider a population of  $N$  neurons. Consider neuron  $i$ . We use the following update for the membrane potential  $V_i$  (leaky integrate and fire with conductance-based synapses):

$$C \frac{dV_i(t)}{dt} = G_0(V_i(t) - V_0) + \left[ \frac{V_E - V_i(t)}{V_E - V_0} G_{i,E}^E(t) - \frac{V_i(t) - V_I}{V_0 - V_I} G_{i,I}^I(t) \right] + I_i(t)$$

where  $C$  is the membrane capacitance,  $V_0$  is the resting potential,  $V_E$  is the reversal potential on excitatory synapses,  $V_I$  is the reversal potential on inhibitory synapses,  $I_i$  is an external input current,  $G_{i,E}$  is the total excitatory conductance and  $G_{i,I}$  is the total inhibitory conductance. For simpler parameter settings, we use a normalized neuron i.e.  $V_0 = 0$ ,  $\theta = 1$ ,  $V_I = -1$  and  $V_E = 6$ . The membrane time constant is  $\tau_m = \frac{C}{G_0}$ . Except for the sign of their target synapses, the neurons are all similar (same membrane time constant  $\tau_m = 10$  ms). When the potential crosses the firing threshold  $\theta_i$ , the potential is immediately reset to 0, i.e.  $V(t) \leftarrow 0$  and a spike is emitted i.e. an instantaneous Dirac impulse is emitted. We note  $S_i(t)$  the output of the neuron at time  $t$ , where  $S_i(\cdot)$  is a sum of all past impulses until  $t$ . We use a refractory period  $\tau_r = 1$  ms which is the minimal time interval between two spikes. The synaptic weight from neuron  $i$  to neuron  $j$  is  $w_{ij}$  and  $\frac{G_{i,E}(t)}{G_0} = \sum_{j|w_{ij}>0} w_{ij}S_j(t - d_{ij})$  and is  $\frac{G_{i,I}(t)}{G_0} = -\sum_{j|w_{ij}<0} w_{ij}S_j(t - d_{ij})$ , where  $d_{ij}$  is the axonal transmission delay.

In addition to the standard update, we implement the following spike-frequency adaptation (see [17]) based on threshold adaptation:

$$\tau_{SFA} \frac{d\theta_i}{dt} = -\theta_i + k_{SFA} S_i(t)$$

with a linear threshold decay (with time constant  $\tau_{SFA} = 300$  ms) compensated at each spike (with shift parameter  $k_{SFA} = 100$ ), increasing the excitability of the inactive neurons and decreasing the excitability of the active ones.

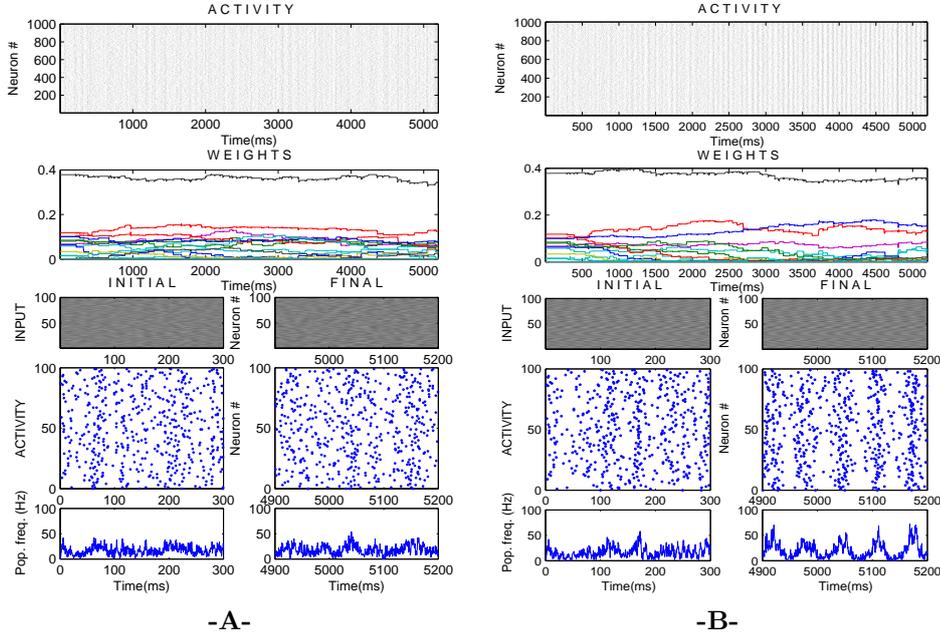


Fig. 1: Excitatory population activity during STDP learning. -A- Non-periodic input -B- Periodic input.

place when a post-synaptic spike is associated with a strong pre-synaptic trace (recent pre-synaptic excitations). The second term corresponds to the synaptic depression: the weight significantly decreases when a pre-synaptic pulse is associated with a strong post-synaptic trace (i.e. after post-synaptic excitation). The result is a global potentiation of pre-post sequences of excitation and a depression of post-pre sequences of excitation. This rule is strictly antisymmetrical, i.e. the potentiation compensates the depression, so it can be considered as “balanced”. From a computational point of view, the storage of a trace is not very expensive, strictly local and as such biologically plausible.

There is no upper bound, but in order to prevent uncontrolled weight drift, the total amount of synaptic weights  $W_i = \sum_j w_{ij}$  is maintained constant with the following scaling factor:  $\beta_i(t) = \frac{\sum_j w_{ij}(t-dt)}{\sum_j w'_{ij}(t)}$  so that:  $w_{ij}(t) = \beta_i(t)w'_{ij}(t)$ . Moreover, the sign change is forbidden (the lower bound is thus zero in the case of excitatory weights).

## 2 Simulations

We take a network composed of  $N_E = 1000$  excitatory neurons and  $N_I = 400$  inhibitory neurons. The average number of afferent synapses is equal to  $K = 100$ , giving a 10% density for the excitatory inputs and 25% density for the inhibitory

inputs. The average synaptic sum from the different synaptic tracts are  $A_{EE} = A_{IE} = 10$  and  $A_{EI} = A_{II} = -10$ , with variance  $H_{EE} = H_{EI} = H_{IE} = H_{II} = 2$ . The excitatory and inhibitory tracts are thus expected to balance each other on average, with a variance of the total synaptic sum equal to 4.

Networks are then randomly drawn and initialized. Then, the activity is observed for several milliseconds under different input conditions. The parameters have been carefully chosen in order to favour a low firing rate<sup>2</sup>. In the absence of any external input, the network activity collapses to 0 in few steps<sup>3</sup>. As observed in many other simulation setups, a small external signal is enough to maintain a realistic low-firing rate regime (often considered as the default activity in the cortex). In order to avoid unexpected synchronization, we take a broad distribution of synaptic delays : the delays  $d_{ij}$  are Poisson distributed. The minimum delay is 6 ms. The average delay is 10 ms, i.e.  $d_{ij} \sim 6 + \mathcal{P}(\lambda)$  with  $\lambda = 4$  ms.

The external input represents the activity from distant areas. For the purpose of plasticity and learning, we build specific input signals, having the possibility to contain reproducible features. We thus construct time-varying signals

$$\mathbf{I}(t) = \mathbf{P}_1(t) \sin(2\pi ft) + \mathbf{P}_2(t) \sin(2\pi ft + \frac{\pi}{2})$$

with  $\mathbf{P}_1(t)$  and  $\mathbf{P}_2(t)$  being renewable gaussian spatial patterns and  $f$  (angular speed), equal to 30 ms in simulations. In the periodic case,  $\mathbf{P}_1$  and  $\mathbf{P}_2$  are drawn once according to  $\mathcal{N}(0, \frac{1}{\sqrt{\tau_m}})$  and remain constant throughout learning, so that the period of the signal is  $f$ . In the non-periodic case,  $\mathbf{P}_1$  is redrawn when  $2\pi ft = 0 \pmod{2\pi}$  and  $\mathbf{P}_2$  is redrawn when  $2\pi ft = \frac{\pi}{2} \pmod{2\pi}$ , which introduces novelty and cancels the periodicity.

After a short relaxation (200 ms), the STDP plasticity is activated for 5 s *on the excitatory synapses of excitatory neurons only*. We represent in figure 1 the population response in both cases (aperiodic and periodic input signals). The two simulations are done on the same network. Only the input signals differ. The two upper panels present the global activity of the excitatory population during the whole simulation. The initial activity is weak (approx. 20-30 Hz), non-periodic in both cases, whereas small  $\gamma$ -like oscillations can be detected with deeper inspection. During learning, the weights change in a similar manner in

<sup>2</sup>The framework of balanced neural networks is very popular for it gives account for the statistics of the spike trains observed in vivo in cortical layers, and also for the high variability observed in the membrane potential of neurons. Balanced neural networks are now considered as the canonical form of connectivity for the simulation of realistic large scale ‘‘cortical’’ neural networks. For building such a model, two populations of neurons must be defined, one of excitatory neurons, the other of inhibitory neurons, each population owning self-feeding recurrent connection and sending axons to the other population. The ‘‘balance’’ is ensured as soon as the expectation of the total synaptic sum is zero, i.e.  $A_{EE} = -A_{EI}$  and  $A_{IE} = -A_{II}$ , where  $E$  represents the excitatory population,  $I$  represents the inhibitory population and  $A_{\alpha\beta}$  represents the  $\beta \rightarrow \alpha$  amplification, with  $\alpha, \beta \in \{E, I\}$ . Strong values for the amplifications drive the networks to ‘‘high conductance’’ states where the network is expected to have shorter reaction times and a linear response to external signal.

<sup>3</sup>The network is however close to a bifurcation point ( $H_{tot} > 2.2$ ) where a robust spontaneous activity can emerge from the self-feeding connections (without external stimulation).

both cases, without saturation or drift. In the first case (non-periodic input), the final activity is very similar to the initial one : same average population frequency, same non-periodicity, light gamma oscillations. In the second case, a clear enhancement of the response is observed. The average frequency remains approximately the same, but the population response becomes periodic at the population level, whereas individual neurons keep firing irregularly. This global synchronization takes place in the beta range, with a 60 ms period. In more detail (i) the small gamma oscillations are kept embedded in the larger beta oscillations and (ii) the beta synchronous response is temporally coupled to the input, where 60 ms is exactly the double of the input 30 ms period.

### 3 Discussion

A first and interesting observation, that was already noticed in [16] is the almost absence of any effect in the non-periodic case. Knowing that in a network of pure Poisson neurons, the STDP potentiations and the depressions globally compensate, the absence of global effect obtained in our first simulation suggests a very irregular underlying activity (i.e. close to pure random) without emerging structure.

On the contrary, the global population effect obtained in the second case suggests that a periodic response is indeed present in the initial activity, whereas difficult to detect from the chaotic self-sustained activity. Only the reiteration of the same pattern allows the network to capture the hidden sequential regularities contained in the activity. The effect of the Hebbian rule being to increase the sequential chains of activation, a specific activity build-up is thus rapidly obtained. This activity build-up can be interpreted as a specific response or “action” at the population level. Interesting to this regard is the activity decrease that systematically follows the increase. This effect is probably a population effect due to the balance of strong excitation and strong inhibition in the network. In a balanced setting, a stronger than usual activation is expected to be followed by a compensatory response of the inhibitory population, leading to a slow oscillation regime, as observed in [2]. Interesting from this perspective is the coupling of the slow oscillations to the periodic input, the network being visibly “paced” by the input.

Finally, we hope those results clarify the role of STDP in recurrent neural networks, as a putative mechanism for shaping the population response (or population “action”) when reproducible sequences are present in the intrinsic activity. The presented results are fully reproducible with different initial draws, but a more systematic study remains to be done in order to clarify the range of parameters under which the considered mechanism is expected to take place. Those simulation results constitute a first insight toward a more systematic study of collective action as a large-scale equivalent of small-scale cellular action, in particular regarding network-wide decision making with respect to accumulated sensory (or sensori-motor) evidence.

## References

- [1] S. Amari. A method of statistical neurodynamics. *Kybernetik*, 14:201–215, 1974.
- [2] N. Brunel. Dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons. *Journal of Computational Neuroscience*, 8:183–208, 2000.
- [3] A. Lechner, M. Ahmadi, and J. Hertz. High conductance states in a mean field cortical network model. *Neurocomputing*, 58-60:935–940, 2004.
- [4] A. Kumar, S. Schrader, A. Aertsen, and S. Rotter. The high-conductance state of cortical networks. *Neural Computation*, 20:1–43, 2008.
- [5] W. Softky and C. Koch. The highly irregular firing of cortical cells is inconsistent with temporal integration of random epsps. *J. of Neuroscience*, 13(1):334–450, 1993.
- [6] A. Riehle, S. Grün, M. Diesman, and A. Aertsen. Spike synchronization and rate modulation differentially involved in motor cortical function. *Science*, 278:1950–1953, 1997.
- [7] E. Rodriguez, N. George, J.-P. Lachaux, J. Martinerie, B. Renault, and F.J. Varela. Perception’s shadow: long-distance synchronization of human brain activity. *Nature*, 397(6718):430–433, 1999.
- [8] C.A. Skarda and W.J. Freeman. How brains make chaos in order to make sense of the world. *Behav. Brain Sci.*, 10:161–195, 1987.
- [9] K. MacLeod and G. Laurent. Distinct mechanisms for synchronization and temporal patterning of odor-encoding cell assemblies. *Science*, 274:976–979, 1996.
- [10] J. Cassenaer and G. Laurent. Hebbian stdp in mushroom bodies facilitates the synchronous flow of olfactory information in locusts. *Nature*, 448:709–713, 2007.
- [11] Guo-Qiang Bi and Mu-Ming Poo. Synaptic modifications in cultured hippocampal neurons : Dependence on spike timing, synaptic strength, and postsynaptic cell type. *The Journal of Neuroscience*, 18(24):10464–10472, 1998.
- [12] L.F. Abbott, Sen Song, Kenneth D. Miller. Competitive hebbian learning through spike-timing dependent synaptic plasticity. *Nature*, 2000.
- [13] Rudy Guyonneau, Rudy VanRullen, and Simon Thorpe. Neurons tune to the earliest spikes through stdp. *Neural Computation*, 17:559–879, 2005.
- [14] Eugene M. Izhikevich, Joseph A. Gally, and Gerald M. Edelman. Spike-timing dynamics of neuronal groups. *Cerebral Cortex*, 14:933–944, 2004.
- [15] Eugene M. Izhikevich. Polychronization: Computation with spikes. *Neural Computation*, 18:245–282, 2006.
- [16] Abigail Morrison, Ad Aertsen, and Markus Diesmann. Spike-timing-dependent plasticity in balanced random networks. *Neural Computation*, 19:1437–1467, 2007.
- [17] Boris Gutkin and Fleur Zeldenrust. Spike-frequency adaptation. *Scholarpedia*, under review.
- [18] J.-P. Pfister and W. Gerstner. Triplets of spikes in a model of spike timing-dependent plasticity. *J. Neurosci.*, 26:9673–9682, 2006.