Spike timing analysis in neural networks with unsupervised synaptic plasticity

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Spike timing analysis in neural networks with unsupervised synaptic plasticity

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Abstract. The synaptic plasticity rules that sculpt a neural network architecture are key elements to understand cortical processing, as they may explain the emergence of stable, functional activity, while avoiding runaway excitation. For an associative memory framework, they should be built in a way as to enable the network to reproduce a robust spatio-temporal trajectory in response to an external stimulus. Still, how these rules may be implemented in recurrent networks and the way they relate to their capacity of pattern recognition remains unclear. We studied the effects of three phenomenological unsupervised rules in sparsely connected recurrent networks for associative memory: spike-timing-dependent-plasticity, short-term-plasticity and an homeostatic scaling. The system stability is monitored during the learning process of the network, as the mean firing rate converges to a value determined by the homeostatic scaling. Afterwards, it is possible to measure the recovery efficiency of the activity following each initial stimulus. This is evaluated by a measure of the correlation between spike fire timings, and we analysed the full memory separation capacity and limitations of this system.

Keywords: unsupervised synaptic plasticity; spiking neurons; homeostasis; STDP; STP.
PACS: 87.18.Sn, 87.19.lg, 87.19.lj, 87.19.lp, 87.19.lv, 87.19.lw

INTRODUCTION

The ever-going changes in synaptic connections are thought to play a major role in a neural network's memory capacity. In this work we investigate how experimentally derived plasticity rules may affect their dynamics within a recurrent architecture, and whether they might give rise to some kind of associative memory. More specifically, we focused on homeostatic scaling[1, 2, 3] with pre-synaptic dependency[4] and spike-timing-dependent-plasticity[5, 6, 7] and their relation to the recovery of patterns of precise timing of spikes, as observed for example in some layers of the visual cortex[8].

SIMULATION DETAILS

We used two models for neural dynamics: an analytically solvable integrate-and-fire with Dirac's delta interactions and an Izhikevich model for pyramidal neurons[9] with conductance-based synapses, and added short-term-plasticity (as in [9]) in both for stability. The system consists of $N = 500$ neurons, being 80% excitatory and 20% inhibitory, randomly connected with probabilities taken from the literature [10].

The simulation starts with a quiescent network to which is imposed a pattern of neuronal spikes. The synaptic weights are updated after the transient activity ceases, in a trial scheme (such as [4]). As the synaptic weights evolve, the network starts to

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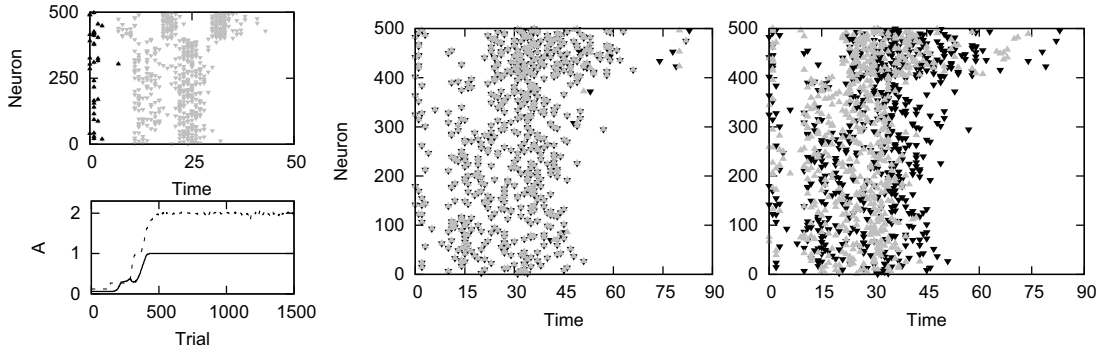

FIGURE 1. On the left: development of activity during learning time on the bottom (full line is the mean A for excitatory neurons, dotted is for inhibitories), and on top, raster plot of the untrained input (black) and subsequent response after learning (grey). On the centre and on the right, juxtaposition of raster plots from two different trials (black and grey symbols): same learned input and different learned inputs respectively. Note that neurons 1-400 are excitatories and 401-500 are inhibitories.

develop activity following the input (Fig. 1), and it converges to a mean value A_{Goal} defined by the homeostatic scaling rule,

$$W_{ij}^{\nu+1} = W_{ij}^{\nu} + \alpha_W A_j^{\nu}(A_{Goal} - A_i^{\nu})W_{ij}^{\nu}$$

where W_{ij}^{ν} is the synaptic weight from the neuron j to the neuron i and A_i^{ν} is an accumulated mean value of the number of spikes S_i^{ν} of the neuron i in the trial ν:

$$A_i^{\nu+1} = A_i^{\nu} + \alpha_A(S_i^{\nu} - A_i^{\nu}) \quad .$$

A_j is the pre-synaptic dependence factor, used as a mechanism to further stabilize the activity. A_{Goal} was set to 1 spike per trial for excitatories and 2 for inhibitories.

Correlation measure

The correlation measured between the activities of two trials

$$C^{\nu\nu'} = \frac{1}{\gamma}\sum_i \sum_{k,l=1}^{S_i^{\nu}, S_i^{\nu'}} e^{-\left(\frac{t_{i,k}^{\nu} - t_{i,l}^{\nu'}}{2\sigma^2}\right)^2}$$

$$\gamma = \left[MAX(S^{\nu}, S^{\nu'}, N)\right]$$

compares the k^{th} spike time of the neuron i in the trial ν with the l^{th} time, the closest one, in the trial ν', with a tolerance of $\sigma = 1$ms. $C^{\nu\nu'}$ is normalized by γ, the largest value within either the number of spikes of one of the trials, or N. This is to take into account that the activity should obey the homeostasis constraint of 1 spike for each excitatory neuron in one trial. Fig. 2 shows the simulation results up to 15 trained patterns. It is possible to see that it recovers the activity if a trained pattern is presented again ($C^{\nu\nu'} \approx 1$), and that it is different from the other trained ones ($C^{\nu\nu'} \approx 0$).

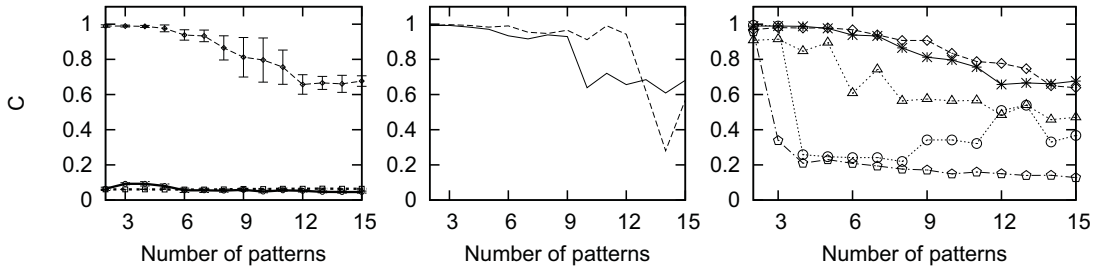

FIGURE 2. On the left, averages for correlation between the same input (dashed line), two different trained inputs (full) and a trained and an untrained one (dotted). On the centre, correlation measures for two presentations of the same pattern with (full line) and without (dashed) STDP. On the right, averages for correlation with the same pattern with varying network connectivity values. The stars are the same of the other graphs, excitatory→excitatory: 0.12, excitatory→inhibitory: 0.2 and inhibitory→excitatory: 0.2. For the others, connectivities are scaled by: 0.75 (diamonds), 0.5 (triangles), 1.5 (circles) and 2 (pentagons).

CONCLUSIONS

We searched for associative memory of spike patterns and presented results of timing correlation as a function of increasing trained patterns, which also enhances the network level of recurrence. There were no major differences of activity behaviour or correlation capacities between the two models of synapses used (static charges for integrate-and-fire or conductance-based for Izhikevich model). The pre-synaptic dependence factor A_j introduces a Hebb-like relation to the connections, making the homeostatic scaling sufficient for the recovery of spike timings. The system without STDP actually yields similar activity and correlation results for a small number of learned inputs. We also checked that the network connectivity sparseness heavily influences this capacity, and there is an optimal range for it (Fig. 2).

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