Unsupervised learning in neural networks with short range synapses

L. G. Brunnet, E. J. Agnes, B. E. P. Mizusaki and R. Erichsen Jr.

Instituto de Física, Universidade Federal do Rio Grande do Sul, Caixa Postal 15051, 9150-970 Porto Alegre, RS, Brazil.

Abstract. Different areas of the brain are involved in specific aspects of the information being processed both in learning and in memory formation. For example, the hippocampus is important in the consolidation of information from short-term memory to long-term memory, while emotional memory seems to be dealt by the amygdala. On the microscopic scale the underlying structures in these areas differ in the kind of neurons involved, in their connectivity, or in their clustering degree but, at this level, learning and memory are attributed to neuronal synapses mediated by long-term potentiation and long-term depression. In this work we explore the properties of a short range synaptic connection network, a nearest neighbor lattice composed mostly by excitatory neurons and a fraction of inhibitory ones. The mechanism of synaptic modification responsible for the emergence of memory is Spike-Timing-Dependent Plasticity (STDP), a Hebbian-like rule, where potentiation/depression is acquired when causal/non-causal spikes happen in a synapse involving two neurons. The system is intended to store and recognize memories associated to spatial external inputs presented as simple geometrical forms. The synaptic modifications are continuously applied to excitatory connections, including a homeostasis rule and STDP. In this work we explore the different scenarios under which a network with short range connections can accomplish the task of storing and recognizing simple connected patterns.

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INTRODUCTION

Since the original works on artificial neuron networks in the last century [1] the scientific contact among physicists and biologists has increased considerably. These attempts converged to a more realistic description of the phenomena and has enriched the knowledge of this field. Versions of model neurons [2] adapted to specific needs and conditions have been proposed along the last fifty years, but just rather recently experiments have advanced to allow for a detailed description of Hebbian like synapses [3, 4] and on mechanisms to regulate network homeostasis [6, 7]. Detailing the connections is also a hard task and it is frequently supposed that they happen involve many neurons. Network models then usually assume a fraction of random connections among neurons and search for properties related to pattern learning, associative memory and storage capacity. This random connections construction is quite artificial since neurons are physical entities that will contact their closest neighbors with a greater probability than farther ones. Here we ask how far should neurons be connected in order to reproduce the expected network properties. The aim of this work is to construct a model neuron lattice with local synaptic connections and to search for the conditions under which spatially induced pattern memories may be recovered.

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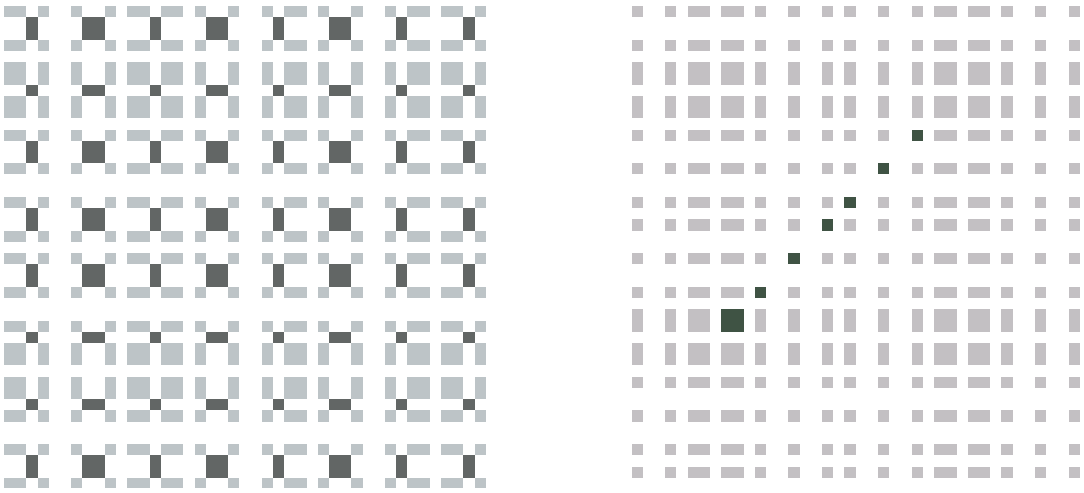

FIGURE 1. Left: lattice topology. Excitatory neurons (pale gray) and inhibitory neurons (dark gray). Right: Representation of excitatory neurons, typical pattern marked on excitatory neurons (dark gray)

THE NEURON LATTICE

The lattice nodes are composed by integrate and fire Izhikevich [8] neurons which are either regular spiking excitatory neurons or fast spiking inhibitory neurons in a proportion of 1 inhibitory to 4 excitatory. Excitatory synaptic currents include both AMPA and NMDA modeling terms and the inhibitory ones contain $GABA_A$ and $GABA_B$ terms. The regular lattice used in the simulations (Fig. 1) has 256 excitatory and 64 inhibitory neurons. In this work we explore two types of connections: i) first neighbors; ii) first and second neighbors.

Presynaptic dependent scaling (PSD) [7] is one of the mechanisms used for synaptic modification. Here it is presented in a continuous version. First we define activity A_i for a neuron i;

$$\tau_A \frac{dA_i}{dt} = (S_i - A_i) \tag{1}$$

where $S_i \rightarrow \sum_k \delta(t - t^k)/t_{max}$ is related to the number of spikes of neuron i in the interval t_{max}. With this definition the weight between the presynaptic neuron i and the postsynaptic neuron j are modified by

$$\tau_w \frac{dW_{ij}}{dt} = \frac{A_i}{A_{GOAL}} \frac{(A_{GOAL} - A_j)}{A_{GOAL}} W_{ij} \ . \tag{2}$$

The second mechanism used for synaptic modification is spike time dependent plasticity (STDP) which increases (decreases) synaptic intensity when the presynaptic neuron fires before (after) the postsynaptic one. This can be modeled [7] (also in a continuous version) by the expression:

$$\frac{dW_{ij}}{dt} = 1 + \sum_{k=1}^{K} \sum_{l=1}^{L} F(t_l^j - t_k^i - \delta_{ij}) \tag{3}$$

where t_l^j is the l^{th} spike of neuron j, δ_{ij} is the synaptic delay and

$$F(\Delta t) = \begin{cases} c_p \exp(-\Delta t/\tau_p), & \Delta t > 0 \\ -c_d \exp(\Delta t/\tau_d), & \Delta t \leq 0 \end{cases} \quad (4)$$

RESULTS AND CONCLUSIONS

We analyzed the two mechanisms for synaptic modification separately. The protocol for the input is to use low frequency (0.2 Hz) injection of current to a line of eight neighboring neurons. We have chosen 4 different directions for these lines: vertical, horizontal and two diagonal lines with $\pm 45^\circ$ (cross pattern). When studying PSD only these currents are presented simultaneously; in the case of STDP they are separated by 6 ms. Yet for STDP, the procedure is repeated until a first neuron reaches the maximal synaptic weight. After that only the first neuron of the line is excited to test if the pattern has been learned. In both cases the initial synaptic weighs were fixed do half the maximal value. The inhibitory weights were kept constant during all simulations.

In the case of PSD being the only mechanism the input protocol is applied constantly at 0.2 Hz and we observe the lattice response. Two distinct behaviors are found for first neighbor synapses: either the initial wave produced by the neurons line induces spiking in some near by neurons and fades out or it produces a wave that propagates by whole system. The parameter governing the transition is the maximal weight. This behavior happens for any of the four excitation lines.

In the case of STDP only the lattice response depends on geometric details: only vertical or horizontal patterns are learned with first neighbor synapses. Obviously diagonal lines cannot be memorized since their neighboring neurons lines are not connected. When considering second neighbors only for excitatory neurons not just these patterns are memorized but also some nearby ones are incorrectly excited. The correct pattern reproduction for some diagonal lines (not all of them) only happens when second neighbors are considered for inhibitory neurons and the patterns are not simultaneously presented. If simultaneous patterns crossing at some point are presented, the part after the crossing is not retrieved. Future work should consider both PSD and STDP and also extend the synapses neighborhood and gradually test the effect of long range connections.

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REFERENCES

1. W. McCulloch, and W. Pitts, *B*ull. Math. Biophys. **7**, 115-133 (1943); S. Grossberger, *J*. Stat. Phys. **1**, 319-350 (1969); J.J. Hopfield, *P*roc. Nat. Acad. Sci. USA **8**, 2554-2558 (1982).
2. A. L. Hodgkin, and A. F. Huxley, *J. Physiol.* **117** 500-544 (1952); J. L. Hindmarsh, and R. M. Rose, *P*roc. R. Soc. London, Ser. B **221** 87-102 (1984);
3. C. C. Bell,V.Z. Han, Y. Sugawara, and K. Grant, *Nature* **387** 278-281 (1997).

4. Bi G-Q, and Poo M-M, *J Neurosci* **18** 10464-10472 (1998).
5. W. Gerstner, et al., *Nature* **383**, 76–78 (1996).
6. G. G. Turrigiano, *Trends Neurosci.* **22**, 221–227 (1999).
7. J. K. Liu, and D. V. Buonomano, *J. Neurosci.* **29**, 13172–13181 (2009).
8. E. M. Izhikevich, and G. M. Edelman, *Proc. Natl. Acad. Sci. USA* **105**, 3593–3598 (2008).