Emergence of Oriented Cell Assemblies Associated with Spike-Timing-Dependent Plasticity

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Abstract. We studied the emergence of cell assemblies out of a locally connected random network of 10,000 integrate-and-fire units distributed on a 100×100 2D lattice. The network was composed of 80% excitatory and 20% inhibitory units with balanced excitatory/inhibitory synaptic weights. Excitatory–excitatory synapses were modified according to a spike-timing-dependent synaptic plasticity (STDP) rule associated with synaptic pruning. In presence of a stimulus and with independent random background noise (5 spikes/s), we observed that after $5 \cdot 10^5$ ms of simulated time, about 8% of the exc–exc connections remained active and were reinforced with respect to the initial strength. The projections that remained active after pruning tended to be oriented following a feed-forward converging–diverging pattern. This result suggests that topologies compatible with synfire chains may appear during unsupervised pruning processes.

1 Introduction

Massive synaptic pruning following over-growth is a general feature of mammalian brain maturation [1]. Pruning starts near time of birth and is completed by time of sexual maturation. Trigger signals able to induce synaptic pruning could be related to dynamic functions that depend on the timing of action potentials. Spike-timing-dependent synaptic plasticity (STDP) is a change in the synaptic strength based on the ordering of pre- and post-synaptic spikes. This mechanism has been proposed to explain the origin of long-term potentiation (LTP), i.e. a mechanism for reinforcement of synapses repeatedly activated shortly before the occurrence of a post-synaptic spike [2]. STDP has also

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been proposed to explain long-term depression (LTD), which corresponds to the weakening of synapses strength whenever the pre-synaptic cell is repeatedly activated shortly after the occurrence of a post-synaptic spike [3]. The relation between synaptic efficacy and synaptic pruning [4], suggests that the weak synapses may be modified and removed through competitive "learning" rules. Competitive synaptic modification rules maintain the average neuronal input to a post-synaptic neuron, but provoke selective synaptic pruning in the sense that converging synapses are competing for control of the timing of post-synaptic action potentials [5].

The originality of our study stands on the size of the network, 10,000 units, the duration of the experiment, 500,000 ms, and the application of an original bio-inspired STDP modification rule compatible with hardware implementation [6]. In this study the synaptic modification rule was applied only to the exc–exc connections. This plasticity rule might produce the strengthening of the connections among neurons that belong to cell assemblies characterized by recurrent patterns of firing. Conversely, those connections that are not recurrently activated might decrease in efficiency and eventually be eliminated. The main goal of our study is to determine whether or not, and under which conditions, such cell assemblies may emerge from a large neural network receiving background noise and content-related input organized in both temporal and spatial dimensions.

2 Model

The complete neural network model is described in details in [7] and we present here only a sketch description of the model. 10,000 integrate-and-fire units (80% excitatory and 20% inhibitory) were laid down on a 100×100 2D lattice according to a space-filling quasi-random Sobol distribution. Sparse connections between the two populations of units were randomly generated according to a two-dimensional Gaussian density function such that excitatory projections were dense in a local neighborhood, but low probability long-range excitatory projections were allowed. Edge effects induced by the borders were limited by folding the network as a torus. The state of the unit (spiking/not spiking) was a function of the membrane potential and a threshold. The states of all units were updated synchronously and the simulation was performed at discrete time steps corresponding to 1 ms. After spiking, the membrane potential was reset, and the unit entered a refractory period lasting 2 time steps. For the simulation runs presented here each unit received a background activity following an independent Poisson process and the "spontaneous" mean firing rate of the units was $\lambda = 5$ spikes/s.

It is assumed *a priori* that modifiable synapses are characterized by discrete activation levels that could be interpreted as a combination of two factors: the number of synaptic *boutons* between the pre- and post-synaptic units and the changes in synaptic conductance as a result of Ca^{2+} influx through the NMDA receptors. In the current study we attributed a fixed activation level (meaning no synaptic modification) $A_{ji}(t) = 1$, to exc–inh, inh–exc, and inh–inh synapses

while activation levels were allowed to take one of $A_{ji}(t) = \{0, 1, 2, 4\}$ for exc–exc synapses, $A_{ji}(t) = 0$ meaning that the projection was permanently pruned out.

3 Simulation

The simulator was a custom, Open Source, C program that relies on the GNU Scientific Library (GSL) for random number generation and quasi-random Sobol distribution implementations. With our current implementation and setup at the University of Lausanne, a 10,000 units network simulation for a duration of $5 \cdot 10^5$ time steps lasted approximatively 3 hours, depending on the network global activity. A complete simulation lasted for $5 \cdot 10^5$ discrete time steps, corresponding to about 8.5 minutes of simulated time. After a stabilization period of 1000 ms without any external input, a 200 ms long stimulus was presented every 2,000 ms for the rest of the simulation duration, i.e. 250 presentations of the stimulus. The stimulus was composed of 10 vertical bars uniformly distributed over the 100×100 2D lattice surface, each bar being 1 column wide. At each time step, the 10 bars were simultaneously moved one column to the right, such that each bar slipped over the complete surface of the network twice per presentation. The stimulus applied on a particular unit provoked a strong depolarization that always induced the unit to discharge except if the stimulation occurred during the refractory period. For each input unit, one stimulus presentation corresponded to a sequence of 20 external inputs regularly distributed in time every 10 ms. At network level, each stimulus presentation resulted in 10 groups of about 80 synchronously spiking excitatory units repeating 20 times a 10 ms long spatio-temporal sequence. The two following presentation protocols have been applied:

Fixed stimulus. Before simulation started, 10% of the excitatory units (800 units) were randomly chosen to become permanently the input units of the network for the entire simulation.

Random stimulus. Before each stimulus presentation, 10% of the excitatory units (800 units) were randomly chosen to become the input units of the network for that particular presentation only.

4 Results

The complete status of the network was dumped at fixed intervals, providing information on the strength of the connections after the STDP–driven synaptic plasticity and pruning. Network activity was recorded as a multivariate time series formatted like a multi site spike train recordings at a resolution of 1 ms. The firing pattern of each unit could be characterized by first- and second-order time domain analyses using the programs and tools accessible from the OpenAdap.Net project[1]. We performed all simulations with both fixed and random

[1] `http://www.openadap.net/`

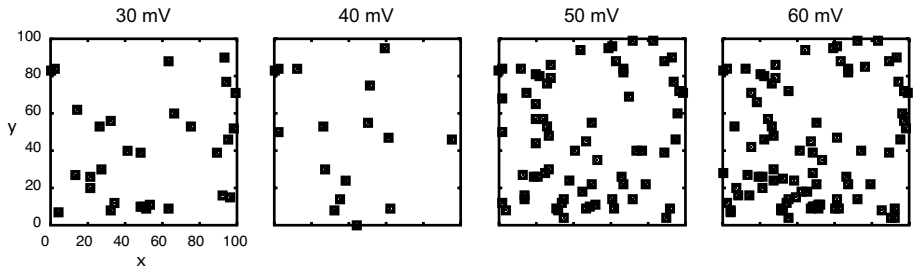

Fig. 1. Example of the location of strongly interconnected units as a function of the intensity of the fixed stimulation. Some units of this pool appeared already at low stimulation intensities but an increasing number of units appeared with an increase of the stimulation intensity.

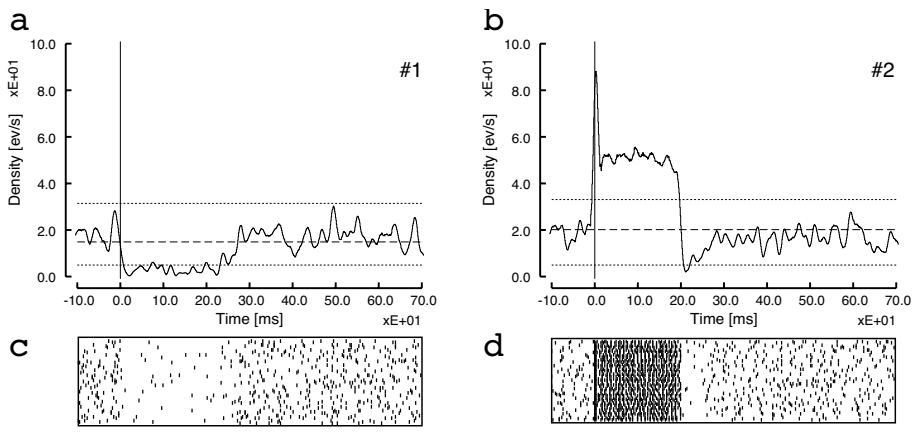

Fig. 2. Response of 2 sample strongly interconnected units responding to 50 presentations of the fixed stimulation between time $t = 450$ and $t = 500$ seconds from the simulation start. (a) and (b) peri-event densities (PSTH) for the last 50 presentations of the stimulus; (c) and (d) corresponding raster plots.

stimuli, using identical parameters and pseudo-random number generator seed. The emergence of stimulus-driven cell assemblies was determined by the comparison of the networks obtained after fixed stimulation vs. random stimulation. We considered the 7,200 excitatory units that were not directly stimulated. Among these units we could identify a group that maintained throughout the whole simulation run at least one strong (*i.e.*, $A_{ji} = 4$) incoming and one strong outgoing projection from and to other units showing the same properties. We dubbed these units *strongly interconnected units*. The count of strongly interconnected units was strongly dependent on the type of stimulation. An increase of the intensity of the fixed stimulation provoked a increase in the count of strongly interconnected units as shown in Fig. 1. On the opposite, the intensity of the random stimulation had a weak effect on the count of strongly interconnected units.

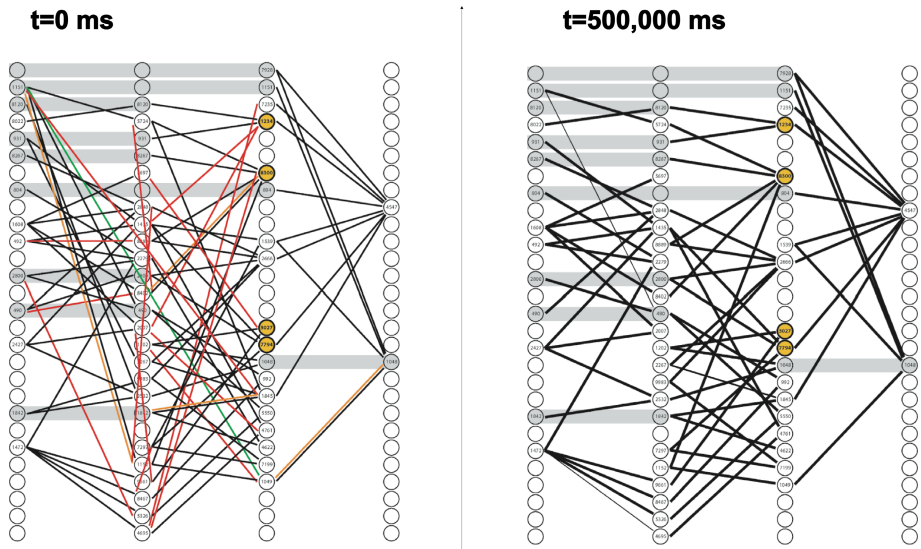

Fig. 3. Selected strongly interconnected units appear to be embedded into a layered circuit. The left panel shows the connections at the begin of the simulation and the right panel at the end. See text for more details.

The majority of the strongly interconnected units (79%) were excited by the fixed stimulation (*e.g.* Fig. 2b) despite none of these units was directly stimulated. The remaining strongly interconnected units (21%) showed a significant inhibition during the presentation, followed by an offset inhibition generally extending nearly 50 ms after the offset of the stimulus (*e.g.* Fig. 2a). The other units, that were not directly stimulated neither belonging to the strongly interconnected group, were mainly inhibited during the stimulation presentation, as the balanced network reacted to the large stimulation input by increasing the inhibition.

Fig. 3 shows the evolution of the interconnections among a group of strongly interconnected units, two of which were represented at Fig. 2. The left panel shows that at the begin of the simulation the assembly is interconnected by a mixture of feed-forward and feed-back projections. These projections were indeed set at random according to the topographic rules described elsewhere [7]. After 500,000 ms the effect of synaptic pruning driven by the stimulus and by STDP let emerge an oriented feed-forward topology because only feed-forward connections remained active.

5 Discussion

Synfire chains are diverging / converging chains of neurons discharging synchronously to sustain the propagation of the information through a feed-forward

neural network [8]. This theoretical model has proven to be very efficient for the transmission of precisely timed information through the cerebral cortex but the mechanisms that may underlie its appearance in the mature brain have never been deeply investigated. This work is aimed at investigating whether synfire chains partially embedded in a large circuit characterized by initial "random" connections may emerge following activity-driven mechanisms. The rationale is that selected synaptic pruning may drive the emergence of synfire chains following certain stimulus patterns. Our results suggest that topologies compatible with synfire chains may appear during unsupervised pruning processes but further investigation is required to determine if self-sustained synfire activity may appear in the emerging networks that we have observed [9].

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References

1. Rakic, P., Bourgeois, J. P., Eckenhoff, M. F., Zecevic, N., Goldman-Rakic, P. S.: Concurrent overproduction of synapses in diverse regions of the primate cerebral cortex. Science **232** (1986) 232–5
2. Bi, G. Q., Poo, M. M.: Synaptic modifications in cultured hippocampal neurons: dependence on spike timing, synaptic strength, and postsynaptic cell type. J Neurosci. **18** (1998) 10464-72
3. Karmarkar, U. R., Buonomano, D. V.: A model of spike-timing dependent plasticity: one or two coincidence detectors? J Neurophysiol. **88** (2002) 507–13
4. Chechik, G., Meilijson, I., Ruppin, E.: Neuronal Regulation: A Mechanism for Synaptic Pruning During Brain Maturation. Neural Computation **11** (1999) 2061–80
5. Song, S., Abbott, Larry F.: Cortical Development and Remapping through Spike Timing-Dependent Plasticity. Neuron **32** (2001) 339–50
6. Eriksson, J., Torres, O., Mitchell, A., Tucker, G., Lindsay, K., Rosenberg, J., Moreno, J.-M., Villa, A.E.P.: Spiking Neural Networks for Reconfigurable POEtic Tissue. Lecture Notes in Computer Science **2606** (2003)
7. Iglesias, J., Eriksson, J., Grize, F., T., Marco, Villa, A.E.P.: Dynamics of Pruning in Simulated Large-Scale Spiking Neural Networks. Biosystems **79** (2005) 11–20
8. Abeles, M.: Corticonics: Neural Circuits of the Cerebral Cortex. Cambridge University Press (1991)
9. Tetzlaff, T., Morrison, A., Geisel, T., Diesmann, M.: Consequences of realistic netowrk size on the stability of embedded synfire chains Neurocomputing **58-60** (2004) 117–21