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Model architecture for associative memory in a neural network of spiking neurons

Everton J. Agnes *, Rubem Erichsen Jr., Leonardo G. Brunnet

Instituto de Física, Universidade Federal do Rio Grande do Sul, Caixa Postal 15051, 91501-970 Porto Alegre, RS, Brazil

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ABSTRACT

A synaptic connectivity model is assembled on a spiking neuron network aiming to build up a dynamic pattern recognition system. The connection architecture includes gap junctions and both inhibitory and excitatory chemical synapses based on Hebb's hypothesis. The network evolution resulting from external stimulus is sampled in a properly defined frequency space. Neurons' responses to different current injections are mapped onto a subspace using Principal Component Analysis. Departing from the base attractor, related to a quiescent state, different external stimuli drive the network to different fixed points through specific trajectories in this subspace.

1. Introduction

A living being interacts with the world receiving external stimuli like light, sound or chemical molecules. The whole set of possible stimuli must be encoded and identified by the brain. Encoding has been the subject of several works that relate responses from animal brains to external influences [1–5]. However, knowledge about the way information is handled by the neural network remains incomplete.

Relevant information concerning sensory input processing in natural neural networks can be extracted when high-dimensional experimental data is projected onto a low-dimensional subspace using reduction methods. Principal Component Analysis (PCA) [6] is one of these methods. It allows the separation of correlated variables in a way such that it is possible to order the highest variance with the first axis, the second highest variance with the second axis and so on. We will detail its use in this work later in the result analysis.

One example of sensory input processing is given in the work by Lin et al. [2]. There, the time response of individual neurons to external stimuli on the mouse hippocampus has been recorded, and using a dimension reduction method known as Multiple Discriminant Analysis (MDA), they have found that there are fixed points on a specific subspace, which represent a set of episodic experiences. Nevertheless, due to its relative simplicity and well-known anatomical characteristics, the olfactory system of insects is a natural model to analyze sensory information processing. Using PCA, Mazor and Laurent [7] studied the projection neurons of the locust olfactory system, specifically, the Antennal Lobe (AL) ones. They have found that, without stimulus, the system remains at a quiescent state. At the onset of odor presentation, the system starts a stimulus-dependent trajectory in the principal component subspace. If the stimulus remains, the system approaches a stimulus-dependent fixed point, and at the stimulus offset the system retrieves the quiescent fixed point. In recent works, Namiki and Kanzaki [8,9] have found a similar behavior concerning the moth olfactory system.

* Corresponding author. Tel.: +55 51 3308 6503; fax: +55 51 3308 7286.
E-mail addresses: everton.agnes@ufrgs.br (E.J. Agnes), rubem@if.ufrgs.br (R. Erichsen Jr.), leon@if.ufrgs.br (L.G. Brunnet).

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Although the usage of reduction methods like PCA is common in experimental works, they also emerge in theoretical and computational research. Early works on linear neuron models have shown that PCA may result from the learning process on feed-forward networks [10,11]. Recently these assumptions have been used in the context of spiking neurons to show that pattern discrimination capabilities may emerge under unsupervised learning [12].

Experimental results relate stimulus to response with no wiring specification, that is, without details about the encoding process. In this work, we propose a simple one-layer neural encoding system based on Hebb's rule. The odors (or any other external stimulus), intended to be recognized and discriminated by the system, are represented by random activation patterns. The model network recognition capability is evaluated through a configuration space reduction method. The main question we address is whether the simple wiring model linking spiking neurons proposed here is able to produce the same kind of stimulus–response relationship observed in experiments.

This paper follows with three sections. In Section 2, the connection architecture is presented, as well as the local neuron model to simulate it. Section 3 presents the numerical results obtained using the model described in Section 2. In Section 4, we conclude with a brief discussion.

2. The model

It is widely accepted that, in order to recognize an external stimulus, neurons within a network activate/deactivate each other via chemical synapses. Our construction starts following the simple hypothesis established by Hebb [13]: neurons responding similarly to a given stimulus have probability C of being connected to each other via excitatory synapse. This is extended to the negative form: neurons that do not respond similarly to a given stimulus have a probability D of being connected through inhibitory synapses. An example of an implementation of Hebb's rule on a network of spiking neurons using a symmetric coupling matrix between excitatory cells and global inhibition can be found in Ref. [14]. Another example of a distinct implementation of Hebb's rule on a network of realistic spiking neurons can be found in Ref. [15], where neuron synchronization based on a modular architecture provides associative retrieval of memory patterns. Ref. [16] shows an example of a neural network with fully random connections among the neurons.

In order to formally implement the connection hypotheses above, we introduce the concept of pattern as a neuron set responding to a specific external stimulus [11]. We suppose that the network recognizes p different input patterns (memories, odors, episodic experiences) $\{\eta_i^\mu\}$, with $i = 1, \ldots, N$, representing the neuron ensemble and $\mu = 1, \ldots, p$, the pattern indexes. Variable η_i^μ may assume values one or zero whether the neuron i responds or not to pattern μ. For simplicity, neurons are assumed to respond to each of the p stimuli with a homogeneous probability a. This means that, since the whole network has N neurons, each pattern is coded approximately by aN neurons chosen randomly. To ensure the robustness of the process, the quantity aN must be large. Supporting this argument, experimental results have shown that a large fraction of neurons initiates an intense active regime in the AL immediately after odor presentation [7]. This also implies that the patterns are formed by overlapping groups of neurons.

We propose that the net effect of chemical synapses on each neuron pair is a superposition of excitatory and inhibitory currents. Each neuron belonging to a pattern excites, on average, $\sim NaC$ neurons and inhibits, on average, $\sim N(1-a)D$ neurons due to this pattern. Furthermore, we want to handle excitation and inhibition between two neurons independently. This requires the definition of two synaptic matrices: an excitatory,

$$W_{ij}^e = \frac{1}{Na^2C} \sum_{\mu=1}^{p} C_{ij}^\mu \eta_i^\mu \eta_j^\mu \tag{1}$$

and an inhibitory,

$$W_{ij}^i = \frac{1}{Na(1-a)D} \sum_{\mu=1}^{p} D_{ij}^\mu \eta_j^\mu (1-\eta_i^\mu), \tag{2}$$

where the variable C_{ij}^μ (D_{ij}^μ) assumes values one or zero whether pattern μ increases or not excitation (inhibition) between neurons i and j with probability C (D). Since C_{ij}^μ and D_{ij}^μ are independent variables, the synaptic matrices are asymmetric.

In addition to the long-range chemical synapses, a neuron also interacts through gap junctions in its close neighborhood. As will be shown below, they play an important role in the recognition process. These connections, as well as the synaptic ones, will be introduced below in our network design following the Rulkov implementation.

A biologically adequate neuron model should reproduce the real well-known spiking/quiescence behavior. The map neuron model introduced by Rulkov [17] presents this property with a low computational cost. A review of the usage of map-based models can be found in Ref. [18].

The model neuron proposed by Rulkov is defined by the set of equations

$$x_i(t+1) = f(x_i(t), x_i(t-1), y_i(t) + \beta_i(t)) \tag{3}$$

and

$$y_i(t+1) = y_i(t) - \mu(x_i(t)+1) + \mu(\sigma_i(t) + \sigma), \tag{4}$$

where i is the neuron label, t is the discrete time and

$$f(x,\tilde{x},u)=\begin{cases}\alpha(1-x)^{-1}+u, & x\leqslant 0 \text{ and } \tilde{x}\leqslant 0\\ \alpha+u, & 0<x<\alpha+u \text{ and } \tilde{x}\leqslant 0\\ -1, & x\geqslant \alpha+u \text{ or } \tilde{x}>0.\end{cases} \tag{5}$$

Here, $x_i(t)$ is a fast variable which represents an appropriately scaled membrane voltage and $y_i(t)$ is a slow variable that introduces a second time scale, since the parameter μ has a small value ($\mu = 0.0005$). Different values of α and σ change the behavior of the individual map, while $\beta_i(t)$ and $\sigma_i(t)$ represent external input. The system is composed by a 2D square network $\aleph$ of $N = L \times L$ neurons.

Gap junctions are modeled by

$$I_i^{gap}(t)=\frac{g}{|\mathcal{V}|}\sum_{j\in\mathcal{V}_i}(x_j(t)-x_i(t)), \tag{6}$$

where $\mathcal{V}_i$ is the neighborhood of neuron i, defined as $\{j\in\aleph : |\mathbf{r}_j-\mathbf{r}_i|\leqslant R\}$, g is the conductivity, $|\mathcal{V}|$ is the number of neighbors, and R is the interaction range. The synaptic current injection to neuron i is written as [19]

$$I_i^{syn}(t+1)=\gamma I_i^{syn}(t)-I_i^{e}(t)-I_i^{i}(t). \tag{7}$$

Chemical contributions are given by

$$I_i^{k}(t)=g^k(x_i(t)-x_{rp}^k)\sum_{j\in\aleph}W_{ij}^k\chi_j(t) \tag{8}$$

where k indicates the type, $k = e$ for excitatory and $k = i$ for inhibitory ones, and $\chi_j(t) = 1$ if $x_j(t) > 0$ and zero otherwise. The excitatory and inhibitory reverse potentials are, respectively, $x_{rp}^e = 0$ and $x_{rp}^i = -1.1$. The parameter γ in (7) controls the relaxation rate of synaptic input ($0\leqslant\gamma<1$). Parameters g^e and g^i are conductivities, allowing a fine adjustment of excitatory and inhibitory current injections, respectively.

With the above definitions, the external parameters in (3) and (4) become

$$\beta_i(t)=\beta^e(I_i^{gap}(t)+I_i^{syn}(t)+I_i^{ext}(t)), \tag{9}$$

$$\sigma_i(t)=\sigma^e(I_i^{gap}(t)+I_i^{syn}(t)+I_i^{ext}(t)), \tag{10}$$

where β^e and σ^e are coefficients which set the balance between the couplings for the fast and slow processes in the cells, respectively. The term $I^{ext}(t)$ in (9) and (10) is the external current, which determines the external input.

The single neuron map has a well-known parameter diagram (α,σ) (see Ref. [17] for more details) and the line which divides the quiescent from the active behavior has the analytical form $\sigma\approx 1-\sqrt{\alpha}$. In order to keep the isolated neuron in a quiescent state but close to the activity edge by a small amount ζ, we set $\alpha = 3.5$, $\sigma = 1-\sqrt{\alpha}-\zeta$. This way, increasing parameter σ by ζ, the neuron enters the activity region with both tonic spiking and chaotic behavior [20]. Here we assume $\zeta = 0.05$. Finally, the parameters related to external currents on the isolated neuron are assumed as $\beta^e = 0.133$, $\sigma^e = 1.0$, $\gamma = 0.9$, $g^e = 0.04$, $g^i = 0.048$, $g = 0.1$ and $R = 2$.

Simulations were performed with periodic boundary conditions, imposed to electrical couplings. All neurons are initially set to the quiescent state.

It is known that the Rulkov model is capable of presenting two specific behaviors besides quiescence: spiking and spiking-bursting. We will not use spiking-bursting behavior, since the actual networks we are taking as the base do not present it. Moreover, the parameters are set in order for the system to act as an excitable media. In other words, it is conceived to present no self-sustained activity. Other works treating the Rulkov model specifically in a spiking-bursting regime can be found in Refs. [20,21].

3. Numerical results

In order to investigate the network ability for pattern recognition we follow a simple strategy: the external stimulus activating a pattern is simulated by choosing a fraction P_{ext} of neurons, among those encoding the pattern, to receive external input. That is, a pattern is presented to the network only through $aP_{ext}N$ neurons. This way we may test if the network is able to identify different stimuli to a given pattern.

The current injected into one of the chosen $aP_{ext}N$ neurons follows a Poisson process: at each map iteration, the neuron has probability p_c to start receiving an external current pulse lasting a time interval θ. The method intends to simulate the input coming from other network layers, as the AL receives it from the olfactory sensory neurons.

To proceed with the numerical simulations we set the remaining parameter values as follows: the number of patterns, $p = 20$, the probability for a neuron to encode a pattern, $a = 0.3$, the excitatory and inhibitory wiring probabilities, $C = D = 0.1$, and the stimulation probability, $P_{ext} = 0.5$. The total number of neurons is 1024 (32×32). This means, on average, 300 neurons per pattern, where 150 neurons receive external stimulus and each cell excites (inhibits) 30 (70)

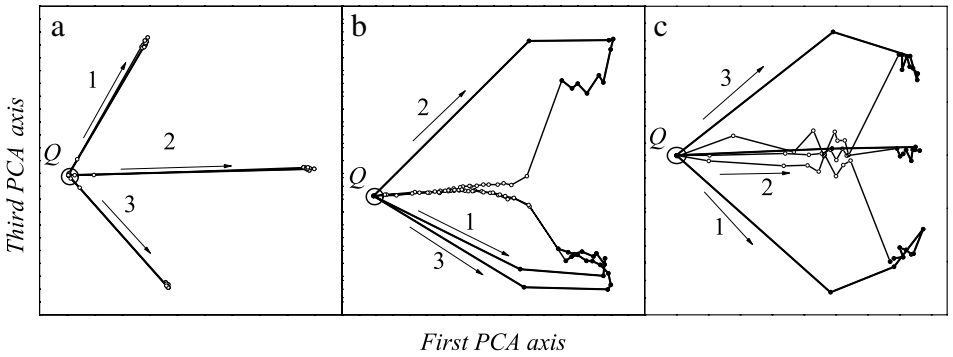

Fig. 1. Plot of the first (abscissa) and the third (ordinate) principal components from the Principal Component Analysis of the frequency vectors **M**. In (a), simulation without connections; in (b) both chemical and electrical connections; in (c) fully random connections. Each circle corresponds to a 50-iteration time bin with external input during 500 iterations. Full circles and thick lines correspond to input time intervals.

neurons due to one pattern. The three types of synapses are required to achieve the desired results: excitatory, to guarantee the activity of the same neurons within a pattern, independently of which are externally excited; inhibitory, to avoid the activity of neurons that do not belong to the activated pattern; and gap junctions, to stabilize the global quiescence state (baseline). One can consider 20 patterns a small number compared with the network size (1024 neurons), but here we intend to show just the architecture and the method.

The outcome of the simulation is a matrix, M_{ij}, with columns representing neuron indexes and rows representing time bins. These bins are defined within $(i-1)\tau < t < i\tau$. The value assigned to the matrix element is defined as the number of spikes generated by neuron j in this interval. Principal Component Analysis is applied to this matrix. Initially a covariance matrix is evaluated as

$$X_{jk} = \langle (M_{ij} - \langle M_{ij} \rangle)(M_{ik} - \langle M_{ik} \rangle) \rangle, \tag{11}$$

where

$$\langle M_{ij} \rangle = \frac{\tau}{T} \sum_{i=1}^{T/\tau} M_{ij} \tag{12}$$

and T is the simulation period. Then, the PCA transformation matrix, **R**, is composed by the eigenvectors of the matrix **X** taken as columns and arranged side by side in the decreasing order of its eigenvalues, that is, from maximal to minimal values. The resulting subspace is determined by the transformation $\mathbf{S} = \mathbf{R} \cdot \mathbf{M}$, where S_{ij} is the jth component of the ith vector in the subspace. The same procedure is done on experimental works [7,9] for the frequency space of the real AL. In this manner, we may compare the experimental trajectories projected on the main axes, produced during odor presentation, with our simulation of this process. This would also test the Hebbian-like encoding procedure proposed here.

A typical simulation procedure is the following: neurons are externally excited, as described above, by three different stimuli separated by periods without stimulation. Pattern $\mu = 1$ is presented along the time interval [501, 1000]. Then, pattern $\mu = 2$ is presented along the time interval [2501, 3000]. Finally, during time interval [4501, 5000] pattern $\mu = 1$ is presented once more, but now to a different choice of neurons. (Remember that during pattern presentation, external current is injected into a fraction $P_{ext} = 0.5$ of neurons belonging to the pattern.)

Fig. 1 shows PCA projection space trajectories corresponding to the simulations on the first (abscissa) and the third (ordinate) axes. Each circle corresponds to a 50-iteration time bin. Full circles and thick lines represent input time intervals. The system starts from the quiescent baseline state (Q), follows a loop under the presentation of the external stimulation and returns to the baseline state. There are three loops in each figure, numbered from 1 to 3, corresponding to the three stimulations. In the absence of connections (Fig. 1(a)), the three loops are uncorrelated, only representing the activation paths of neurons receiving current injection. In the connected network (Fig. 1(b)), loops numbered 1 and 3 are much closer to each other than the loop numbered 2, since the former follow stimulation of pattern $\mu = 1$. This means that the network is able to recognize pattern 1 and to distinguish it from pattern 2, even if the neurons receiving current injection are different.

Fig. 1(c) shows the simulation with fully random connections, i.e., excitatory or inhibitory connection between two neurons are independent from the patterns. Although the network dynamics follows closed loops like 1(b), all the three loops are uncorrelated. This means that the system is unable to recognize the same pattern if different sets of neurons are stimulated.

Now we investigate the system's stability against different injection times. Fig. 2 shows four simulations using external stimuli on the same neurons, but with different input time intervals Δt. In (a) $\Delta t = 100$, in (b) $\Delta t = 800$, in (c) $\Delta t = 1600$ and in (d) $\Delta t = 3200$. Squares and thick lines represent the input time interval. As can be seen in the figure, the trajectory remains nearly the same and, as the injection time increases, there is an accumulation of points around a fixed point (FP). In the absence of current injection this attractor destabilizes and the system returns to the baseline state (Q), following the

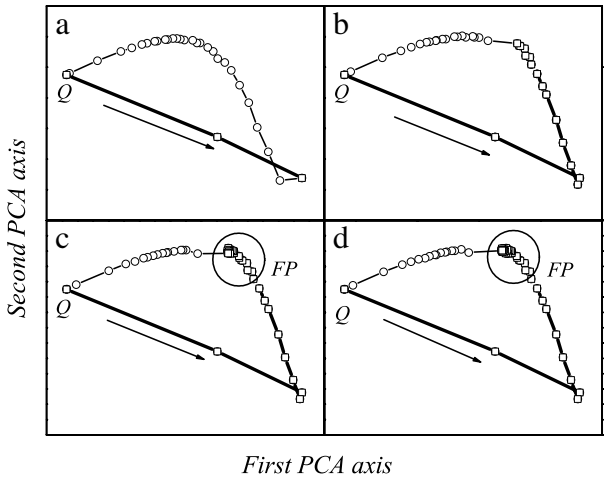

Fig. 2. Plot of the first (abscissa) and the second (ordinate) principal components from Principal Component Analysis of four simulations. The external input is injected into one pattern and into the same neurons, differing only in the time input interval (Δt). In (a), (b), (c) and (d), $\Delta t = 100$, $\Delta t = 800$, $\Delta t = 1600$ and $\Delta t = 3200$ iterations, respectively. Squares and thick lines represent the input time intervals. The number of circles increase with the increase of Δt. In (c) and (d) the regions with accumulation of circles, indicated as FP, are the fixed points of this pattern.

same trajectory, independently of the injection time. This way we recover the same features of the trajectories observed in the work by Mazor and Laurent [7].

4. Conclusion

A synaptic architecture providing pattern recognition abilities to a network of spiking neurons is proposed in this article. PCA analysis of simulations shows that the synapses modify the trajectories in the frequency space in a way that trajectories corresponding to the same input pattern are close to each other, while injection corresponding to some other pattern describes a very different trajectory. This means that the network has clear discriminative capabilities. The response to different current injection times was also investigated showing the presence of a fixed point. Furthermore, trajectories linking the baseline to this attractor forward and backward are injection time independent. This scenario is consistent with two alternatives: either the recognition of a pattern is more related to describing a trajectory than just reaching an attractor, or the attractor itself is the discriminative element for the pattern. The first idea allows a fast pattern recognition of, e.g., an odor, which is a desired biological feature, while the second idea makes the whole process robust.

Here we have shown abilities, emerging from a network ensemble of local maps and connectivity architecture, to discriminate among a small and fixed number of patterns. Further work is required in order to implement a quantitative measure of the distance among the trajectories. Also, the relationship between the number p of patterns, the size N, and the network performance should be investigated. Finally, the whole space of parameters and different recipes for the connectivity matrices should be explored.

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