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

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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.

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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

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