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Synaptic depression enlarges basin of attraction

Narihisa Matsumoto^{a,b,*}, Daisuke Ide^c, Masataka Watanabe^c, Masato Okada^{a,d,e}

^aPRESTO, Japan Science and Technology Agency, Saitama 351-0198, Japan ^bNeuroscience Research Institute, AIST, Tsukuba Central 2,1-1-1 Umezono, Tsukuba, Ibaraki 305-8568, Japan ^cFaculty of Engineering, University of Tokyo, Tokyo 113-0033, Japan ^dRIKEN Brain Science Institute, Saitama 351-0198, Japan ^eGraduate School of Frontier Sciences, University of Tokyo, Chiba 277-8561, Japan

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Abstract

Neurophysiological experiments show that synaptic depression controls a gain for presynaptic inputs. However, the functional roles of this gain control remain unknown. We propose that one of the functional roles is to enlarge basins of attraction. To verify this, we employ an associative memory model. An activity control is requisite for the stable retrieval of sparse patterns. We investigate a storage capacity and the basins of attraction. Consequently, the basins of attraction are enlarged while the storage capacity does not change. Thus, the synaptic depression might incorporate the activity control mechanism. © 2004 Elsevier B.V. All rights reserved.

Keywords: Synaptic depression; Attractor network; Storage capacity; Basin of attraction

1. Introduction

Neurophysiological experiments show that high-frequency inputs induce decrease of synaptic weights [10]. This process is known as short-term synaptic depression.

^{*}Corresponding author. Neuroscience Research Institute, AIST, Tsukuba Central 2,1-1-1 Umezono, Tsukuba, Ibaraki 305-8568, Japan.

E-mail address: xmatumo@ni.aist.go.jp (N. Matsumoto).

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The synaptic depression is known to control a gain for presynaptic inputs [1,12]. However, it is still a controversial issue what are functional roles of this gain control. To elucidate the functional roles, some information can be embedded in synaptic connections. We employ an associative memory model that stores memory patterns in the synaptic connections. Only a few works have investigated how the synaptic depression affects the performance of the associative memory model [4,8,11]. Memory patterns embedded by Hebb rule [5] become fixed points, i.e., attractors [3]. The retrieval of a memory pattern corresponds to a convergence to an attractor. Bibitchkov et al. [4] found that the synaptic depression does not influence the fixed points but reduces a storage capacity. Torres et al. [11] found that the storage capacity decreases with the degree of the depression in the thermodynamical limit. We investigate how the synaptic depression influences basins of attraction which express the regions where the system converges to attractors.

2. Associative memory model with depressing synapses

We investigate a network that consists of N neurons mutually connected. We consider the thermodynamics limit: $N \to \infty$. The neuron *i* is characterized by its binary state $s_i(t) = \{0, 1\}$ and discrete time *t*. The internal potential $h_i(t)$ follows $h_i(t) = \sum_{j \neq i}^{N} J_{ij}(t)s_j(t)$, where $J_{ij}(t)$ represents a synaptic weight at time *t* from the presynaptic neuron *j* to the postsynaptic neuron *i*. The synaptic connections are dynamically changed and the specific value of $J_{ij}(t)$ is discussed later. The neuron state $s_i(t)$ updates the synchronous rule, $s_i(t+1) = \Theta(h_i(t) - \theta)$, where θ is a uniform threshold, and the function $\Theta(\cdot)$ is a step function. If the neuron *i* fires at *t*, its state is $s_i(t) = 1$; otherwise, $s_i(t) = 0$. Each element ξ_i^{μ} of the μ th memory pattern $\xi^{\mu} = (\xi_1^{\mu}, \xi_2^{\mu}, \dots, \xi_N^{\mu})$ is generated independently by $\text{Prob}[\xi_i^{\mu} = 1] = 1 - \text{Prob}[\xi_i^{\mu} = 0] = f$. The expectation of ξ^{μ} is $E[\xi_i^{\mu}] = f$, and thus *f* is a coding level of the memory pattern. The memory pattern with a small coding level *f* is called a sparse pattern, and this coding scheme is called sparse coding. The initial synaptic weight $J_{ij}(0)$ is determined according to a Hebbian-like rule, i.e., a covariance rule:

$$J_{ij}(0) = \frac{1}{Nf(1-f)} \sum_{\mu=1}^{p} (\xi_i^{\mu} - f)(\xi_j^{\mu} - f).$$

The self-connection $J_{ii}(0)$ is assumed to be nonexistent. The value *p* denotes the number of memory patterns and $\alpha = p/N$ is defined as a loading rate. When the loading rate α is larger than a critical value $\alpha_{\rm C}$, the retrieval of memory patterns become unstable. The critical value $\alpha_{\rm C}$ is known as a storage capacity.

The synaptic weight $J_{ij}(t)$ incorporating the synaptic depression is determined according to a phenomenological model of a synapse [1,12]. The initial synaptic weights $J_{ij}(0)$ is multiplied by a dynamic amplitude factor $x_j(t)$ ($0 < x_j(t) \le 1$): $J_{ij}(t) =$ $J_{ij}(0)x_j(t)$. When the synapses transmit the input signals, they exhaust a finite amount of resources, e.g., neuromodulators. The factor $x_j(t)$ denotes the fraction of available resources. After each spike the resources are assumed to decrease by a

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