

Synaptic Depression Enlarges Basin of Attraction

Narihisa Matsumoto ^{a,*} Daisuke Ide ^b Masataka Watanabe ^b

Masato Okada ^{c,a}

^a*PRESTO, Japan Science and Technology Agency, Saitama 351-0198, Japan*

^b*Faculty of Engineering, University of Tokyo, Tokyo 113-0033, Japan*

^c*RIKEN Brain Science Institute, Saitama 351-0198, Japan*

Abstract

Neurophysiological experiments show that synaptic depression controls a gain for presynaptic inputs. However, it remains a controversial issue what are functional roles of this gain control. 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.

Key words: synaptic depression, attractor network, storage capacity, basin of attraction

* Corresponding author.

Email address: xmatumo@brain.riken.jp (Narihisa Matsumoto).

1 Introduction

Neurophysiological experiments show that high-frequency inputs induce decrease of synaptic weights [10]. This process is known as short-term synaptic depression. 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]. The memory patterns embedded by Hebb rule [5] become fixed points, i.e., attractors [3]. The retrieval of the memory pattern corresponds to a convergence to an attractor. Bibitchkov et al. found that the synaptic depression does not influence the fixed points but reduces a storage capacity [4]. Torres et al. found that the storage capacity decreases with the degree of the depression in the thermodynamical limit [11]. However, the main targets of these works were the steady states of the model. It is necessary to investigate the dynamical properties of the model because the synapses change dynamically. One of the important dynamical properties is basins of attraction. They express the regions where the system converges to attractors. We investigate how the synaptic depression influences the basins of attraction.

2 Associative Memory Model with Depressing Synapses

We investigate a network that consists of N neurons mutually connected. We consider the thermodynamics limit: $N \rightarrow \infty$. 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 $\boldsymbol{\xi}^\mu = (\xi_1^\mu, \xi_2^\mu, \dots, \xi_N^\mu)$ is generated independently by $\text{Prob}[\xi_i^\mu = 1] = 1 - \text{Prob}[\xi_i^\mu = 0] = f$. The expectation of $\boldsymbol{\xi}^\mu$ 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 α_C , the retrieval of memory patterns become unstable. The critical value α_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 $x_j(t)$ ($0 < x_j(t) \leq 1$): $J_{ij}(t) = J_{ij}(0)x_j(t)$. 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 certain fraction U_{SE} and to recover with a time constant τ . If the recover lags behind the interval of the high-frequency presynaptic inputs, the amount of resources decrease and the synapses are depressed. The factor $x_j(t)$ updates following dynamics [8, 12]:

$$x_j(t+1) = x_j(t) + \frac{1 - x_j(t)}{\tau} - U_{SE}x_j(t)s_j(t). \quad (1)$$

When $x_j(t) = 1$, the synapses are not depressed.

3 Mean-Field Equations

Here, we derive mean-field equations at the steady state, i.e., $t \rightarrow \infty$. For simplicity, each value at the steady state is written by $x_j(\infty) = x_j$, $h_i(\infty) = h_i$, and $s_i(\infty) = s_i$. The factor $x_j(t)$ reaches its steady-state value by $t \rightarrow \infty$ in the equation (7) [4]: $x_j = \frac{1}{1+\gamma s_j}$ and $\gamma = \tau U_{SE}$. The value γ indicates the level of the synaptic depression. Since s_j takes binary values, i.e., $s_j = \{0, 1\}$, the value $x_j s_j$ is written as $x_j s_j = \frac{s_j}{1+\gamma s_j} = \frac{1}{1+\gamma} s_j$. Using this relationship, the internal potential h_i is written as $h_i = \sum_{j \neq i}^N J_{ij}(0) \frac{s_j}{1+\gamma} = \frac{1}{1+\gamma} \sum_{j \neq i}^N J_{ij}(0) s_j$. Then, the neuron state s_i is written as $s_i = \Theta\left(\frac{1}{1+\gamma} \sum_{j \neq i}^N J_{ij}(0) s_j - \hat{\theta}\right)$, where $\hat{\theta}$ indicates the neuron threshold when the synaptic depression is incorporated into the model. Hereafter, the threshold with the synaptic depression is written as $\hat{\theta}$ while the threshold without the synaptic depression as θ . The closeness between $\mathbf{s}(t)$ and $\boldsymbol{\xi}^t$ at time t is characterized by an overlap $m^\mu(t) = \frac{1}{Nf(1-f)} \sum_{i=1}^N (\xi_i^1 - f) s_i(t)$. If the overlap is close to 1, i.e., $m^\mu(t) \approx 1$, the memory pattern $\boldsymbol{\xi}^\mu$ is retrieved. For simplicity, the overlap at the steady

state is written as $m^\mu(\infty) = m^\mu$. Hereafter, the target pattern for the retrieval is the first memory pattern ξ^1 . The internal potential h_i is represented as

$$h_i = \frac{1}{1+\gamma} \frac{1}{Nf(1-f)} \sum_{\mu=1}^p \sum_{j \neq i}^N (\xi_i^\mu - f)(\xi_j^\mu - f)s_j = \frac{1}{1+\gamma} \left\{ (\xi_i^1 - f)m^1 + z_i \right\}, \quad (2)$$

where $z_i = \sum_{\mu=2}^p (\xi_i^\mu - f)m^\mu - \alpha s_i$. The first term of the equation (2) is a signal term for the retrieval of pattern ξ^1 . The second term is a cross-talk noise term, which represents contributions from non-target patterns and prevents the target pattern ξ^1 from being retrieved. According to the extended mean-field theory [7, 9], the cross-talk noise obeys a Gaussian distribution whose mean is Γs_i and variance is σ^2 . Using this theory, the mean-field equations are obtained by the following equations. For simplicity, the overlap m^1 is written as m .

$$m = \frac{1}{2} \operatorname{erf} \left(\frac{(1-f)m - \frac{\hat{\theta}}{1+\gamma} - \frac{\Gamma}{2}}{\sqrt{2\alpha\sigma^2}} \right) + \frac{1}{2} \operatorname{erf} \left(\frac{fm + \frac{\hat{\theta}}{1+\gamma} + \frac{\Gamma}{2}}{\sqrt{2\alpha\sigma^2}} \right), \quad (3)$$

$$U = \frac{f}{\sqrt{2\pi\sigma^2}} \exp \left(- \frac{\left((1-f)m - \frac{\hat{\theta}}{1+\gamma} - \frac{\Gamma}{2} \right)^2}{2\alpha\sigma^2} \right) + \frac{1-f}{\sqrt{2\pi\sigma^2}} \exp \left(- \frac{\left(fm + \frac{\hat{\theta}}{1+\gamma} + \frac{\Gamma}{2} \right)^2}{2\alpha\sigma^2} \right), \quad (4)$$

$$q = \frac{1}{2} + \frac{f}{2} \operatorname{erf} \left(\frac{(1-f)m - \frac{\hat{\theta}}{1+\gamma} - \frac{\Gamma}{2}}{\sqrt{2\alpha\sigma^2}} \right) - \frac{1-f}{2} \operatorname{erf} \left(\frac{fm + \frac{\hat{\theta}}{1+\gamma} + \frac{\Gamma}{2}}{\sqrt{2\alpha\sigma^2}} \right), \quad (5)$$

where $\sigma^2 = \frac{q}{(1-U)^2}$, $\Gamma = \frac{\alpha U}{1-U}$, $\operatorname{erf}(y) = \frac{2}{\sqrt{\pi}} \int_0^y \exp(-u^2) du$. Because of the page restriction, the detailed derivation of these equations is shown elsewhere.

4 Results

At first, we investigate how the synaptic depression influences the steady state of the model. Since the function $\Theta(\cdot)$ is a step function, each neuron state is

determined by the sign of the input. Then, the state s_i can be expressed by

$$s_i = \Theta\left(\frac{1}{1+\gamma}\left(\sum_{j \neq i}^N J_{ij}(0)s_j - (1+\gamma)\hat{\theta}\right)\right) = \Theta\left(\sum_{j \neq i}^N J_{ij}(0)s_j - (1+\gamma)\hat{\theta}\right). \quad (6)$$

If the threshold is set at $\hat{\theta} = \theta/(1+\gamma)$, the neuron state s_i is written as $s_i = \Theta\left(\sum_{j \neq i}^N J_{ij}(0)s_j - \theta\right)$. Therefore, the steady state of the model with the synaptic depression coincides with the steady state without the synaptic depression when the threshold is set at $\hat{\theta} = \theta/(1+\gamma)$.

Solving the mean-field equations (3-5), the steady state of the model can be analyzed. Fig.1(a) shows the dependency of the overlap $m^1(100)$ on the loading rate α without the synaptic depression ($\gamma = 0$) at $f = 0.1$. The dashed lines are obtained from the mean-field equations (3-5) while the error bars indicate medians and quartile deviations obtained by the numerical simulations in 11 trials. The initial state is set at the first memory pattern, i.e., $\mathbf{s}(0) = \boldsymbol{\xi}^1$, and the threshold is fixed at $\theta = 0.51$, which is optimized to maximize the storage capacity. The storage capacity α_C is 0.44. Fig.1(b) shows the dependency of $m^1(100)$ on α with the synaptic depression ($\gamma = 1$, $\tau = 2.0$, $U_{SE} = 0.5$, $x_j(0) = 0.5$) at $\hat{\theta} = 0.255$. The storage capacity α_C is 0.44. Therefore, the steady states do not change.

Next, we investigate how the synaptic depression influences the basins of attraction. When the loading rate α is less than the storage capacity α_C , the critical overlap m_C exists [2]. When the initial overlap $m^1(0)$ is larger than the critical overlap m_C , the retrieval of the memory pattern $\boldsymbol{\xi}^1$ is successful. In other words, the system converges to the attractor $\boldsymbol{\xi}^1$. Therefore, the region of $m^1(0) > m_C$ is known as the basins of attraction.

Using the numerical simulations, we calculate the basins of attraction. Fig.2(a)

shows the basin of attraction without the synaptic depression ($\gamma = 0$) at $f = 0.1$. The regions between the solid lines and the symbols $(-, \square, \times, *)$ are basins of attraction. When the threshold θ is small, the basins of attraction are enlarged, but the storage capacity decreases. Fig.2(b) shows the basins of attraction with the synaptic depression in four cases: $\hat{\theta} = 0.51$ and $\gamma = 0$ case, $\hat{\theta} = 0.425$ and $\gamma = 0.2$ case, $\hat{\theta} = 0.34$ and $\gamma = 0.5$ case, and $\hat{\theta} = 0.255$ and $\gamma = 1$ case. In these four cases the relationship of $(1 + \gamma)\hat{\theta} = 0.51$ is kept. Therefore, the storage capacity does not change: $\alpha_C = 0.44$. The basins of attraction are enlarged compared to in the case without the synaptic depression $(-)$.

5 Summary

We investigated how the synaptic depression influenced the performance of the associative memory model in terms of the storage capacity and the basins of attraction. Using the extended mean-field theory and the numerical simulations, we found that the stable retrieval of the sparse patterns is achieved and the basins of attraction are enlarged. Furthermore, the storage capacity does not decrease. In the sparse coding scheme the threshold increases gradually to maintain the overall activity at a constant level [2, 6, 7]. Thus, the synaptic depression might incorporate the mechanisms that the threshold effectively increases in the progress of the retrieval.

References

- [1] L.F. Abbott, J.A. Varela, K. Sen, and S.B. Nelson. Synaptic depression and cortical gain control. *Science*, 275:220–224, 1997.

- [2] S. Amari and K. Maginu. Statistical neurodynamics of various versions of correlation associative memory. *Neural Network*, 1:63–73, 1988.
- [3] D.J. Amit. *Modeling brain function*. Cambridge university press, 1989.
- [4] D. Bibitchkov, J.M. Herrmann, and T. Geisel. Pattern storage and processing in attractor networks with short-time synaptic dynamics. *Network*, 13:115–129, 2002.
- [5] D.O. Hebb. *The Organization of Behavior: A Neuropsychological Theory*. Wiley, New York, 1949.
- [6] N. Matsumoto and M. Okada. Self-regulation mechanism of temporally asymmetric hebbian plasticity. *Neural Computation*, 14:2883–2902, 2002.
- [7] M. Okada. Notions of associative memory and sparse coding. *Neural Networks*, 9:1429–1458, 1996.
- [8] L. Pantic, J.J. Torres, H.J. Kappen, and S.C.A.M. Gielen. Associative memory with dynamic synapses. *Neural Computation*, 14:2903–2923, 2002.
- [9] M. Shiino and T. Fukai. Self-consistent signal-to-noise analysis and its application to analogue neural networks with asymmetric connections. *Journal of Physics A*, 25:L375–L381, 1992.
- [10] A.M. Thomson and J. Deuchars. Temporal and spatial properties of local circuits in neocortex. *Trends in Neuroscience*, 17:119–126, 1994.
- [11] J.J. Torres, L. Pantic, and H.J. Kappen. Storage capacity of attractor neural networks with depressing synapses. *Physical Review E*, 66:061910, 2002.
- [12] M.V. Tsodyks and H. Markram. The neural code between neocortical pyramidal neurons depends on neurotransmitter release probability. *Proceedings of the National Academy of Sciences*, 94:719–723, 1997.

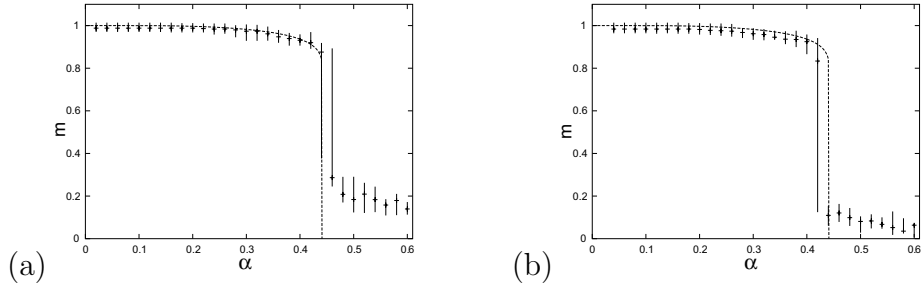


Fig. 1. The dependency of $m^1(100)$ on α at $f = 0.1$. The dashed lines are obtained from the mean-field equations while the error bars indicate medians and quartile deviations obtained by the numerical simulations in 11 trials. (a):the case without the synaptic depression at $\theta = 0.51$. $\alpha_C = 0.44$. (b):case with the synaptic depression ($\gamma = 1$, $\tau = 2.0$, $U_{SE} = 0.5$, $x_j(0) = 0.5$) at $\hat{\theta} = 0.255$. $\alpha_C = 0.44$.

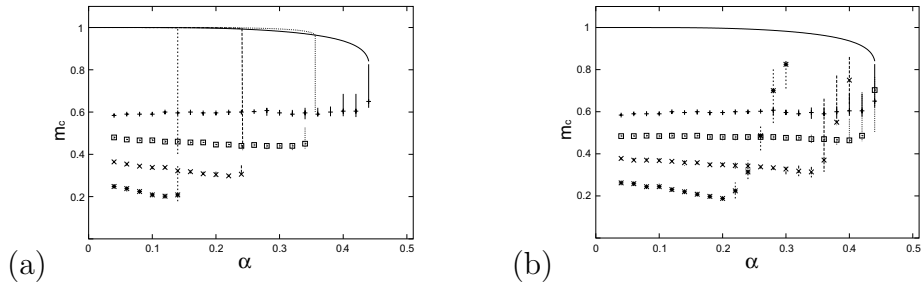


Fig. 2. The dependency of m_C on α at $f = 0.1$. The dashed lines are obtained from the mean-field equations while the error bars indicate medians and quartile deviations obtained by the numerical simulations in 11 trials. (a):the synapses are not depressed. $-:\theta = 0.51$. $\square:\theta = 0.425$. $\times:\theta = 0.34$. $*:\theta = 0.255$. (b):the synapses are depressed at $x_j(0) = 1.0$. $-:\hat{\theta} = 0.51$, $\gamma = 0$. $\square:\hat{\theta} = 0.425$, $\gamma = 0.2$, $\tau = 1.2$, $U_{SE} = 0.167$. $\times:\hat{\theta} = 0.34$, $\gamma = 0.5$, $\tau = 1.5$, $U_{SE} = 0.333$. $*:\hat{\theta} = 0.255$, $\gamma = 1$, $\tau = 2$, $U_{SE} = 0.5$.