Neuronal Normalization Provides Effective Learning Through Ineffective Synaptic Learning Rules

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1 Introduction

This paper studies the fundamental interplay between Hebbian synaptic changes and neuronally driven processes modifying synaptic efficacies, and its role in associative memory learning. The importance of neuronally driven normalization processes has already been demonstrated in the context of self-organization of cortical maps [1, 2] and in continuous unsupervised learning as in Principal-Component-Analysis networks [3]. In these scenarios such normalization was shown to be necessary to prevent the excessive growth of synaptic efficacies that occurs when learning and neuronal activity are strongly coupled. In this paper we focus on associative memory learning where this excessive synaptic runaway growth is mild [4], and show that even in this more simple learning paradigm, normalization processes are essential. Moreover, while various normalization procedures can prevent synaptic runaway, our analysis shows that a specific neuronally-driven correction procedure that preserves the total sum of synaptic efficacies is essential for effective memory storage. To this end we analyze the effectiveness of Hebbian synaptic learning rules and identify a critical constraint on effective learning. We then describe a neuronal procedure obtaining this constraint and show how it can be implemented via a biologically plausible mechanism.

2 The Space of Synaptic Learning Rules

We study memory storage in a model of associative memory low-activity neural network with binary firing $\{0, 1\}$ neurons similar to the model of [5]. In our model, the synaptic weight W_{ij} is determined by a general Hebbian additive synaptic learning rule

$$W_{ij} = \sum_{\eta=1}^{M} A(\xi_i^\eta, \xi_j^\eta) \quad , \tag{1}$$

where $\{\xi^\eta\}_{\eta=1}^{M}$ are the stored memory patterns and $A(\xi_i^\eta, \xi_j^\eta)$ is a two-by-two synaptic learning matrix

		presynaptic (ξ_j)	
	$A(\xi_i, \xi_j)$	1	0
postsynaptic (ξ_i)	1	x_P "homo synaptic potentiation"	x_D "hetero synaptic depression"
	0	x_{homo} "homo synaptic depression"	x_{hetero}

,

whose four parameters $\{x_P, x_D, x_{homo}, x_{hetero}\}$ determine the modifications to the synaptic efficacy following some combination of pre and post synaptic activity. Utilizing a scaling invariance constraint and the requirement that the synaptic matrix should have a zero mean [6], we reduce the whole four dimensional space of learning rules into a two-dimensional space of possibly effective ones, having two free parameters (x_P,x_D).

A. The Space of learning rules **B. Effective learning rules**

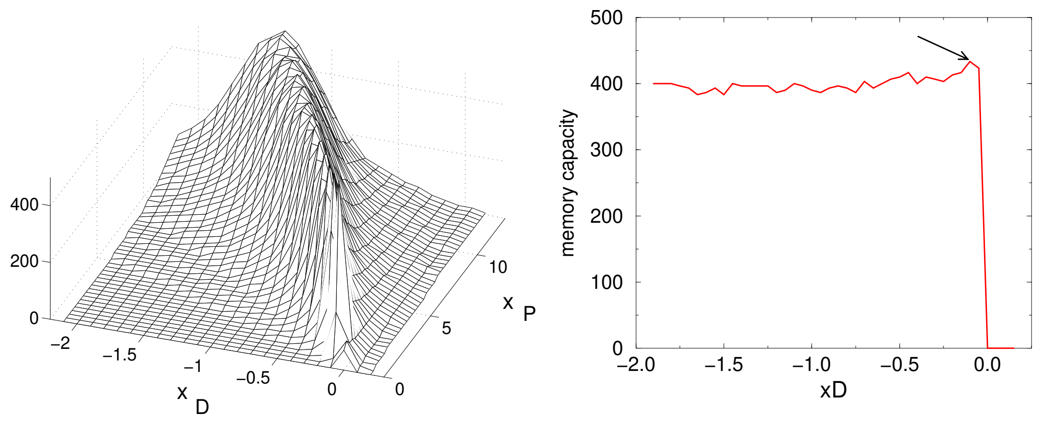

Figure 1: A. Memory capacity of a 1000-neurons network for different learning rules as obtained in computer simulations. Capacity is defined as the maximal number of memories that can be retrieved when presented with a degraded input cue. The coding level (fraction of firing neurons) is $p = 0.05$. B. Memory capacity of the effective learning rules. The peak values on the ridge of Figure A, are displayed by tracing their projection on the x_D coordinate. The optimal learning rule [4] is marked with an arrow.

Figure 1A plots the memory capacity of the network as a function of the two free parameters x_P and x_D. It shows that considerable memory storage may be obtained only along an essentially one dimensional curve, revealing that an additional constraint on the synaptic learning rule must be obeyed. This constraint is identified by signal-to-noise analysis of the neuronal input field f_i during retrieval

$$\frac{Signal}{Noise} = \frac{E(f_i|\xi_i = 1) - E(f_i|\xi_i = 0)}{\sqrt{Var(f_i)}} \propto \frac{\sqrt{N/M}}{\sqrt{Var\left[A(\xi_1,\xi_2)\right] + NpCOV\left[A(\xi_1,\xi_2), A(\xi_1,\xi_3)\right]}}. \quad (2)$$

As evident from Eq. (2), when the **postsynaptic covariance** $COV\left[A(\xi_1,\xi_2), A(\xi_1,\xi_3)\right]$ (determining the covariance between the incoming synapses of the postsynaptic neuron) is positive, the network's memory capacity is bounded. Conversely, when the postsynaptic covariance vanishes an **effective learning rule** is obtained, achieving linear scaling of memory capacity as a function of the network's size. As shown in Figure 1B, all these effective rules are only slightly inferior to the optimal synaptic learning rule ($A(\xi_i,\xi_j) = (\xi_i - p)(\xi_j - p)$ [6]), which maximizes memory capacity.

The vanishing covariance constraint implies a new requirement concerning the balance between synaptic depression and facilitation: $x_D = \frac{-p}{1-p}\, x_P$. Effective memory storage thus explicitly depends on the coding level p which is a **global property of the network**. It is thus difficult to see how effective rules can be implemented at the synaptic level. Moreover, as shown in Figure 1A, Hebbian learning rules lack robustness as small perturbations from the effective rules may result in large decrease in memory capacity. We further prove that the postsynaptic covariance cannot be zeroed by introducing a nonlinear Hebbian rule of the form $W_{ij} = g\left(\sum_\eta A(\xi_i^\eta, \xi_j^\eta)\right)$. These observations show that **effective associative learning with Hebbian rules alone is biologically implausible**.

3 Effective Learning Via Neuronal Weight Correction

We now proceed to show a neuronally-driven procedure that ensures effective memory storage. We propose that during learning, as a synapse is modified, its postsynaptic neuron additively modifies all its synapses to maintain the sum of their efficacies at a baseline zero level. Our analysis shows that this **neuronal weight correction** results in a vanishing postsynaptic covariance and achieves effective memory storage using local information only.

To demonstrate the beneficiary effects of neuronal weight correction we apply it to a common realization of the Hebb rule (with inhibition added to obtain a zero-mean input field) yielding $A(\xi_i, \xi_j) = \xi_i\xi_j - p^2$ [7]. Figure 2 compares the memory capacity obtained with this zero-mean Hebb rule, before and after neuronal weight correction. The memory capacity of the original zero-mean Hebb rule is bounded, but when neuronal weight correction is applied, it turns to scale linearly with the network's size.

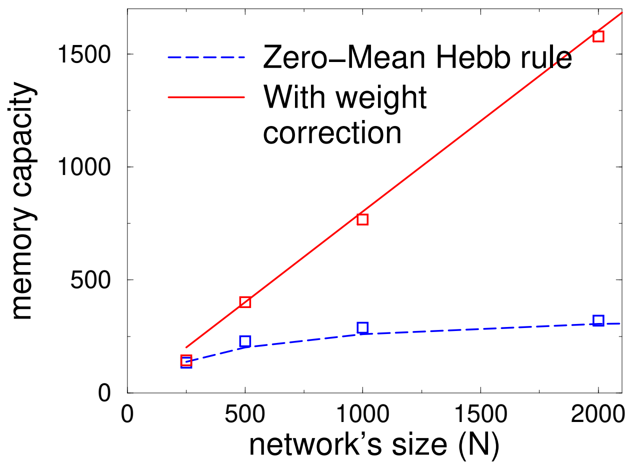

Figure 2: Network memory capacity as a function of the network's size. The lines plot analytical results and the squares designate simulation results ($p = 0.05$).

4 Neuronal Regulation Approximates Neuronal Correction

Several mechanisms for conservation of the total synaptic strength have been proposed [8]. We focus here on one possible mechanism: Neuronal Regulation (NR) [9], whose goal is to maintain the homeostasis of neuronal activity. NR constantly regulates the postsynaptic activity around a baseline level by multiplying the neuron's incoming synaptic efficacies by a common factor. Such activity-dependent modification of excitatory synapses, maintaining the homeostasis of neuronal firing, has been observed in cortical tissues by [10]. Interestingly, NR approximates the additive neuronal weight correction mechanism described above, and succeeds in maintaining a vanishing postsynaptic covariance, thus drastically improving the capacity of memory networks.

Figure 3A plots the memory capacity of networks storing memories according to the Hebb rule $W_{ij} = \sum_{\eta=1}^{M} \xi_i^\eta \xi_j^\eta$, showing how NR succeeds in obtaining a linear growth of memory capacity that is only slightly inferior to that obtained with neuronal weight correction. Figure 3B plots the temporal evolution of the retrieval acuity (overlap) and the average postsynaptic covariance, showing that NR slowly removes the interfering covariance, improving memory retrieval.

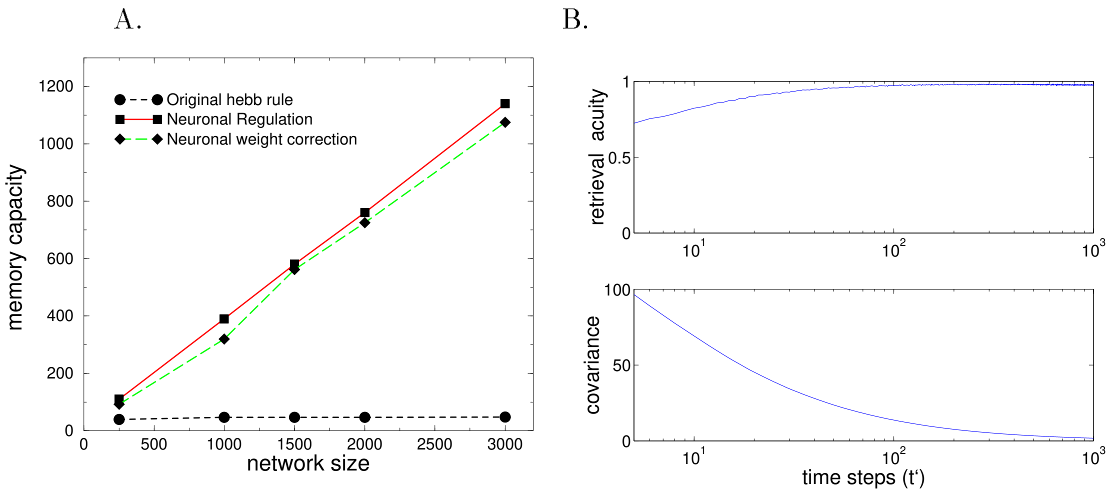

Figure 3: A. Memory capacity at the stable state of networks storing patterns via the Hebb rule, $p = 0.1$. B. The temporal evolution of retrieval acuity and average postsynaptic covariance in a 1000-neurons network storing 250 memory patterns.

5 Conclusions

Analyzing Hebbian learning rules in associative memory network models, we have identified an essential requirement for effective memory storage: a vanishing postsynaptic covariance. While Hebbian learning alone is bound to yield catastrophic postsynaptic covariance, the latter can be removed by a neuronally driven mechanism that preserves the total synaptic sum. This provides effective learning using ineffective synaptic learning rules, as long as they are further modified and corrected by neurally driven weight correction. The resulting improvement in memory capacity is drastic: learning rules yielding bounded capacity are transformed into learning rules yielding linear memory capacity as a function of the network's size. Finally, the normalization mechanism can be carried out by neuronal regulation (NR), a mechanism recently identified in mammalian cortical cultures.

Our results, obtained within the paradigm of autoassociative memory networks, apply also to hetero-associative memory networks. More generally, neuronal weight correction qualitatively improves the ability of a neuron to correctly discriminate between a large number of input patterns. It thus enhances the computational power of the single neuron and may be applied in other memory storage paradigms. With this perspective, it is not surprising that the interplay between correlation-based synaptic changes and normalization processes plays a fundamental computational role in a variety of brain functions such as visual processing and associative learning.

References

[1] K.D. Miller and D.J.C MacKay. The role of constraints in Hebbian learning. *Neural Computation*, 6(1):100–126, 1994.

[2] C. von der Malsburg. Self organization of orientation sensitive cells in the striate cortex. *Kybernetik*, 14:85–100, 1973.

[3] Erkki Oja. A simplified neuron model as a principal component analyzer. *Journal of Mathematical Biology*, 15:267–273, 1982.

[4] A. Grinstein Massica and E. Ruppin. Synaptic runaway in associative networks and the pathogenesis of schizophrenia. *Neural Computation*, 10:451–465, 1998.

[5] M.V. Tsodyks and M. Feigel'man. Enhanced storage capacity in neural networks with low activity level. *Europhys. Lett.*, 6:101–105, 1988.

[6] P. Dayan and D.J. Willshaw. Optimizing synaptic learning rules in linear associative memories. *Biol. Cyber.*, 65:253, 1991.

[7] M.V. Tsodyks. Associative memory in neural networks with Hebbian learning rule. *Modern Physics letters*, 3(7):555–560, 1989.

[8] K.D. Miller. Synaptic economics: Competition and cooperation in synaptic plasticity. *Neuron*, 17:371–374, 1996.

[9] D. Horn, N. Levy, and E. Ruppin. Synaptic maintenance via neuronal regulation. *Neural Computation*, 10(1):1–18, 1998.

[10] G.G. Turrigano, K. Leslie, N. Desai, and S.B. Nelson. Activity dependent scaling of quantal amplitude in neocoritcal pyramidal neurons. *Nature*, 391(6670):892–896, 1998.