Neuronal Regulation and Hebbian Learning

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Introduction

Since its conception half a century ago, Hebbian learning has become a fundamental paradigm in the neurosciences. The idea that neurons that fire together wire together has become fairly well understood, as in the case of NMDA-dependent long term potentiation in the Hippocampus [2]. However, for both computational and biological reasons, this type of plasticity has to be accompanied by synaptic changes that are not synapse specific but neuron specific, i.e. they involve many synapses of the same neuron. Biologically, such interactions are inevitable as synapses compete for finite resources and are subject to common processes of the same neuron to which they all belong. Computationally, neuron-specific modifications of synaptic efficacies are required in order to obtain efficient learning, or to faithfully model biological systems. Hence *neuronal regulation* (NR), defined here as a process modulating all synapses of a postsynaptic neuron, is a general phenomenon that complements Hebbian learning.

There exists evidence for cellular mechanisms resulting in normalization of synaptic efficacies, some of which operate to maintain total synaptic strength and others to regulate mean postsynaptic activity [10]. Among these mechanisms are cellular regulation of the number of synapses or of trophic factors, competition between synapses for some finite resources, changes in presynaptic and post synaptic learning thresholds or activity dependent regulation of conductances. Normalization of synaptic efficacies is also induced by certain types of plasticity as an emergent phenomenon, for example in the case of spike-time dependent plasticity [14]. Of particular interest are the findings by [15] who studied cultures of pyramidal neurons of postnatal rats. They observed slow postsynaptic up- or down-regulation of AMPA-mediated synaptic currents in a way that maintains the mean firing activity of the neuron. This scaling resulted in overall synaptic normalization through a multiplicative factor that is inversely related to the neuron's activity.

What are the computational consequences of such neuronal level processes? It turns out that learning through Hebbian learning alone raises many theoretical difficulties and questions, such as: What stops the positive feedback loop of Hebbian learning and guarantees some normalization of the synaptic efficacies of a neuron? How can a neuron acquire specificity to particular inputs without being prewired? How can memories be maintained throughout life while synapses suffer degradation

due to metabolic turnover? As we will see, neuronal regulation provides a possible answer to all of the above.

We may divide the computational problems to be looked at according to the traditional dichotomy of supervised and unsupervised learning. Within the latter, the important role of NR is to allow for *competition* between the various synapses and lead to *normalization* of the synaptic efficacies. This will be further explained in the next section, where we review shortly some basic learning paradigms, discuss the difference between multiplicative ($\Delta \mathbf{w} \propto \mathbf{w}$) and additive ($\Delta \mathbf{w} = const$) scaling, and mention some applications to biological systems. We will then turn to supervised learning paradigms, and show that NR improves the *capacity* of associative memory models, and can be used to guarantee the *maintenance* of biological memory systems.

Unsupervised Learning

When Hebbian plasticity operates in a network in an unsupervised manner, a positive feedback loop is created. To illustrate the problem, think of a presynaptic cell A that caused the firing of a postsynaptic cell B. Due to Hebbian plasticity the efficacy of the synapse w_{BA} from A to B is strengthened. This leads to an increase in the ability of cell A to activate cell B, which in turn leads to strengthening of the same synapse again. When this positive feedback is unconstrained it leads to synaptic runaway, i.e. divergence of synaptic efficacies. It seems reasonable to assume that synaptic values are limited by some upper bound that stops this process. Even then a problem emerges: different afferents will activate different synapses of the target neuron B. When all of them get saturated at their upper bounds, the neuron will not have any discrimination ability. This problem may be solved by introducing constraints such as limiting the total synaptic strength of a neuron, $\sum_i w_i^2 = const.$ This results in competition between synapses: the increased strength of one synapse causes the decrease of another, preventing saturation of all synapses.

Multiplicative vs Additive Constraints

Normalization prevents synaptic divergence, thus the combined operation of Hebbian learning and normalization induces new dynamics of synaptic efficacies. This combined dynamics was described by [12], showing that multiplicative weight normalization of a neuron with real valued stochastic

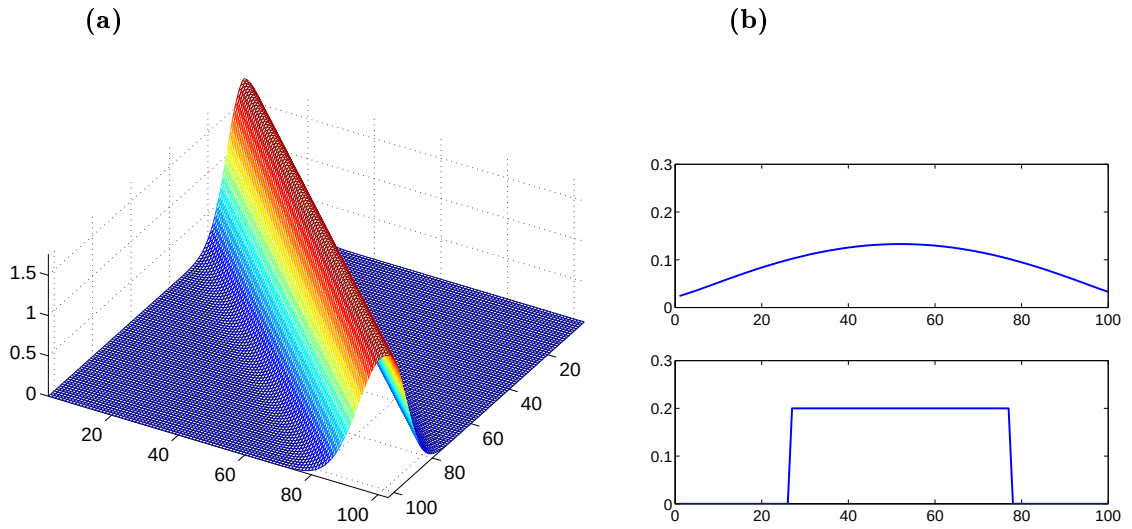

Figure 1: Steady states of synaptic weights under multiplicative and additive normalization in a toy example of a neuron with 100 inputs. **(a)** The correlation matrix of all inputs. **(b)** Under multiplicative constraints the weights converge onto the first principal component (upper plot), while an additive rule leads to a binary separation of weights that reach their extremal values (bottom plot).

input extracts the first principal component of the input distribution (also known as PCA (q.v.) or Karunen Leove feature extraction).

Whereas PCA extraction follows for multiplicative normalization, the results change if other types of constraints, such as additive normalization are imposed [11]. While multiplicative normalization leads to a graded weight vector that represents even weak correlations in the input (upper plot of figure 1b), additive normalization yields a sharpened receptive field, where weights saturate at their lower and upper bounds in a way that only reflects the maximally correlated inputs (figure 1b, bottom). Similar results were obtained for competitive learning where only the weights of the winning unit are changed [7].

Neuronal Regulation and Synaptic Normalization

What is the relation between synaptic normalization and neuronal regulation ? Normalization of synaptic efficacies involves all synapses of a postsynaptic neuron, thus it requires neuronal-level

computation. Moreover, in some cases synaptic normalization is an emergent result of synaptic changes that depend on neuronal activity. We approach this idea by discussing two learning models: the Oja learning rule discussed above and the BCM model.

Using the linear perceptron $V = \sum_i w_i x_i$, Oja's learning rule can be implemented by $\Delta w_i = \eta V(x_i - V w_i)$. Here, NR is explicitly manifested by the second term, which provides a multiplicative correction that is independent of the specific input x_i but is determined by the neuronal output V. Interestingly, this neuronal regulation term guarantees $\sum_i w_i^2 = 1$.

The BCM (q.v.) [1] model is another example of complex interplay of neuronal regulation and synaptic competition. In the BCM approach, both Hebbian potentiation and depression are used in defining the synaptic learning rule: Synapses are potentiated when the presynaptic neuron fires frequently, while depressed otherwise. The boundary between potentiation and depression is determined by the activity of the postsynaptic neuron, which is where neuronal regulation comes in. This NR component leads to competition between synapses and introduces statistical correlations [9] that are higher than the second order used in PCA . Thus, BCM captures high order statistical structures in the input, and tunes the efficacies of incoming synapses accordingly.

Biological Models

The BCM model was developed to describe the emergence of orientation selective cells and ocular dominance in the visual cortex and their dependence on the stimuli that the visual system receives during its critical developmental stages. A study that discusses the same issues with particular emphasis on the dynamics of synaptic efficacies under additive and multiplicative normalization schemes is that of Miller [11]. He shows that when the inputs to a neuron have positive correlations only, additive normalization leads to the convergence of weights to an on-center-off-surround receptive field, or to a bi-lobed receptive field, depending on the parameter regime. When the cell receives inputs from both eyes, additive normalization leads to ocular dominance through the sharpening of receptive fields. Multiplicative normalization can lead in this case to ocular dominance only if the inputs from both eyes are negatively correlated.

A convenient system for the study of NR is the vertebrate neuromuscular junction (NMJ). Its

development is characterized by an initial stage of super-innervation (each muscle fiber is innervated by several motor neurons) followed by withdrawal of axon terminals until a state of single innervation is reached. Modeling the NMJ, Willshaw [16] has shown that competition for postsynaptic resources explains the decrease in innervation, yet fails to account for other experimental findings such as incomplete innervation after artificial partial denervation during development. However, a combination of post and presynaptic competition provides good account of the data. Indeed, the efficacy of the neuro-muscular synapse during the period preceding axon withdrawal was traced by Colman et al.[5], who found changes in quantal content and efficacy. These lead to continuous strengthening of some synapses with parallel weakening of the rest, suggesting the operation of a cascade of pre- and postsynaptic processes regulating synaptic efficacies. This is therefore an interesting example where both pre- and postsynaptic normalization cooperate during early development.

Finally we wish to point out that competition may arise also through effects other than NR. An example is the case of spike-time dependent synaptic plasticity (STDP) in which potentiation of excitatory synapses occurs when the presynaptic spike shortly precedes the post synaptic spike, and depression occurs when the opposite temporal order holds. Whan assuming that spike dependent synaptic potentiation is weaker than depression [14], in a neuron that is driven by net positive input, the excitatory synapses will be weakened on average, leading eventually to a balanced input in which the more relevant excitatory synapses will be strengthened. Thus, effective competition between synapses may occur even in the absence of an explicit NR term, one that depends on the post-synaptic neuron only.

Supervised Learning

We saw that in unsupervised learning, neuronal regulation solves the problem of synaptic runaway and guarantees specificity of neuronal response through synaptic competition. In supervised learning we will show that normalization constraints introduced by NR provide both maintenance of memory systems and high memory capacity.

Memory Maintenance

The concept of NR was introduced by [8], in the context of associative memory networks, while developing a model that can account for the stability of memory systems in the face of continuous metabolic turnover of synapses. This repetitive process of synaptic degeneration and buildup occurs on a time scale of few days. Under these conditions one wonders how memories can be stored in synaptic connections for prolonged periods. It turns out that NR may play an important role in bringing about the necessary homeostasis of this system, i.e. account for its ability to continue to both learn and retrieve memories [8].

To understand this issue consider an associative memory system that is tested through activation by random inputs. Neurons that belong to memories with large basins of attraction will be much more active than those who participate only in memories with small basins of attraction. Introduce now NR through multiplicative synaptic corrections that are inversely proportional to the activity of the postsynaptic neuron. This will up-regulate weak memories and down-regulate strong ones. The multiplicative nature of the correction guarantees that the relative weights of different memories on the same neuron are maintained. The result of this procedure is depicted in Figure 2. As can be seen, repeated synaptic degradation and neuronal regulation leads to normalization of the basins of attraction.

The homeostasis strategy suggested by [8] involves repeated sessions of random activation, synaptic degradation and neuronal regulation, that provide the required maintenance of the network after it goes through some period of Hebbian learning. It can be shown that the combined effect of synaptic degradation and neuronal regulation also results in the removal of weak synapses, due to emerging synaptic competition [3]. This can provide insight into the phenomenon of synaptic pruning that is believed to occur during early mammal development (see [13] for review of the constructive vs. selectionist approaches to brain development).

Learning Capacity

Normalization of synaptic efficacies plays a crucial role in producing effective Hebbian learning: Without normalization, Hebbian learning leads to poor associative memory capacity that does not

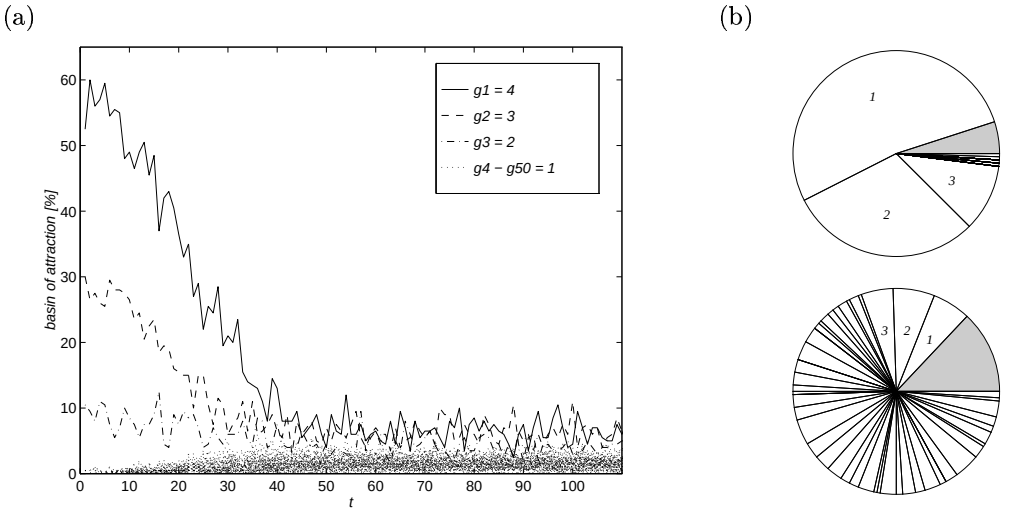

Figure 2: (a) Size of basins of attraction as measured by the percentage of retrievals of specific memories. Fifty memories are stored in a system of thousand neurons. Three of the memories are stronger (parametrized by g) than the rest, overshadowing all others before the corrective dynamic action of NR is introduced. (b) Shares of memory space (relative sizes of basins of attraction) at the beginning (upper figure) and the end (lower figure) of the simulation that consists of repeated cycles of synaptic degradation and neuronal regulation. Random inputs lead either to encoded memories or to the null attractor (gray shading) in which all activity stops. Taken from [8].

grow with the size of the network.

Several authors (e.g. [6, 3]) have studied the space of additive Hebbian learning rules for associative memory networks with low activity patterns (i.e. patterns where only a low fraction of the neurons fire). Such learning rules determine the changes in synaptic efficacy when storing a memory pattern, and may be formally written as $\Delta w_{ij} = aS_iS_j + bS_i + cS_j + d$ where $S_i \in \{0, 1\}$ is the activity of the ith neuron of the stored pattern. Analysing the associative memory capacity of such learning rules shows that only a constrained subspace of learning rules leads to effective memory storage. Figure 3 illustrates this phenomenon showing the capacity resulting from such rules within a subspace of two parameters. Most learning rules are ineffective and lead to low memory capacity because they create correlations between synaptic weights even when the stored memory patterns are uncorrelated. Moreover, the set of effective learning rules all fulfill a constraint that depends

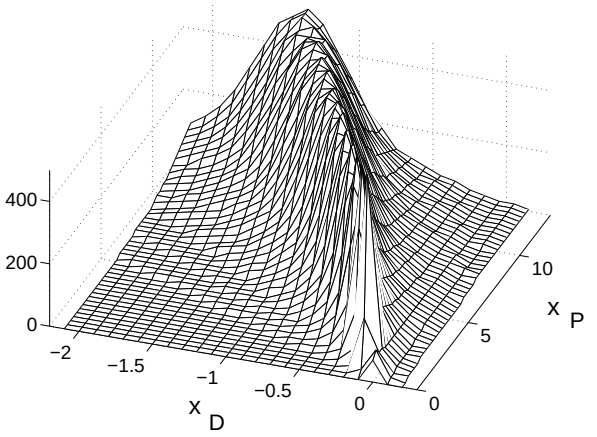

Figure 3: The memory capacity of an associative memory network for various learning rules. The number of memories that can be stored in a 1000-neurons network and be later retrieved from distorted cue is plotted as a function of two parameters: the strength of synaptic potentiation (x_P) and hetero-synaptic depression (x_D). These two parameters span the two-dimensional subspace of learning rules $\Delta w_{ij} = x_P S_i S_j + x_D S_i (1 - S_j)$ where $S_i \in \{0, 1\}$ is the activity of the ith neuron. Apparently, only a one dimensional set of learning rules provides effective learning. Taken from [3]

on the fraction of firing neurons within the stored memory patterns (a global network parameter). Unfortunately, small perturbation in the learning rule parameters lead to violation of this constraint, and consequently to memory capcacity collapse.

Interestingly, learning with effective learning rules lead to a vanishing sum of synaptic efficacies for each neuron. This is true for example for the learning rules on the ridge in Figure 3. More importantly, the converse also holds: a vanishing synaptic sum guarantees effective learning. Thus, enforcing through NR the condition that the sum of synaptic efficacies vanishes, yields high memory capacity irrespective of the generalized Hebbian rule one starts with [3].

Discussion

Hebbian mechanisms per-se fail to provide robust and effective learning, both in supervised and unsupervised scenarios. Although some synapse-specific mechanisms may provide partial remedies for these problems, the current article focused on neuronal regulation of synaptic efficacies and its role

in complementing Hebbian learning. Experimental evidence exists for such cellular mechanisms that regulate synapses, to maintain global constraints on activity or total synaptic strengths [15]. This evidence suggests that neuronal regulation and Hebbian learning are distinct mechanisms: they are mediated through different receptors (NMDA vs. AMPA) and operate on different time scales. From a computational standpoint, the combined operation of Hebbian learning and neuronal regulation provides powerful learning capabilities, ranging from PCA and ICA extraction to robust associative memory learning. We conclude that the functional interplay between synaptic and neuronal mechanisms plays a fundamental role in biological neural networks.

References

[1] E. L. Bienenstock, L. N. Cooper and P. W. Munro. Theory for the development of neuron selectivity: Orientation specificity and binocular interaction in visual cortex. *Journal of Neuroscience* 2, 32-48, 1982.

[2] T.V.P. Bliss and G.L. Collingridge. Synaptic model of memory: long-term potentiation in the hippocampus. *Nature*, 361:31–39, 1993.

[3] G. Chechik, I. Meilijson, and E. Ruppin. Neuronal regulation: A mechanism for synaptic pruning during brain maturation. *Neural Computation*, 11(8):2061–2080, 1999. Effective Neuronal learning with Ineffective Hebbian learning rules. *Neural Computation*,13(4):817-840, 2001 .

[4] H. Colman, J. Nabekura, and J. W. Lichtman. Alterations in synaptic strength preceding axon withdrawal. *Science*, 275(5298):356–361, 1997.

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

[6] G.J. Goodhill and H.G. Barrow. The role of weight normalization in competitive learning. *Neural Computation*, 6:255–269, 1994.

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

[8] N. Intrator and L.N. Cooper. Objective function formulation theory of visual cortical plasticity: Statistical connections, stability conditions. *Neural Networks*, 5, 3–17, 1992.

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

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

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

[12] S.R. Quartz and T.J. Sejnowski. The neural basis of cognitive development: a constructivist manifesto. *Behav Brain Sci* , 20(4):537–556, 1997.

[13] S. Song and K.D. Miller and L.F. Abbott. Competitive Hebbian Learning Through Spike-Timing Dependent Synaptic Plasticity. *Nature Neuroscience*, 3(9),919–926, 2000.

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

[15] D.J. Willshaw. Presynaptic and postsynaptic competition in models for the development of neuromuscular connections. *Biol. Cybern.*, 61(5):85–93, 1993.