Post-Hebbian learning algorithms

Péter Érdi †,‡ and Zoltán Somogyvári †
†Department of Biophysics, KFKI Research Institute for Particle and Nuclear Physics of the Hungarian Academy of Sciences
H-1525 Budapest P.O. Box 49, Hungary
‡Center for Complex System Studies,
Kalamazoo College, Kalamazoo, MI 49006 , U.S.A
Contact author: Péter Érdi
Phone: (36-1)39-2742 : summer; (616)-337-5720 : spring, fall
Fax: (36-1)395-91-51

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Post-Hebbian learning rules in retrospective

Hebb marked a new era by introducing his learning rule and resulted in the sprouting of many new branches of theories and models on the mechanisms and algorithms of learning and related areas.

Two characteristics of the original postulate [Hebb, 1949] played key role in the development of post-Hebbian learning rules. First, in spite of being biologically motivated, it was a verbally described, *phenomenological* rule, without having view on detailed physiological mechanisms. Second, the idea seemed to be extremely convincing, therefore it became a widespread theoretical framework and a generally applied formal tool in the field of neural networks. Based on these two properties, the development of Hebb's idea followed two main directions. First, the postulate inspired an intensive and long lasting search for finding the *molecular* and *cellular* basis of the learning phenomena - which have been assumed to be Hebbian - thus this movement has been absorbed by neurobiology. Second, because of its computational usefulness, many variations evolved from the *biologically inspired* learning rules, and were applied to huge number of very different problems of artificial neural networks, without claiming any relation to biological foundation. Several families of rules sprouted from the original idea will be discussed. Before doing it, we have to note that what we qualify as Hebbian, post-Hebbian, and non-Hebbian learning rule is may be subjective and time-varying.

While there is a broad overview: A PHENOMENOLOGICAL OVERVIEW OF HEBBIAN SYNAPTIC PLASTICITY in this book, here we focus our attention dominantly to computational implementations of the Hebbian rules. However, in the first part of the article the different roots and new sprouts related to Hebbs hypothesis, such as psychological motivated conditioning, neural development and physiological realistic cellular level learning phenomena are discussed, respectively. Than families of formal Hebbian learning —*algorithms* are reviewed.

Variations on the Hebbian theme: motivations

Conditioning

It had been known since the end of the nineteenth century that mature

nerve cells cannot divide. Thus learning could not result from the proliferation of new neurons, therefore the locus of learning *must be the connections between cells*. Such kinds of phenomena are related to the neural basis of classical conditioning. The first attempt to model conditioning in terms of synaptic change was due to Hebb.

Hebb's original intention was to connect the behaviour of whole organisms to neural mechanisms by using concepts represnted by cell assemblies. Specifically, classical conditioning involves the development of an association between two otherwise unrelated events over number of trails in which the events are temporally paired. Typically, the presentation of a neutral stimulus - one that does not naturally provoke behavior - is immediately followed with the presentation of unconditioned stimulus – an event that does not require training to produce a response-resulting in the eliciting of an unconditioned response.

Classical conditioning has been described by the Rescorla and Wagner's model [Rescorla and Wagner, 1972]. They gave a formal model of conditioning which expresses the capacity a conditional stimulus (CS) has to become associated with an unconditional stimulus (US) at any given time. The central idea of the Wagner-Rescola model is that learning occurs if when events violate expectations. More specifically, whenever the actual US level received on a trial differs from the level expected. The Rescola-Wagner rule can be interpreted that the discrepancy between expected and actual values determines the measure of reinforcement. So, the rule and its many later modifications, are over the the "unsupervised learning" paradigm. One drawback of of the Rescorla - Wagner model that it completely ignores the temporal sequence of information.

Development

The formation and refinement of neural circuits involve both the establishment of new, and the elimination of already existing connections. Specifically, the mechanism for leading synaptic elimination is called axonal or synaptic competition. Neuromuscular junctions and the visual system are the two best investigated examples, where synaptic competition plays an important role. A large variety of different generalized Hebbian learning rules applied for neural development was reviewed by [van Ooyen, 2001].

The different mechanisms of competition elaborated in population biological context have been adopted in neural context.

In *consumptive competition*, in systems of consumers and resources (e.g. predators and preys, respectively), each individual consumer tries to avoid the others and hinders the others solely by consuming resources that they might otherwise have consumed; in other words, consumers hinder each other because they share the same resources. In neurobiology, competition is commonly associated with this dependence on shared resources.

In *interference competition*, instead of hindrance through dependence on shared resources, there is direct interference between individuals. This occurs, for example, if there are direct negative interactions e.g. aggressive or toxic interactions between individuals. In axonal competition, nerve terminals could seek to destroy each other by releasing proteases.

Long term potentiation - long term depression

Long-term potentiation (LTP) was first discovered in the hippocampus and is very prominent there. LTP is an increase in synaptic strength that can be rapidly induced by brief periods of synaptic stimulation and which has been reported to last for hours *in vitro*, and for days and weeks *in vivo*.

The LTP (and later the LTD) after their discovery, have been regarded as the physiological basis of Hebbian learning. Subsequently, the properties of the LTP and LTD became more clear, and the question arises, whether the LTP and LTD could really be considered as the microscopical basis of the phenomenological Hebb type learning. Formally, the question is that how to specify the general functional F to serve as a learning rule with the known properties of LTP and LTD. Recognizing the existence of this gap between biological mechanisms and the long-used Hebbian learning rule, there have been many attempts to derive the corresponding phenomenological rule based on more or less detailed neurochemical mechanisms.

The time-course of LTP may be insufficient to sustain long-term memory, but there appear to be multiple LTP mechanisms, and one dependent on protein synthesis might serve long-term memory: inhibition of protein synthesis

disrupts the maintenance of LTP, but leaves the induction of LTP relatively or totally intact. It is possible to relate properties and mechanisms of long-term synaptic plasticity in the mammalian brain to learning and memory.

An example for the new synaptic bidirectional Hebbian rules was introduced [Grzywacz and Burgi, 1998]. This rule was compared with physiological homosynaptic conditions in the hippocampus, with the results indicating the consistency of this rule with LTP and LTD phenomenologies. The phenomenologies considered included the reversible dynamics of LTP and LTD and the effects of N-methyl-D-aspartate blockers and phosphatase inhibitors.

Timing

Studies in cortical and hippocampal slices have shown that back-propagating action potentials may contribute to induce persistent synaptic potentiation or depression. The timing of presynaptic and postsynaptic action potentials play a decisive role in determining the sign of synaptic modification [Markram et al., 1997]. The temporal order of the synaptic input and the postsynaptic spike within a narrow temporal window determines whether LTP or LTD is elicited, according to a temporally asymmetric Hebbian learning rule.

Bi and Poo [Bi and Poo, 1998] showed that postsynaptic spiking that peaked within a time window of 20ms *after* synaptic activation resulted in LTP, while spiking within a window of 20ms *before* synaptic activation led to LTD. They suggested that a narrow and asymmetric window for the induction of synaptic modification should be taken into account.

The majority of the generalized Hebbian rules are based on statistical properties of presynaptic and postsyanptic activity (e.g. activity product, activity covariance etc.) without considering the detailed temporal structure of the spike patterns. Relative time spiking, however, has been taken into account even earlier (e.g. [Sutton and Barto, 1981].

Since changes in synaptic efficacy can depend on the precise timing relations of pre- and postsynaptic spikes, phenomenological 'temporal learning rules' generate opposite change in synaptic efficiency depending on whether the postsynaptic spike in advance of, or follow, the presynaptic spike. There

is an attempt to show that differential Hebbian learning could be a proper framework to take into account the timing effects [Roberts, 1999].

Generalized Hebbian rules and their phenomenological derivations

Hebb's idea has been formalized in many variations. The first, and simplest versions of the Hebbian-learning rule have the important properties of being *local, interactive (specifically conjunctional, and time-dependent*, as we will now explain. If we accept these requirements, these properties should be attempted to preserve in course of its generalization.

The most general form of Hebb's rule to express the idea above, is that the synaptic weight from neuron i to neuron j changes according to:

$$\frac{d}{dt}w_{ij}(t) = F(a_i, a_j) \tag{1}$$

where F is a functional, and a_j and a_i are presynaptic and postsynaptic activity functions (i.e., they may include activity levels over some period of time and not just the current activity values. To define specific learning rules, i.e., the form of F, a few points should be clarified.

1) What are the assumptions about the *locality* of the modifying signal? In many cases, the modification of a synapse between neurons i and j depends on the state of these two cells alone, i.e., the mechanism is local. In this case teacher or external reinforcement signals are not explicitly involved: local synapses are the bases of the unsupervised learning.

2) How, if at all, do the presynaptic and postsynaptic cells *interact*? Consider first the potential answers for the "if at all" part of the question. The modification can be interactive, if both the pre- and postsynaptic cells are involved, and non-interactive, if either the pre- or postsynaptic cell alone influences the modification. The mechanism of the interaction may be conjunctional or correlational. In the first case, the co-occurrence of the pre- and postsynaptic activity is sufficient to cause synaptic change, while in the second case the covariance of the two activities has to be taken into account. (From a formal point of view, additive interactions - e.g., given with the function $F(a_i \pm a_j)$ - could have been defined, but they are considered as non-interactive rules. In other words, not only an entire rule, but even each

term of it can be evaluated as interactive or non-interactive.)

3) What are the assumptions about the form of the *time-dependent* activity functions? In the simplest case, only the actual activity values are involved. In somewhat more complex situations, short-term averaged activity values determine the synaptic change. More generally, the history of the activity values plays a role in the modification process.

The simplest Hebbian learning rule can be formalized as:

$$\frac{d}{dt}w_{ij}(t) = k\, a_i(t)\, a_j(t),\ \ k > 0 \tag{2}$$

This rule expresses the conjunction among pre- and postsynaptic elements (using neurobiological terminology) or associative conditioning (in psychological terms), by a simple product of the actual states of pre- and post- synaptic elements, $a_j(t)$ and $a_i(t)$.
A characteristic and unfortunate property of the simplest Hebbian rule (1) is that the synaptic strengths are ever increasing.

$$\frac{d}{dt}w_{ij}(t) = k\, g(a_i(t))\, h(a_j(t)) \tag{3}$$

where g and h, functions of the actual activity, serve as some measure of the post- and presynaptic activity (i.e., $g, h > 0$); and

$$\frac{d}{dt}w_{ij}(t) = k\, g(a_i(\cdot))\, h(a_j(\cdot)) \tag{4}$$

where g and h are now functionals of the activity function. A special case of (5) is

$$\frac{d}{dt}w_{ij}(t) = k \int_0^t a_i(t)dt \int_0^t a_j(t)dt \tag{5}$$

which takes into account the total activity history.

There is a particular time-dependent, local and conjunctional rule, which does not increase the synaptic weight. This is the case when the pre- and postsynaptic activities are negatively correlated:

$$\frac{d}{dt}w_{ij}(t) = k\, a_i(t)\, a_j(t),\ \ k < 0 \tag{6}$$

This "anti-Hebbian" rule (there is some confusion in the literature concerning this terminology, here it is used in the sense that $k < 0$) or "decorrelation" rule was suggested to describe features of dissociations of patterns [Barlow and Földiak, 1989].

There are both brutal and sophisticated methods to eliminate the unpleasant property of ever-increasing weights which, unless compensated for, yields a network with saturated synaptic weights and thus no effective pattern discrimination. The qualification "brutal" was adopted for the situation when some external constraint (taking into account somehow the finiteness of resources) is applied to the internal mechanism. First, a predetermined upper bound can be given, such as the maximal value of the synaptic strength. Second, the so-called normalization procedure (which appeared already in [Rochester et al., 1956] gives a finite-sum constraint on all synaptic strengths, and can be interpreted as a competition of the presynaptic elements for postsynaptic resources (therefore it violates locality). Such rules may explain some aspects of neural development

More sophisticated methods decrease the synaptic strengths selectively. [Brown et al., 1990] use the expression generalized Hebbian synaptic mechanism for cases where interactive synaptic increase is combined with activity-dependent synaptic depression. The underlying mechanism behind synaptic depression may be of interactive or non-interactive type.

Instead of giving a formal derivation of the rules which are able to describe selective decrease, two important special cases are mentioned. First, the rule

$$\frac{d}{dt} w_{ij}(t) = k\, g(a_i(t))\, (h(a_j(t)) - \theta(t)) \tag{7}$$

implements synaptic increase only if the $h(a_j(t))$ presynaptic activity is larger than the $\theta(t)$ modification threshold. If presynaptic activity is smaller than the threshold, the synaptic weight decreases. Second,

$$\frac{d}{dt} w_{ij}(t) = k\, (g(a_i(t)) - \theta(t))\, h(a_j(t)) \tag{8}$$

implements a postsynaptic control mechanism on the modification process.

The learning rules (8) and (9) can be written in the forms of $kgh - k\theta g$ and $kgh - k\theta h$, respectively. Each of these expressions may be interpreted as the sum of a Hebbian interactive term and a non-interactive term. In the first case the decrease is due to the postsynaptic activity g and is called "heterosynaptic" depression, while in the second case it depends on the presynaptic activity h and is called "homosynaptic" depression. Learning rules of the form (9) were suggested by [Bienenstock et al., 1982] and thus sometimes referred as the "BCM theory" - and used to model the plasticity of visual cortex. $\theta(t)$ was identified with a nonlinear function of the averaged postsynaptic activity:

$$\theta(t) = [g(t)]^2 \tag{9}$$

where $[\cdot]$ is the average taken for a period of time. The suggestion that the occurrence of either homosynaptic long-term potentiation (LTP) or long-term depression (LTD) depends on the strength of the depolarizing current induced by an NMDA blocker (which increases the modification threshold) in the visual cortex seemed to be justified experimentally.

The learning expression has also been described in the form $\phi(g, [g])h$, where the two-variable function ϕ depends on an "actual value" and an "averaged" quantity, so an underlying microscopic stochastic mechanism should exist behind the phenomenological and deterministic formalism.

The weaker form of the interactive rule (namely when correlational and not conjunctional interactions were assumed), namely

$$\frac{d}{dt} w_{ij}(t) = k\,(a_i(t) - [a_i(t)])(a_j(t) - [a_j(t)]) \tag{10}$$

was offered by [Rochester et al., 1956]. Depending on the sign of the correlation, the rule is capable of describing either synaptic enhancement or decrease. Covariance was suggested to induce associative LTD in the hippocampus.

Another way to describe the decrease of synaptic weights is the introduction of a spontaneous decay (or "forgetting") term. The original Hebbian rule (2) supplemented with a decay term reads as

$$\frac{d}{dt} w_{ij}(t) = -k_1 w_{ij}(t) + k_2 a_i(t) a_j(t) \tag{11}$$

(Instead of first order decay, a quadratic forgetting term was also introduced and studied to improve the stability properties of the learning rule.) If the decay is not spontaneous, but modulated with the postsynaptic activity, the rule has the form

$$\frac{d}{dt}w_{ij}(t) = -k_1 w_{ij}(t)a_i(t) + k_2 a_i(t)a_j(t) \equiv a_i(t)(k_2 a_j(t) - k_1 w_{ij}(t)) \quad (12)$$

and describes the phenomenon called "competitive learning". Postsynaptic neurons compete for incoming resources: the larger the postsynaptic activity, the larger the measure of learning.

$$\frac{d}{dt}w_{ij}(t) = k\frac{d}{dt}a_i(t)\frac{d}{dt}a_j(t) \quad (13)$$

This rule is an example of differential learning mechanisms [Klopf, 1986]. The rate of change of activities obviously may be positive or negative, i.e., both synaptic increase and decrease may occur. The differential competitive rule.

$$\frac{d}{dt}w_{ij}(t) = \frac{d}{dt}a_i(t)(k_2 a_j(t) - k_1 w_{ij}(t)) \quad (14)$$

implements the "learn only if change" principle.

In some cases, the time delay due to signal transmission is explicitly taken into account, consequently earlier presynaptic activities, rather than current activities, are in conjunction:

$$\frac{d}{dt}w_{ij}(t) = k\, a_i(t)\, a_j(t-\tau) \quad (15)$$

This spirit of "timing sensitivity" is materialized in the rule

$$\frac{d}{dt}w_{ij}(t) = k_1\frac{d}{dt}a_i(t)[a_j(t)] \quad (16)$$

used to describe conditioning (see e.g., [Sejnowski and Tesauro, 1990].

Hebbiam mechanisms and Hebbian algorithms

Hebb proposed that the connection between two neurons will be increased if activity in the neurons is temporally paired. More specifically, the Hebbian model proposes that the strength of a particular connection will increase if

the use of the synapse contributes to the occurrence of an action potential in the postsynaptic neuron. This account critically depends upon coincidence detectors in the postsynaptic neuron.

The underlying biophysical mechanisms and algorithms of even generalized Hebbian synaptic modification were reviewed by [Brown et al., 1990]. In the subsequent years system level computational models of the neural bases of learning and memory started also to proliferate.

The general question has been, and still it is, whether how to connect the formal algorithms of the neural basis of learning phenomena. Although many commonly used learning rules lead to successful models of plasticity and learning, they are inconsistent with neurophysiology. Other rules, more physiologically plausible, fail to specify relevant properties, such as bidirectionality and the biological mechanism that prevents synapses from changing from excitatory to inhibitory, and vice versa. Newer attempts try to overcome these difficulties.

Discussion: Over the Hebbian paradigm

It is certainly not true that all learning rules could be interpreted in (even generalized) Hebbian sense. It is difficult, however, to draw the borderline between the Hebbian and "non-Hebbian" frameworks. One possible choice is to consider a learning rule is Hebbian, if only two elements (one presynaptic and one postsynaptic) are involved. If we accept these limitations, we can determine what is labeled as non-Hebbian learning rule. Many types of supervised learning rules used in the ANN, such as delta rules, and its variations certainly belong to this category. Heterosynaptic plasticity and modifiability of synaptic triads and glomeruli - where *more than two cells* are explicitly involved in the modification process - could be understood also, as non-Hebbian. Such choice, however, would also exclude rules with the normalization procedure.

What is the relationship between the homosynaptic (or Hebbian activity-dependent), and heterosynaptic (or modulatory input-dependent) plasticity? It was suggested that Hebbian mechanisms are used primarily for learning and for forming short-term memory traces but they are not sufficient to recruit the events required to maintain a long-term memory [Bailey et al., 2000].

In contrast, heterosynaptic plasticity commonly recruits long-term memory mechanisms that lead to transcription and to synaptic growth. When jointly recruited, homosynaptic mechanisms assure that learning is effectively established and heterosynaptic mechanisms ensure that memory is maintained.

The spirit of the Hebbian idea survived more than a half century. It will be interesting to see whether what kinds of phenomenological learning rules will be derived in the next several years starting from cellular level experimental and modeling studies of synaptic modifiability.

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