

BIOLOGICALLY INSPIRED ARTIFICIAL NEURAL NETWORK ALGORITHM WHICH IMPLEMENTS LOCAL LEARNING RULES

Ausra Saudargiene^{1,2}, Bernd Porr¹ and Florentin Wörgötter¹

¹Department of Psychology, University of Stirling, Stirling FK9 4LA, Scotland

²Department of Informatics, Vytautas Magnus University, Kaunas, Lithuania

<ausra,bp1,worgott>@cn.stir.ac.uk

ABSTRACT

Artificial neural networks (ANNs) are usually homogeneous in respect to the used learning algorithms. On the other hand, recent physiological observations suggest that in biological neurons synapses undergo changes according to local learning rules. In this study we present a biophysically motivated learning rule which is influenced by the *shape* of the correlated signals and results in a learning characteristic which depends on the dendritic site. We investigate this rule in a biophysical model as well as in the equivalent artificial neural network model. As a consequence of our local rule we observe that transitions from differential Hebbian to plain Hebbian learning can coexist at the same neuron. Thus, such a rule could be used in an ANN to create synapses with entirely different learning properties at the same network unit in a controlled way.

1. INTRODUCTION

Learning rules used to update the weights in artificial neural network algorithms are the same for all inputs and units. However, recent physiological experiments suggest that in biological neurons synaptic modifications depend on the location of the synapse (1) i.e. synaptic strength is regulated by local learning rules.

The same synapse may be strengthened and weakened depending on the temporal order of the pre- and postsynaptic activity. The weight grows if the presynaptic signal precedes the postsynaptic signal, and shrinks if the temporal order is reversed. Such form of synaptic modifications is called spike-timing-dependent plasticity (STDP) (2). However, not only the timing of the pre- and postsynaptic activity, but also the shapes of the signals may define the properties of synaptic plasticity. This claim is supported by the fact that strong depolarization, necessary to induce synaptic changes, has a different origin and a varying shape along the dendritic tree. Close to the soma learning is driven by steep and short back-propagating spikes which become more shallow and longer in duration while back-propagating

into the dendritic tree (3). In distal parts, where back-propagating spikes fail to invade, slow and wide local Na^+ - and Ca^{2+} channel-dependent dendritic spikes provide the necessary depolarization (1). These observations suggest that synaptic modifications are location-dependent.

In this paper we present a biophysical model of STDP which captures the dependence of synaptic changes on the membrane potential shape. The model uses a differential Hebbian rule to correlate the NMDA synaptic conductance and the derivative of the membrane potential at a synapse. We will show that the model reproduces the STDP weight change curve in a generic way and is sensitive to the different shapes of the membrane potential. The model predicts that learning depends on the synapse location on the dendritic tree. Then we will describe the equivalent circuit diagram and discuss the model referring to system-theory, presenting it in the context of filter transfer functions at the end of this article.

2. BIOPHYSICAL MODEL

The model represents a dendritic compartment with a single NMDA synapse (Fig. 1 A). The NMDA channels are essential in inducing synaptic plasticity as their blockade to a large degree prevents STDP (1). It is believed that NMDA channel-mediated Ca^{2+} influx triggers the chain reactions involving CaMKII, calmodulin, calcineurin and in this way affects the synaptic strength (4). The NMDA synaptic conductance, regarded as a presynaptic signal, is given by :

$$g(t) = \bar{g}\hat{g}(t) = \bar{g} \frac{e^{-t/\tau_1} - e^{-t/\tau_2}}{1 + \kappa e^{-\gamma V(t)}} \quad (1)$$

where V is the membrane potential, $\bar{g}_N = 4 nS$ peak conductance, $\bar{g}_N = 4 nS$, $\tau_1 = 40 ms$, $\tau_2 = 0.33 ms$ time constants and $\eta = 0.33/mM$, $[Mg^{2+}] = 1 mM$, $\gamma = 0.06/mV$ (5). The membrane potential is expressed as:

$$C \frac{dV(t)}{dt} = \rho g(t)[E - v(t)] + i_{dep}(t) + \frac{V_{rest} - V(t)}{R}, \quad (2)$$

where ρ is the synaptic weight of the NMDA-channel, g its conductance, $E = 0 \text{ mV}$ its equilibrium potential. The current i_{dep} is used to account for the depolarization caused by other sources than synaptic inputs, such as back-propagating spikes or local dendritic regenerative potentials. The last term represents the leakage current. The resting potential $V_{rest} = -70 \text{ mV}$, membrane capacitance $C = 50 \text{ pF}$ and the membrane resistance to $R = 100 \text{ M}\Omega$.

The differential Hebbian learning rule for the synaptic change is defined as:

$$\frac{d\rho}{dt} = \hat{g}(t)V'(t), \quad (3)$$

where \hat{g} is the normalized conductance function of the NMDA channel, the pre-synaptic influence quantity, and V' is the derivative of the postsynaptic membrane potential.

The depolarizing membrane potentials, which trigger synaptic plasticity, vary along the dendritic tree. We use a short and steep back-propagating action potential to model the synaptic changes close to the soma, and long and shallow dendritic spike to account for synaptic modifications in the distal parts. The back-propagating spike and the dendritic spike, measured $210\mu\text{m}$ and $860\mu\text{m}$ from the soma, respectively, are presented in Fig. 1 B and have been taken from (6; 7). The depolarization coming from these spikes is very strong, therefore we may neglect the contribution of the NMDA synaptic input. Instead of using Eq. 2 we calculate the change of the membrane potential using the given shape of the spike and then substitute its derivative in the learning rule (Eq. 3).

We obtain an asymmetrical weight change curve if the depolarization is provided by a steep back-propagating spike (Fig. 1 C). The synapse is weakened if $T < 0$ and strengthened if $T > 0$, where T is the temporal difference between the presynaptic activity and the postsynaptic activity. $T > 0$ means that the postsynaptic signal follows the presynaptic signal at the NMDA channel and vice versa. However, we observe a shifted curve if the depolarization comes from the shallow dendritic spike. The synaptic weight grows even for negative values of $T > -20\text{ms}$. Thus, we get plain Hebbian learning between -20ms and ∞ .

The model reproduces the STDP curve in a generic way. The shape of the weight change curve is strongly influenced by the shape of the depolarizing membrane potential, which induces plasticity. The slow rising flank of this signal is the essential factor of the transition from an asymmetrical to a symmetrical weight change characteristic. As the depolarizing potentials vary in different parts of the dendritic tree, these results suggest that learning rules are local and depend on the location of the synapse in biological neurons.

The electrical circuit equivalent to the model described above is presented in Fig. 2. Elements R_1, C_1 define the shape of the presynaptic signal g . R_3 corresponds to the in-

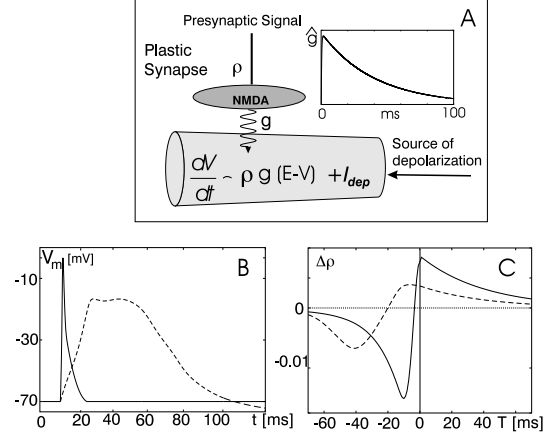


Fig. 1: Schematic diagram of the model. A) Components of the membrane model. The inset shows the NMDA synaptic conductance function. B) Depolarizing membrane potentials: steep back-propagating spike and shallow dendritic spike $210\mu\text{m}$ and $860\mu\text{m}$ from the soma, respectively C) The resulting weight change curves. The shallow depolarizing potential leads to potentiation even for negative values $0 < T < -20\text{ms}$ values.

tracellular resistance, R_2 and C_2 describe the passive membrane properties and altogether determine the shape of the postsynaptic signal v . The derivative of v , obtained after the filtering in the last R_2 and C_2 circuit, is multiplied by g . The resulting so called weight change is fed to a gain-controlled amplifier and influences the postsynaptic signal v . Various shapes of the postsynaptic signal v may be obtained by adjusting the values of R_2, R_3 and C_2 and would lead to different learning characteristics.

3. BIOLOGICALLY INSPIRED SITE-SPECIFIC LEARNING ALGORITHM

We represent a further step of abstraction in Fig. 3. This block-diagram is not directly equivalent to the circuit in Fig. 2 but it captures the main observation emerging from the biophysical model. Namely that learning depends on the location of the synapse, i.e. is driven by the derivative of a postsynaptic signal specific at a given site. In an artificial neural network system this would mean that output signal undergoes a transformation specific for each input and only then its derivative is applied to update the weight of a given input. The diagram of such an algorithm is presented in Fig. 3. We can still roughly associate the NMDA characteristic to the pathways x_1, \dots, x_n representing many (possibly different) inputs and the source of depolarization (e.g., the back-propagating spike) to the pathway x_0 . Hence this pathway enters the summation node with an unchangeable weight ρ_0 . This circuit is a modified version of the ISO learning circuit (8). ISO learning is a drive-reinforcement

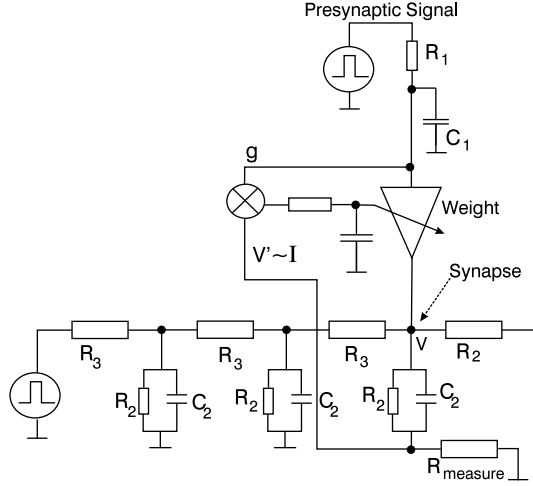


Fig. 2: Equivalent electrical circuit of the learning algorithm. Postsynaptic signal v is differentiated by $R_2 C_2$ circuit and multiplied by the presynaptic signal g to obtain the weight which influences the postsynaptic signal v via a gain-controlled amplifier.

algorithm for temporal sequence learning where the weights change according to the relative timing of the input signals. All inputs x_0, x_1, \dots, x_n are filtered using bandpass filters h_0, h_1, \dots, h_n , weighted by $\rho_0, \rho_1, \dots, \rho_n$ and summed to produce the output v : $v = \rho_0 u_0 + \sum_{i=1}^n \rho_i u_i$, where $u = x * h$. Different from the ISO learning, here the output is also filtered with the filters h_{11}, \dots, h_{nn} , and only then the derivatives of the obtained signals v'_1, \dots, v'_n are used to change the weights of the corresponding inputs:

$$\frac{d}{dt} \rho_i = \mu u_i v'_i \quad \text{where } v_i = v * h_{ii}, \mu \ll 1. \quad (4)$$

We assume that the input x_0 is dominating the output and its weight ρ_0 is fixed. We apply the analytical solution derived for the ISO learning (8) to calculate the weight change curve for different shapes of the filtered output signal (for details see Appendix). For a steep output signal entering the learning rule we obtain differential Hebbian learning, and for a shallow one we get a curve similar to plain Hebbian learning (Fig. 3B,C). The parameters of the filters h_{11}, \dots, h_{nn} which transforms the output signal determines this transition.

4. DISCUSSION

The biophysical model of STDP inspired an artificial neural network algorithm with site-specific learning rules. The biophysical model is based on a differential Hebbian learning rule which correlates the NMDA synaptic conductance with the derivative of the membrane potential. The results show that the weight change curve strongly depends on the

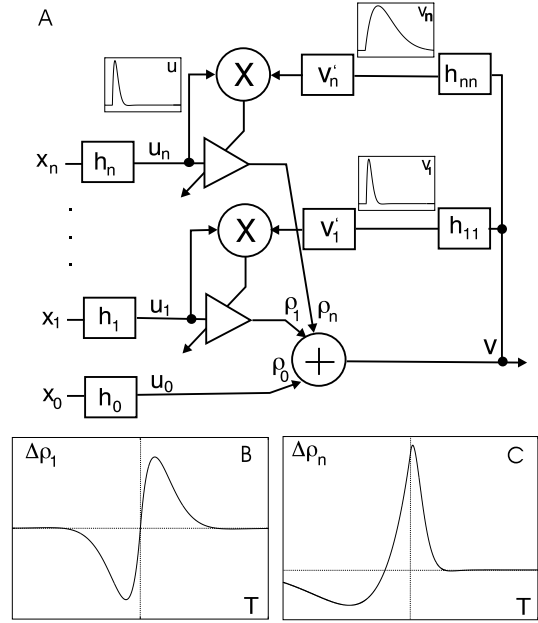


Fig. 3: A) Algorithm for site-specific learning. Transfer functions are denoted as h , changing weights ρ as an amplifier. All inputs are filtered. Weight ρ_0 is fixed. Weights ρ_1, \dots, ρ_n are updated using the derivatives v'_1, \dots, v'_n of the filtered output v . Filter functions h_{11}, \dots, h_{nn} differ for each input. B) Analytically calculated weight change curve if the filtered output has a steep rising flank C) Analytically calculated weight change curve if the filtered output has a shallow rising flank.

shapes of the depolarizing membrane potential at the location of the synapse. This signal changes its shape along the dendrite and may be provided by different mechanisms such as back-propagating spikes close to the soma and dendritic spikes in the distal parts. Therefore we predict that learning rules are location-dependent. Close the soma, where learning is driven by short back-propagating spikes, the synaptic modifications are bidirectional, described by an asymmetrical STDP curve. In the distal parts, where synaptic changes are induced mainly by long-lasting dendritic spikes, synapses undergo potentiation even for negative values of T . The same learning rule leads to different synaptic modifications and it is self-adjusting following the shapes of the depolarization source in different locations of the dendritic tree.

The typical approach to model STDP is to assume a certain weight change curve which does not depend on the local properties of the cell, e.g. (9). A few more detailed models take into consideration the postsynaptic signal which is associated with the membrane potential, e.g. (10; 11; 12) and observe that its shape influences the shape of the weight change curve. These models differ from our as the rule of (10) is based on TD learning, while (11; 12) rely on the absolute Ca^{2+} concentration in the weight updating algo-

rithm.

Our algorithm offers the possibility to easily define a parameter-controlled learning rule in an artificial neural network. We have just now started trying to solve an instrumental conditioning problem, where the actions of the learner influence its inputs and hence the learning with such an architecture. A small network of sub-compartmentalized neurons is linked to a simple agent that reacts to stimulus presentation with an orienting behaviour following the stimulation of the right neuronal subset. The goal is to train it with one unconditioned stimulus (US) and several conditioning stimuli (CS) only one of which is correlated to the unconditioned stimulus. The US will always trigger the correct output neurons to elicit the orienting response. Conversely, each CS elicit a response in many input neurons, some of which are better correlated with *each other* than others. Hebbian learning *between* these CS inputs will “extract” and strenghten the better-correlated neurons. This leads, after learning, to a drive from *all* CS regardless of their correlation with the US. Now we get reliable (but mostly *wrong*) behavioural reactions. Since all but one CS are temporally uncorrelated to the US, differential Hebbian learning will lead to a weakening of all “wrong” CS. By the end, the system has learned to drive a small subset of only a few neurons in a feed-forward way, eliciting a response that will lead to the desired behaviour. This is work in progress and no results exist so far. Nevertheless, it clearly shows how such sub-compartmentalized learning rules could be used for behavioral control.

5. APPENDIX

The weight change curves are calculated using the analytical solution obtained for ISO learning (8). We assume that the output is dominated by x_0 and the contribution of other inputs is negligible ($\rho_i|_{t=0} = 0, i > 0$). Then the pairs of the filter functions h_0 and h_{11} , h_0 and h_{22} , etc., h_0 and h_{nn} can be considered as single filter functions h_{01}, \dots, h_{0n} . These filters are specific for each input x_1, \dots, x_n pathway and shape the output signal v_i whose derivative enters the learning rule. The filters h are described by: $h(t) = \frac{1}{b} e^{at} \sin(bt)$ with $a := -\pi f/Q$ and $b := \sqrt{(2\pi f)^2 - a^2}$, where f is the center frequency and Q is the damping. Then the cumulative weight change at the i -th pathway is given by: for $T \geq 0$

$$\rho_i(T) = \mu \frac{b_i M_i \cos(b_i T) + (a_i P_i + 2a_{0i} |p_i|^2) \sin(b_i T)}{b_i (P_i + 2a_i a_{0i} + 2b_i b_{0i}) (P_i + 2a_{0i} a_i - 2b_i b_{0i})} e^{-T a_i}$$

and for $T < 0$

$$\rho_i(T) = \mu \frac{b_{0i} M_i \cos(b_{0i} T) + (a_{0i} P + 2a_i |p_{0i}|^2) \sin(b_{0i} T)}{b_{0i} (P_i + 2a_{0i} a_i + 2b_{0i} b_i) (P_i + 2a_{0i} a_i - 2b_{0i} b_i)} e^{-T a_i},$$

where $M_i = |p_i|^2 - |p_{i0}|^2$, $P_i = |p_i|^2 + |p_{i0}|^2$ and $p_i = |a_i|^2 - |b_i|^2, p_{0i} = |a_{0i}|^2 - |b_{0i}|^2, i > 0$. The parameters for the weight change curves presented in Fig. 3 are: $f_{01} = 0.01, Q_{01} = 0.6, f_{0n} = 0.002, Q_{0n} = 0.6, f_1 = f_n = 0.01, Q_1 = Q_n = 0.6$.

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