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Revisited: Hebbian Postulate under Homeostatic Plasticity

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Abstract: We propose a new method to model Hebbian Postulate with the intension of developing new learning algorithm based on it. The propose method integrate the Hebbian Postulate with Homeostatic Plasticity to avoid the node saturation of the conventional Hebbian based learning algorithms. Moreover the completely novel view of the brain as a network of agents with large number of constituent components, signal propagation within the network and complete elimination of weight components are main features that distinguish our method with the conventional approaches while making our model much closer to the biologically supported learning environment.

Keywords: Hebbian Postulate, Homeostatic Plasticity, Artificial Neural Network

I. INTRODUCTION

With the advancement of Information Technology, the world has being dreaming of imitating human cognitive system on machines. As an attempt of it, Artificial Neural Network, Fuzzy Logic, and Collective Intelligence are some of those technologies which have been evolved in mainly with this aspect. Artificial Neural Network is still considered as the main technology that reasonably imitates the human learning and memory formation. Artificial Neural Network has composed and evolved with many learning algorithms thereafter. Among many other learning rules, Hebbian learning rule is considered as the most effective learning rule that is supported by the biological findings. Hebbian learning rule is derived from the Hebbian Postulate which is based on the correlated activities of presynaptic neuron and postsynaptic neuron. In Hebbian Postulate, Hebb [1] says that "*When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process and metabolic change takes place in one or both cells such that A's efficiency, as the one of the cells firing B, is increased*".

It is very interesting to note that it has not been mentioned anything about what would happen when the cell A is not correlated with the cell B. One can argue that either it may decrease the correlated firing rate of the cell A and the cell B or there might be no significant changed to the current correlated firing rate of the cell A and the cell B when they become uncorrelated. The Hebbian learning algorithm is based on the first assumption and its basic mathematical formula is shown in "(1)".

$$\Delta w_{ij}(t) = \eta x_i x_j \tag{1}$$

x_j is the output of the presynaptic neuron, x_i is the output of the postsynaptic neuron, η is the learning rate and w_{ij} is the strength of the connection between presynaptic neuron and postsynaptic neuron.

It can be seen that correlated input patterns will influence the neuron's weight and eventually produce the largest output. On the other hand weight strength of the uncorrelated neurons will tend to zero for uncorrelated input patterns. Either this unbounded increase of the strength of presynaptic and postsynaptic connectivity or the decrease of strength of the connectivity makes to lose the sensitivity of these neurons to external inputs. This issue is known as node saturation, and it can be seen in many Hebbian Postulate based learning algorithms, Williams and Noble [2]. To overcome this critical issue, many versions of Hebbian learning have been derived, some of those are Rate-based Hebbian learning, Spike based Hebbian learning, Gerstner and Kistler [3] and Differential Hebbian learning rule, Kosko [4]. Critical analysis on these learning algorithms, we can identify that they are based on six important factors, namely, locality, co-operativity, synaptic depression, boundedness, competition and long-term stability, Gerstner and Kistler [3]. The factor boundedness has been introduced mainly to eliminate this node saturation issue and it has been implemented as the concept of weight normalization. Basically it is an adjustment to weight components by a calculated parameter to bring it back a saturated node to unsaturated status, Williams H. and Noble[2], Abbott and Nelson [5], even though these updating significantly improve the signal propagation within the

network, it has also damaged to the learning of the neural network and its performances, Williams H. and Noble [2], Williams [6]. Therefore, it is still a research challenge for researches to find appropriate learning algorithm based on Hebbian Postulate which is also supported by biological findings. In our article we propose a new approach to study Hebbian Postulate with the aim of developing more effective learning algorithm. Our study concentrate on the latter assumption that is there might be no significant changed (decreased) to the current correlated firing frequency of the cell A and cell B when they become uncorrelated. We propose to integrate Homeostatic Plasticity with the Hebbian Postulate instead of introducing weight normalization concept as a boundednes factor.

According to biologist while extremely large stimuli take neurons' firing frequency into very high firing frequency, extremely low stimuli may take neurons' into very low firing frequency, however as per biology, although fluctuations to external and internal stimuli are necessary for learning, it is also required to maintain the neuron's firing frequency in a feasible range. Homeostatic Plasticity is the mechanism that helps neurons to maintain their fluctuations in a feasible range, Turrigiano [7]. The significant feature of this process is it decreases the firing frequency of the neuron when it is extremely high and similarly it increases the firing frequency of neuron when its firing frequency is very low. This is supposed to be achieved through the change of neurons' electrical and, morphological properties and ionic concentrations. For an example, when a neuron is in high firing frequency, Homeostatic Plasticity closes down ca+2 ion channels to decrease the ca+2 ion concentration which in turn reduces the amount of neurotransmitter release and thereby the firing frequency of the neuron. On the other hand, when the neuron firing frequency is very low, it opens up ca+2 ion channels to increase the ca+2 ion concentrations in order to increase the amount of neurotransmitter release, Nicholls and Martin [8].

II. METHOD

We understand nervous system as a network of neurons; each neuron is an agent consists of a large number of constituent elements which work as synapses. A synapse can be either a transmitter or a receptor. A model neuron, in our study is shown in fig. 1. The propose model neuron enables synapses to have two dynamic statuses, either active or inactive. When a receptor receives a signal from a transmitter in another neuron, the receptor propagates the signal to a transmitter in the same neuron if the receptor is in an active status at the time of receiving. Similarly, a transmitter can transmit a signal to a receptor in other neuron, if the transmitter is in an active status at the time of signal transmitting. If the selected receptor or transmitter is in an inactive status at the time of receiving and transmitting respectively then the signal is dropped. Further, receptors in a neuron are grouped and number of receptor-groups within a neuron is equal to the number of neurons in the network -1. Number of receptors in a group may be different within a neuron and among neurons. Therefore, number of receptors and active number of receptors of a given connection are critical parameters that determine the strength of the connection between two neurons at a given time. These active and inactive statuses of constituent components are determined by an integrated process, which can be decomposed into two processes, synaptic computation and homeostatic plasticity. Our proposed network consists of 4 neurons because it has been proven that network with 4 neurons is capable enough to simulate learning effectively, Izquierdo-Torres and Harvey [9]. The network structure according to our approach is shown in fig 2. Receptors are grouped into three groups to establish the connection with other three neurons. For an example, if an active transmitter in neuron A wants to transmit a signal to a receptor in neuron B, then it selects a receptor from A-receptor group in neuron B.

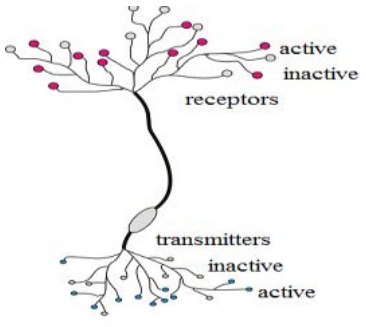

Fig. 1. A typical structure of a model neuron.

1. Signal Propagation within the Network

When a brief train of stimuli is applied to a pre-synaptic neuron, during the train, amplitude of the resulting pre-synaptic potential may either increase (called synaptic facilitation) or decease (synaptic

depression). According to biologist, the amount of neurotransmitter release from pre-synaptic terminals is subject to these two relatively short-term modifications. At the facilitation, the amount of neurotransmitter release is very high and it decays along the time. This phenomenon can be easily explained using internal ca+2 ion concentration. Arrival of train of stimuli increases the amplitude of pres-synaptic potential which opens up ca+2 ion gates. The growth of internal ca+2 ions concentration increases the amount of neurotransmitter release, however, as time goes, the appearance of antagonized ions into pre-synaptic terminal such as magnesium, cadmium, nickel, etc reduces the internal ca+2 ions concentration. This decreases the amount of neurotransmitter release. Similarly, continuous stimulus may also reduce pre-synaptic potential which makes the amount of neurotransmitter release is minimal at the depression.

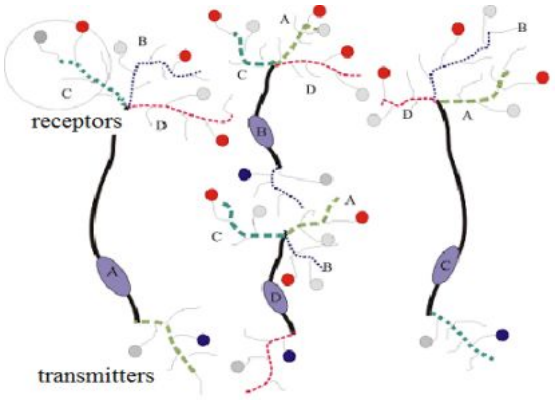

Fig.2. Structure of the network with four model neurons.

These two phenomena have been considered in the model proposed by Maass and Zador [10]. They have modeled the behavior of a synapse as a stochastic process with two finite statuses, i.e. R and F. This model is used in our approach to control signals propagation within the network. We map these two statuses, into active status and inactive status of our constituent components respectively. In their model, for each spike in spike train t, the output of a synapse consists of the sequence S(t) of those $t_i \in t$ on which neurotransmitters are released by S. Thus, $t \in S(t)$ becomes a stochastic process, computed by synapse S, with output sequence $q = q_1, q_2, q_3, \ldots, q_n \in \{R,F\}$. $P_s(t_i)$ defines, see "(2)", the probability that i^{th} spike in the pre-synaptic spike train $t = (t_1, t_2, \ldots, t_k)$ triggers the release of a signal at time t of the synapse S. If $P_s(t_i) > 0$ then spike excites synapse and releases the neurotransmitters, so the output is R, otherwise the output is F. Non-negative functions C(t) and V(t), defined in "(3)" and "(5)" model facilitation and depression. Function C(s) in "(4)", models the response of C(t) to a pre-synaptic spike that had reached to the synapse S at t-s. Moreover function V(s) in "(6)" models the response of V (t) to a proceeding release of the synapse S at time $t\text{-}s \leq t$. Whilst non-negative parameters α, τ_c and τ_v model the magnitude of the signal and decay constants of facilitation and depression respectively. C_0 and V_0 model the parameters for facilitation and depression at the equilibrium. So that, C_0 is the internal ca+2 ions concentration at the equilibrium and V_0 is the maximum amount of neurotransmitters can be released by a synapse.

$$P_s(t_i) = 1 - \exp(-C(t_i) * V(t_i)) \quad (2)$$

$$C(t) = C_0 + \sum_{t_i < t} C(t - t_i) \quad (3)$$

$$C(s) = \alpha . \exp(-s/\tau_c) \quad (4)$$

$$V(t) = \max(0, V_0 - \sum_{t_i < t \text{ and } t_i \in S(t)} V(t - t_i)) \quad (5)$$

$$V(s) = \exp(-s/\tau_v) \quad (6)$$

This stochastic process has been developed and tested only for a one synapse, but in our research it is applied into thousands of individual synapses. Therefore, we modified the model by introducing a θ as a threshold value, if $P_s(t_i) > \theta$ then spike excites synapse, so the output becomes R otherwise it is F. This threshold value is determined in the training phase under homoeostatic plasticity process.

2. Homeostatic Plasticity

Each model neuron has got four threshold values. One threshold value is for transmitters and other three threshold values are for three receptor-groups. These threshold values are determined to ensure the stability of the network within a particular range. So that, when firing rate of a neuron increases, it also increases the threshold values of the relevant groups to derive the network towards stability. Similarly, when the firing rate of a neuron is very low, it decreases threshold values of the relevant groups, in order to increase the firing frequency of the neuron. Let RIJ is J-receptor-group of I-neuron. For an example, RAC is the C-receptor-group of the A-neuron. XAC is the output of the RAC receptor-group. TI is the transmitter group of I-neuron

and OI is the output of the transmitter group of I-neuron. For an example, TA is the transmitters in neuron A and OA is the output of the transmitter group of neuron A, see fig. 3.

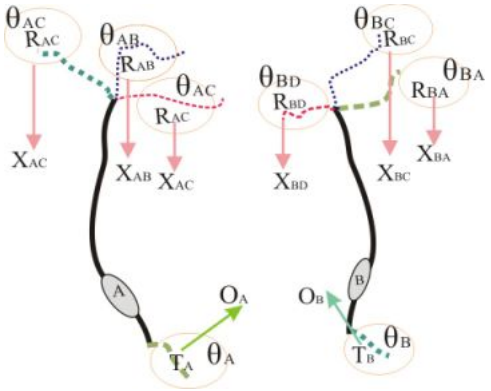

Fig..3. Signal transmission among neurons.

θ_I is the threshold value for the transmitters in neuron I. θ_{IJ} is the threshold value for the J-receptor-group of the I-neuron. We define θ_I as in "(7)". Instance of "(7)" is shown in "(8)". The output of IJ-receptor-group, X_{IJ}, can be expressed in terms of active receptors in the IJ-group as define in "(9)". Similarly, O_I can be defined in terms of active transmitters in I-neuron as shown in "(10)". f(.) is the threshold calculation function, defined in "(11)".

$$\theta_I = f(O_I * [X_{IJ_1} + X_{IJ2} + X_{IJ3}]) \quad (7)$$

$$\theta_B = f(O_B * [X_{BA} + X_{BC} + X_{BD}]) \quad (8)$$

$$X_{IJ} = \frac{\text{Act.Recp in } R_{IJ}}{\text{Recp.in } R_{IJ}} \quad (9)$$

$$O_I = \frac{\text{Act.Trans.in } T_I}{\text{Trans.in } T_I} \quad (10)$$

$$f(x) = 1/(1+\exp(-x)) \quad (11)$$

Similarly, we calculate threshold values for each receptor-group, θ_{IJ}, in terms of active number of constituent components in relevant neurons, as defined in "(12)". Calculated threshold values are then use as constant values in the testing phase.

$$\theta_{IJ} = f\left(\frac{X_{IJ}}{O_J}\right) \quad (12)$$

III. DISCUSSION

In our study instead of defining weight component to represent synaptic efficacy we define small programmable computational units which change their statuses, active and inactive, according to the amount of signal processing. These programmable units are attached to the neurons which are defined as agents and their communication enable through the message passing which in turn represents the internal and external signals to the network. Thus number of active computational units defines the strength of the connectivity between presynaptic and postsynaptic neurons at a given time. The active and inactive statuses of these small programmable units are subjected to the Homeostatic plasticity process and Zador and Mass approach. The most significant feature of our method is no initialization of weight components but the introduction of threshold increment process to the training phase. Bipolar status of constituent components at the site of neurotransmitter release, i.e. R or F, and integration of Homeostatic plasticity as a stability supportive mechanism and the complete elimination of weight components are other significant features that distinguish our method from others while making us much closer to the biological findings than existing learning environments in Artificial Neural Network.

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