New Threshold Updating Mechanism to Stabilize Activity of Hebbian Neuron in a Dynamic Stochastic 'Multiple Synaptic' Network, similar to Homeostatic Synaptic Plasticity Process

Subha Fernando, Koichi Yamada and Ashu Marasinghe
Nagaoka University of Technology, Niigata, Japan

ABSTRACT
Unconstrained growth of synaptic activity and lack of references to synaptic depression in Hebb's postulate have diminished its value as a learning algorithm. The existing synaptic scaling mechanisms such as weight normalization, threshold updating, and spike time dependent plasticity have greatly contributed to address these issues in Hebb's postulate. However, all these mechanisms are based on the networks with single synaptic connection between neurons which process according to a central clock. This article presents a new threshold updating mechanism which behaves similar to the biological process called Homeostatic synaptic plasticity process and helps Hebb's presynaptic neuron to stabilize its activity in a dynamic stochastic multiple synaptic network. Our modeled network had neurons that (1) processed signals in different time scales, (2) having dynamic and multiple synaptic connections between neurons. And these synapses were modeled as stochastic computational units which had computational power to calculate their own signal release probability as a function of signal arrival time to these synapses, and (3) neurons regulated their own local excitation through the threshold updating mechanism. Under these significant features of the modeled network, the examined neuron exhibited the behavior similar to the presynaptic neuron of Hebb's postulate when its activity synchronized with the postsynaptic neuron's activity. And it demonstrated the behavior similar to the Stent's and Lisman's anti-Hebbian postulates when its activity asynchronized with the activity of the postsynaptic neuron.

General Terms
Neural Networks

Keywords
Hebb's Postulate, Stent's anti-Hebbian Postulate, Stochastic Computational Synapse.

1. INTRODUCTION
Although it has been more than a half a century since Hebb's postulate was introduced by the Donald Hebb [1], it still deserves careful analysis about its behavioral explanation about the two neurons when their association account for learning; though it has been widely applied in the field of neural network, the lack of references to synaptic depression and unconstrained growth of synaptic gain have diminished its value as a learning algorithm. The issue of unconstrained gain of synapses in Hebbian learning based neural networks called node saturation abolishes the sensitivity of the entire network to the external updates. The approaches that have been taken so far to overcome the issues in Hebb's postulate can be classified mainly into three categories; techniques based on weight scaling or weight normalization [2, 3, 4, and 5], threshold updating mechanisms such as BCM theory [6] and algorithms based on Spike Time Dependent Plasticity (STDP) [7, 8, 9]. Weight normalization in unsupervised competitive Hebbian learning algorithm receives great attention because of their mathematical plausibility of explaining the effect of weight constraints very clearly on the behavior of the networks. However it has been shown that dynamic behavior [3] and learning ability [4 and 5] of these networks highly depends on the enforced weight constraints. BCM theory is another significant theory based on Hebb's postulate. This theory explains the synaptic activity as a temporal competition between input patterns and explains the dynamics of a neuron as a function of postsynaptic response. Synaptic inputs that drive postsynaptic firing to higher rate than a threshold value result in an increase of synaptic strength by inducing long-term potentiation (LTP) while synaptic inputs that drive postsynaptic firing to lower rate than the threshold value result in a decrease of synaptic strength by inducing long-term depression (LTD). Even though biological evidences support the sliding of threshold in neurons according to the sensitivity of the input it received, sliding of the threshold merely based on the postsynaptic activity as defined in the BCM theory is not directly biologically supported [10]. In addition, BCM theory considers instantaneous postsynaptic firing frequencies for its threshold updating mechanism rather than the effect of spike arrival time to the synapses. According to the latest biological findings, generation of LTP and LTD mainly depends on the spike arrival time to the synapses and the probability of spike release at these synapses is also controlled at synaptic levels than at neuronal levels [11, 12]. In contrast, STDP mainly depends on the timing between presynaptic and postsynaptic spikes. STDP is described by the window function that determines how the strength of synapses is modified by a pair of spikes. If presynaptic spikes occur before the postsynaptic spikes it strengthens the synaptic strength; if presynaptic spike occur after the postsynaptic spike it weakens the synaptic strength [13]. Although all these concepts are very important for a computational model, strengthening and weakening of synaptic connections further depend on other factors as well. Among them the most significant factors are: the number of synaptic connections between two neurons, type of postsynaptic neuron, the size of the postsynaptic depolarization and the probability of spike release at each synapse [14, 11, and 15]. In order to understand the effects of some of these factors on the

behavior of Hebb's presynaptic neuron, we modeled a fully connected network with four neurons. Our network structure supported four significant features: First, The modeled neurons processed signals in different timescales, namely, slow, fast and medium; this arrangement allowed us to analysis the effect of different time scales on the behavior of Hebb's neurons. Second, each neuron was attached a large number of stochastic computational units, called receptors and transmitters. And the receptors in a neuron were grouped to establish to dedicated connections between the transmitters of a particular presynaptic neurons. Thus, the transmitters of the presynaptic neuron and the receptors of the corresponding receptor group of the postsynaptic neuron build an artificial synaptic connection between the two neurons. Third, these stochastic computational units updated their signal releasing probability through their own local feedback process which sensed the excitation of the synapse they attached. Furthermore, the mathematical process of these computational units modeled the biological process that occurs in a single synapse. Doing so we assumed that above second and third properties enable multiple and dynamic synaptic connectivity between two neurons. Finally, to calculate the signal release probability at each signal, a stochastic computational unit accounted all the preceding released signals and summed their contributions. This approach drove the network to a saturated state (similar behavior is discussed in [16]). Therefore in order to stabilize the network activity a new threshold updating mechanism was introduced. The introduced threshold updating mechanism helped the neurons to measure its own excitation in terms of its receptors and transmitters. These four features successfully regulated the excitation of Hebb's presynaptic neuron while demonstrating the behaviors characterized in fundamental theorems in the learning process, namely Hebb's postulate, Stent's anti-Hebbian postulate and Lisman's anti-Hebbian postulate. Further, the introduced new threshold updating mechanism demonstrated the behavior similar to the homeostatic synaptic plasticity process, a biological process that helps neurons to maintain their firing frequencies within a feasible range through synaptic redistribution [17, 18].

The synaptic depression in Hebb's postulate has been physiologically discussed by Stent [19] as a complementary statement of Hebb's postulate, "When the presynaptic axon of cell *A* repeatedly and persistently fails to excite the postsynaptic cell *B* while cell *B* is firing under the influence of other presynaptic axons, metabolic change takes place in one or both cells such that *A*'s efficiency, as one of the cells firing *B*, is decreased". As per Stent, neuron *A*'s activity is decreased when it fails to excite the postsynaptic cell *B*. This occurs when cell *A* fails to synchronize its activity with the other presynaptic neurons of the postsynaptic cell *B*. Stent explanation to *A*'s synchrony can be quoted as follows, "The activity of the synapse of cell *A* upon cell *B* is manifestly asynchronous with the activity of synapses of other cells converging on cell *B* if most the impulses that arise in cell *B* occur while the synapse of cell *A* is inactive". He has further added that this asynchrony between the presynaptic neuron and postsynaptic neuron is detected at postsynaptically. Lisman [20] called the Stent's synaptic depression 'post-not-pre' anti-Hebbian process and he has discussed another possibility of synaptic depression in Hebb's postulate called 'pre-not-post' anti-Hebbian process. Lisman's synaptic depression occurs "when the presynaptic input is active but the postsynaptic cell is not active because of inadequate excitation by other inputs or too much inhibition by other neurons".

2. NETWORK MODEL

A fully connected network was developed with four artificial neurons; namely two input layer neurons *A* and *B*, and two hidden neurons *C* and *D* (no signals were externally fed into these hidden neurons). Each modeled neuron had thousands of artificial stochastic units. These units were classified into two classes based on the role they played in the network. An artificial unit in a neuron was called a receptor if it took signals into the neuron otherwise it was called a transmitter which transmitted signals to its postsynaptic neurons (the neuron that received the signal). Moreover the receptors attached to a neuron were grouped so that when a presynaptic neuron (the neuron that transmitted the signal) wanted to contact a postsynaptic neuron, a transmitter in the presynaptic neuron could contact a receptor in the corresponding receptor group of the target postsynaptic neuron, see figure 1. Thus neurons connected with other neurons through an artificial synaptic connection which mediated the communication between transmitters of the presynaptic neuron and the corresponding receptor group of the postsynaptic neuron.

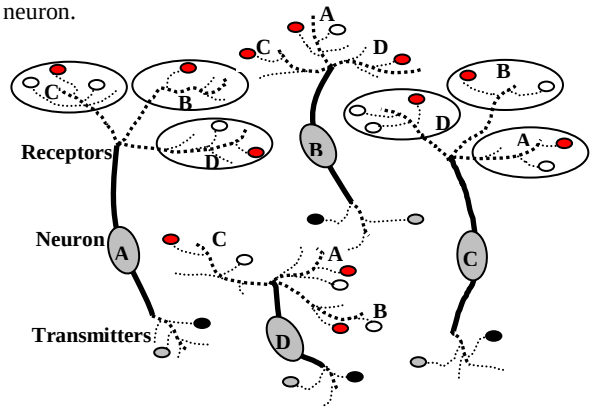

Fig 1: Architecture of the network and neurons. The number of receptor groups in a neuron was equal to the number of presynaptic neurons the neuron communicated with. For example, if a transmitter in neuron C wants to contact neuron A, the transmitter should contact a receptor in the receptor-group C of neuron A.

2.1 Signal Transmission in Computational Stochastic Units.

The dynamic behavior of these artificial units was modeled as a two state (i.e., active and inactive) stochastic process and according to a predefined behavioral rule, **Rule1**: When a receptor receives a signal from the corresponding presynaptic neuron at time step *t*, the signal is propagated within the network according to following two conditions. **Cond.1:** Once the received signal is applied to the receptor, if the receptor updated itself to an inactive state then the received signal is not propagated any further. Otherwise the signal is propagated to a randomly selected transmitter of the same neuron. **Cond.2:** Once a transmitter of a particular neuron receives a signal at time step *t*, if the transmitter updated itself to an active state after receiving the signal, the signal is transmitted to a randomly selected receptor of the relevant receptor group of a randomly

selected postsynaptic neuron. Otherwise the received signal is dropped. This behavioral rule defined the underlying mechanism of signal transmission between a presynaptic neuron and a postsynaptic neuron; i.e., the signal was successfully transmitted, only when the connection between the transmitter of the presynaptic neuron and the receptor of the postsynaptic neuron were in active state. The total amount of input a particular neuron received at a given time step *t* was determined by the total number of active receptors in that neuron at the given time step *t.* Similarly, the total amount of output a neuron produced at a given time step *t* was determined by the number of active transmitters in that neuron at the given time step *t.* Moreover active and inactive states of these stochastic units were determined by a combined process of signal transmission and the newly introduced threshold updating mechanism.

Dynamic stochastic states of these units were determined by adapting to the theory proposed by Maass and Zador [21] for a single synapse. Here we have mapped the process of a single synapse to a single stochastic unit (which allowed multiple and dynamic synaptic connectivity between neurons). Maass and Zador defined the single synapse response to its signal arrival times by a two state stochastic process. If $Ps(t_i)$ is the probability that signal is released by the synapse S at time t_i and train $\underline{t} = \{t_1, t_2, ..., t_n, ..\}$ consists of exact signal releasing times of the synapse S, $S(t)$ consists of the sequences of times where the synapse S has successfully released the signals. The map $\underline{t} \rightarrow S(t)$ at the synapse S forms a stochastic process with two states; i.e., Release (R) for $t_i \in S(t)$ and Failure of Release (F) for $t_i \notin S(t)$. The probability $Ps(t_i)$ in Eq.(1) describes a signal release probability at time t_i by the synapse S as a function of facilitation $C(t) \geq 0$ in Eq.(2) and a depletion $V(t) > 0$ in Eq.(4) at time *t.* C_0 and V_0 are the facilitation and depression constants respectively. Function $C'(s)$ given in Eq.(3) defines the response of $C(t)$ to presynaptic signal that had reached to the synapse S at time $t-s$; α is the magnitude of the response. Similarly $V'(s)$ given in Eq.(5) models the response of $V(t)$ to the preceding releases of the synapse S at time $t-s \leq t$ and, τ_c and τ_v are time decay constants of facilitation and depression. Maass and Zador allowed the synapse S to release the signal at time *t,* if $Ps(t_i) > 0$. We updated this rule by introducing a new θ threshold value. So that if $Ps(t_i) > \theta$, an artificial stochastic unit S is allowed to release the received signal. And we called it is in *active state*.

$$Ps(t_i) = 1 - \exp(-C(t_i) \times V(t_i)) \quad (1)$$

$$C(t) = C_0 + \sum_{t_i < t} C'(t - t_i) \quad (2)$$

$$C'(s) = \alpha . \exp(-s/\tau_c) \quad (3)$$

$$V(t) = \max(0, V_0 - \sum_{t_i < t \text{ and } t_i \in S(t)} V'(t - t_i)) \quad (4)$$

$$V'(s) = \exp(-s/\tau_v) \quad (5)$$

2.2 Stabilizing Neuronal Activity

A new threshold updating mechanism was introduced as an anti-Hebbian mechanism to the network which increased the threshold values of responsible groups of a neuron when the neuron was excited (or decreased when the neuron was depressed). A neuron had four threshold values; one for each receptor-group (a modeled neuron had 3 receptor groups) and one for the transmitters. Let R_{JI} denotes the receptor group in neuron J that communicates with transmitters of the presynaptic neuron I, and let $x_{JI}(t)$ be the output and $\theta_{JI}(t)$ be the threshold value of R_{JI} at time step *t*. Similarly let T_I denotes the transmitters in neuron I and let $O_I(t)$ be the output and $\theta_I(t)$ be the threshold value of T_I at time step *t*. The threshold value of the receptor-group R_{JI} was defined as in Eq.(6) and it was exponentially increased as the activity of R_{JI} to T_I is increasing (or decreased when the activity of R_{JI} to T_I is decreasing). Threshold value for transmitters in neuron I, i.e. T_I was defined as a function of total synaptic inputs from all its presynaptic neurons into the total output of the neuron I as in Eq. (7). Figure 2 shows these terms in abstract form.

$$\theta_{JI}(t) = f\left(X_{JI}(t)/O_I(t)\right) \quad (6)$$

$$\theta_I(t) = f\left(O_I(t).(X_{IJ_1}(t) + X_{IJ_2}(t) + X_{IJ_3}(t))\right) \quad (7)$$

$f(x) = 1/\left(1 - e^{-x}\right)$ $X_{JI}(t) = \left|R_{JI}^{Act}(t)\right| / \left|R_{JI}\right|$, $O_I(t) = \left|T_I^{Act}(t)\right| / \left|T_I\right|$, and $\left|G\right|$ is the number of components in G and $\left|G^{Act}(t)\right|$ is the number of active components in G at time *t.*

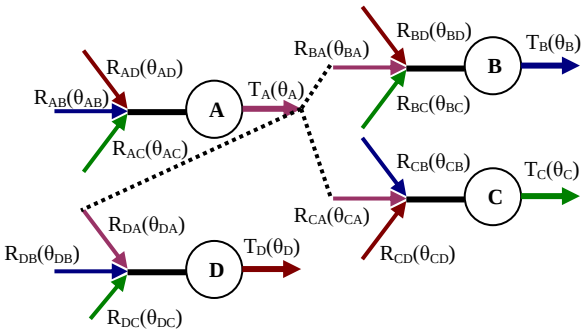

Fig 2: Signal Transmission between neurons. The developed network had four neurons, each neuron had three 'receptor-groups', and set of 'transmitters'. $T_I(\theta_I)$ denotes the 'transmitters' in neuron I and its threshold value. Similarly, $R_{JI}(\theta_{JI})$ denotes the 'receptor-group' in neuron J that is contacted by the 'transmitters' in neuron I. For simplicity the figure illustrates the communication between the 'transmitters' in neuron A with relevant the 'receptor-groups' of postsynaptic neurons only. The dotted connected lines in the network indicate the dynamicity of the synaptic connection between neurons since it depends on the number of 'active-transmitters' in neuron A, and the number of 'active-receptors' in the relevant receptor-group of the corresponding postsynaptic neurons.

3. EXPERIMENT

Sine waves with variable frequency *f*, amplitude 0.2, $\pi/4$ offset and $[0,\pi]$ length of time were applied to the input layer neurons externally. The frequency of the sine waves that was applied to neuron *A*, denoted by f'_A,was set to 1Hz while the frequency of the sine waves that was applied to neuron *B*, denoted by f'_B, was randomly selected from the ranges ((0.95,1.05), (0.5,0.8), (1.2, 1.5)) depending on the session neuron *B* was in (discuss later). Each generated sine wave was then digitized into square wave as a stream of one and zero. Each binary value in this stream represented a signal. Signals represented by binary one were applied to the network and signals represented by binary zero were neglected by the input layer neurons as defined in Rule 2. **Rule 2:** If an input layer neuron receives a signal which holds value zero at time step *t*, the signal is not fed to its receptor-groups at that time step *t*. If the received signal has value one and $r < p$, the signal is fed to each receptor-group of that input neuron. *r* is a random number in between 0 and 1 which is generated at each time step *t*. *p* is the proportion of the number of active receptors to the total number of receptors in the target receptor-group.

The parameter values of the stochastic computational units were set to model the effect of three different timescales on the developed neural network; slow, fast and medium. Generally a biological synapse exhibits mainly three different synaptic plasticities at a given time. A synapse is called a slow synapse if it exhibits long-term plasticity [14]. We can call a synapse is operating under a medium phase if it exhibits short-term plasticity [22], if a synapse operates in a fast time scale, it is called a fast synapse [23]. The stochastic computational units attached to neuron *A* and neuron *B* processed the signal similar to slow synapses while computational units in neuron *C* worked as fast synapses and computational units in neuron *D* processed the signals as medium phase synapses. This network arrangement was necessary to evaluate the Hebb's postulate because both presynaptic neuron and postsynaptic neuron in Hebb's postulate (in our case it is neurons *A* and *B*), require to have internal characteristics that support long-term plasticity (which is the main substrate for the formation of long-term memory). Therefore the values for the parameters τ_c and τ_v of theses computational units of the four neurons were taken from Maass and Zador [24] and are summarized in table 1. The values for the other parameters, i.e. C_0 , V_0 and α were determined according to [21] which grants a higher probability to both neuron *A* and neuron *B* to remain in an active state while neuron *C* and neuron *D* demonstrate counter and neutralize behavior to these two input layer neurons respectively.

Table 1. Neurons' parameter values

Neurons	τ_c and τ_v	C_0	V_0	α
A and *B*	30 min	1.5	0.5	0.7
C	100 ms	0.5	1.5	0.7
D	15 min	1.0	1.0	0.7

Each experiment consisted of three uniform stages and each stage had two phases; namely correlated phase and uncorrelated phase. Further, each phase consisted of two sessions called training session and testing session. In between stages, in between phases and in between sessions, a random delay was introduced. The structure of a stage in an experiment is given in table 2. In correlated training sessions, f'_B was randomly selected from the interval (0.95, 1.05) Hz while at uncorrelated training sessions f_B was randomly selected either from interval (0.5, 0.8) Hz or from interval (1.2, 1.5) Hz. At all the testing sessions no signals were externally applied to input layer neuron *B* and threshold updating process was also not executed on the network. At the end of each training session, the threshold values of the receptors and the transmitters of all the four neurons were fixed to their final threshold values and these values were taken as constants throughout the corresponding testing session.

Table 2. Structure of a stage

Phase	Correlated			*RD*	Uncorrelated		
Session	*Tr.*	*RD*	*Tst.*	*RD*	*Tr.*	*RD*	*Tst.*

RD stands for random delay, *Tr.* stands for Training and *Tst.* stands for Testing.

Two experiments were conducted by switching the order of the stages to observe the effect of one stage on another and the consistency of the behaviors generated. Stages were named according to the signal processing time step value of the neuron *A* as given in table 3-4 (Each neuron was modeled using a Java thread by setting the neuron's signal processing time step value as the thread activation time. For example, when neuron *A*'s signal processing time step was 1000 ms, *A*'s thread was activated at every 1000 ms. When neuron *A*'s thread got activated, it either received a signal to its receptor or its transmitter transmitted a signal to a postsynaptic neuron.). As given in table 3, at stage α each neuron had unique firing rate 1Hz, $f = 1/t$; *t* is the time step. At stages β and γ signal processing time steps of neuron *A* were 2000 ms (0.5 Hz) and 500 ms (2 Hz) respectively. Therefore, at stage β , neuron *A* was slower than the rest of the other neurons in the network. Moreover, each neuron had 60,000 artificial stochastic units which were equally distributed between transmitters and receptors of that neuron. And receptors in a neuron were uniformly distributed between receptor groups. Initially one percent of the number of receptors in each receptor-group was set to active state. Similarly, one percent of the transmitters in a neuron were initially set to active state to study the neuronal growth and its stabilization. Moreover, within each session 20 sine waves were externally applied to both input layer neurons, only if it was permitted by the relevant session. And at the end of each sine wave, θ values of the four neurons were updated if it was allowed by the relevant session. Finally the number of active receptors and the number of active transmitters in neuron *A* at every 1000 ms were recorded to generate the outputs.

Table 3. Signal processing time step value (*t*) of the neurons

	Name of the stage		
Neurons	α	β	γ
A	1000 ms	2000 ms	500 ms
B, C and *D*	1000 ms	1000 ms	1000 ms

Table 4. Order of the stages in each experiment

	Order of the stages		
Experiment number	**1st**	**2nd**	**3rd**
1	α	β	γ
2	α	γ	β

4. RESULTS

The distributions of the number of active receptors and the number of active transmitters of neuron *A* at each stage of the two experiments are shown in figure 3-8. Each of these figures consists of two subfigures, (*a*) and (*b*). Sub figure (*a*) shows the distributions of the number of active receptors in neuron *A* while sub figure (*b*) illustrates the distributions of the number of active transmitters in neuron *A*. The boundary lines in these subfigures, i.e. *CTr*, *CTs*, *UTr* and *UTs*, specify the end of "correlated training", "correlated testing", "uncorrelated training" and "uncorrelated testing" sessions respectively. When discussing the behavior of neuron *A* at a given stage, the mean number of active transmitters in the neuron *A* at that stage was taken as the measurement because it denotes the average output produced by neuron *A* at the stage. Similarly the average input that neuron *A* received at a stage is discussed in terms of the mean number of active receptors the neuron *A* had at that stage.

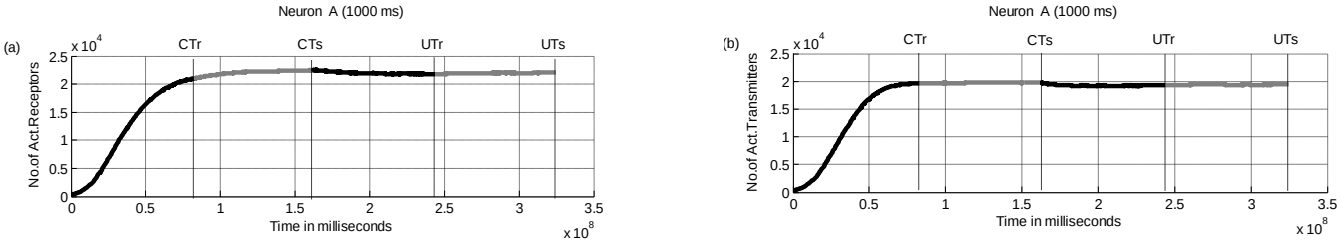

Fig 3: Distributions of (a) the number of active receptors (b) the number of active transmitters, of neuron *A* at stage 1 of exp.1.

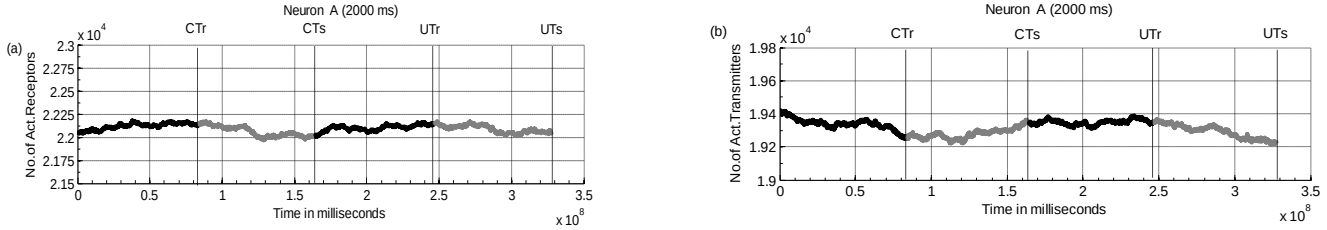

Fig 4: Distributions of (a) the number of active receptors (b) the number of active transmitters, of neuron *A* at stage 2 of exp.1.

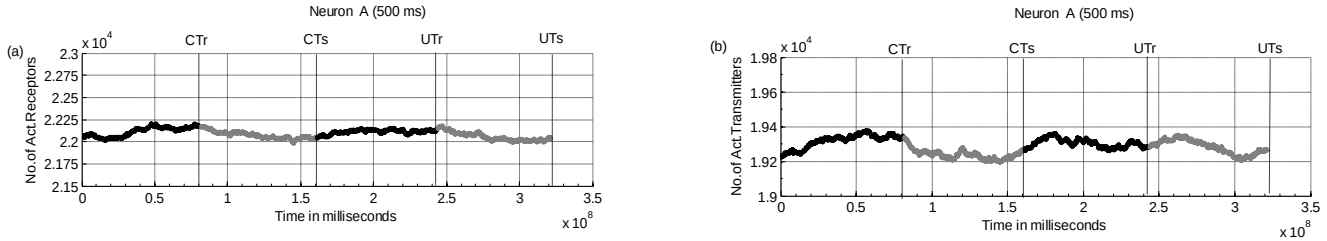

Fig 5: Distributions of (a) the number of active receptors (b) the number of active transmitters, of neurons *A* at stage 3 of exp. 1.

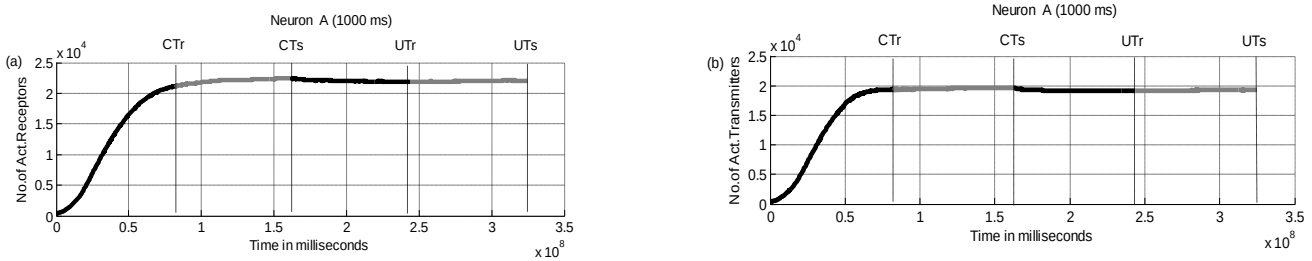

Fig 6: Distributions of (a) the number of active receptors (b) the number of active transmitters, of neuron *A* at stage 1 of exp.2.

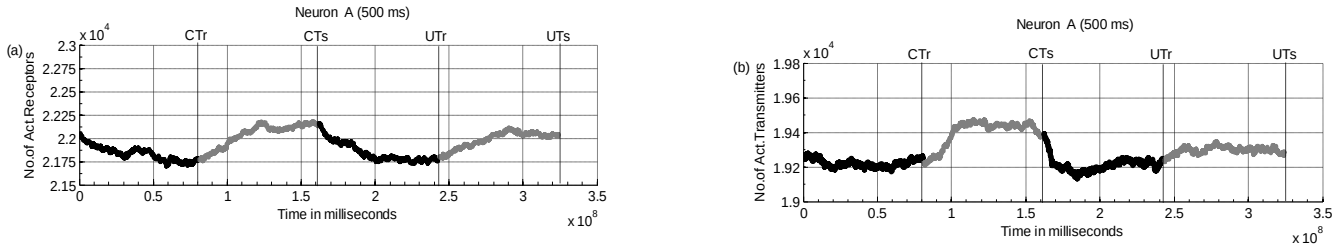

Fig 7: Distributions of (a) the number of active receptors (b) the number of active transmitters, of neuron *A* at stage 2 of exp. 2.

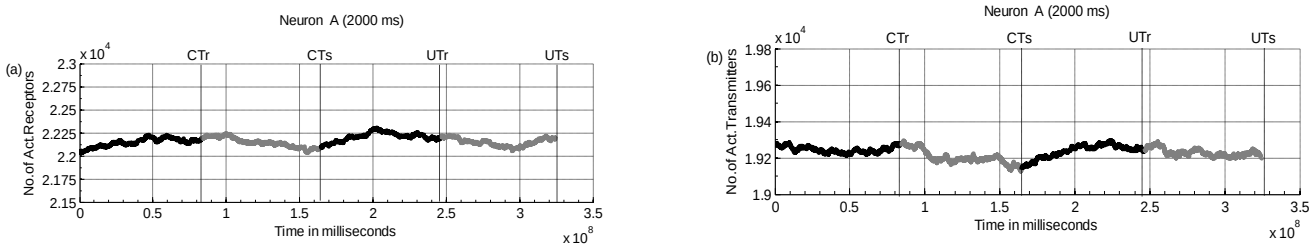

Fig 8: Distributions of (a) the number of active receptors (b) the number of active transmitters, of neuron *A* at stage 3 of exp.2.

5. DISCUSSION

5.1 Behavior of neuron *A* at Correlated Phases

Findings of the two experiments support fact that at all the correlated training sessions in spite of the signal processing time step of neuron *A*; i.e. t_A , neuron *A* has increased its average output (in terms of the mean number of active transmitters) when we moved from the 2nd stage to the 3rd stage in both experiments as summarized in table 5 (we could not evaluate the behavior of neuron *A* at the correlated training session of the 1st stage because throughout the session neuron *A* was in a growth state). Conversely, at correlated testing sessions, we could see a continuous decrease in average output that was produced by neuron *A* in terms of the mean number of active transmitters when we moved from 2nd stage to 3rd stage of the both experiments, see table 5. Although neuron *A* has stabilized its activity in terms of the output that it produced in both correlated training and correlated testing sessions, see figure 3-8, we could not see the properties of Hebb's postulate or its anti-Hebbian postulates at any correlated testing sessions as expected. This could be because of the strong binding of the activity between neuron *A* and neuron *B* due to the correlation of the sine waves applied to both neurons at the correlated training sessions of the two experiments.

Table 5. Behavior of neuron *A* at Correlated Phase

Experiment Number		Stages		
		1st stage	2nd stage	3rd stage
1	Name	$\alpha(f_A = 1Hz)$	$\beta(f_A = 0.5Hz)$	$\gamma(f_A = 2Hz)$
	Session	Correlated Training		
	μ_T	N/A	19324.3	19341.4
	Session	Correlated Testing		
	μ_T	19774.3	19286.2	19227.4
2	Name	$\alpha(f_A = 1Hz)$	$\gamma(f_A = 2Hz)$	$\beta(f_A = 0.5Hz)$
	Session	Correlated Training		
	μ_T	N/A	19216.7	19238.5
	Session	Correlated Testing		
	μ_T	19601.1	19437.0	19184.4

The notation μ_T denotes the mean number of active transmitters. *N/A* is used when neuron *A* showed a growth continuously throughout the stage.

5.2 Behavior of neuron *A* at Uncorrelated Training Sessions

In contrast to the correlated phase, behavior of neuron *A* at uncorrelated phase was different. The analysis of neuron *A*'s behavior at the uncorrelated training session in experiment 1 (summarized in table 6) shows that when we externally decreased the firing rate of neuron *A* when moving from stage α to stage β , neuron *A* has increased its average output in terms of the mean number of active transmitters to stabilize its behavior. Moreover, when we externally increased the neuron *A*'s firing rate from 0.5 Hz to 2 Hz at the uncorrelated training session of the experiment 1 when moving from stage β to stage γ , neuron *A* has decreased its average output in terms of the mean number of active transmitters to stabilize its behavior. Furthermore when we analyze the behavior of neuron *A* in experiment 2, we could see that, when we moved from stage α to stage γ while externally increasing neuron *A*'s firing rate from 1 Hz ms to 2 Hz (see table 6) it has further increased its average output in terms of the mean number of active transmitters instead of decreasing it. This behavior of neuron *A* contradicts with the observations at the uncorrelated training sessions of the 1st experiments. Nevertheless when we externally decreased neuron *A*'s firing rate by moving further from stage γ to stage β , neuron *A* has increased its average output in terms of the mean number of active transmitters to stabilize its activity as expected. The behavior that was shown by neuron *A* when moving from stage α to stage γ in experiment 2 may be because neuron *A* could not significantly detect the change we made to its signal

processing time step value as we discuss in section 5.3. However in general at all the uncorrelated training sessions of both experiments, neuron *A* has shown a resistance to the external manipulations and has been able to stabilize its behavior within each session. This resilient behavior of neuron *A* to external manipulations has been governed by the newly introduced threshold updating mechanism as discussed in section 5.4.

5.3 Behavior of neuron *a* at Uncorrelated Testing Sessions

At testing sessions we did not apply the threshold updating process to any neuron; however neuron *A* has stabilized its activity in terms of the mean output it produced while being responsive to the external updates that we made to its firing rate, see figure 3-8. Especially at uncorrelated testing sessions, neuron *A* has not only stabilized its activity but also has been able to demonstrate the behavior that is explained in Hebbian postulate and its anti-Hebbian postulates. In experiment 1, when we decreased firing rate of neuron *A* from 1Hz to 0.5Hz when moving from stage α to stage β under the uncorrelated testing sessions, neuron *A* has decreased its average output in terms of the number of active transmitters, see table 6. Moreover when we increased the firing rate of neuron *A* from 0.5 Hz to 2 Hz when moving from stage β to stage γ , the average output of neuron *A* has further decreased. We postpone the interpretation of this behavior of neuron *A* for a later discussion and move to analyze the neuron *A*'s behavior in experiment 2. In experiment 2, when we increased the neuron *A*'s firing rate from 1 Hz to 2 Hz when moving from stage α to stage γ under the uncorrelated testing sessions, neuron *A* has increased its average output in terms of the mean number of active transmitters, see table 6. This observation and the decrease of the average output produced by neuron *A* at uncorrelated testing sessions of experiment 1 (when moving from stage α to stage β of experiment 1) confirm the Stent's anti-Hebbian postulate.

As per Stent neuron *A* which is the presynaptic neuron of the postsynaptic neuron *B*, decreases its activity (the mean output it produced) when its firing rate is asynchronous with the firing rate of other presynaptic neurons (*C* and *D*) of the postsynaptic neuron *B*. Stent has further described that this asynchrony is detected by the postsynaptic neuron *B*. Thus, according to Hebb's postulate and Stent's anti-Hebbian postulate, neuron *A* should always increase its output when we increase neuron *A*'s firing rate from 0.5 Hz to 1 Hz or from 0.5 Hz ms to 2 Hz. And neuron *A* should always decrease its output when we decrease its firing rate from 2 Hz to 0.5 Hz or from 1 Hz to 0.5 Hz (see table 6-7). When we increased the firing rate of neuron *A* from 1 Hz to 2Hz, neuron *A* continuously increased its average output in our experiment 2 because neuron *B* could not detect any asynchrony from this change.

Why did neuron *A* decreased its average output at uncorrelated testing sessions when moving from stage β to stage γ in experiment 1 (see table 6)? This behavior of neuron *A* may have supported the Lisman's postulate of synaptic depression. According to Lisman, neuron *A* should decrease its average output when neuron *A* is in active state and postsynaptic neuron *B* is in inactive state due to inadequate excitation or inhibition by other neurons. We can see that at uncorrelated testing session of experiment 1, when moving from stage α to stage β , neuron *A* has decreased its average output (see table 6). Although we did not update the neuron *B*'s signal processing time step value, this behavior of neuron *A* has influenced the neuron *B* to decrease its average output when moving from stage α to stage β in experiment 1 (see table 8). Here neuron *B* has decreased its average output by increasing the average input of the receptor-group R_{BA} that communicated with the transmitters of neuron *A* (see table 8). As discuss in the section 5.4, generally at uncorrelated testing sessions neuron *A* increased its average output by decreasing its average input in terms of the number of active receptors (refer table 6 under experiment 2). Therefore, we presume that the synaptic connections between neurons *A* and *B* was in a depression at stage β in experiment 1. This depression may have affected the neuron *A* to decrease its average output when moving from stage β to stage γ .

Table 6. Behavior of neuron *A* at Uncorrelated Phase

Experiment Number		Stages		
		1st stage	2nd stage	3rd stage
1	Name	$\alpha(f_A = 1Hz)$	$\beta(f_A = 0.5Hz)$	$\gamma(f_A = 2Hz)$
	Session	Uncorrelated Training		
	μ_T	19234.7	19347.7	19294.8
	μ_R	21873.5	22108.6	22121.3
	Session	Uncorrelated Testing		
	μ_T	19391.4	19269.1	19250.7*
	μ_R	21994.5	22060.9	22018.8
2	Name	$\alpha(f_A = 1Hz)$	$\gamma(f_A = 2Hz)$	$\beta(f_A = 0.5Hz)$
	Session	Uncorrelated Training		
	μ_T	19166.5	19213.4	19257.2
	μ_R	21941.3	21779.3	22228.5
	Session	Uncorrelated Testing		
	μ_T	19246.5	19300.0	19219.9
	μ_R	22050.4	22048.7	22129.3

The notations μ_R denotes the mean number of active receptors, *Because of Lisman's effect neuron *A* might be in depression.

Table 7. Asynchrony detection at postsynaptic neuron B

t (ms)	Neuron *A*'s firing rate			Neurons C and D's firing rate
	2 Hz (1/500 ms)	1 Hz (1/1000 ms)	0.5 Hz (1/2000 ms)	1 Hz (1/1000 ms)
t + 500	√			
t + 1000	√	√		√
t +1500	√			
t +2000	√	√	√	√

Let *t* is an arbitrary time. Neuron *B* receives signals from neurons *A*, *C* and *D* concurrently at all the times only when *A*'s firing rate is 2 Hz or 1 Hz

Table 8. Neuron *B* at Uncorrelated Testing Session

Experiment Number		Stages		
		1st stage	2nd stage	3rd stage
1	Name	$\alpha(f_A = 1Hz)$	$\beta(f_A = 0.5Hz)$	$\gamma(f_A = 2Hz)$

	μ_T	19454.0	19358.3	19383.8
	μ_R	22299.5	22279.5	22226.8
	$\mu_{R_{BA}}$	7423.5	7456.9	7417.7
	$\mu_{R_{BC}}$	7365.7	7348.6	7356.5
	$\mu_{R_{BD}}$	7510.3	7473.9	7452.6

5.4 Properties of the New Threshold Updating Mechanism

The introduced threshold updating mechanism helped the neuron *A* to stabilize its activity throughout both experiments. It has demonstrated the behavior similar to homeostatic synaptic plasticity process [17, 18] identified in biological synapses only at the uncorrelated training sessions. During uncorrelated training sessions, the threshold updating mechanism helped neuron *A* to increase its average output when we decreased neuron *A*'s firing rate from 1 Hz to 0.5 Hz and from 2 Hz ms to 0.5 Hz (see table 6). Also when we increased neuron *A*'s firing rate from 0.5 Hz to 2 Hz, the threshold updating mechanism helped neuron *A* to decrease its average output (see table 6). Although we did not execute the threshold updating process on neurons at uncorrelated testing sessions, neuron *A* demonstrated the behavior characterize in Hebb's postulate and its anti-Hebbian postulates accordingly. Neuron *A* has shown these behaviors by appropriately updating its average input in terms of the mean number of active receptors. As shown in table 6 under uncorrelated testing sessions, when we externally decreased the firing rate of neuron *A* while moving from stage α to stage β in experiment 1 and while moving from stage γ to β in experiment 2, neuron *A* has decreased its average output by increasing its average input. Conversely, when we externally increased the firing rate of neuron *A* while moving from stage α to stage γ in experiment 2, neuron *A* has increased its average output by decreasing the average input. This internal metabolic changes of neuron *A* in terms of the number of active receptors and the number of active transmitters to generate appropriate output according to the external fluctuations under Hebb's postulate and its anti-Hebb's postulate has been defined in the Levy and Desmond excitatory synaptic rules [25] and further biologically observed under the process of Synaptogenesis [17].

6. CONCLUSION

Neuron *A* which is the Hebb's presynaptic neuron of the postsynaptic neuron *B* stabilized its activity without any predefined constraints. Meanwhile it demonstrated the behavior characterized in Hebb's postulate and Stent's anti-Hebbian postulates during the uncorrelated phase with the support of newly introduced threshold updating mechanism which demonstrated the behavior similar to homeostatic synaptic plasticity mechanism. Therefore, we presume that the distinguished factors incorporated to our network, i.e. (1) neurons with different signal processing time scales, (2) the role of dynamic stochastic computational units which played the role of a single synapse allowing dynamic and multiple connectivity between neurons, and (3) the introduced threshold updating mechanism, have significantly contributed to regulate the Hebb's presynaptic neuron's excitation as defined in the basic learning postulates.

7. REFERENCES

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