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Synaptic redistribution and variability of signal release probability at hebbian neurons in a dynamic stochastic neural network

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Abstract: This paper presents the finding of the research we conducted to evaluate the variability of signal release probability at Hebb's presynaptic neuron under different firing frequencies in a dynamic stochastic neural network. According to our results, synaptic redistribution has improved the signal transmission for the first few signals in the signal train by continuously increasing and decreasing the number of postsynaptic 'active receptors' and presynaptic 'active-transmitters' within a short time period. In long-run at low-firing frequency it has increased the steady state efficacy of the synaptic connection between Hebbian presynaptic and postsynaptic neuron in terms of the signal release probability of 'active-transmitters' in the presynaptic neuron. However, this 'low-firing' frequency of the presynaptic neuron has been identified by the network when compared it to the ongoing frequency oscillation of the network.

Keywords: Dynamic stochastic network, Hebbian neurons, Synaptic redistribution, Release probability

1 INTRODUCTION

As per recent biological findings a synaptic connection between two neurons is strengthened by increasing the number of postsynaptic receptor channels or by increasing the probability of neurotransmitter release at presynaptic neuron. This functional behavior at synapses is varied if long-term plasticity interacts with the short-term depression, Abbott and Nelson [1]. Short-term depression is an activity dependent reduction of neurotransmitters from the readily releasable pool at presynaptic neurons, Zucker [2]. When long-term plasticity interacts with the short-term depression, the effect is called synaptic redistribution. This synaptic redistribution increases the probability of neurotransmitter release at presynaptically; and subsequently increases the efficiency of signal transmission between neurons and decreases presynaptic readily releasable pool size, Abbott and Nelson [1]. Therefore, the high-frequency dependent increase in synaptic response for the first few spikes in the spike-train is caused because of the redistribution of the available synaptic efficacy and not because of the increase of synaptic efficacy at the steady-state. However, at low-firing-frequencies of presynaptic neuron such as < 10 Hz, an increase of synaptic efficacy at steady state has been observed. This increase of synaptic efficacy depends on the short-term plasticity factors such as probability of neurotransmitter release and time constant of recovery, Markram and Tsodyks [3]. Conversely Okatan and Grossberg [4] suggest that pairing of Hebbian neuron to reach the steady state, might be frequency-dependent and as a result the time taken to reach to the steady-state at high-firing-frequency of presynaptic neuron may be longer than at lower-presynaptic-firing frequencies; therefore even there is steady-state increase of the synaptic efficacy at high-firing frequency of presynaptic neuron, it is not well observed due to the effect of short-term synaptic plasticity factors on the synaptic efficacy. Lisman and Spruston [5] have further added that this increase or decrease of synaptic efficacy at steady state is not merely based on spike arriving time to the synapses but also on the level of postsynaptic depolarization, rate of synaptic inputs and the phase of synaptic input relative to the ongoing frequency oscillations.

2 METHOD

Our modeled neuron had thousands of artificial units, named 'transmitters' and 'receptors'. The 'transmitters' in a presynaptic neuron contacted the corresponding 'receptors' in postsynaptic neuron dynamically according to the presynaptic activity by forming an artificial synaptic connection between the two neurons. Therefore, one can presumes that synaptic connection between the presynaptic neuron and postsynaptic neuron in our network as a dynamic connection which mediates the intercommunication between the presynaptic 'transmitters' and the corresponding postsynaptic 'receptors'. Furthermore these artificial units were two-state stochastic computational units which stochastically updated their signal release probability at time t, i.e. $P(t)$, according to the history of their activity by adapting to the mathematical model of Maass and Zador [6] which describes the signal

release probability as a function of spike arrival time and the effect of short-term plasticity. The active state of a given artificial unit at a specified time t was determined by the release probability at that time and a threshold value *theta* (θ). The threshold θ was updated according to the feedback of a stability promoting mechanism which sensed the neuronal local excitation and the corresponding postsynaptic excitation as defined in eq. (1) and eq. (2). Thus, an artificial unit was in *active state* at time t if $P(t) > \theta$ and was allowed to transmit/receive signals between neurons; otherwise it was in *inactive state*.

3 EXPERIMENT

An experiment was conducted on fully connected neural network which had four neurons namely *A*, *B*, *C* and *D*. The neuron *A* and neuron *B* were set to process similar to the long-term plasticity by setting time decay constants to 30 min while neuron *C* and *D* simulated the short-term effects on the network with time decay constants 100 sec and 15 min respectively. We have shown that with this setting of the network, the neurons *A* and *B* formed a Hebbian pairing and was capable of demonstrating the characteristics explained in fundamental learning theories; Hebb's postulate and Stent's anti-Hebbian postulate, see Fernando, Yamada and Marasinghe [7]. The experiment had three stages where each stage consisted of two phases; correlated and uncorrelated, and each phase consisted of two sessions, namely training and testing sessions. In between sessions, a random delay was introduced. Throughout the experiment, the firing frequency of neurons *B*, *C*, and *D* remained in a constant firing rate at 1 Hz while neuron *A* updated its firing frequency accordingly as it was moved from one stage to the other. At stage-1, the firing frequency of neuron *A* was at 1 Hz, at stage-2 it was 2 Hz and stage-3 it was 0.5 Hz. Therefore, comparatively neuron *A* was in high-firing frequency at stage-2 and was in low-firing frequency at stage-3. At stage-1 the network was allowed to stabilize its activity as a one unit because all the neurons were firing at 1 Hz. Signals in terms of sine waves were externally fed to the network through neuron *A* and neuron *B* based on the phase and the session they were in. The phases were named according to the frequency of the sine waves applied to neuron *B*. Throughout the experiment the frequency of the sine waves applied to neuron *A* was at $f'_A = 1$ Hz and the frequency of the sine waves applied to neuron B (f'_B) was randomly selected from interval (0.95, 1.05) Hz at the correlated phase. At the uncorrelated phase f'_B was randomly selected either from interval (0.5, 0.8) Hz or from interval (1.2, 1.5) Hz. At the training sessions, the external signals were fed to the both neurons but at the testing sessions signals were not externally fed to the neuron *B*. At testing sessions, threshold values of all the four neurons were not updated; the threshold values trained at the training sessions were taken as constants at the corresponding testing sessions. Each neuron had 60,000 artificial units which were uniformly distributed between 'receptors' and 'transmitters'; and subsequently 'receptors' were distributed uniformly between 'receptor-groups'. Initially 1% of the receptors in each receptor group and 1% of the 'transmitters' in each neuron were set to an active state. Fig.1 shows the architecture of the network in abstract.

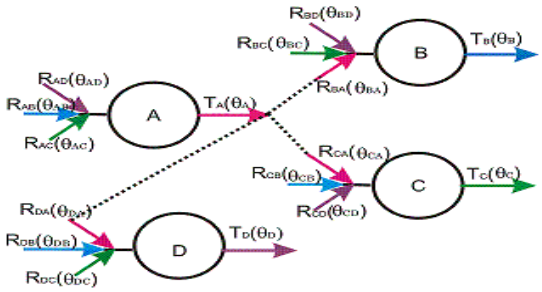

Fig.1. Architecture of the network with dynamic synaptic connections. The developed network had four neurons, each neurons had three 'receptor-groups', and set of 'transmitters'. $T_I(\theta_I)$ denotes the 'transmitters' in neuron *I* and its threshold value. Similarly, $R_{JI}(\theta_{JI})$ denotes 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 'receptor-groups' of the postsynaptic neurons only. The dotted connecting 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 their signal release probability, and the number of 'active-receptors' in the relevant receptor-group of the corresponding postsynaptic neuron.

$$\theta_{JI}(t) = f\left(X_{JI}(t)/O_I(t)\right) \quad (1)$$

$$\theta_I(t) = f\left(O_I(t).(X_{IJ_1}(t) + X_{IJ_2}(t) + X_{IJ_3}(t))\right) \quad (2)$$

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

4 RESULTS

The distribution of the number of 'active-transmitters' in neuron *A* and their probability of signal release at each stage, and the distribution of the number of 'active-receptors' in the receptor group R_{BA} that is contacted by the

'transmitters' in neuron *A* through the artificial synaptic connection between neuron *A* and neuron *B* are shown from fig.2 to fig.5.

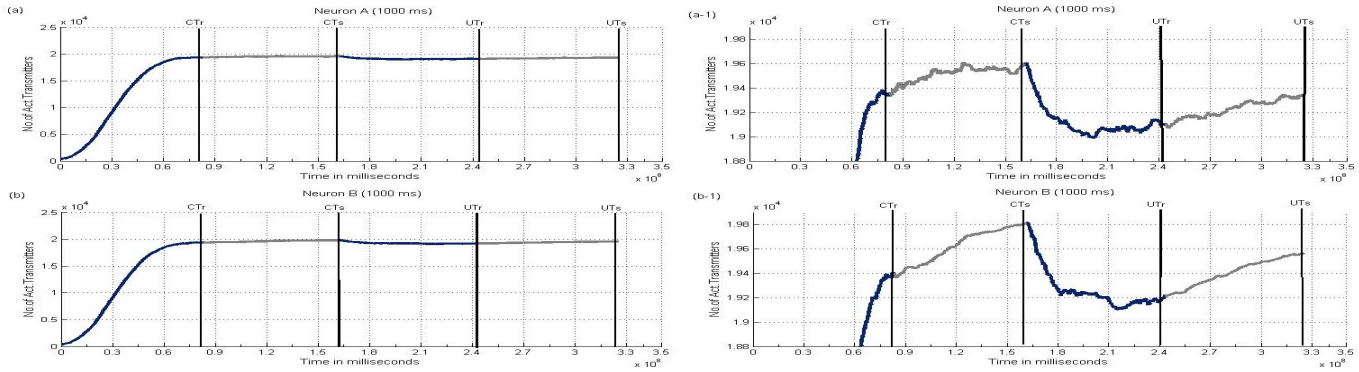

Fig.2. Distributions of 'active-transmitters' in neurons *A* and *B* at stage-1 over the time (a): 'active-transmitters' in neuron *A*, **(b):** 'active-transmitters' in neuron *B*, **(a-1):** Enlarged version of subfigure (a), and **(b-1):** Enlarged version of subfigure (b).

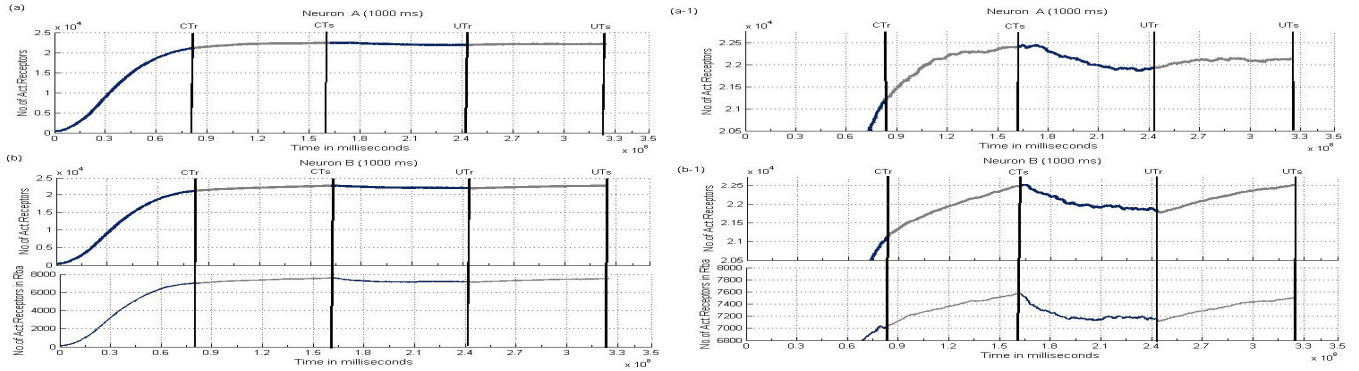

Fig.3. Distributions of 'active-receptors' in neurons *A* and *B* at stage-1 over the time (a): 'active-receptors' in neuron *A*, **(b):** 'active-receptors' in neuron *B* and in R_{BA} respectively, **(a-1):** Enlarged version of subfigure (a), and **(b-1):** Enlarged version of subfigure (b)

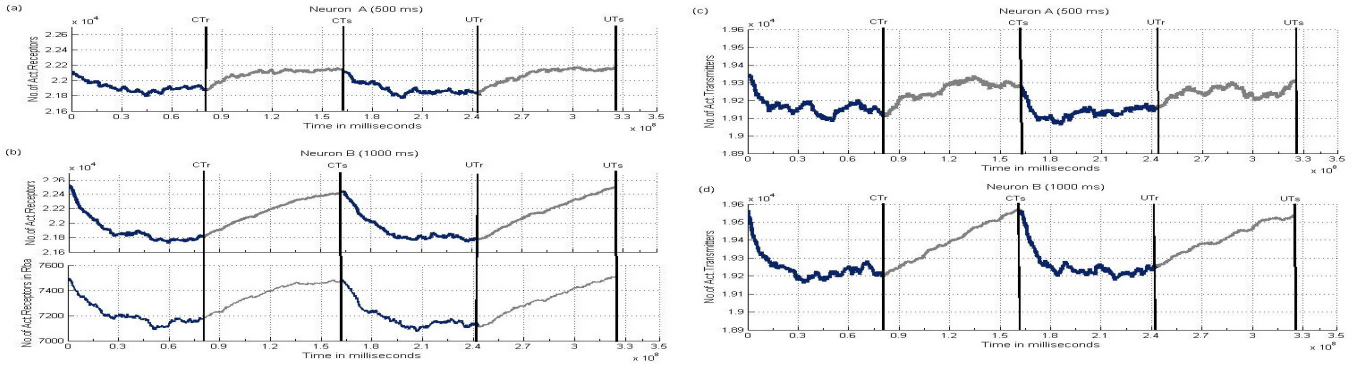

Fig.4. Distributions of active components in neurons *A* and *B* at stage-2 over the time (a): 'active-receptors' in neuron *A*, **(b):** 'active-receptors' in neuron *B* and in R_{BA} respectively, **(c):** 'active-transmitters in neuron *A*, **(d):** 'active-transmitters' in neuron *B*

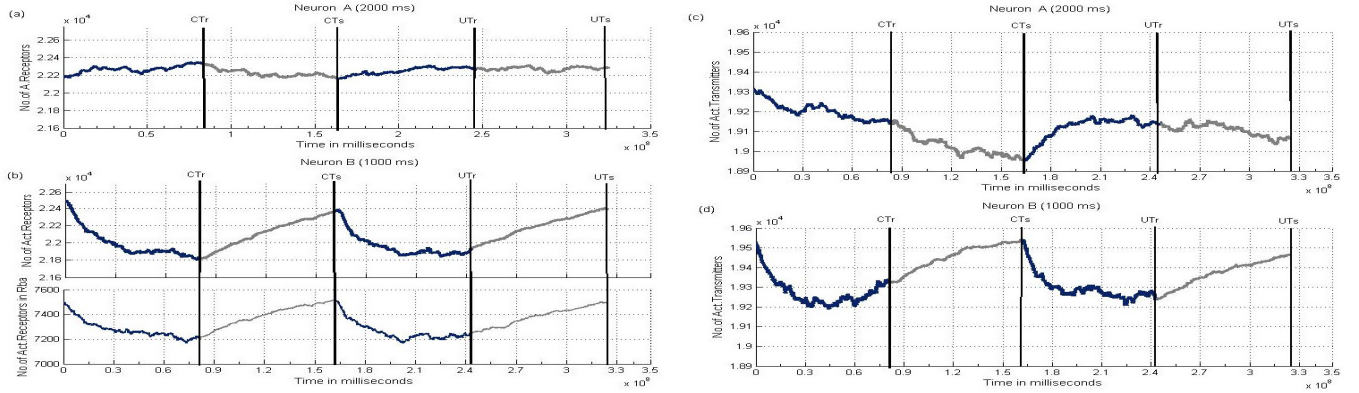

Fig.5. Distributions of active components in neurons *A* and *B* at stage-3 over the time (a): 'active-receptors' in neuron *A*, **(b):** 'active-receptors' in neuron *B* and in R_{BA} respectively, **(c):** 'active-transmitters in neuron *A*, **(d):** 'active-transmitters' in neuron *B*

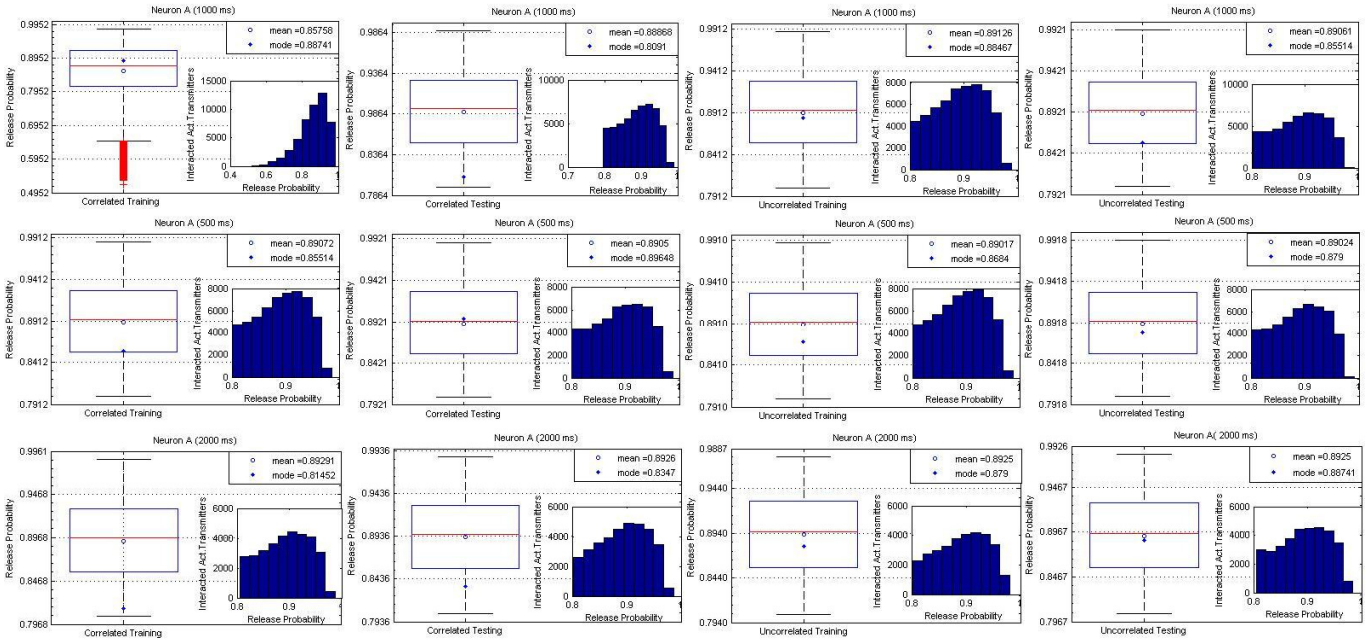

Fig.6. Box plot in each subfigure illustrates the variation of the release probability of the interacted 'active-transmitters' of neuron *A* (i.e., 'active-transmitters' that communicated with 'receptors' in R_{BA} receptor-group during the session). The inner histograms depict the variation of the number of interacted 'active-transmitters' in neuron *A* over the time against the signal release probability at the corresponding session of the stage.

5 DISCUSSION

According to the results of, from fig.2 to fig.5, the increase or decrease of the response of neuron *A* is a continuous insertion and deletion of the presynaptic 'active transmitters; and the redistribution of the number of 'active receptors' of the receptor group R_{BA} in the postsynaptic neuron *B*. Further this dynamic update to the number of presynaptic 'active transmitters' and the number of postsynaptic 'active receptors' in R_{BA} was remained in very short time period, and subsequently presynaptic neuron *A* attained to the steady state. However in long-run we could see that although neuron *A* at high-firing frequency (i.e. at 2Hz in stage-2) increased the number of 'active transmitters', it has not increased the average (mean) signal release probability at the steady state of the synaptic connection compared to when presynaptic neuron *A* fired at low-firing frequency(i.e. 0.5Hz in stage-3, see fig.6). In contrast, at the low-firing frequency of neuron *A,* even though the number of 'active-transmitters' in neuron *A* has decreased (compared to when *A* was at high-firing frequency), the average signal release probability of 'active transmitters' at the steady state has increased, see fig.6. Therefore, synaptic redistribution has improved the transmission for the first few signals in the signal train by dramatically increasing or decreasing the number of postsynaptic 'active receptors'. In long-term at low-firing frequency synaptic redistribution has increased the steady state synaptic efficacy of the synaptic connection (i.e. increase of the signal release probability of 'active-transmitters' in neuron *A*). Further, in our experiment the firing frequency of neuron *A* was always < 10 Hz. Therefore, according to Markram and Tsodyks [3] we were supposed to observe an increase in the efficacy at the synaptic connection between *A* and *B* at all the stages. But, we could observe an increase of the synaptic efficacy only at stage-3, i.e. when neuron *A* was in 0.5 Hz. Therefore the increase or decrease of synaptic efficacy at a steady state may not only base on rate of synaptic inputs but also on the phase of the synaptic input relative to the ongoing frequency oscillations of the network.

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