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Agent based approach for homeostatic plasticity in neuronal activities

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Abstract: **The capability of re-establishing to its normal rhythm over an excitation while adapting to external or internal stimuli is a process of complexity. We propose an agent based framework to model the homeostatic plasticity in neuronal activity incorporating the concept of self-organization. Our model provides the ability for neuro-agents to adapt themselves as a series of activities over the excitements of synaptic inputs as similar to the nervous systems, hence allowing the creation of diversification and competitive environment.**

Keywords: Agent, Homeostasis, Self-organization

I. INTRODUCTION

The capability of re-establishing to its normal rhythm over an excitation while adapting to external or internal stimuli is a process of complexity which is characterized by self-organization, non-linearity, chaos dynamics and emergent properties, Kirshbaum [1]. According to the researches, if the system is complex enough it will trigger changes automatically and naturally within the system in order to improve the system efficacy. These changes are mainly accomplished by its elements as a responds to the external and internal inputs. Especially these elements reorganize and resettle them-selves, to accomplish the overall system goal. This process is known as self-organization of a complex system.

As similar to other complex systems like social system, echo system, politic systems etc, nervous systems also adapt to the changes in the environment over a time period. Depending on the time scale of this adaption, different terms, such as plasticity, potentiation, augmentation, etc have been used. Apart from the short-term adaption, long-term adaptions are also possible in the nervous system which can be varied from couple of seconds to couple of days, Burrone and Murthy [2]. However, all these adaption or self-organization is required when the nervous system is being pushed away from its operational range, to bring back to its normal rhythm. So we can interpret, this behavior as a behavioral rule of the nervous system, refer rule 1,

Rule 1: neurons reorganize and resettle them-selves, to accomplish the overall system goal.

According to the empirical researches, this is accomplished through process of homeostatic plasticity, where neurons monitor their own activity levels and transduce the information to regulate changes in excitability. This adaption should not prevent the neuronal excitements or changes because it is identified that neurons are learning through these excitability, Davis and Bezprozvanny [3]. Therefore, nervous systems are subject two opposite requirements, adaption to the change and maintain the stability. Researches into biology have identified that; these changes are accomplished in the form of activity dependent alteration, by modifying,

Rule 2: The number of synaptic connections, and

Rule 3: The strength of synaptic connections,

to regulate the synaptic firing rate, Turrigiano [4]. According to the researchers, Turrigiano [4] and Burrone and Murthy [2], Homeostatic plasticity allows,

Rule 4: continuous and steady modifications to neural network elements by making neurons to stabilize their excitability while keeping relative differences in individual strengths of synapses.

Rule 5: The overall activity of the network is controlled as a set of rules; these rules either can be local or global rules.

As such, neurons fluctuate constantly their electrical, morphological and synaptic properties, Turrigiano [4]. According to the biology, neurons electrical properties are determined by the ca^{+2} ionic concentrations. For example,

Rule 6: if the synaptic firing rate is very low then neurons close their ca^{+2} gates which in turn increase the

synaptic firing rates. On the other hand, as similar to the previous scenario,

Rule 7: neurons open their ca^{+2} ionic gates to bring down the synaptic firing rates when it is high,

Gazzaniga, Ivry and Mangun [5]. Apart from the regulations of ionic channels, neurons also regulate their synaptic strength in order to control their synaptic firing rates. According to the Turrigiano [4],

Rule 8: As the firing rates increase, associated activities of homeostatic plasticity put down synaptic strength by allowing pre and post synaptic neurons to grow and contact others to form trial synapses.

Rule 9: If the firing rate is very low, neuron strength and stabilize the appropriate connections by making inappropriate connections to be lost, and this will be achieved through by halting synaptic growth.

Information about neuronal activity (synaptic strength) and neuronal size, (number of synaptic connections) are known to be monitored through neurons monitors, which are seem to be located inside the neuronal cell.

Rule 10: These sensors, inside the neurons report the deviation of a particular neuron from its equilibrium point.

The deviation will be fed back to the neuron as an error signal to bring it back to the equilibrium point Davis and Bezprozvanny [3] and Davis [6]. Simply, homeostatic plasticity can be viewed as a feedback system, Turrigiano [1].

The rest of the paper organized as follows: section II discusses the applicability of homeostatic plasticity in neural networks while highlighting the issues in applications. Section III describes our approach of implementing homeostatic plasticity and section IV evaluates the first result of the approach and paper concludes with section V discussing the applications, limitations, advantageous and further work.

II. APPLICATIONS OF HOMEOSTATIC PLASTICITY

According the current review on Homeostatic plasticity, current researches are now attempting to apply this mechanism as a supportive plasticity mechanism for Hebbian synaptic plasticity, because Hebbian rules allows (a) unconstraint growth of synaptic weights in neural networks when correlation of input and output is positive and (b) when correlation is negative, it causes strength of synaptic to move towards the zero level. Finally this is ended up in losing the network sensitivity. The network to be sensitive to the input, the signals generated by that input should propagate through the network, Vogels, Rajan and Abbott [7]. Therefore researches are now attempting to implement the homeostatic plasticity as a feedback system, either (a) to modify excitability of total synaptic strength at a constant level or (b) to modify the synapse number to produce changes in synaptic strength. Especially these researches are into applications of Continuous Time Recurrent Neural Network (CTRNN), which are capable of approximate output of any dynamical system when appropriately parameterized, Funahashi and Nakamura [8].

In CTRNN each node is described in terms of differential equation as shown in (1), where u_i represents the internal state of the i^{th} neuron, τ_I – the time constant of the i^{th} neuron, I_i – external input (or threshold) of the i^{th} neuron, w_{ij} – connection weight from j^{th} neuron to the i^{th} neuron and σ -output function as shown in (2).

$$\frac{du_i(t)}{dt} = -\frac{1}{\tau_i} u_i(t) + \sum_j w_{ij}\sigma(u_j(t)) + I_i \quad (1)$$

$$\sigma(x) = \frac{1}{(1+\exp(-x))} \quad (2)$$

The shape of the sigmoid function causes the node firing rate to be saturated when the neuron potential is very high and low. When the firing rate is saturated there are no fluctuations in firing rate, therefore the sensitivity of the network is lost. To avoid this saturation, researches are now trying to apply homeostatic plasticity on CTRNN in terms of weight plasticity or as a process that affecting the excitability of individual neurons, Williams and Noble [9] and Williams [10]. Some of these researches are being concluded with even homeostatic plasticity improves the signal propagation; it acts as barrier for creating evolvers, Williams [10].

Our interpretation on these conclusions are, either (a) approach for modeling homeostatic plasticity might be wrong or (b) current view on CTRNN may not be supported to the homeostatic plasticity. With align to the part (b) some researchers are being carried out to solve the evolvability of CTRNN with fixed weight concepts by viewing the learning as interactions between the multiple timescale dynamics, Izquierdo-Torres and Harvey [11]. On their research they are being able to demonstrate, for the smallest CTRNN network, with the

concept of two-time scale, slow-time and fast-time scale, that it is possible to make the CTRNN to be evolved. However, still, they are not being able to exhibit it for large neural networks with proper time delays. Therefore, developing reliable and evolvable CTRNN networks are not being solved. With align to part (b) we understand the nervous system as a network of network, where each node on the network has self-organization ability. Our approach is Artificial Life approach which define the complexity as it could be rather than as we know it.

III. OUR APPROACH

We understand the nervous system, as a network of self-organizing network, in other terms, as a network of simple agents, called neuro-agents, where they organize and re-settle themselves to accomplish the overall system goals, refer to rule (1). As a process of organizing, it has the ability to control its own excitation by changing the size which in turn changes its strength, refer to rules (2) and (3). This is promoted through the structure of the neuro-agent, which consists of large number of receivers and transmitters which can be in either active or inactive status. At the same time, neurons are able to monitor their own deviations through the monitors inside the soma, refer rule (10). Based on the retrieved information, neuron can change the selected number of active transmitters and receivers, since information can be transferred from active transmitter to active receivers. So if neuron is over excited it changes the selected number of inactive receivers and inactive transmitters to be active to weak the strength as similar to the rule (8). And if the neurons is inhibited it make selected number of active transmitters and active receivers to be inactive as similar to the rule (9). As such, our model neuron can be viewed as in figure 1.

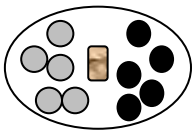

Fig. 1: The model of neuro-agent, light-color circles represent the receivers, dark-color circles represent the transmitters, and square represent the soma.

However, over all behavior of the system as a self-organizing network is controlled through the set of global rules imposed to the entire network, refer to rule (5). As a mechanism of signal propagation, all the neurons should always adhere to the following four rules, Fernando, Matsuzaki, Nakamura, et al [12].

Rule A: Only active transmitter can transmit the signal to the active receiver.

Rule B: Only active receiver can receive the signal.

Rule C: Once active transmitter transmit the signal it should become inactive (as similar to the refectory period) and inactive receiver in the same neuron become active (similar to Na^{+2} ionic concentration).

Rule D: Once active receiver receives the signal it should becomes inactive and inactive transmitter in the same neuron becomes active.

Therefore, the number of active transmitters within the neuron determines the volume of the signal that is transmitted by a particular neuro-agent. There are neither fixed connections between receivers and transmitters nor among the neurons. The active transmitter can transmit the signal to the active receiver in the same neuron or active receiver in another neuron. Therefore, our network somewhat looks like Recurrent neural network (RNN) as shown in figure 2.

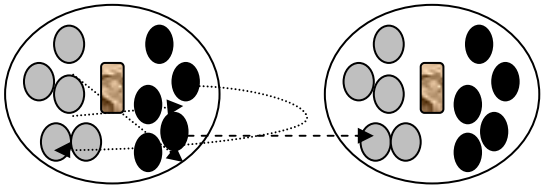

Fig. 2: The model of the proposed neural network, signal can be transmitted through active transmitters to active receivers on the same or different neuron.

As communication mechanisms, neurons are monitored through the concepts of central controller, which implements as a set of rules, in order to guarantee homeostatic plasticity is achieved. As a first step of the proposed model, we developed a network of network, simply having three model neurons, where each one having only receivers and transmitters. We evaluate the system, in terms of implementing above for rules as described in the next section.

IV. EVALUATION OF THE SYSTEM FROM FIRST RESULT

We model the system with three neurons (N1, N2 and N3) where each neuron consists of 10,000 receivers and 10,000 transmitters. At the onset only half of the transmitters and receivers are set to active. The number of active transmitters and receivers may be changed

accordingly as neuro-agents response to the external or internal stimuli. Active transmitters within the neuron emit signals in iterations of 100 steps. Experiment was divided into five stages; each stage consists of 5000 steps. Emission interval for N2 was set to 200 steps in stage 4, indicating that activity of N2 was decreased in that stage. Similarly, the interval for N3 was set to 50 steps in stage 3 and 10steps in stage 3 and 4 indicating that activity was increased.

When the neurons attained to its dynamic equilibrium, signaling patterns between the neurons was observed to fluctuate in different stages as shown figure 3, while individual neurons change their active transmitters and receivers in response to the manipulations.

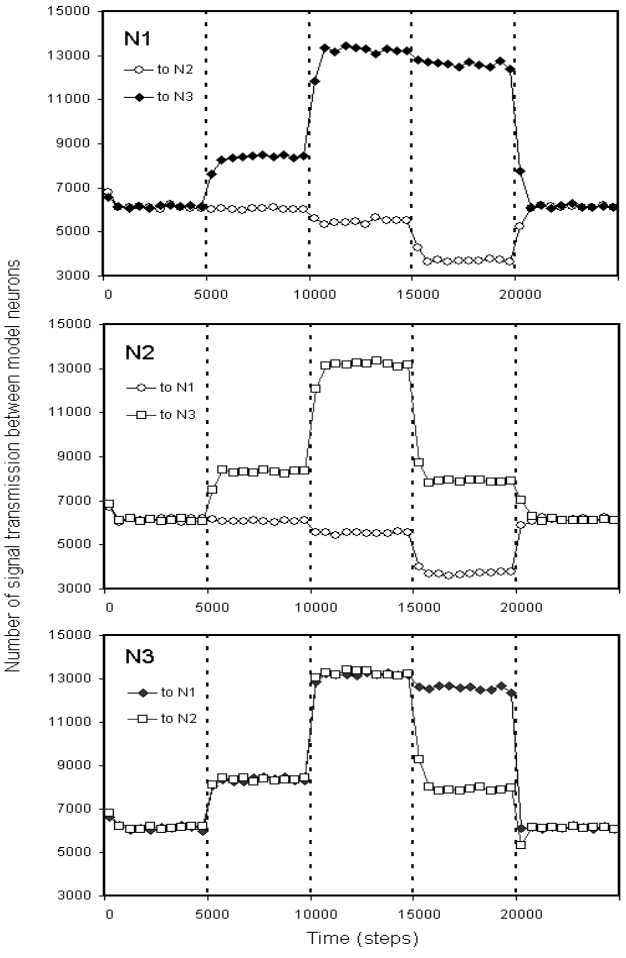

Figure 3: Signal propagation from one neuron to others.

IV. CONCLUSION

We demonstrated the preliminary version of homeostatic plasticity as a communication between self-organizing neuro-agents. The self-organization, in this case was merely achieved through very simple four rules, which set up as a global or environmental rules. The system has sown it is possible to achieve the homeostatic plasticity as a means of signal propagation without updating of internal parameters.

However, real implementation of homeostatic plasticity attach to the environmental input as a self-organizing agent networks still to be tested. If the propose model able to demonstrate the homeostatic plasticity as a smooth process then it will open up the new direction for the modeling nervous system. At the same time, it will open up new challenge for the research; demonstration of learning, evolving with the homeostatic plasticity.

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