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MODELING LEARNING ON DYNAMIC BEHAVIOUR OF SYNAPSES

Learning is a process involved in multiple timescales. As per biology, changes which last from milliseconds to seconds and hours to days are the main mediators for the formation of short-term and long-term memory. It is obvious that, memory formation is neither static nor it is restricted into a one phase of life. Every step we keep in our life, even it succeed or fail or no matter what happen, we learn from them and acquire invaluable knowledge on that, which makes us easy manipulation on similar events in future. Thus continuous learning in a dynamic environment is a necessary qualification for the researches which are interested in studying phenomena, such as addiction, stress, noise, etc on such a dynamic learning environments. This research proposes a new approach of modelling our nervous system with the intention of implementing learning on dynamic environment.

1. INTRODUCTION

Artificial neural network, the present day example of learning and memory formation has demonstrated its applicability in many fields including medicine, engineering, science, management, etc. However, it is still unable to demonstrate learning on dynamic environment which is rich with temporal information [1]. The static representation of learning effect has weakened neural network being evolved and worked in a dynamic environment. This has being a greater barrier for the researches which are interested in studying or simulating phenomenon on dynamic learning environment.

For example, medical phenomenon like addiction which is supposed to be aroused as a result of pleasure causing activities such as eating, drinking, sex, etc is difficult to simulate with the present neural network architecture. As a result of engaging in pleasure causing activities, also known as beneficial behaviour, dopamine is released by special neurons in reward pathway. In addition to making us feeling better when engaging in beneficial behaviour, rewarding pathways are also responsible for, encouraging us to repeat the activity again and again, rewarding pathways strength the connection of that behaviour [6]. Normally in our active brain, inhibitory neurotransmitters are active in synapses. These neurotransmitters inhibit dopamine being released. When we engage in beneficial behaviours, anadamide is released, which stops the release of neurotransmitters and lets the dopamine to be released. Anadamide is known to be involved in removing unnecessary short-term memory. However, anadamide breaks down very quickly in the body. If you take drug, like marijuana, it has THC (delta-9-tetrahydrocannabinol) chemical substance that mimics the role of anadamide and stops the release of inhibitory neurons. Thus, THC allows the dopamine to be released. THC does not break down very quickly like anadamide, and it may effect to the loosing of short-term and long-term memory [11, 12].

Thus, it is interested to know, the role of dynamic synapses in an addiction environment and how addiction damages to the short- and long-term memory formation. Further, the researches into similar areas may willing to find the answers for the research questions, such as, what are the main characteristics of the particular behaviour, which neurons participating in the circuit produce that behaviour, what is the connectivity among those neurons, and how those neurons and their connections give rise to those behaviour. To answer these questions accurately, it is necessary to simulate such an interesting phenomenon in a similar environment where it is exist in the real world. Thus, in this particular case, it is necessary to simulate addiction on a dynamic learning environment to find the accurate answers for those questions.

Therefore, in our research we propose a new approach to implement a neural network, which enables the short- and long-term memory formation on dynamic environment. In our research, we define nervous system as a dynamic network where each neuron is model as an agent with large number of constituent elements, known as constituent agents, which play the role of synapses. These synapses can either be transmitters or receptors, and also they can either be in active or inactive statuses. These two dynamic statuses are controlled through short- and long-term plasticity mechanisms. The active number of receptors in a given connection defines the strength of the connection at a given time, thus making the strength dynamic rather than static.

2. SYNAPTIC PLASTICITY

Our human brain is a network of millions of neurons. Each neuron consists of millions of synapses which convey information whilst contributing the memory formation and learning. A typical structure of a neuron is shown in fig. 1. As shown in the figure, at the end of axon terminals there are special cleft called synapses, locations where information is

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really exchanged from one neuron to another. In synapses, information is exchanged as a means of neurotransmitter release. The amount of neurotransmitter released depends on the synapses type, location and its status, see fig. 2.

Basically neuron which transmits the signal is known as pre-synaptic neuron, and neuron which receives the signal is known as post-synaptic neuron. Thus, synapses in pre-synaptic neuron play the role of transmitters and synapses in post-synaptic neurons play the role of receptors. The response to the pre-synaptic action potentials of post-synaptic receptors depends on their statuses and locations. Therefore synapses in the same neuron exhibits different plasticity based on their statuses, location and density; plasticity of a synapse is the adaption of the synapse according to the external or internal stimuli. This variation is considered to be aroused because of large number of underlying mechanisms, collectively known as synaptic plasticity. Synaptic plasticity is mainly classified under three plasticity processes, namely short-term plasticity, long-term plasticity and homeostatic plasticity.

Fluctuations in synapses, which release neurotransmitters, are highly dynamic and show the plasticity in wide range of time scales. This plasticity can vary from milliseconds to seconds and hours to days. The plasticity which last from milliseconds to seconds are known as short-term plasticity whilst plasticity that lasts from hours to days are known as long-term plasticity. Short-term plasticity is considered as the main correspondent for short-term memory formation, and long-term plasticity is for long-term memory formation and learning.

Very large external stimuli may take the neuron into high firing frequency, On the other hand very low frequency take it to very low firing rate. However, neurons are also required to maintain its firing rate within a specified operational range to avoid any physical damage. Therefore, neurons are subject to two opposition requirements, i.e. need to change, and need for stability. The process, which brings a neuron to its operational range when the firing rate of the neuron is very high or low, is known as homeostatic plasticity [3, 4, 8, 9]. In our paper, we mainly focus on the processes of short- and long-term plasticity, and memory formation.

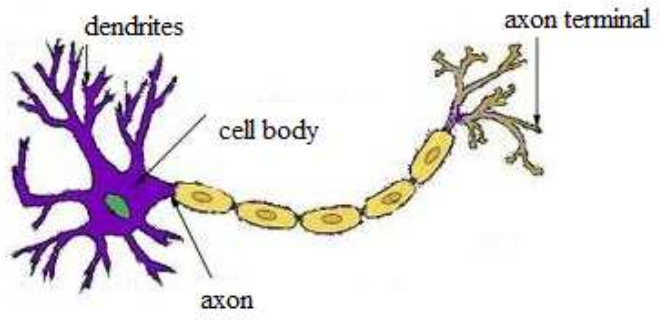

Fig. 1. **A structure of a typical neuron** consists of dendrites, cell body and axon. Dendrites take signals from other neurons, process them in cell body and finally signals are propagated trough the axon to axon terminals.

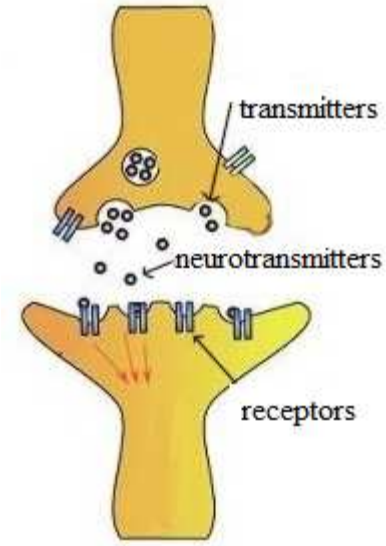

Fig. 2. **Release of neurotransmitters**. At the end of axon terminals, there are special location called synapse, the location where the information exchange takes place. At the synapse, signals are transmitted as a release of neurotransmitters. The amount of neurotransmitter release depends on the signal strength, pre-synaptic neuron status and its location. Furthermore, the response of the post-synaptic neuron also depends on its status, location, and density.

2.1. SHORT-TERM PLASTICITY

As per biology, short-term plasticity can alter response of receptors in post-synaptic neuron thereby change the overall activities of the neural circuit. The responses of receptors mainly depend on their size, locations and statuses. However, in our research, we are interest on studying how the short-term plasticity changes the synapse status over time. Synapses with low-initial-probability-release can later (after couple of milliseconds) exhibit as high-pass-filters; similarly synapses with high-initial-probability-release can later exhibit as low-pass-filters. Moreover, synapses with intermediate probability of neurotransmitter release, also known as band-filters, can later change either to high-pass-filters or low-pass-filters, based on the pre-synaptic neuron inhibition. As such we can argue that, the status of synapse depends on the pre-synaptic neuron activity,

which in turn depends on the post-synaptic response. Thus we argue that, pre-synaptic neuron activities and the previous status of the synapse determine the current status of the synapse [2, 5]. Therefore, dynamic status of synapse can be explained as a stochastic process as described in the next section.

2.2. MODELING SHORT-TERM PLASTICITY AS STOCHASTIC PROCESS

When a brief train of stimuli is applied to a pre-synaptic neuron, during the train the amplitude of the resulting pre-synaptic potential may either increase (called synaptic facilitation) or decease (called synaptic depression). A relatively long, high-frequency train of stimuli, known as tetanus, usually results in synaptic-depression, but after a few seconds, it is followed by an increase in synaptic potential amplitude that can last for tens of minutes. This is called post-tetanic-potentiation of transmitter release. Meanwhile facilitation appears instantly and lasts for milliseconds to seconds during the tetanic build-up. Fig.3 shows the normalized form of amount of neurotransmitter release corresponding to the simultaneous effect of facilitation, depression and potentiation.

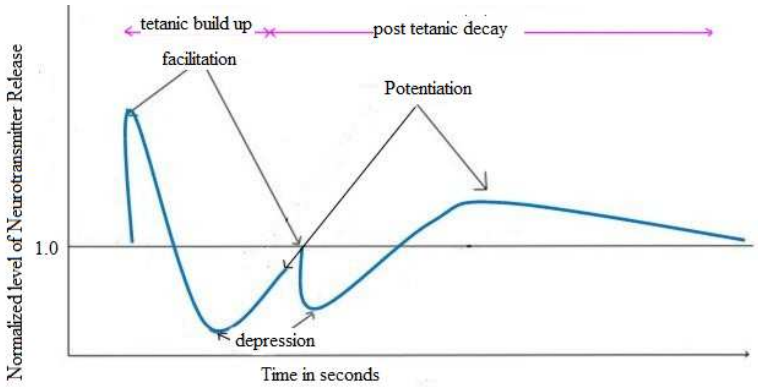

Fig. 3. **Frequency dependent changes in Synaptic status,**
the effect of simultaneous facilitation, depression and potentiation on transmitter release [10].

To model the short-term plasticity, we use the model proposed by Mass and Zador which improves the computational power of synapses with two statuses, i.e. Release (R) or Failure of release (F) whilst describing their dynamic status under stochastic process [7]. Synapses change their statuses from R to F or F to R based on the stochastic probability, which is defined in eq.1. For each spike in spike train t, the output of the synapse consists of the sequence S(t) of those $t_i \in t$ on which neurotransmitters are released by S. Thus, $t \rightarrow S(t)$ becomes a stochastic process, computed by synapse S, with output sequence $q= q_1, q_2, q_3, ..., q_n \in \{R,F\}$. $P_s(t_i)$ defines the probability that i^{th} spike in the pre-synaptic spike train $t = (t_1, t_2,... t_k)$ 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 eq.2 and eq.4., model facilitation and depression. Function C(s) in eq.3, 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 eq.5, models the response of V(t) to a proceeding release of the synapse S at time $t-s \leq t$. Whilst non-negative parameters α, τ_c and τ_v model the magnitude of the signal and the decay constant of facilitation and depression respectively. C_0 and V_0 model the parameters for equilibrium statuses. Summary of the output of the synapse S on a given time spike-train is shown in fig.4. It is interesting to note that all spike do not cause the release of neurotransmitters. And arise of facilitation may lead to the depression and may stop the neurotransmitters being released.

$$p_s(t_i) = 1 - e^{-C(t_i)\cdot V(t_i)} \quad (1)$$

$$C(t) = C_O + \sum_{t_i < t} C(t - t_i) \quad (2)$$

$$C(s) = \alpha \cdot e^{-s/\tau_c} \quad (3)$$

$$V(t) = \max\left(0, V_O - \sum_{t_i : t_i < t \text{ and } t_i \in S(t)} V(t - t_i)\right) \quad (4)$$

$$V(s) = e^{-s/\tau_v} \quad (5)$$

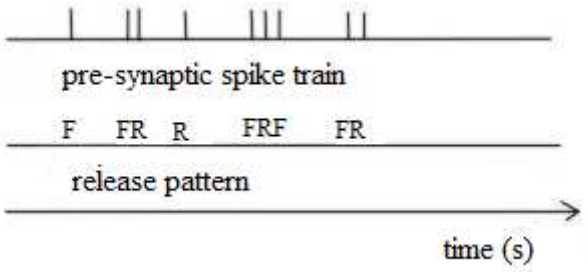

Fig. 4. **Dynamic status of synapse on a spike train t**,
synapse S releases the neurotransmitter only if the output is R, otherwise it does not release the neurotransmitters [7].

Thus, neurotransmission can be excitatory, i.e. increase in post-synaptic neuron firing on an action potential, or inhibitory, the decrease in post-synaptic neuron firing. Excitation can be considered as the release of R for further propagation of a signal. Furthermore when F is the output the signal propagation is stopped and it is called inhibition. Another important aspect of this modelling is the two time decay constants for facilitation and depression i.e. τ_c and τ_v. For the short-term plasticity these two constants vary from milliseconds to seconds.

2.3. LONG-TERM PLASTICITY

In our nervous system, repetitive activities can produce changes in synaptic efficacy which lasts from hours to days. The long duration of these changes suggests that they may be associated with long-term memory formation. These repetitive activities can induce two types of changes, namely, long-term potentiation and long-term depression. As per biology, long-term potentiation can be induced by applying high-frequency stimulation of inputs which ultimately produces a subsequent increase in the amplitude of excitatory synaptic potential that lasts for hours to days. Long-term potentiation appears to involve in both the insertion of new receptors and increase in receptor sensitivity in post-synaptic neuron. On the other hand, long-term depression is a prolonged depression produced by previous repetitive activities in the same pathway or different pathway to the same cell. This long-term depression can be induced by prolonged low-frequency stimulation and it appears to mediate a decrease in receptor numbers and sensitivity.

2.4. MODELING LONG-TERM PLASTICITY WITH SHORT-TERM PLASTICITY

Synaptic facilitation and depression occur on multiple timescales, including short-term or long-term. Therefore we are interested in integrating short-term plasticity with the long-term plasticity, which provide an underline framework for short- and long-term memory formation. Activities that last for milliseconds to seconds involve in short-term memory formation whilst repetitive calling of these activities or signals with high frequencies may mediate the facilitation and depression which could last for hours to days.

Thus, we can extend the modal proposed by Mass and Zador to incorporate the long-term plasticity. This extension must attribute the factors which induce the long-term-potentiation and -depression, namely high-frequency stimulation of inputs, repetitive calling of activities and low-frequency stimulation of inputs which could last for hours to days. Once we assign larger values like hours to days, to τ_c and τ_v, we can modal the long-term -potentiation and -depression. The repetitive calling of short-term activities can mediate the formation of long-term memory as well. An update of the above theory is shown in eq.6 to eq.12. Modifications to eq.7 is to integrate both short- and long-term facilitation, similarly, modification to eq.10 is to accommodate both short- and long-term depression. τ_{cs}, τ_{vs}, τ_{cl} and τ_{vl} are the time decay constants for short- and long-term facilitation and depression respectively. The parameters α_s and α_l model the magnitude of the signal for short- and long-term facilitation.

$$P_s(t_i) = 1 - e^{-C(t_i)\cdot V(t_i)} \tag{6}$$

$$C(t) = C_O + \sum_{t_i<t} C_s(t-t_i) + \sum_{t_j<t} C_l(t-t_j) \tag{7}$$

$$C_s(s) = \alpha_s \cdot e^{-s/\tau_{cs}} \tag{8}$$

$$C_l(s) = \alpha_l \cdot e^{-s/\tau_{cl}} \tag{9}$$

$$V(t) = \max\left(0, V_O - \sum_{t_i:t_i<t \text{ and } t_i \in S(t)} V_s(t-t_i) - \sum_{t_j:t_j<t \text{ and } t_j \in S(t)} V_l(t-t_j)\right) \tag{10}$$

$$V_s(s) = e^{-s/\tau_{vs}} \tag{11}$$

$$V_l(s) = e^{-s/\tau_{vl}} \tag{12}$$

3. AGENT MODELLING ON DYNAMIC SYNAPSES

In our research, nervous system is modelled as a dynamic neural network, where each neuron, an agent, consists of large number of constituent elements, known as constituent agents playing the role of synapses. A synapse can either be a transmitter or a receptor. Therefore, typical structure of a neuron agent, in our approach can be shown as in fig. 5.

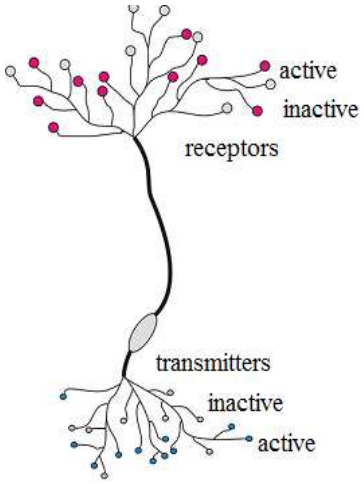

Fig. 5. **A typical structure of a model neuron**. A neuron consists large number constituent elements, either play the role of transmitters or receptors. The statuses of these synapses can change according to the input stimulus. When the output of the synapse is R, it is considered as an active synapse and when output is F, it is considered as inactive.

The propose agent structure enables the synapses to have two dynamic statuses, either active or inactive. Only active receptors can receive signals from other neurons, and propagate the signal to transmitters in the same neuron. Similarly, active transmitters can only transmit signals to the receptors in other neurons. If the selected receptor or transmitter is in the inactive status subsequently the signal is dropped. In addition, receptors in a neuron are grouped and number of receptor groups within the neuron is equal to the number of neurons in the network -1. Also the number of receptors in each group may change based on the connection strength. Therefore, number of receptors, and active number of receptors of a given connection are the critical factors which determine the strength of the connection between two neurons. Thus, the propose architecture enables to model the synapses with two dynamic statuses which are manipulated by short- and long-term plasticity mechanisms. For example, if we consider a network with four neurons, the typical structure of the network can be shown as in fig.6.

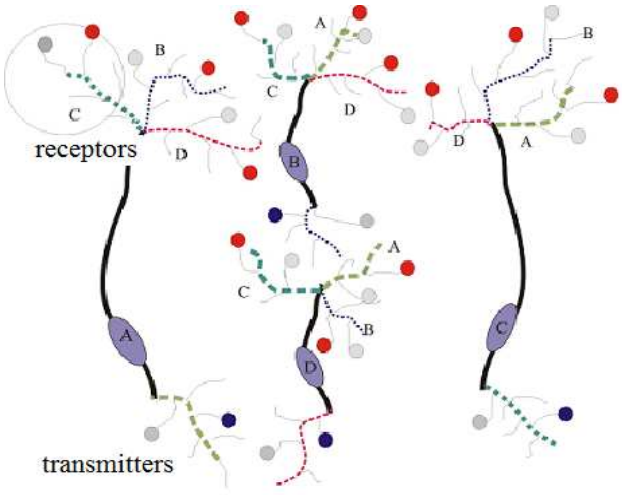

Fig. 6. **Structure of the network with four model neurons**. Each neuron has three receptor groups corresponding to the other three neurons. Transmitters are not grouped, but for the randomly selected neuron, a transmitter can transmit the signal to a randomly selected receptor in the corresponding receptor group of the selected neuron. Within the network, signal is propagated in terms of messages. A, B, C and D are neurons in the network. Receptors in each neuron are grouped to communicate with the corresponding neuron. For example, receptor group C in neuron A can communicate with only the transmitters in C neuron

Receptors are grouped into three groups to establish the connection with the other three neurons. Once the external stimulus is given to the input layer neurons, a receptor in each receptor group may receive the signal. Then the signals are propagated to the transmitters in the same neuron. Transmitters those are in active statuses randomly select a neuron, and transmit the signal to a receptor in the corresponding receptor group of the selected neuron. For instance, if active transmitter

in neuron A wants to transmit the signal to a receptor in neuron B, then it selects the receptor from A-receptor group in neuron B.

4. DISCUSSION

Memory continuously improves while adapting to external and internal stimuli. Changes that occur due to these adoptions could last for milliseconds to seconds, and hours to days. Thus based on their duration, changes are classified into two main phenomena, i.e. short-term plasticity and long-term plasticity. According to biology, these two processes are correlated so that short-term manipulations affect to the long-term behaviours. However, behaviours of these two processes can be explained by dynamic behaviour of synapses and vice versa. The location of synapses, statuses and their size define the response to the external or internal stimuli which ultimately define the overall activity of the nervous system.

In this paper, we proposed a new approach to model the learning process by mainly concentrating on the dynamic behaviour of synapses. The approach identifies the nervous system as a dynamic network where connections among neurons are subjective to the behaviours of synapses. The given model defines the neuron as an agent with large number of constituent agents playing the role of synapses either as transmitters or receptors. Receptors within the neuron are grouped therefore number of receptors in a group, and the number of active receptors within the group determine the strength of the connection at a given moment.

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