Effect of Multiple Synaptic delays in Neural Plasticity

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Abstract

Neurons are the fundamental units of the nervous system, responsible for receiving sensory input from the external world, processing them in a non-linear manner and transmits information to neighbouring neurons connected to each other with *synapses* via electrochemical signals. That is, they are both the source and transmission cells of the same order. Together with the brain's malleability, one of its most remarkable features, that provides sophisticated learning capabilities, it results in one of the most complex systems that have ever existed.

In this work, a generalised plasticity model based on synaptic weights using the local traces method will be formulated and compared with the classic one. This model enables *multi-synapses* and *propagation delay* characterization in a *spiking neural network*. The main objective of this project is to study how the *delay distribution* of a network determines its topological features after a *learning process*. This study is made, both, for small motives and large networks in order to understand the internal machinery of the model and to study its effect on a larger scale. Furthermore, the steady-state large network results are compared with the steady-state results obtained in situations where specific neurons are stimulated. This last configuration represents the learning process of one specific *stimulus* which feeds the network.

This project opens the door to future plasticity studies with propagation delay implementation, or in other words, allows the study of *plasticity in spatial arrangements* of neurons using the concept of *local traces*.

1 - Introduction

The brain is a complex organ that controls thought, memory, emotion, touch, motor skills, vision, breathing, temperature, hunger, and every process that regulates our body. Neurons consitute its funcitoning unit. This type of cell is highly specialized for generating electrical signals in response to inputs and transmitting them to other cells. The neuron is basically constituted by: Axons, Dendrites, and Soma (see Figure 1).

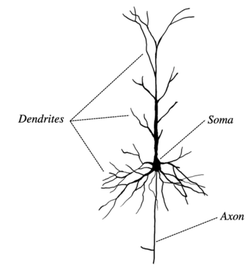

Figure 1. Drawing of a Pyramidal Cell [1].

Signals are passed from one neuron to the next at junctions called synapses. The synapse includes the end of an axon of the emitting neuron (presynaptic), the dendrite of a receiving neuron (postsynaptic), and a space between the two called the synaptic cleft.
The whole process of transmission is based on the action potential, which is a fast depolarization of the membrane's voltage due to a sudden increase of positive ion inflow towards the cell. The membrane potential rapidly goes from its resting -70 mV value to a positive value, around 30 mV , returning to the resting state in roughly 1 ms (see Figure 2).

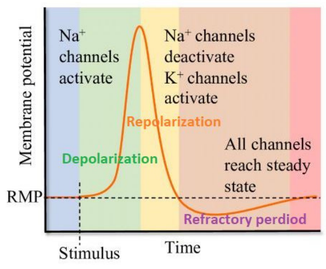

Figure 2. Action potential stages [2].

Depending on the nature of the ion flow, the synapses can have either an excitatory or inhibitory, having a depolarizing or hyperpolarizing effect, respectively, on the postsynaptic neuron. Thus, we can classify neurons as excitatory and inhibitory.

The most important homeostatic plasticity mechanism corresponds to physical changes in the synapses. The synaptic current, in charge of transmitting action potential to neighbouring neurons, can be modulated by the strength of the synapse. This term is going to be refered as synaptic weight.
Long term Spike Timing Dependent Plasticity (STDP), known for inducing persistent long-lasting effects on the synapse physiology, can be summarised as neurons that fire together wire together and neurons that fire apart wire apart . That is, if presynaptic neuron spikes before the postsynaptic neuron the synapse is potentiated (Long Term Potentiation) and if the presynaptic neuron spikes after the postsynaptic one the synapse is depotentiated (Long Term Depression) (see Figure 3).

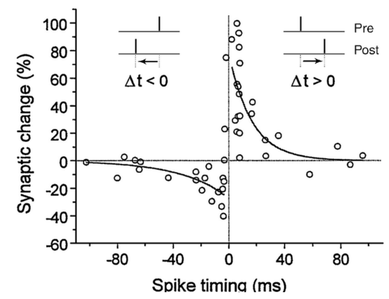

Figure 3.The critical window for the induction of synaptic potentiation and depression [3].

2 - Methods

2.1 - Spiking model

The spiking model selected for the study has been Izhikevich's [4] because of its high biological plausibility while keeping very low the computational cost.

$$\begin{cases} v' = 0.04v^2 + 5v + 140 - u + I_{ext} \\ u' = a(bv - u) \quad \text{if } v \geq 30\text{mV, then } \begin{cases} v \leftarrow c \\ u \leftarrow u + d \end{cases} \end{cases}$$

The variable v represents the membrane potential of the neuron and u represents a membrane recovery variable, which accounts for the activation of K+ ionic currents and inactivation of Na+ ionic currents, and it provides negative feedback to v (see Figure 4). Both variables are dimensionless. I_{ext} is the external current which excites each neuron, given in mA units.The rest of parameters enable the model to reproduce several spiking dynamics observed in real neurons.

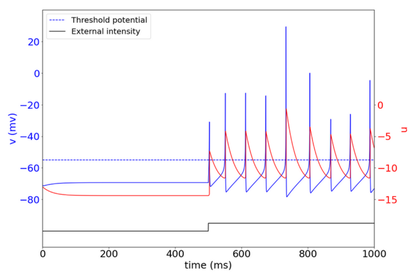

Figure 4. Values of variables from the model.

2.2- Synapse model

Chemical synapse is a complex signal transduction device that produces a postsynaptic response when an action potential arrives at the presynaptic terminal. These will be described by a conductance-based model:

$$I_{\text{syn}}(t) = g_{\text{syn}}(t)\,(V(t) - E_{\text{syn}})$$

In an attempt to model such a complex process which takes the arrival of action potential from previous presynaptic unit, recycling and releasing of vesicles, and it's propagation though the synaptic cleft into account, $g_{syn}(t)$ has been shaped with an alpha function.

$$g_{\text{syn}}(t) = \bar{g}_{\text{syn}} \frac{t - t_s}{\pi_\alpha} \exp\left(-\frac{t - t_s}{\pi_\alpha}\right)$$

Being t_s the time of arrival of a presynaptic spike, π_α a decay constant and $\bar{g}_{syn}$ the maximal synaptic conductivity, a constant value. Finally, with the consideration of synaptic weight, w_{syn} , acting as a percentage value of the maximal synaptic conductivity which will evolve according to the plasticity model, the final expression for the synaptic intensity is:

$$I_{\text{syn}}(t) = -\sum_{pre}\sum_{i} \bar{g}_{\text{syn}}\, w_{syn}\alpha(t - t_{i_{pre}}, \pi_\alpha)\,(V_{post}(t) - E_{syn_{pre}})$$

Where pre stands for the in-degree of the postsynaptic neuron and i for each presynaptic neuron spike. That is, the total I_{syn} of a postsynaptic neuron is the algebraic sum of timeshifted alpha-functions over all the spike times of the presynaptic cell, for every presynaptic cell it is linked to (see Figure 5).The reversal potential E_{syn} of the presynaptic neuron is set to a value of 0 mV if the neuron is excitatory and -80 mV if the neuron is inhibitory, common values when modelling synapses.

2.3- Plasticity model

STDP plasticity has been implemented using local variables called traces, a popular option when dealing with spiking neural networks. Each neuron is associated with a variable quantity x such that it is updated with each spike by a unit impulse and decays between spikes with a time constant π_{trace}.

$$\frac{dx}{dt} = -\frac{x}{\pi_{trace}} + \sum_{t^s} \delta(t - t^s)$$

Traces left by presynaptic spikes need to be combined with postsynaptic spikes. The spikes of presynaptic neuron j leave a trace $x_j(t)$ and the spikes of the postsynaptic neuron i leave a trace $y_i(t)$ (see Figure 6).

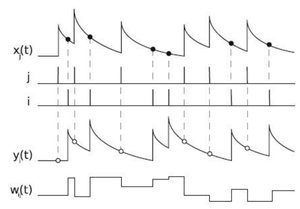

Figure 6.Implementation of pair-based plasticity by local variables.[5]

In order to implement the change of synaptic weights, the following update rule is proposed, where, τ , standing for dendritic and axonal propagation delay, has been taken in consideration and is always positive defined (see Figure 7).

$$\Delta w_{ij} = \begin{cases} -\lambda\,(1 - w_{ij})\; y_i(t_{pre\ spike} + \tau), & \text{a time } = \tau \text{ after presynaptic neuron spikes} \\ \lambda\; w_{ij}\; x_j(t_{post\ spike} + \tau), & \text{a time } = \tau \text{ after postsynaptic neuron spikes} \end{cases}$$

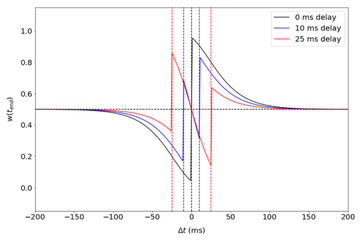

Figure 7. Weight stationary value for different Δt using deterministic spike trains in both pre and post synaptic excitatory neurons.

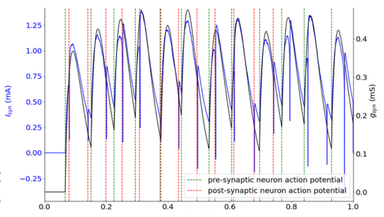

Figure 5. g_{syn} and I_{syn} dynamics in a pre post psynaptic neuron pair simulation.

3 - Results

3.1 - Small motives

The synaptic plasticity exploration is started by studying the simplest case, a pre post synaptic neuron pair and tuning the propagation delay.

For τ = 0 the maximal synaptic potentiation is reached for this particular neuron configuration. As the delay increases the positive contributions, mainly due to the term $y_i(t_{pre\ spike} + \tau)$ become smaller while the $x_j(t_{post\ spike} + \tau)$ becomes larger, reaching the synapse depression. When the delay is larger than the average spiking inter-spike interval, this trend is reversed. In fact this behaviour is repetead multiple times as the delay value increases.

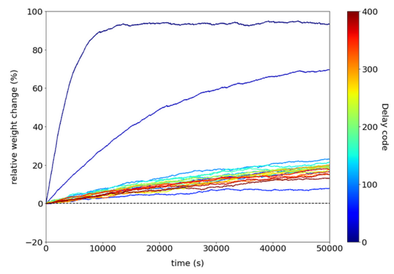

Figure 8. Proposed update rule with a wide range of synaptic delays.

The most simple case of multi-synapses is studied next. A pre and post synaptic neuron pair with two synapses, a short one and a long one in terms of the delay range used in the project. For comparison purposes both synaptic weights are set to the same value initially, 0.5.

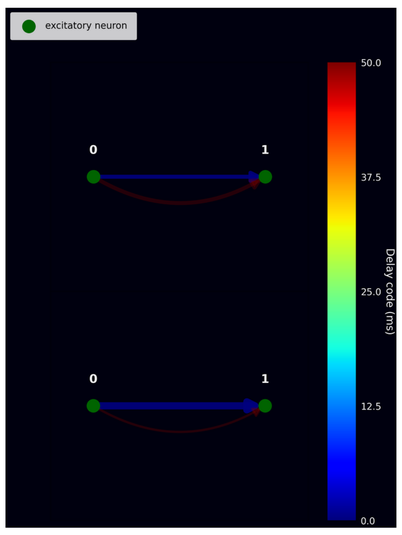

Figure 9.Configuration before and after the learning process. The red connection gets depressed and the blue one gets potentiated.

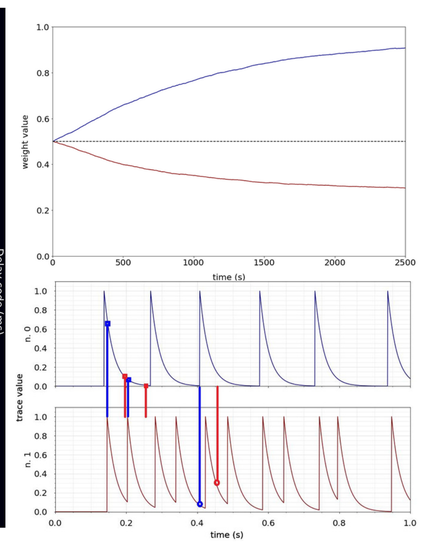

Figure 10. (a) Synaptic weight evolution and (b) local traces diagram.

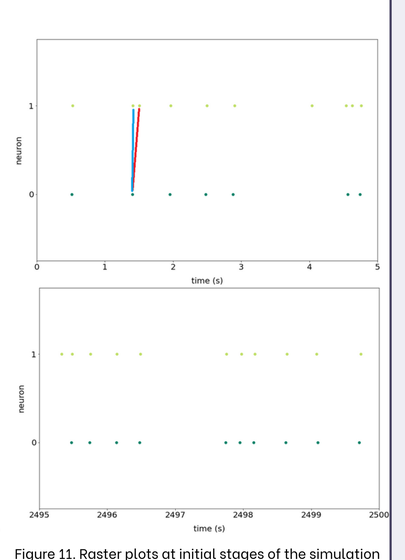

Figure 11. Raster plots at initial stages of the simulation and at final ones. Triangular shape joining two postsynaptic spike and the presynaptic one can be found in the first raster plot but not in the second one as a result of weight evolution.

Each time the postsynaptic neuron spikes postive increments added to each synaptic weight. These are greater in the shorter delay synapse than in the longer one. The same takes place regarding presynaptic spikes and negative increments. However, those are greater in the longer synapse than in the shorter one (see Figure 10 b).

The essence of why some synapses get potentiated and some depressed lies, firstly on how neurons communicate, which translated on how local traces interact between each other. The delay propagation characterisation is involved twice in this porcess, in the time of action potential propagation and also in the trace evaluation, following the proposed synaptic weight update rule.

3.2 - Larger Networks

Several delay distributions are used in order to characterise the synapses of the same network. These are uniformly distributed, normally distributed centered in the middle range and a bivalued distribution using two gausians, one centered at the low range and one at the high range (not shown). Statistical tools, as histograms and inter-spike intervals, lead to more visual intuition of the synaptic weight stationary state.
In addition, two cofiguration with respect the powering of the neurons are compare between each other, one with homogeneous supra-threhsold intensities for every neuron of the network and another in which, only a given subset of neurons of the network, are driven by a supra-threshold external intensity which ultimately powers the whole network. This second configuration, mimicks a stimulus-powered learning process. Results differ significantly and are commented in the manuscript of the memory.

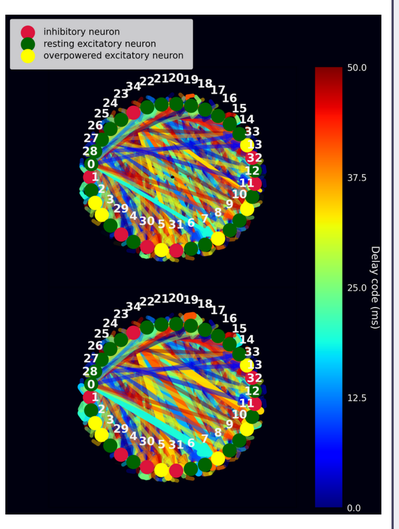

Figure 12. Topology of the network, before and after, the evolution the weights. The network is formed by 35 neurons connected by 595 synapses. In this case, delays are distributed uniformly.

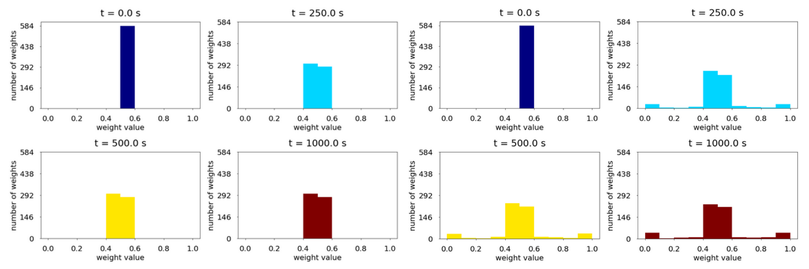

Figure 13. Histogram of synaptic weight state comparison of how the learning stimulus infulences the network after learning with middle range cenetered distributed delays.
Homogeneous supraspike stimulation (Left). Only stimulating certain excitatory neurons over the spiking threshold (Right).

4-Conclusion

The proposed model successfully yields synaptic weight evolution according to it's delay characterisation. It also allows to study plasticity in multi-synaptic connections, a field in which not many computational contributions have been made. A preliminar study where the effect of delays has been done and small motives composed of small sets of neurons have been studied (not shown), understanding the internal machinery of the plasticity model. A larger network has been used in order to study the effect of multiple synaptic delays in neural plasticity with and without specific stimulation, resulting in different outputs in each simulation. The final state of the network has been proven to be affected by the neurons which are stimulated, thus the network has gone through a learning process of this stimulation.

5-References

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