

A Review on VLSI Implementation of Neuromorphic Spike Time Dependent Plasticity Algorithms for Different Neuron Models

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ABSTRACT

Background: The Neuromorphic VLSI is concerned with the development of electronic systems in order to mimic neural ones, mostly for brain simulation purposes. The brain has a unique ability to continuously evolve and adapt to changing environment. *Methods:* The brain has a large amount of synapse which has ultra-high integration density and ultra-low power consumption as they are operated under sub-threshold region. Neurons produce the action potentials according to the event and synapses are the connections among neurons. It is believed that synapses are the part of the brain where learning and memory take place. The strength of the synapse is dependent on synaptic efficacy or weights. *Findings:* These weights alter according to synaptic plasticity rule which is dependent on the pre- and post-synaptic activities. This rule can be timing and rate dependent. We study the some spike timing dependent plasticity rule like triplet or pair based and BCM rule which is rate dependent. BCM like behavior emerges from triplet based plasticity rule. There are various neuron models mainly HH, MN, and IZ. *Conclusion:* The spike timing dependent circuits perform learning with reasonable low silicon area consumption and better energy efficiency. The spike neuromorphic circuits lead to the advancement in the field of robotics, brain machine interfaces and many more.

Keywords: spike timing dependent plasticity; Bienenstock-Cooper-Munro component; rate and time-based synapticity

1. INTRODUCTION

Our brain has an ability to continuously evolve itself and it adapts continuously changing the environment. This ability to change and adapting environmental changes over time is called plasticity. This concept of plasticity is present and implemented at the level of connections between two neurons and this connection is called synapse. So it is required to study the synaptic plasticity to learn how the brain evolves and how it store memory.

D.O. Hebb (1949) postulated that When an axon of cell A is near enough to excite cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased.

Also, according to this postulate, correlated activity of neuron A and B increases the strength of the synapse (or weight) between A and B. Even if the notion of correlation-based learning has been formulated before Hebb (James 1890), it is often called Hebbian learning. An important aspect of Hebb's postulate is its intrinsic notion of locality. Only locally available information, namely the pre- and postsynaptic activities can trigger a weight change. Without such an unsupervised mechanism which adapts continuously the weights, it would be impossible to tune individually each of the 10¹⁴ synapses in the human brain [1].

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The paper is been organized as follows: In section 2, we discussed basics of a neural network like biological neural networks, silicon neural networks and basic of spike timing dependent plasticity. In section 3 we discussed neuron models and synaptic plasticity models including protocols of pairing. In section 4 we discussed mathematical modeling of synaptic plasticity.

2. NEURAL NETWORKS

2.1. Biological Neural Networks

Biological Neural Networks Neural networks are inspired by our brains. The human brain has about 10^{11} neurons and 10^{14} synapses. A neuron consists of a soma (cell body), axons (sends signals), and dendrites (receives signals). A synapse connects an axon to a dendrite. Given a signal, a synapse might increase (excite) or decrease (inhibit) electrical potential. Neuron fires when its electrical potential reaches a threshold. Learning might occur due to changes in the synapse.

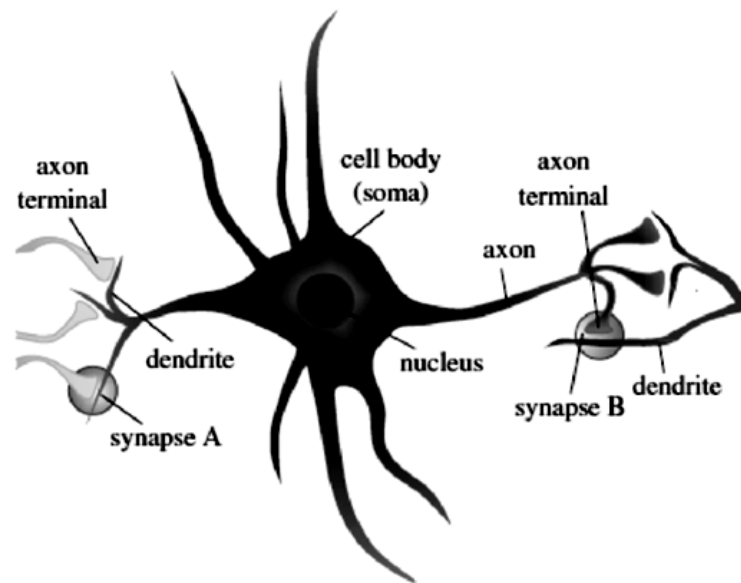


Figure 1: Biological neuron[20]

Above diagram shows biological neuron and the main four parts are highlighted which are dendrites, soma, synapse, and axon. For left synapse A, the yellow axon is from the Presynaptic neuron and the blue neuron is the postsynaptic neuron, while in synapse B the blue neuron is the Presynaptic neuron and the pink dendrite is from the postsynaptic neuron Adapted from [20].

2.2. Silicon Neural Network

Neuromorphic engineers have proposed a variety of problem-solving techniques which are both human and nature inspired like Genetic Algorithm [2] and Artificial Neural Network[3]. These have resulted in three different generations of neural networks based on three different types of neurons.

The First generation was proposed by McCulloch and Pitts (1943) [4]. These were composed of threshold neurons also known as binary units. This was the most simple neuron model. When the summation of all the inputs to a neuron is more than a predefined threshold, neuron fires, i.e. a Pulse which is used to represent a binary state. Basic Boolean operations can be performed by this type of neuron but for computational problems this model was completely impractical.

After this came the new model (second generation) which was more realistic than the first generation as it uses the continuous output function rather than binary phase.

The first two generation models were concerned about the rate of carrying information and were independent of input and output timings. These input and output timings were very important to be considered as these were responsible for the learning and recognition. This input-output relation brings timing into action which makes a transition model of neurons known as spiking neurons. These neurons are building blocks for the third generation of artificial neural network. The third generation of the artificial neural network called SNN. The SNN are composed of spiking neurons and synapses [4]. The spike carries information to other neurons and synapses in the network. These spikes are nothing but action potentials. Propagation these spikes carry information and this information is transferred in the SNN

From a functional point of view, an artificial neuron consists of one or more than one *synapse* blocks which are said to be responsible for receiving signals(spikes) from other neurons and integrating all the signals all together over time and converting them into current signals as shown in fig.2 . There is another important block known as a *soma* block, responsible for the spatiotemporal integration of the input signals and also, it generates the output analog action potentials and/or digital spike events. In addition, to both these synapse and soma blocks, there are units which model the neuron's structure(spatial) and implement the signal processing that takes place in dendritic trees and axons, respectively.

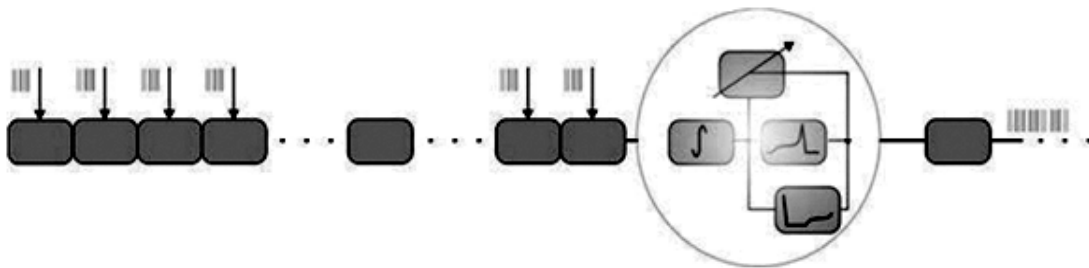


Figure 2: Artificial neuron [39]

Table 1
Comparison of biological and artificial neural network

<i>Biological neural network</i>	<i>Artificial neural network</i>
Soma	Unit
Axon, dendrite	Connection
Synapse	Weight
Potential	Weighted sum
Threshold	Bias weight
Signal	Activation

Spiking neurons as the name suggest are the neurons which use a spike (electric pulse) to transfer information. Silicon Neural network (SNN) is made up of spiking neurons and synapses. There is a special relation on the basis of information coding in the forms of rate, timing and hybrid of both by the means of which neurons communicate to each other. Along with spiking neurons, synapses are one of the most important building blocks in SNN. These synapses are nothing but the connection between two neurons. Through this synapse or change in weight (ANN), transfer of information takes place inside the neural network.

2.3. Synaptic Plasticity Rules

Many neuroscientist researchers on their hypothesis show how the brain performs different tasks of learning or judging and according to that, they propose different hypothetical models and experimental observations which are further tested with real world stimuli. This approach is used to for both realizations of hardware systems and also to get a deeper understand the concepts. Currently, many of such models are implemented practically by developing VLSI 'neuromorphic' systems [4], [5]

Investigating synaptic plasticity experimentally, it leads to some diverse results which are mainly depending on the animal preparation studied, in the area of the part of the brain analyzed, on the protocol used, and on many other important factors required for the same [3], [6], and [7]. Due to all these variations, researchers have come out with many different results which are both inconsistent and also controversial. Continuous study of all these has led to the development of an extremely large numbers of models called synaptic plasticity models. Some of these are abstract, some are very elaborate and some are presenting synaptic plasticity in detail [8].

Models of synaptic plasticity are aimed to reproduce the basic concept of learning. This learning as said earlier depends on weight change. This change in weight is due to different factors. One of the important factors is the timing of spikes which is been produced by presynaptic and postsynaptic neurons [9]–[12]. Some of the most common Examples of such rules include spike-timing-dependent plasticity (STDP) [6], [7], [13] which further includes pair-based STDP (PSTDP) and the triplet-based STDP (TSTDP) [14], [15] models. One other important model which explains the data obtained in neuroscience experiments take into account different neuron and synapse state variables, some examples of such models include the spike-driven plasticity model, voltage based STDP model, and membrane potential based models.

Generally, there are many factors which influence synaptic plasticity but the most factors which influence is the timing of pre and postsynaptic spikes. This timing based plasticity is generally termed as spike timing dependent plasticity. The two terms considered in STDP are

- 1) Potentiation: If a presynaptic spike occurs before a post one than it is called potentiation or also called up regulated.
- 2) Depression: If a postsynaptic spike occurs before a pre-one than it is termed as depression or also called downregulated.

3. MODELS OF NEURON AND PLASTICITY

3.1. Spiking neuron

As discussed earlier, basic building block of a spiking neuron network is neuron and synapse. These neurons on the basis of their shapes and bio-physic characteristic can be divided into various types. As the synapse

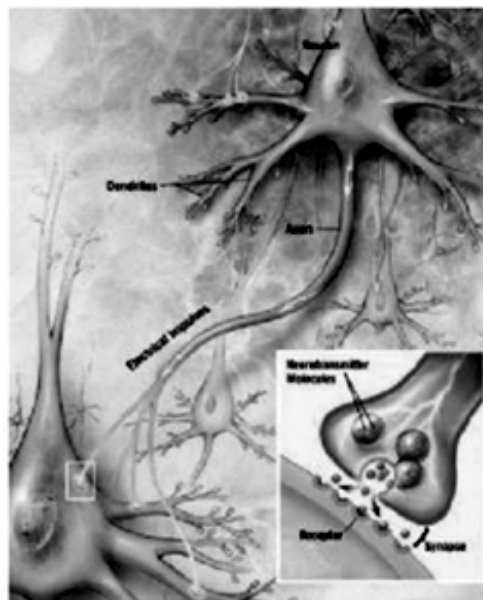


Figure 3: Various parts of neuron and also shows the synapse structure

of neuron depends on electrical pulses (spikes), these spikes are produced by every type of cortical neuron and a general shape of such neurons is shown below

In addition, to neurons, the other major part is synapse which is also shown in fig 3. These synapses also possess complex structure and are responsible for the learning and memory storage in brain neural network. As in fig, a synapse is a site where various neurotransmitter molecules are transmitted to the postsynaptic neuron.

There are three types of ions present in the neuron structure which are Na^+ , K^+ , and Ca^+ . The concentration of these ions is responsible neuron function like the unbalanced concentration of these ions inside and outside of neuron results in charging and discharging of input and output potential. And this difference results in the formation of electric pulse signal also referred as a spike.

Some of the most common neurons model available in studies are:-

- 1) Hodgkin-Huxley model (HH model)
- 2) Integrate and fire neuron model
- 3) Izhikevich and Wilson model (IZ model)

Hodgkin-Huxley Model: - This is a conductance-based model. The first neuron model based on HH model was designed in 1991. This model attempts to match the sodium and potassium currents by using Analog components and Very Large Scale Integration. This model helps in obtaining neuronal behavior including spike generation,

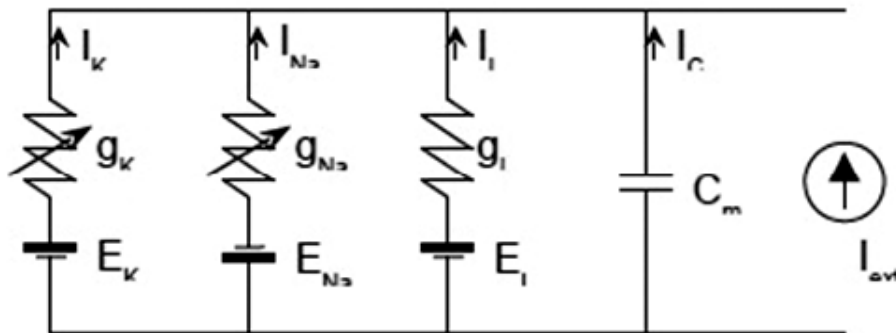


Figure 4: Electrical circuit of HH model[38]

Currents are carried inside through membrane capacity (C_m) or by carrying ions in the resistance parallel to the membrane capacity. This ionic current is divided into two parts carried by sodium and potassium ions and also a small part of leakage current.

These different currents are represented as following equation

$$I_k = g_k n^4 (V_m - E_k)$$

$$I_{Na} = g_{Na} m^3 h (V_m - E_{Na})$$

$$I_l = g_l (V_m - E_l)$$

Though this HH model can describe the activity of a neuron accurately but the main problem with this model was its complexity. This complexity makes it hard to perform the intuitive analysis. Also, this model is depending on too many parameters, currents and bias voltages which are difficult to fabricate on a single chip when considering a large neural network.

Integrate and fire neuron model: - This specific model is the most powerful model for representing a spiking neuron. Also, the model is simplest model available [20], [21], [23]–[25]. This model has been

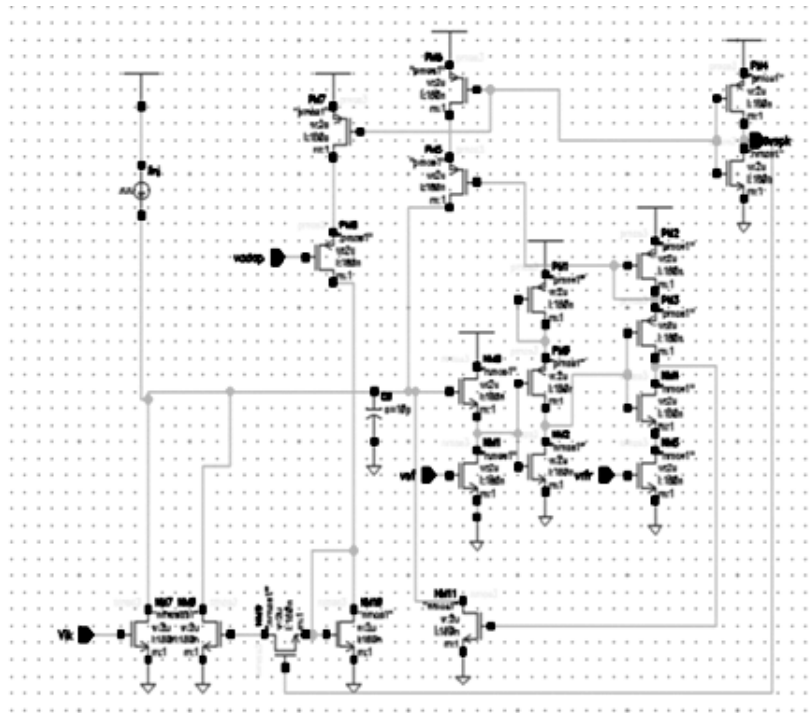


Figure 5: Integrate and fire neuron

implemented many times in silicon [26]-[30]. Integrate and fire neuron, in general, are mixed mode circuits in which when analog membrane voltage exceeds the pre-specified threshold voltage a digital pulse is produced which represents a spike required of a spiking neuron. The model by Culurciello et al. is a basic integrate-and-fire neuron, containing no adaptation or explicit leak resistance. The neuron circuit is simple and compact. It has only seven transistors and a capacitor, with a photodiode which acts as a synaptic input for the circuit. After this, a simple and effective, leaky integrate-and-fire neuron was designed by van Schaik [27]. The main required leak was implemented using a current source. This current source draws the neuron's membrane voltage to its resting potential. To get an output spike, it was required that the Excitatory inputs are larger than this leakage current. If this happens then only the membrane capacitor will get charged up and it will elicit a spike. The neuron by Indiveri is an optimized for low power neuron adapting integrate-and-fire model [29]. This circuit consists of 20 transistors and one capacitor. In this circuit basic current mirror configuration is used and it helps in implementing spike frequency adaptation. The circuit was implemented in cadence virtuoso using 180nm technology.

Each spike increments the voltage at the node which results that the amount of current that gets subtracted from the input which hence effectively reduces the firing rate of the neuron [29]. This integrate-and-fire neuron is very easy to implement in CMOS processes. However, the main disadvantage of this model is, it lacks many of the features of cortical spiking neurons. It is said that Ninety percent of such cortical neurons of the body have a nonlinear oscillatory behavior. This behavior cannot be captured by integrating and fire neurons [34].

Izhikevich and Wilson model: In 2003, Eugene Izhikevich presented a neuron model which is computationally simple and capable of producing the firing patterns exhibited by real biological neurons. It is described as a two-dimensional system of ordinary differential equations of the form

$$\frac{dv}{dt} = 0.04v^2 + 5v + 140 - u + I$$

$$\frac{du}{dt} = a(bv - u)$$

With the auxiliary after-spike resetting

$$\text{if } v \geq 30mV, \text{ then } \{v < -c \text{ and } u < -u + d\}$$

where v represents the membrane potential of the neuron and u represents a membrane recovery variable, which accounts for the activation of K^+ ionic current and inactivation of Na^+ ionic current. After the spike reaches its upper limit (+30mV), the membrane voltage and the recovery variable are reset. Synaptic currents or injected DC currents are delivered via the variable I . a and b are dimensionless parameters which govern behaviors of u . c and d are also dimensionless parameters which define reset value of v and u . The mathematical complexity of Hodgkin-Huxley model helps to fully describe the firing nature of a real neuron. Unfortunately, the same feature can be a burden in silicon neuron implementation of a neuron and possibly of a neural network in the future, due to increased complexity of the circuit. Hence, for the circuit implementation, it is required to have a model which is simple enough, yet is able to describe the firing behavior of a neuron without much deviation or error. Izhikevich's model is simpler than Hodgkin-Huxley model, and can express different neuron characteristic by varying the parameters in the equation. [35]

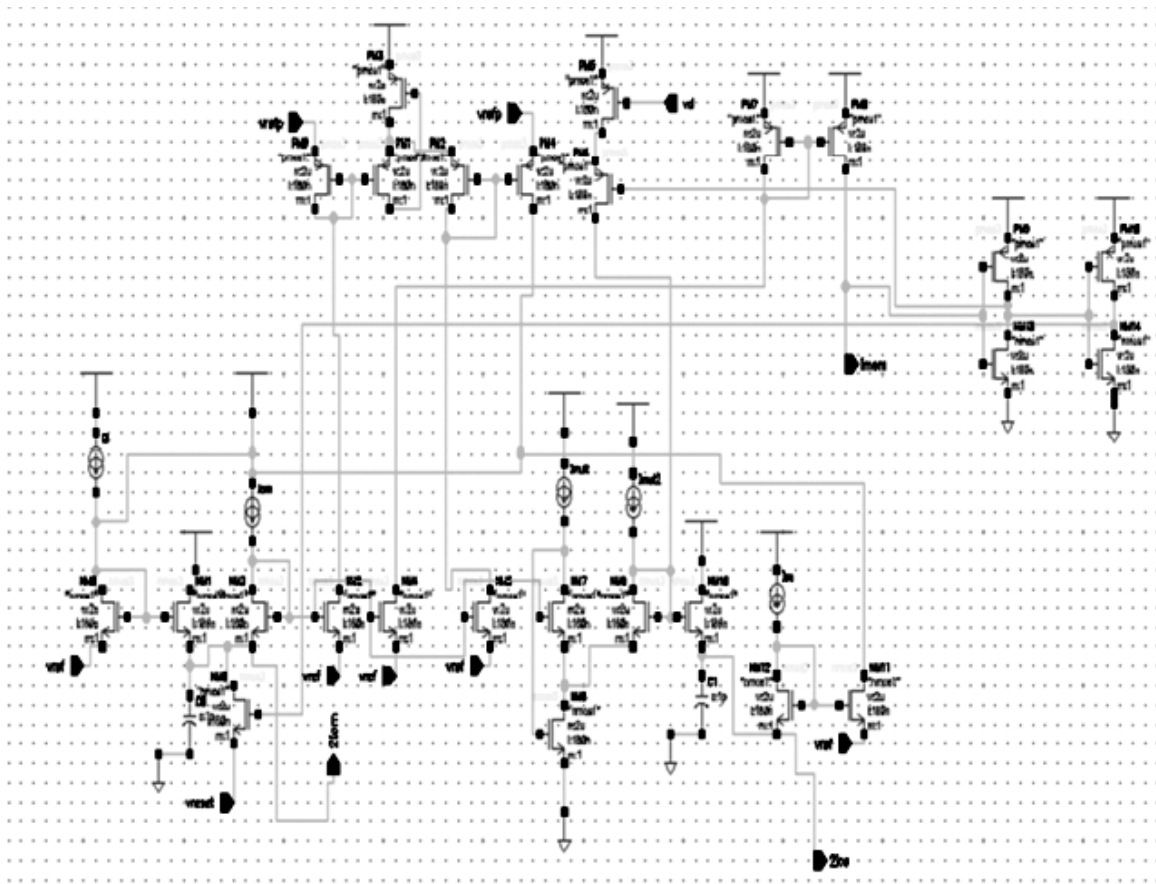


Figure 6: IZ Model

3.2. Spike Timing dependent plasticity

Synaptic plasticity is the study of change of weights on synapse which is responsible for learning and other brain functions. Like different neuron models, there are various synaptic plasticity rules which have a relationship with real biological synapse. The one which is closer to biology are complex and some other models which only approximate the biological experiments are easy and are mathematical modeling based models. The second group of plasticity is used to implement simple rules and to obtain as many synaptic rules as possible. There are different synaptic plasticity rules/protocols available which are differentiated on the following basis:

- 1) Simplicity
- 2) Strength of reproducing particular synaptic experiments
- 3) Suitability when to be implemented on large scale hardware.

On the basis of pairing which is discussed in next chapter of this report, there are different protocols available. Three widely used protocols are explained below:

3.3. Protocols of plasticity

- 1) Pairing protocols
- 2) Frequency dependent pairing protocol
- 3) Triplet protocol

Pairing protocol: This is the old protocol of plasticity earlier used in electrophysiological experiments and simulation-based studies. In this protocol 60 pairs of pre and postsynaptic spikes with a delay of

$$\Delta t = t_{\text{pre}} - t_{\text{post}}$$

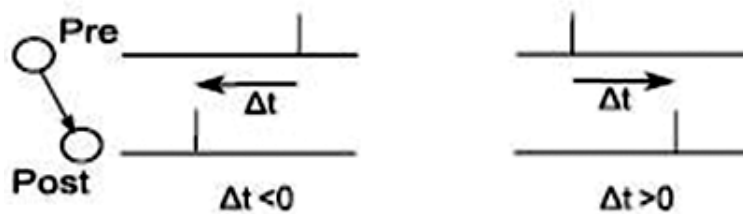


Figure 7: Spike pairing protocol

This delay is required to time for reproducing an STDP window. These pre and post spikes are conducted at a repetition with a frequency of 1 Hz (approximately).

Frequency dependent pairing protocol: In pairing protocol frequency was fixed at 1 Hz and hence spike pairs were kept constant. Studies show that changing the pairing frequency results in total weight change of the synapse. In Higher frequency order of pre and post-spike pairs does not matter and both cases will lead to potentiation whereas in low pairing frequency the pairing do matter. A pre-post pair results in potentiation and a pre-post pair results in depression[36].

Triplet protocol: - there are two types of triplet protocol which are used to compute the change in synapse/weight. Both these patterns consist of 60 triple of the spike. These spikes repeat itself at a frequency of 1Hz.

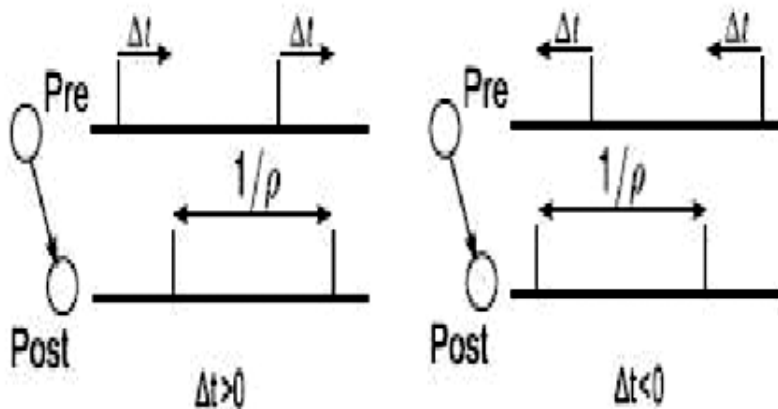


Figure 8: Frequency dependent protocol showing potentiation and depression

The first pattern is composed of two pre-synapses and one post-synapse in a configuration of pre-post-pre-configuration, where the second pattern is composed of 2 posts and a pre-spike in a configuration of post-pre-post.

Due to this configuration of spikes, there are two different delay that comes given as

For pattern 1:

$$\Delta t_1 = t_{\text{post}} - t_{\text{pre1}}$$

$$\Delta t_2 = t_{\text{post}} - t_{\text{pre2}}$$

For pattern 2:

$$\Delta t_1 = t_{\text{post1}} - t_{\text{pre}}$$

$$\Delta t_2 = t_{\text{post2}} - t_{\text{pre}}$$

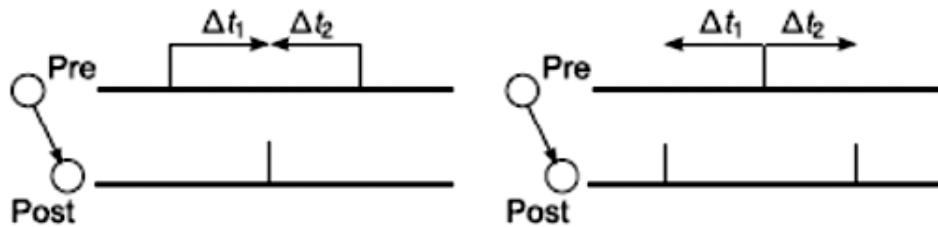


Figure 9: Both Triplet protocol

3.4. Models of Synaptic Plasticity

All the available models of STDP can be divided widely into two major groups.

- 1) Abstract models or phenomenological models
- 2) Biophysics of synapse

The abstract model shows how the learning and memory inside the brain are dependent on synaptic plasticity. These models mimic the output of plasticity independent of molecular mechanism observed in the synapse. Whereas biophysics models are depending on practical cellular and molecular mechanism presented in the synapse and demonstrate how the synapse change occurs biologically in neurons.

Phenomenological models can be classified further into different groups like spike timing dependent based models such as pair based and triplet based, rate-based models such as Bienenstock-cooper-Munro(BCM).

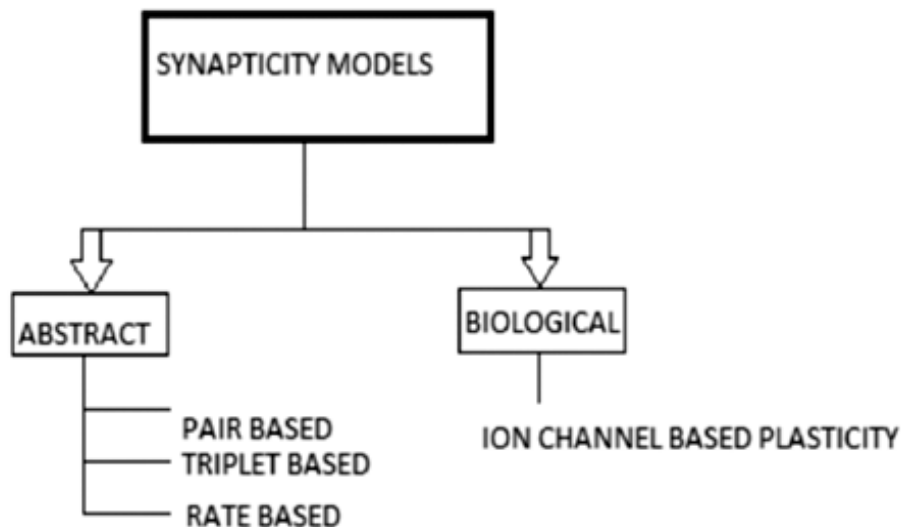


Figure 10: Various plasticity models

3.4.1. Abstract models

Pair based STDP: pair based stdp is the classical approach and description of STDP. It is been widely used in various VLSI implementation of neural network theory. The original rule is expressed by following equation [37]:

$$\Delta w = \begin{cases} \Delta w^+ = A^+ e\left(\frac{-\Delta t}{\tau_+}\right), & \text{if } \Delta t < 0 \\ \Delta w^- = -A^- e\left(\frac{\Delta t}{\tau_-}\right), & \text{if } \Delta t \geq 0 \end{cases}$$

Pair based synaptic plasticity is the classical method which depends on the arrival of spikes produced by pre and postsynaptic neurons.

Here Δt is a timing difference between a pair of the post and presynaptic synapses. According to the model specified above, the synaptic weight will be up-regulated if a pre-synaptic spike arrives before the occurrence of a post-synaptic spike in a specified time window (τ_+). Analogously, down-regulated will occur if a presynaptic spike occurs after the post-synaptic spike [39]. The amount of up/down regulation will be determined as a function of the timing difference between pre- and post-synaptic spikes, their relevant amplitude parameters (A^+ and A^-) and their temporal order [38].

Triplet based STDP: In TSTDTP timings of triplet combination of spikes from pre and post neurons determines the changes in synaptic weight [19]. A higher order temporal pattern of spikes are used to modify the weights of synapses and is called triplet based spike timing-dependent plasticity (TSTDTP) [38]. Mathematical representation of TSTDTP learning rule is given by

$$\Delta w = \begin{cases} \Delta w^+ = e\left(\frac{-\Delta t_1}{\tau_+}\right)\left(A_2^+ + A_3^+ e\left(\frac{-\Delta t_1}{\tau_+}\right)\right) \\ \Delta w^- = -e\left(\frac{\Delta t_1}{\tau_-}\right)\left(A_2^- + A_3^- e\left(\frac{-\Delta t_3}{\tau_x}\right)\right) \end{cases}$$

Where $\Delta w = \Delta w^+$ for $t = t_{\text{post}}$ and if $t = t_{\text{pre}}$ then the weight change is $\Delta w = \Delta w^-$. A_2^+ , A_2^- , A_3^+ , and A_3^- are potentiation and depression amplitude parameters, $\Delta t_1 = t_{\text{post}}(n) - t_{\text{pre}}(n)$, $\Delta t_2 = t_{\text{post}}(n) - t_{\text{post}}(n-1) - \epsilon$ and $\Delta t_3 = t_{\text{pre}}(n) - t_{\text{pre}}(n-1) - \epsilon$, are the time differences between combinations of pre- and post-synaptic spikes. Here, ϵ is a small positive constant which ensures that the weight update uses the correct values occurring just before the pre- or post-synaptic spike of interest, and finally τ_+ , τ_- , τ_x and τ_y are time constants [38].

Rate-based synaptic plasticity rules: The rate of spikes is one of the most important characteristics of a spike train. These rate dependent spikes are basics for first and second generation of a spiking neuron. This is because many researchers from the past have considered this as a main and essential cause of synaptic plasticity [5]. Two most used and well-known synaptic plasticity rules, which operate according to the rate of pre- and postsynaptic action potentials, are (i) the one proposed by Oja (1982) and (ii) the experimentally verified Bienenstock–Cooper–Munro (BCM) rule originally proposed by Bienenstock et al. (1982). The second rule which is BCM rule is capable of producing both Long Term Potentiation (LTP) and Long-Term Depression (LTD). Acc to BCM rule the relative proportions of synaptic potentiation and depression can be controlled by a sliding threshold, which also overcomes issues of positive feedback. BCM rule proves that change in weight in a synapse depends linearly on potentiation and non-linearly on depression [38], [39].

3.4.2. Biological model

The main problem with abstract models was that none of the model so far truly maps to a biophysical characteristic of synapse and biochemical reaction taking place inside and outside the synapse to induce synaptic weight changes. These biological models bring out the outcomes of many biological experiments. These include the study of protein-protein interaction using a unified mathematical expression. Due to the analogy of these rules with dynamics of the synapse, these are called biological models. The most widely used vlsi implemented biological rule available is modified ion channel based plasticity.

Modified ion channel based plasticity: This rule includes both calcium and level of calcium inside the synapse and also the effect of other channels and receptors as the pathways for calcium to change in post-synaptic neuron and hence it result in potentiation and depression. The disadvantage of this model is its complexity and requirement of a large number of state variables.

4. MATHEMATICAL MODELLING OF TRIPLET STDP

STDP data has too much variability. Due to this, it is difficult to develop a canonical model such as Hodgkin and Huxley model. This variability is observed when we compare plasticity experiments with other electrophysiological experiments. So it is required that model must be as simple as possible. This is the approach of the abstract model. Now we will explain a conceptual framework for these models. This framework is modeled using Volterra expansion.

Volterra expansion: This can be done by assuming that the instantaneous weight change w_{ij} is given by the Volterra expansion [21] of an unknown function $H[X, Y]$. This function depends on presynaptic pulse train

$X(t)$ and postsynaptic spike train $Y(t)$.

$$\begin{aligned} X(t) &= \sum t_{pre} \delta(t - t_{pre}) \\ Y(t) &= \sum t_{post} \delta(t - t_{post}) \end{aligned}$$

Where $\delta(s)$ is the Dirac function and t_{pre} and t_{post} are respectively the pre- and postsynaptic spike times:

$$W(t) = H[X, Y]$$

If we further assume that the weight changes are instantaneous and occur whenever a pre- or a postsynaptic spike is emitted, we can write

Note that the upper index in functions represents the type of interaction. For example, G_{xyy}^3 refers to a triplet interaction consisting of 1 pre- and 2 postsynaptic spikes. We remark that the G_{xyy}^3 term could correspond to a pre-post-post sequence as well as a post-pre-post sequence. The interest of such a framework is that it is possible to classify a lot of existing models within this framework. For example, a model of STDP (Gerstner et al. 1996) considers the pair terms, i.e. F_{xy}^2 and G_{xy}^2 as well as a constant term H_0 . Kempter et al. (1999) and Kistler and van Hemmen (2000) modeled STDP with the pair terms F_{xy}^2 and G_{xy}^2 and added a pure presynaptic term F_x^1 as well as pure postsynaptic term G_y^1 . Most of the STDP models, like the ones of Song et al. (2000) and Roberts (1999) only consider the pair terms F_{xy}^2 , G_{xy}^2 .

$$W(t) = H_0 + H[X, Y] + Y(t)G[X, Y]$$

Here F and G are unknown functional parameters for the required pre and postsynaptic rains and H_0 is constant. Let us now perform a Volterra expansion of those functional spike trains of pre and postsynapses. This Volterra expression is as follows:

$$\begin{aligned} F([X, Y]) &= F_1^x + \int_0^{\text{inf}} F_2^{xx}(s) X(t-s) ds + \int_0^{\text{inf}} F_2^{xy}(s) Y(t-s) ds \\ &+ \int \int_0^{\text{inf}} F_3^{xxx}(s, s') X(t-s) X(t-s') ds' ds \end{aligned}$$

$$\begin{aligned}
 & + \int \int_0^{\text{inf}} F_3^{xyy} (s-s') X(t-s') Y(t-s') ds' ds \\
 & + \int \int_0^{\text{inf}} F_3^{xyy} (s-s') X(t-s) Y(t-s') ds' ds \dots \dots
 \end{aligned}$$

Similarly equation of another parameter G is represented as

$$\begin{aligned}
 G([X, Y]) = & G_1^y + \int_0^{\text{inf}} F_2^{xy} (s) X(t-s) ds + \int_0^{\text{inf}} G_2^{xy} (s) Y(t-s) ds \\
 & + \int \int_0^{\text{inf}} G_3^{xyy} (s, s') X(t-s) X(t-s') ds' ds \\
 & + \int \int_0^{\text{inf}} G_3^{xyy} (s-s') X(t-s') Y(t-s') ds' ds \\
 & + \int \int_0^{\text{inf}} G_3^{yyy} (s-s') X(t-s) Y(t-s') ds' ds \dots \dots
 \end{aligned}$$

Since not all STDP models can be presented in this framework, it is possible to broaden it in order to include new classes of STDP models. Whenever there is a pre- or postsynaptic spike, it interacts with all previous pre- or postsynaptic spikes. We will call this the All-to-All interaction scheme. There is no a reason to think that say each presynaptic spike should interact in the same way with all previous postsynaptic spikes and vice versa. This is why other schemes of interaction have been proposed [36]. In a Nearest-Past-Spike interaction scheme, each presynaptic spike interacts only with the previous postsynaptic spike and each postsynaptic spike with the previous postsynaptic one. The Nearest-Future-Spike interaction scheme considers the opposite case. Each presynaptic spike interacts with the next postsynaptic spike and vice versa. In the Presynaptic-Centric case, each presynaptic spike interacts only with the last and the next postsynaptic spikes. Similarly, in the Postsynaptic Centric case each postsynaptic spike interacts with the previous and the next presynaptic spikes [36].

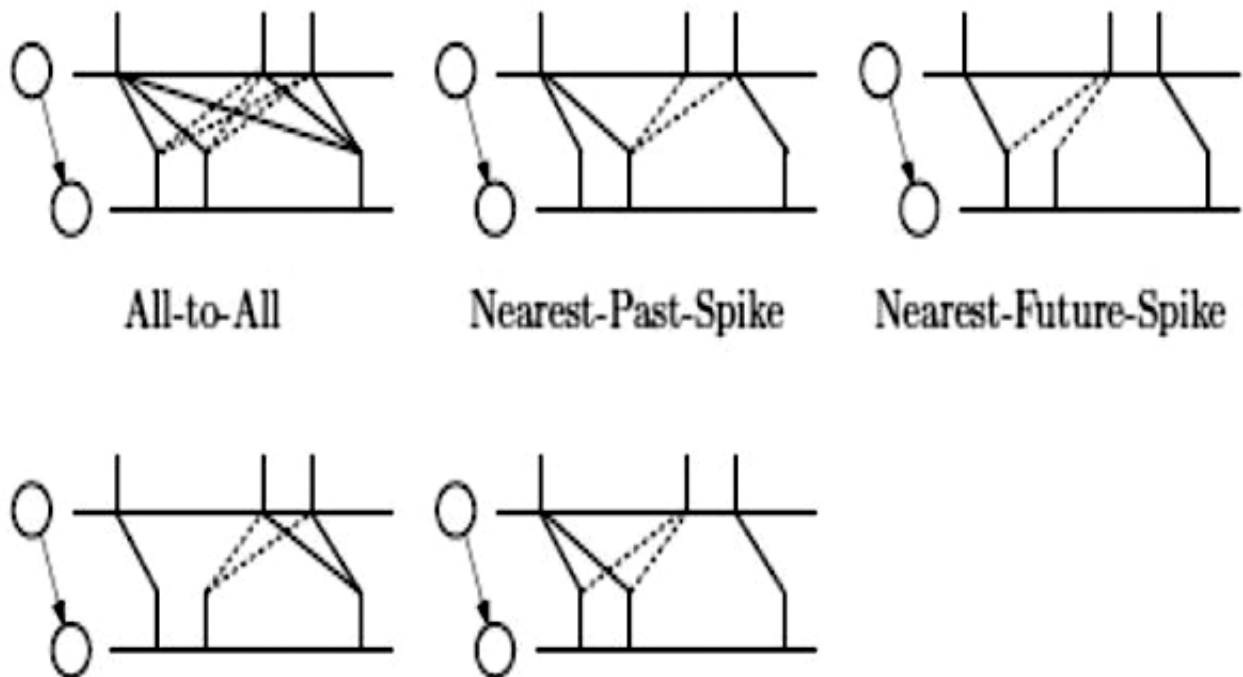


Figure 11: Interaction schemes of pre and postsynaptic trains[36]

5. CONCLUSION

We presented a modular and systematic configurable neuromorphic system with different spiking neuron models and their implementation. Different rules and protocols of spike time dependent plasticity was discussed in detail with their mathematical modeling which can be used for designing and implementing the different STDP protocols.

6. ACKNOWLEDGEMENT

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