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LEARNING STIMULUS-STIMULUS ASSOCIATION IN SPATIO-TEMPORAL NEURAL NETWORKS

N. Yusoff*, F. Kabir Ahmad, N. ChePa, A. Ab Aziz

School of Computing, College of Arts and Sciences, Universiti Utara Malaysia, 06010 UUM Sintok, Kedah, Malaysia

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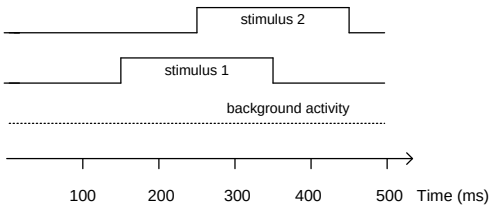
*Corresponding author
nooraini@uum.edu.my

Graphical abstract



Abstract

We propose a stimulus-stimulus association learning by coupling firing rate and precise spike timing encoding for spatio-temporal neural networks. We simulate a generic recurrent network with random and sparse connectivity consisting of Izhikevich spiking neurons. The magnitude of weight adjustment in learning is dependent on pre- and postsynaptic spikes based on their spikes count and time correlation. As a result of learning, synchronisation of activity among inter- and intra-subpopulation neurons demonstrates association between two stimuli. The associations show in spill-over of activity between the two stimuli involved.

Keywords: Associative learning, stimulus-stimulus association, spatio-temporal neural networks, spike-timing dependent plasticity

1.0 INTRODUCTION

There are convincing findings from the neurophysiological experiments that have concluded two types of task-related activity in the brain namely "retrospective" that is related to previously shown stimulus, the recall of past events and "prospective" that is related to a stimulus that the brain expects to appear, e.g. [2-4]. Prospective activity is not triggered directly by external stimuli but could be invoked by activations of other associated events. This indicates the signs of influence of previous information that can facilitate the retrieval of information of a later proceeded stimulus when both are related [5-7, 37]. Such effect as what the psychologists term as the 'spread activation' mechanism in the brain gives us important clues on how relationship between different stimuli could be established through learning with some forms of synaptic plasticity (i.e. learning mechanism) in the brain.

Spatio-temporal Neural Networks or commonly known as the Spiking Neural Networks (SNNs) fall into the third generation models of Artificial Neural Networks (ANNs). In comparison to McCulloch Pitts based models, SNN has more advantages for biological reasonable values of its function parameters, and fast and efficient computation where the timing of input signals carries important information, e.g. [28-29, 31]. From both neuronal network structure and computational properties, learning mechanisms have been improved to closely simulate the features and behaviours of neuron spikes in the brain. Hence, the dynamics of neuronal circuit consisting of spiking neurons with spatio-temporal distribution of spikes have been of interests in most of the recent models. Nevertheless, such spatio-temporal processing pays a tradeoff to complexity of information encoding depending on different neurophysiological parameters. Learning is challenging in a spike based model as neuron may respond differently to even the same repeated

stimuli [1]. Furthermore, individual neurons may show irregular behaviours, hence synchronised activity could provide significant information on particular response encoding and form a basis of associative memory [34-35].

For synaptic plasticity, firing rate has been the standard approach to neural encoding for many years while there are growing evidences that essential information could also be found in the precise timing of spikes [1],[14],[31]. Hence, for learning in the new generation neural network models with spike coding, spike-time dependent plasticity (STDP) [13] can be regarded as a generalisation and refinement of Hebbian learning [14]. In learning with STDP, synaptic efficacy is dependent on the temporal correlation between pre- and postsynaptic neurons.

In STDP based learning, for an unsupervised problem in an SNN, inputs are imposed during training, and the network evolves to a state in which its dynamics determine the output using the current values of the weights. The designed learning algorithms must uncover patterns and synchronicity in the network activity to create causal relationship between triggering input to an interpretable reached desired network state, i.e. the desired output [1]. Meanwhile, in SNN supervised learning, STDP needs to be coupled with appropriate encoding scheme, e.g. [15],[27],[30],[36], [38]. In such learning, synaptic changes are dependent on the direction of the gradient of the timing difference between currently produced output and target spike trains. However, the fidelity of so called 'teacher signals' (i.e. target spike train) operating in the biological network remains a conundrum. As discussed in the review by [20] and [21], there are still questions open to debate; from which part of the brain might such instruction signals come, the consistency of the propagation of error signals with neurobiology and is there such local computation of error terms? It is our concern that, to some extent, a learning algorithm should be biologically plausible. In addition, we as well emphasise on learning with simple mathematical computation.

In this study, we explore the computational significance in combining both firing rate and spike-timing dependent plasticity for learning stimulus-stimulus association. Learning is implemented in a noisy setting using simple learning rules, with no specific spike template required. The spill-over of neuronal activity indicates an association between two stimuli. Such effect reflects the retrospective and prospective activity in the brain. In our simulated network, the associative memory and segmentation of memory patterns are attained via the synchronous periodic firings activity of the neurons population.

2.0 NEURAL NETWORK SIMULATION MODEL

For learning experiments, we simulate a recurrent neural network model, adapted from [10], [11] and [12]. The network is composed of 1000 spiking neurons consisting of 80% excitatory (N_E) and 20% inhibitory (N_I) neurons, following the ratio of pyramidal cells (i.e. excitatory) to interneurons (i.e. inhibitory) in the cortical network [8,9].

Each neuron has random synaptic contacts from 20% of excitatory and 20% of inhibitory neurons. From the population of 800 excitatory neurons, there are groups of stimulus-sensitive neurons (*S*) and the remaining excitatory neurons are non-selective (*NS*) to any stimulus. Neurons 801-1000 are inhibitory (*IH*). Each *S* represents an object for the memory under study, while the inhibitory subpopulation acts as the global network inhibition. The neuronal grouping according to their selectivity properties is based on neurophysiological data, (e.g. [22-26]) from inferotemporal cortex (ITC) or prefrontal cortex (PFC) recordings. Cells that are visually responsive show consistent activation in response to their best stimulus [22]. The schematic diagram of our proposed network model is illustrated in Figure 1.

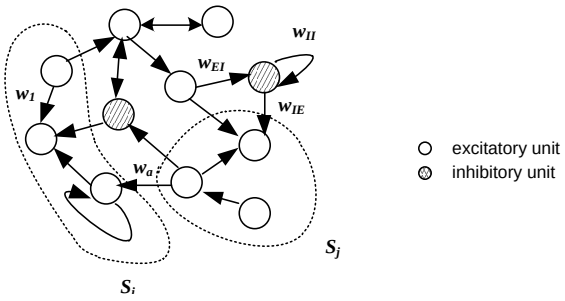


Figure 1 A schematic diagram of a recurrent spiking neural network.

The network consists of 80% excitatory (N_E) neurons and 20% of inhibitory (N_I) neurons, with sparse and random connectivity, p = 0.2. The network comprises of excitatory neurons pool and inhibitory neurons pool. S_i and S_j are subpopulations of excitatory neurons that are selective to a certain object stimulus. The connection strengths of excitatory synapses on excitatory neurons, excitatory synapses on inhibitory neurons, inhibitory synapses on excitatory neurons, and inhibitory synapses on inhibitory neurons, are labelled by $w_{I/a}$, w_{EI}, w_{IE}, *and* w_{II}, respectively. w_I is the synaptic connection within the same subpopulation and w_a is the synaptic connection between two associated subpopulations.

The spiking properties of each neuron are modelled using Izhikevich spiking neuron model (IM) [10], according to (Eq. 1) and (Eq. 2):

$$v' = 0.04v^2 + 5v + 140 - u + I \quad \text{(Eq. 1)}$$

$$u' = a(bv - u) \quad \text{(Eq. 2)}$$

where;

v : Dynamics of membrane potential (v' is the derivation of v over time t)

u : Membrane recovery variable that provides negative feedback to v

I : Synaptic currents (and external currents)

After the spike, v, reaches its peak +30 mV (v_{peak} = +30 mV), the membrane voltage and the recovery variable are reset according to (Eq. 3):

$$\text{if } v \geq +30 \text{ mV, then } u \leftarrow u + d, v \leftarrow c \quad \text{(Eq. 3)}$$

From (Eq. 1) - (Eq. 3), a-d are the model parameters that could lead to particular behaviours of cortical neurons (and also some thalamocortical neurons). The parameter a is the time scale of the recovery variable u, b describes the sensitivity of u to the sub-threshold fluctuations of v, c is the resting potential (in between -70 and -60 mV depending on the value of b), and d is the after-spike reset of the recovery variable u.

In the proposed network model with IM spiking neurons, neurons are classified into two different types of dynamic namely regular spiking (RS) type and fast spiking (FS) type. The excitatory and inhibitory neurons are RS and FS neurons, respectively. The inhibitory neurons with FS dynamics are well suited to suppress some neurons from reaching threshold when stimuli are non-optimal or weak.

3.0 LEARNING SIMULATION METHOD

For leaning simulation, we train a network with the properties described in Section 2.0. The learning protocol is adapted from the neuropsychological experiment in [32]. The learning rules are dependent on firing rate and spike timing.

3.1 Learning Protocol

For learning implementation, in a 500-ms trial, a network is trained to learn a pair of stimuli. The learning trial is run in the following four intervals, see Figure 2.

- *Pre-stimulus*: Each neuron is stimulated with noisy external currents, $\gamma\xi(t)$ pA, where γ is the strength of currents with $\gamma_{N(E)}=3$ and $\gamma_{N(I)}=1$, and $\xi(t)$ is Gaussian noise with $\mu=0$ and $\sigma=1$, injected to neuron *i*. The noisy current reflects the
- thalamocortical input [10] and serves as some background activity with no preferred stimulus.
- *Presentation of the first member of a stimulus pair*: For $t > 150$ to $t \leq 350$ *ms*, we enhance the strength of external currents to the first target stimulus (i.e. target excitatory neurons subpopulation) to $\gamma\zeta_i(t)$ with γ =30 and $\zeta_i(t)$ is the random uniformly distributed current in the range of 0 to 1. The different variability of current is to simulate a stimulus ensemble with enhanced activity in the target neuronal group in the presence of background noise.
- *Stimulus Onset Asynchrony (SOA)*: The delay between the onset of the first stimulus and the onset of the second stimulus.
- *Presentation of the second member of a stimulus pair*: For $t > 250$ to $t \leq 450$ *ms*, we enhance the strength of currents to the second target stimulus (i.e. target excitatory neurons subpopulation) with the same range of currents applied on the first stimulus.

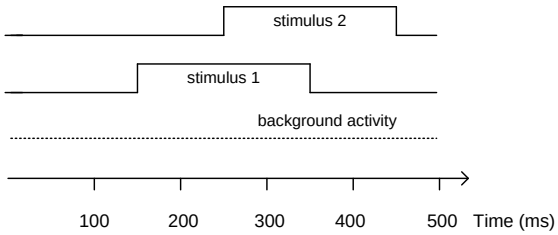


Figure 2 Learning protocol

Within a 500-ms testing window, the network is probed with the learned pairs via stimulation to any of the associated members.

For learning stimulus-stimulus association, in our experiments we define 4 conditions of network activity namely asynchronous activity – neurons in a network fire at random time and there is no target enhanced activity occurs (see Figure 3A), synchronous activity within a subpopulation – neurons in the same group fire in close temporal proximity within a certain time interval (Figure 3B), synchronous activity among inter and intra-subpopulation neurons – neurons from the same group and associated groups fire closely within a certain time interval, in which the activation of neurons in the associated group could be due to some *causal effect* of the *neuronal firing* (Figure 3C), and synchronous activity in the network with co-activation of undesired groups – a network suffers undesired activity due to activation of incongruent pairs (Figure 3D).

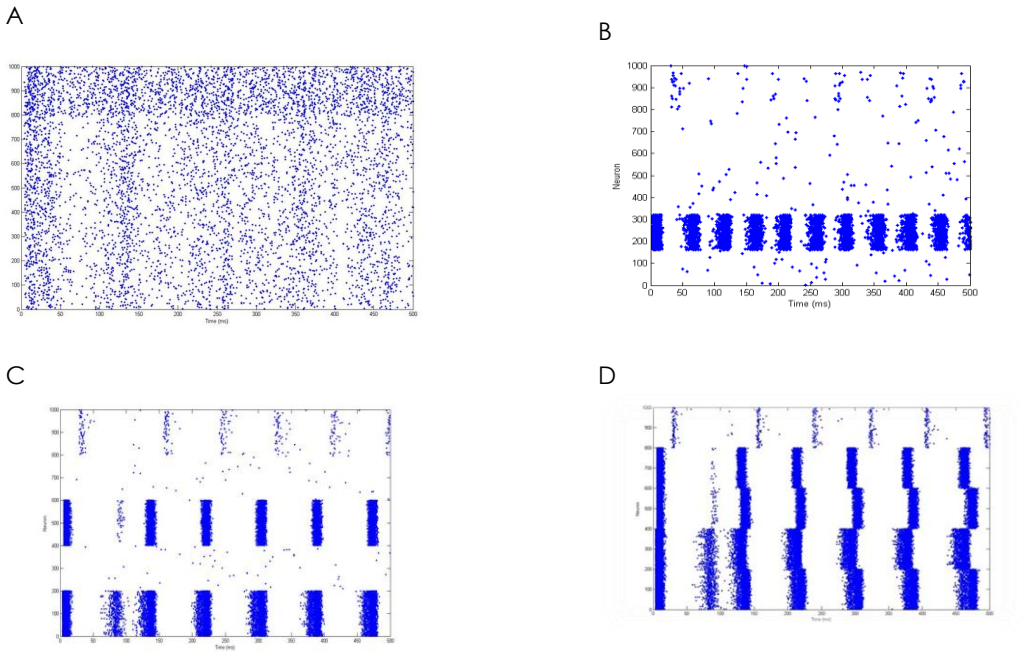


Figure 3 Network activity; (A) asynchronous activity, (B) synchronous activity within a subpopulation, (C) synchronous activity among inter and intra-subpopulationn neurons (i.e. stimuli association), and (D) synchronous activity in the network with co-activation of undesired groups.

3.2 Learning Rules

Learning rules are implemented on excitatory to excitatory synapses only (i.e. $w_{1/a}$), see Figure 1. Other synapses (w_{EI}, w_{IE}, and w_{II}) are set to some optimal values (as in Table 1) derived from a preliminary experiment on a pre-structured network (with fixed range of synaptic weights). In our learning model, inhibitory synapses are not plastic.

Table 1 Initialisation of synaptic connection strengths with ζ, uniformed random values between 0 and 1

Synaptic connection	Coding	Synaptic strength
Excitatory synapses on excitatory neurons (only where initialisation applies)	$w_{1/a}$	ζ
Excitatory synapses on inhibitory neurons	w_{EI}	0.5ζ
Inhibitory synapses on excitatory neurons	w_{IE}	- ζ
Inhibitory synapses on inhibitory neurons	w_{II}	- ζ

Learning is implemented using a semi-supervised approach. The supervision is only through intensification of random external currents to target subpopulations without any specific spike train required as a learning target. The proposed neural encoding is based on an integration of both rate dependent and spike timing of pre- and post-synaptic neurons.

In a learning trial run for 500 ms, the time window is partitioned into a number of overlapping bins with length of 100 ms each (T=100 ms) with ½T increments (Figure 4). For each learning time bin T, we count the number of pre- and post-synaptic spikes, S_{pre} and S_{post}, respectively, as proposed in [12]. We extend their work by incorporating precise spike-time based encoding into our learning rules for more plausible spatio-temporal learning. For brevity, in our approach, weight adjustment is dependent on Δw calculated as a function of time difference (as in [15]), $\Delta t = t_{post} - t_{pre} = t_j^{(f)} - t_i^{(f)}$, where $t_j^{(f)}$ and $t_i^{(f)}$ are the last firing times [14] of post-synaptic neuron j and pre-synaptic neuron i, respectively, within the learning time bin, T (Figure 5).

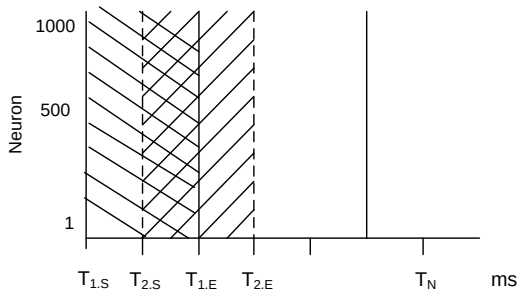


Figure 4 Learning time bins with overlapping window slides, $T_{N.S}$ is a beginning of a time bin which the plasticity ends at $T_{N..E}$ for T=100 ms (i.e. $T_{N..E}$ - $T_{N.S}$) with increment of ½T [12]

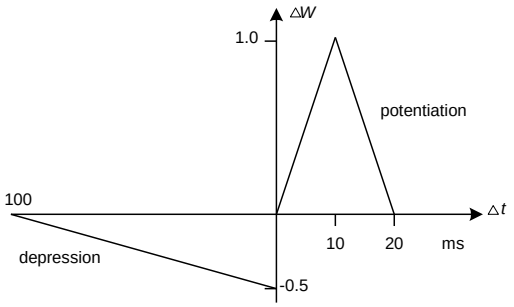


Figure 5 A function of time difference between last firing of pre-, $t_i^{(f)}$, and post synaptic neurons, $t_j^{(f)}$, $\Delta t = t_{post} - t_{pre} = t_j^{(f)} - t_i^{(f)}$, on excitatory neurons; reproduced from [15]

As a combination of the rate dependent and timing based approaches, the synaptic plasticity rules are formulated in (Eq. 4)-(Eq. 6):

i. High potentiation with high threshold, T_+ and maximal synaptic strength, $w_{max} = 3$

$$\text{IF } ((S_{pre} \geq T_+ \text{ AND } S_{post} \geq T_+) \text{ AND } \Delta t \geq 0)$$
$$w_{ij}(t+1) \leftarrow w_{max} \quad \text{(Eq. 4)}$$

ii. Weak potentiation with low threshold, T_a and minimum synaptic strength, $w_{min} = 0$

$$\text{IF } ([(S_{pre} \geq T_+ \text{ AND } T_a < S_{post} < T_+) \text{ OR } (T_a < S_{pre} < T_+ \text{ AND } S_{post} \geq T_+)] \text{ AND } \Delta t > 0)$$
$$w_{ij}(t+1) \leftarrow \max(w_{min}, \min(w_{max}, w_{ij}(t) + \Delta w)) \text{ (Eq. 5)}$$

iii. Depression

$$\text{IF } ([(S_{pre} \geq T_+ \text{ AND } S_{post} \leq T_a) \text{ OR } (S_{post} \geq T_+ \text{ AND } S_{pre} \leq T_a)] \text{ AND } \Delta t < 0)$$
$$w_{ij}(t+1) \leftarrow \max(w_{min}, \min(w_{max}, w_{ij}(t) - |\Delta w|)) \quad \text{(Eq. 6)}$$

For strong weight potentiation in (Eq. 4), a synapse w is set to the maximal synaptic strength (w_{max}=3) if both pre- and postsynaptic neurons fire spikes above the high threshold of spikes count, T_+ and only if the time difference between the pre- and postsynaptic neurons is above 0 ($\Delta t \geq 0$). The w_{max} value is obtained from an initial experiment with a pre-structured network. Meanwhile for weak potentiation in (Eq. 5), the magnitude of weight change Δw is derived from the function of spike time difference (as in Figure 5), if the pre-synaptic neuron fires spikes above T_+ whilst the postsynaptic neuron (or otherwise) fires spikes below T_+ but above the low threshold, T_a. Similarly, in (Eq. 6), the amount of depression is derived from Figure 5, for post-then-pre spikes order (i.e. $\Delta t < 0$). This is applied for the number of pre-synaptic (postsynaptic) spikes above T_+ and the post-synaptic (pre-synaptic) spikes below T_a. To avoid infinite saturation of weight strength values, we keep the weight values within a certain range, 0 to 3, similar approaches of synaptic scaling can also be found in [17] and [19].

To determine the values for T_+ and T_a, we simulate a network with background activity of $\gamma\xi(t)$ where $\gamma_{N(E)}$=5 and $\gamma_{N(I)}$=2, and $\xi(t)$ is Gaussian noise with μ=0 and σ=1 injected into neuron i, randomly for 100 ms. For every ms, we deliver a range of external currents to a single selected excitatory neuron (i.e. RS type neuron). At this stage, no synaptic plasticity is implemented, and weights are initialised according to Table 1. The cut-off value for the high threshold T_+ is calculated as 80% of the averaged spike counts of the stimulated neuron within the 100-ms window, over 100 simulations. Meanwhile, for the low threshold cut-off value T_a, it is 80% above the averaged spike counts of a randomly fired excitatory neuron. For the implementation of synaptic rules, the high threshold T_+ and the low threshold T_a are respectively 5 and 2.

4.0 SIMULATION EXPERIMENTS AND RESULTS

All simulations were implemented in Matlab. The proposed learning rules were applied onto the excitatory-inhibitory network consisting of IM neurons as described in Section 2.

4.1 Preliminary Experiments

We began with a series of preliminary simulations using a simple pre-structured network to investigate the optimal parameters for later use in learning. The parameters included a range of weight values and stimulation to background activity. With the same neuronal connectivity as described in Section 2, we hard coded the association between neurons within the same group, as well as between associated groups. In this mode, all neurons were not plastic as there was no learning (i.e. pre-structured). We tested the pre-structured network with a set of weight values and studied the dynamic properties of our proposed learning model. Next, we implemented the auto-association learning according to (Eq. 4) – (Eq. 6), in which encoding was based on the combination of firing rate dependent and spike-timing dependent plasticity approaches from [15] on a learning task similar to [12]. Learning was performed by pairing two different stimuli through intensified currents to both neuronal group with different onset times. We then observed the synchronisation of network activity within and between subpopulations of neurons.

For every ms in each learning trial, each neuron received background noisy external currents $\xi(t)$, i.e. Gaussian noise with mean μ and standard deviation σ, varied depending on the type of neurons, excitatory or inhibitory where $\sigma_{Ne} > \sigma_{Ni}$. In our simulations, external current to target subpopulation can be classified into three types; Type I – constant currents with probability of 1.0, Type II – uniformly distributed currents in the range of 0 to γpA, and Type III - Gaussian noisy currents with mean μ and standard deviation σ. Examples of the three current types to target excitatory subpopulation are illustrated in Figure 6 A-C.

4.1.1 Maximal Synaptic Strength

Initially we ran an experiment to find an appropriate range of strength of excitatory synapses on excitatory neurons $w_{I/a}$. This phase was necessary for further learning experiments in order to avoid weight saturation to infinite values. In our implementation, other connections, excitatory synapses on inhibitory neurons w_{EI}, inhibitory synapses on excitatory neurons w_{IE}, and inhibitory synapses on inhibitory neurons w_{II}, were set to random values with moduli drawn uniformly the range of 0 to 1 (Table 1), and with signs of connections depending on the type of the neuron (excitatory or inhibitory).

In the experiment with a pre-structured network, the task was to recall an associative memory $S^m(t)$ in response to a presented stimulus $S_i(t)$. The recall was simulated over 500 ms. At times $150 < t \leq 350$ ms, the network was stimulated with an amount of current into a target excitatory subpopulation with Type I, Type II and Type III currents. The fraction of neurons within a group to be stimulated was p=0.75, selected randomly (with uniform distribution). In order to minimise the influence of irrelevant network activity, and only to see the effect of synaptic strength, no background current was present at this stage of experiment.

To find the optimal values of synaptic connections between excitatory neurons among the same subpopulation and two associated subpopulations, we varied the values of $w_{I/a}$ into five different uniformed distributions in the ranges of 0 to 1, 0 to 2, 0 to 3, 0 to 4 and 0 to 5. The network showed no consistent synchronisation of activity with the maximal value of connection strength $w_{max} < 3$ (e.g. Figure 7). In the 500-ms simulation window, neurons only fired asynchronously. Performance of memory recall was only improved with more synchronised activity when $3 \leq w_{max} \leq 5$ (e.g. Figure 8).

The higher range of synaptic scale can cause a network to become more sensitive to external currents. This is to say, only a small number of neurons need to be stimulated in order to have synchronised activity. Nevertheless, the synaptic connection values should not grow infinitively. This is to avoid the network from being too sensitive to any irrelevant noise signal. For this, we have chosen the range of 0 to 3, as the appropriate range of synaptic strengths. With the chosen range, the network shows some synchronised activity with constant current as well as fair performance for noisy currents, i.e. Type II and Type III currents. For a network with noisy currents, the activity could be improved with some other optimal current distribution. In addition to the w_{max} value, depression of excitatory synapses should also be controlled thus the value of $w_{min} < 0$ is to be avoided. This is crucial for plausibility aspect of the network model, not to violate the neurophysiology laws, i.e. excitatory synapses with inhibition effect (negative strength) [22]. In our experiment, we considered w_{min}= 0.

4.1.2 Background Activity

In the following experiment, we examined the effect of background current to network activity. The purpose of the experiment was to find an appropriate range of background current for learning simulations. At this stage, the network was a pre-structured network with fixed connection strengths in between 0 and 3 obtained from the previous experiment (as discussed in Section 4.1.1).

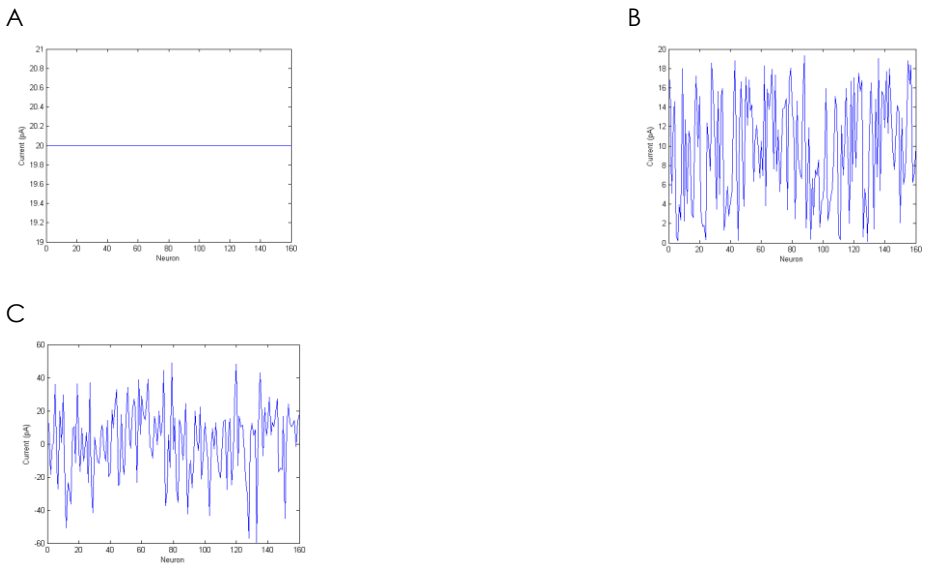


Figure 6 Current stimulation of (A) Type I to an excitatory subpopulation having constant current with probability of 1.0 e.g. 20 pA, (B) Type II to an excitatory subpopulation with uniform distribution from 0 to □□pA, e.g. □=20,and (C) Type III to an excitatory subpopulation having Gaussian distribution with mean □□and stdev □, e.g. □,$_{Ne}$=20

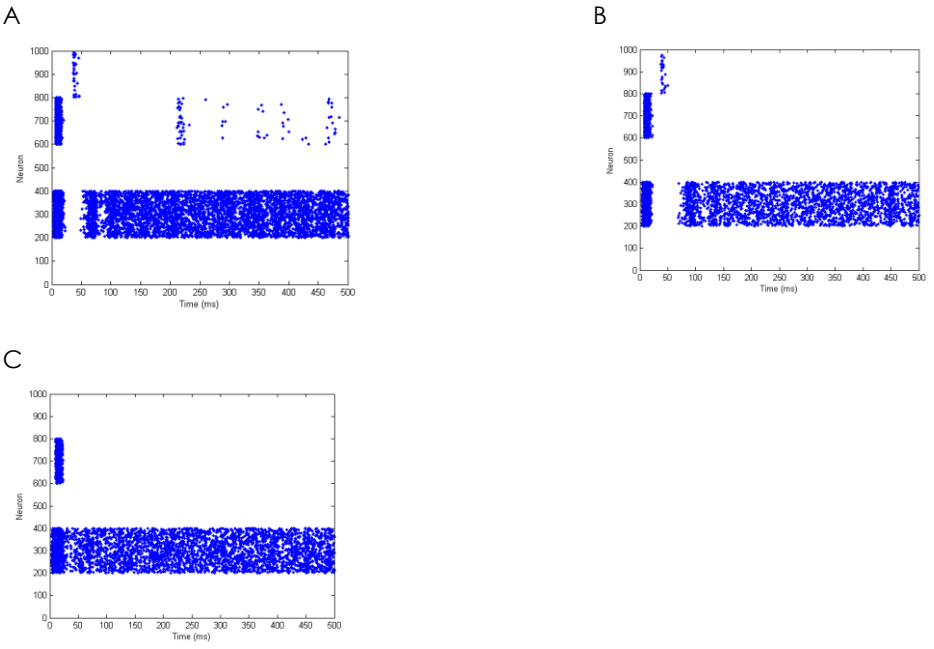


Figure 7 Neuronal network activity in groups S_1 (neurons: 201-400) and S_3(neurons: 601-800) for association of $S_1 \leftrightarrow S_3$ with uniformly distributed $w_{1/a}$ values between 0 and 2 when stimulated with A) Type I, i.e. 20 pA, B) Type II, i.e. □□=20 pA, and C) Type III i.e. □=20 pA currents

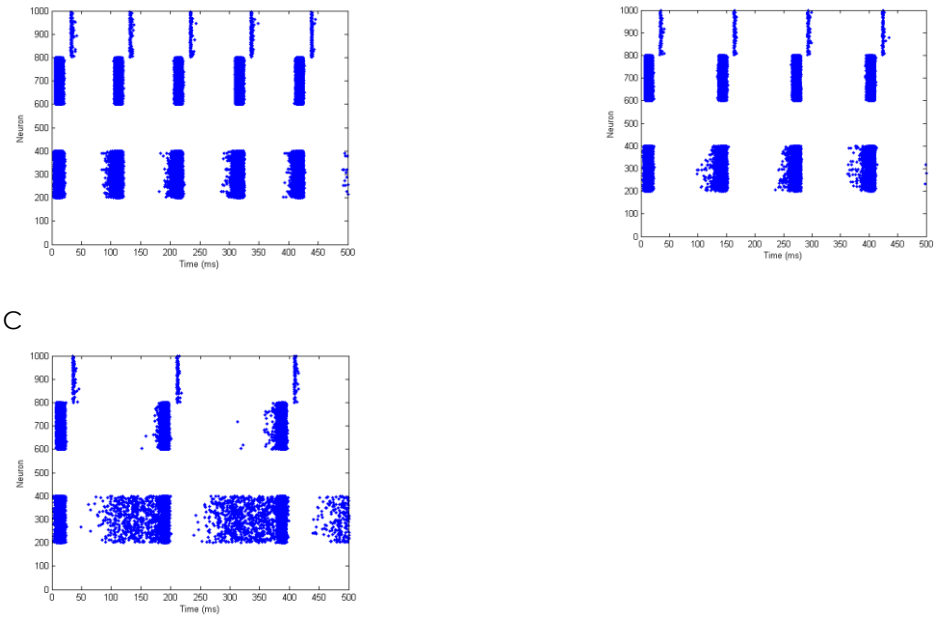


Figure 8: Neuronal network activity in groups S_1 (neurons: 201-400) and S_3 (neurons: 601-800) for association of $S_1 \leftrightarrow S_3$ with uniformly distributed $w_{1/a}$ values between 0 and 4 when stimulated with A) Type I, i.e. 20 pA, B) Type II, i.e. □□=20 pA, and C) Type III i.e. □=20 pA currents

The background activity enriches the dynamics of a network simulating the processing of noisy and chaotic signals in the brain. Moderate level of background activity is important to give balance to the network excitation and inhibition. In an associative memory network, learning with too high background activity level could lead to an interference effect as the network could suffer too much excitation, and a network with low level noise-tolerance is not efficient. Moreover, a noisy input signal is used to model the noisy thalamocortical input.

In our experiments, for background activity, both excitatory and inhibitory neurons received Type III current where $\sigma_{Ne} > \sigma_{Ni}$. We varied the standard deviation σ (mean $\mu=0$) started with a ratio of σ_{Ne}: σ_{Ni} = 5:2 [10]. For memory recall tasks, the excitatory target subpopulation was stimulated with Type II currents in the range of 0 to 30 pA. The networks with corresponding background activities are exhibited in Figure 9.

We have studied four ratios of σ_{Ne}: σ_{Ni} i.e. 5:2, 4:1.6, 3.5: 1.4 and 3:1.2. We observed that, with the background activity (Figure 9A-D Left), when $\sigma_{Ne} \geq 4$ and $\sigma_{Ni} \geq 1.6$, the network seemed to have synchronised pattern with unknown event. Furthermore, when we performed memory recall on S_1 for association $S_1 \leftrightarrow S_3$ (i.e. S_1: neurons 161-320, S_3: neurons 481-640) (Figure 9A-D Right), there were undesired activations of patterns obviously found for $\sigma_{Ne}= 5$, $\sigma_{Ni}= 2$. Meanwhile for $\sigma_{Ne}= 4$, $\sigma_{Ni}= 1.6$, activation of relevant pattern seemed to be distracted. The network performed well for memory recall task when $\sigma_{Ne} \leq 3.5$ and $\sigma_{Ni} \leq 1.4$. Therefore, here we selected the maximum of standard deviation σ for the Gaussian distributed external current to be σ_{Ne} = 3.5 and σ_{Ni} = 1.4.

4.2 Pair-Associate Learning

For our learning simulations, excitatory and inhibitory neurons received external currents with standard deviations $\sigma_{Ne}=3.5$ and $\sigma_{Ni}=1.4$, respectively (i.e. obtained from the findings in Section 4.1.2). There were four subpopulations of excitatory neurons, S_0 (neurons: 1-160), S_1 (neurons: 161-320), S_2 (neurons: 321-480) and S_3 (neurons: 481-600), each represented a stimulus. We trained the network to learn associations of $S_0 \leftrightarrow S_2$ and $S_1 \leftrightarrow S_3$.

For learning the association of $S_0 \leftrightarrow S_2$, during a learning trial, for $t>150$ to $t\leq350$ ms, the external current distribution to target stimulus subpopulation S_0 was changed to a uniform one from range 0 to γ with γ=30. This corresponded to a stimulus (e.g. visual) presentation with intensity of $0 < \gamma \leq 30$ for 200 ms in the presence of the transient background input. Then, for $t>250$ to $t\leq450$ ms, the target stimulus subpopulation S_2 was stimulated with the same range of currents as its subpopulation to be associated. For the implementation of synaptic rules (4-6), the high threshold T_+ and the low threshold T_a were respectively 5 and 2, obtained from a preliminary experiment (as discussed in Section 3.1).

For learning initialisation, 20% of neurons within the same subpopulation were initialised with w_1 values in the range of 0 and 1. The initial values of w_1 represent some random connectivity assumed to be the result from any previous learning. Nevertheless, the initialised synaptic connections were not sufficient to have synchronous activity. Results of association learning with stimuli S_0 and S_2 are depicted in Figure 10.

From Figure 10, during the early phase of learning, after stimulations to S_0 and S_2, the neurons in both subpopulations only fire asynchronously caused by the injected current within $t>150$ to $t\leq350$ ms and $t>250$ to $t\leq$ 450 ms on S_0 and S_2, respectively. A spill-over of activity from S_0 to S_2 and vice-versa could be observed after ten trials. Results of memory recall are exhibited in Figure 11.

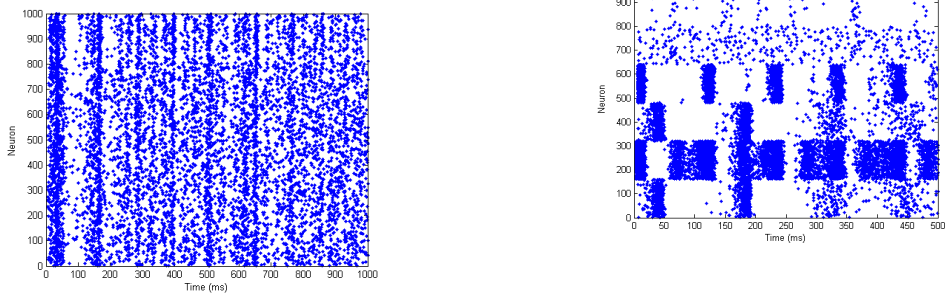
From the results of memory recall, it showed that the network had learned each stimulus. The association of the stimulus pair e.g. $S_0 \leftrightarrow S_2$, was shown by synchronous activity among neurons in the same subpopulations and associated subpopulations in comparison with the recall to unlearned and non-associated stimuli as depicted in Figure 11B. By frequently stimulating S_0 and S_2 pair, and firings that follow the pre-then-post order rule of the STDP, the synaptic connections from those 160 neurons in the paired groups to the fired postsynaptic neurons become eligible for potentiation. However, the connections are strengthened only if both pre- and postsynaptic neurons emit a significant amount of spikes according to (4)-(6) in each 100-ms learning bin for every trial runs for 500 ms. In the case of undesired coincident activation that could probably trigger a small potentiation, due to random spikes (i.e. post-then-pre) and long-term depression (LTD) window of STDP is greater that the long-term potentiation (LTP) window, the amount of the potentiation is compensated by the STDP depression mechanism (Figure 5). Even though, we could observe some co-activation of neurons from the non-associated group during a memory recall (e.g. Figure 11A), we conjecture that could happen only by chance. This also evident by the memory recall to the unlearned stimuli when stimulated with strong pulse of current that resulted in no activation of any neurons ensemble.

5.0 CONCLUSION

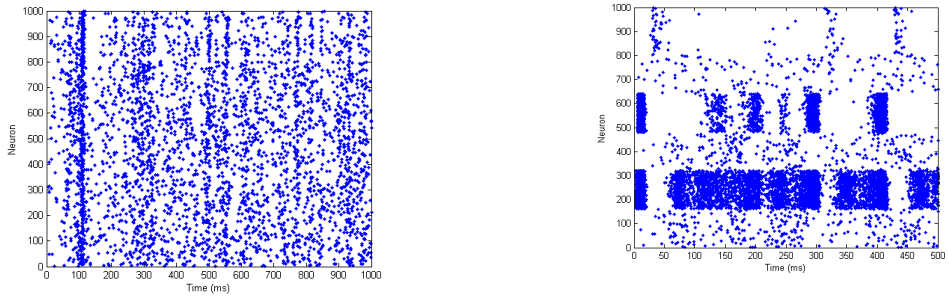
We propose a simple stimulus-stimulus association learning in Hebbian paradigm using both spike rate and precise timing encoding for synaptic plasticity. Our model is inspired by the work in [12] and [15]. The learning rules incorporate pre- and postsynaptic spikes count and time correlation within a window

slide, and the amount of weight change is determined from a function of time difference.

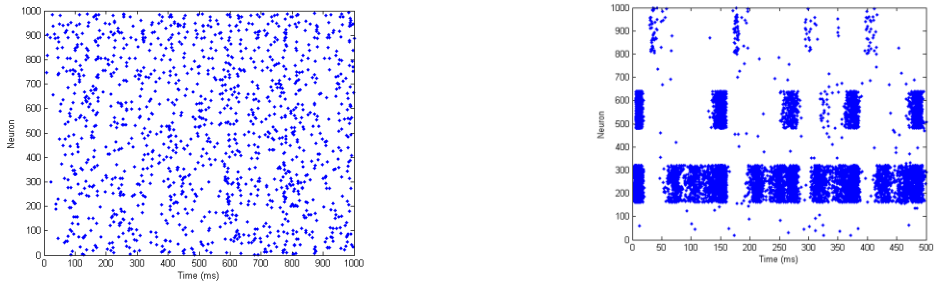
A $\sigma_{Ne}= 5,\ \sigma_{Ni}= 2$



B $\sigma_{Ne}= 4,\ \sigma_{Ni}= 1.6$



C $\sigma_{Ne}= 3.5,\ \sigma_{Ni}= 1.4$



D $\sigma_{Ne}= 3,\ \sigma_{Ni}= 1.2$

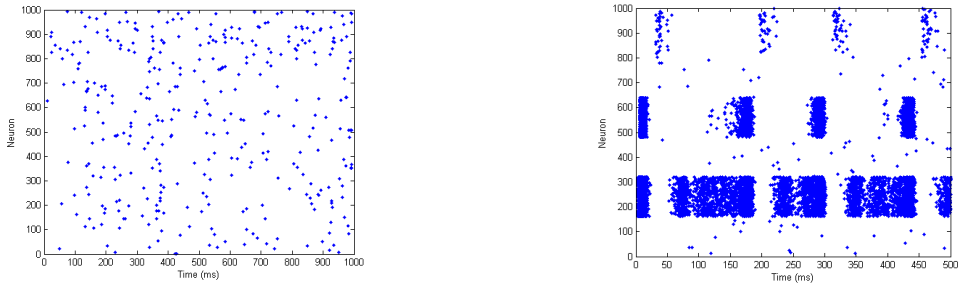


Figure 9: Network with background activity for 4 (A-D) different ratios of σNe:σNi (Left: background activity with noisy currents, mean μ and stdv=σ, Right: recall to target subpopulation S1: neurons 161-320, with corresponding background activity)

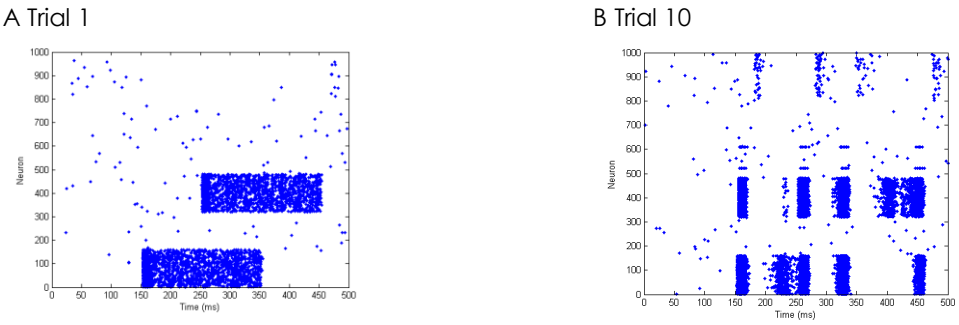


Figure 10: Neuronal network activity after one and ten learning trials for stimulus pair S_0 □ S_2. Currents to excitatory subpopulation neurons of S_0 (neurons: 1-160) is intensified for 200 ms (*t>150 to t□□ 350 ms*), then S_2 (neurons: 321-480) is stimulated for the same duration (*t>250 to t□□ 450 ms*). (A) In the early phase of learning, after one trial, neurons in subpopulations S_0 and S_2 fired asynchronously as both stimuli are novel and activity are only dependent on the external currents. (B) After ten trials, neuronal activity within each subpopulation is more synchronised as the result of learning. Activation of S_2 (within *t>150 to t<= 250 ms*) and activation of S_0 (within *t>400 to t□□ 500 ms*) indicate association of S_0 □ S_2

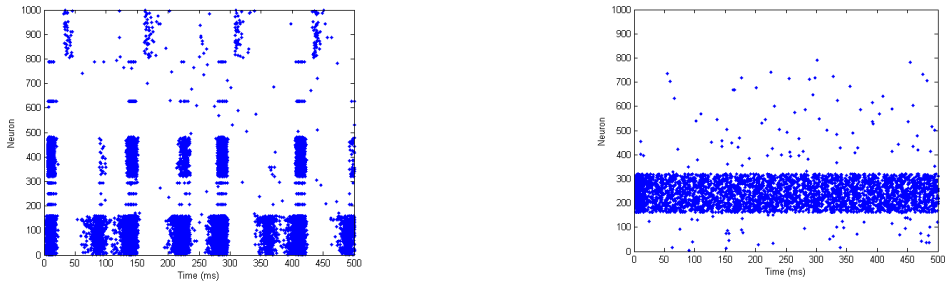


Figure 11: Pattern recall after an associative learning with intensified current to target subpopulation. (A) Recall to learned and associated stimulus pair, S_0 □ S_2. Stimulation of S_0 (neurons: 1-160) triggers activation of S_2 (neurons: 321-480). (B) Recall to novel and non-associated stimuli (unlearned)

In [12], the weight adjustment is dependent on a range of probability values estimated based on the pre- and postsynaptic spikes. In ours, the magnitude of the weight change is based on time correlation between the pre- and postsynaptic spikes. The latter approach has a plausibility advantage in using the precise spike timing as one of the key properties in spatio-temporal neural network. Furthermore, it is also unknown if different parts of the brain may execute different encoding strategies. As neurons work collaboratively to perform a cognitive function [16], [18], we anticipate that the neuronal activity may result from the interaction between the process at local synapses and global network activity derived from the precise spike timing and accumulative firing rate, respectively. Hence, the contribution of our learning model can be attributed to the application of the proposed learning rules using integration of spike-timing dependent plasticity and firing rate in spatio-temporal neural networks, with Izhikevich's spiking neurons. We adapt the proposed learning protocol as suggested in [12] inspired by the popular behavioural GO/NO-GO experiment by Erickson and Desimone [32]. In particular, we implement learning in a spiking neural network in a different experiment paradigm in comparison to learning using the leaky-and-integrate (LIF) neuron properties in [12] (a comprehensive review of spiking neuron model can be found in [33].

To our knowledge, there is no yet such model in the literature. The synchronicity of activity found in a network trained with the visual stimulus-stimulus model demonstrates that it is practical for association tasks. Furthermore, the synchronisation of firing within the same neuronal group simulates the retrospective activity in human cognition as a result of stronger synaptic connections derived from a learning process. This is to say a recall to an individual stimulus. Meanwhile, the spread activation to an associated group demonstrates prospective activity triggered by a recall to another stimulus.

In most goal-directed learning (i.e. supervised) in spatio-temporal neural networks, the objective of learning is to minimise the error between the desired and output spike timings. Even though learning has been proven successfully, it remains unclear on the biophysical mechanism of such synaptic plasticity in the brain [20], [21]. In the defined protocol, we implement learning in a more natural way without any spike template as a learning target.

However, there are some aspects of our learning model could be improved. The model is not feasible to train temporal sequence since its algorithm only performs auto-associative learning. For example once an association of paired stimuli has been obtained, triggering any member of the pair would also invoke the other member. Hence, the model is well-advised for pattern completion problem. For learning temporal sequences, it requires response groups to discriminate representations of stimuli.

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