

Supervised Associative Learning in Spiking Neural Network

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Abstract. In this paper, we propose a simple supervised associative learning approach for spiking neural networks. In an excitatory-inhibitory network paradigm with Izhikevich spiking neurons, synaptic plasticity is implemented on excitatory to excitatory synapses dependent on both spike emission rates and spike timings. As results of learning, the network is able to associate not just familiar stimuli but also novel stimuli observed through synchronised activity within the same subpopulation and between two associated subpopulations.

Keywords: Spiking neural network, Associative learning, Supervised learning, Excitatory-Inhibitory network, Izhikevich spiking neurons.

1 Introduction

There is evidence in neurophysiology that long-term association between stimuli, which involves synaptic plasticity, is triggered by overlapping short-term activity, which only involves activity dynamics (e.g. [1], [7] and [8]), linking neuronal activity and long-term memory.

Associative-based learning can be implemented using unsupervised or supervised approaches [6]. For unsupervised learning, perhaps the temporal variant of Hebbian learning known as spike-timing dependent plasticity (STDP) is the most biologically plausible approach. However, the purely unsupervised approach is often not suitable for goal-oriented applications, so it is used in this paper with prescribed target stimuli as a form of supervision. Such supervisory signal could be assumed to come from another part of the brain [2].

In this study, we explore a supervised associative learning algorithm as a combination of spike emission rate dependent and STDP approaches from [9] on a learning task similar to [8]. Supervision in learning is only through intensified currents into paired target neuron subpopulations. Learning is performed by associating two different stimuli with synchronisation of network activity within and between subpopulations of neurons as the key measure of stimulus association.

2 Simulation Model

For our simulation, the network structure is an adaptation of excitatory-inhibitory neural network model similar to [7]-[8]. The neuron model used with simple computational properties is based on the Izhikevich spiking neuron (IM) with standard parameters governing the dynamics of membrane potential (further details of the IM can be found in [3] and [4]).

The network is composed of 1000 neurons ($N=1000$) with 800 excitatory neurons ($N_E=800$) and 200 inhibitory neurons ($N_I=200$). Each neuron receives synaptic contacts from 20% of excitatory neurons ($C_E=0.2N_E$) and 20% of inhibitory neurons ($C_I=0.2N_I$), randomly. The excitatory neurons population is divided into subpopulations that each represents an object for the memory under study, meanwhile the inhibitory subpopulation acts as the global network inhibition. In our simulations, there are four ($p=4$, P1-P4) subpopulations of excitatory neurons with 160 (selectivity, $f=0.2 \rightarrow fN_E=160$) units each with the following allocation: P1: neurons 1-160, P2: neurons 161-320, P3: neurons 321-480 and P4: neurons 481-640, while neurons from 801-1000 are inhibitory and the remaining excitatory neurons 641-800 are the non-selective pool of neurons. The connection strengths of excitatory synapses on excitatory neurons are denoted $W_{I/O/a}$, excitatory on inhibitory neurons W_{EI} , inhibitory on excitatory neurons W_{IE} , and inhibitory on inhibitory W_{II} . Within $W_{I/O/a}$, W_I are the synaptic connections within the same subpopulation, W_a is the synaptic connection between two associated subpopulations and W_o labels the non-associated subpopulation.

3 Learning Implementation

For our simulation experiments, learning is performed through implementation of synaptic plasticity on excitatory to excitatory synapses ($W_{I/O/a}$). Other synapses (W_{EI} , W_{IE} , and W_{II}) are set to random values with moduli drawn uniformly the range from between 0 and 1 and with signs of connections depending on the type of the neuron (excitatory or inhibitory).

Learning is implemented in a Hebbian paradigm, considering both spike rate and timings of both pre-synaptic and post-synaptic neurons in a learning window [9]. In a learning trial with 500 milliseconds (ms) simulated time, the time window is divided into 100 ms ($T=100$) wide overlapping bins at 50 ms intervals (Fig. 1). For each learning time bin, the average spike rate of every excitatory neuron (S_{pre} and S_{post}) is estimated as the ratio of the number of spikes emitted in the bin divided by T [8]. The weight adjustments, ΔW are calculated as a function of time difference, $\Delta t = t_j^{(j)} - t_i^{(i)}$, where $t_j^{(j)}$ and $t_i^{(i)}$ are the last firing times of post-synaptic neuron j and pre-synaptic neuron i , respectively, within the learning time bin (Fig. 2) [9]. To avoid saturation of synaptic strength values infinitely, we keep the values within the range 0 to 3.

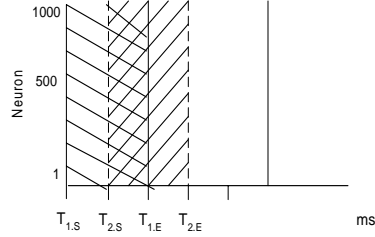


Fig. 1. Learning time bins with overlapping windows, $T_{N,S}$ is the beginning of a time bin which ends at $T_{N,E}$ with $T_{N,E} - T_{N,S} = 100$ ms, and $T_{N,S}$ increasing in steps of 50 ms [8].

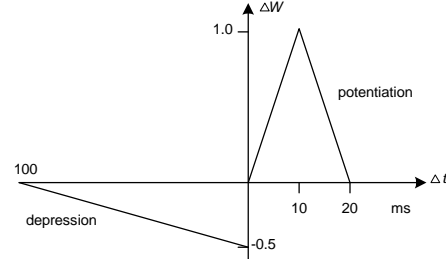


Fig. 2. A function of time difference between last firing of pre-, $t_i^{(j)}$, and post synaptic neuron, $t_j^{(j)}$, $\Delta t = t_{post} - t_{pre} = t_j^{(j)} - t_i^{(j)}$, on excitatory neurons [9], Fig. 2.

A synapse W is highly potentiated (if $W(t) = 0$) to the maximal synaptic strength ($w_{max} = 3$) if both pre- and postsynaptic neurons emit spikes above the high rate threshold T_+ and only if the time difference of the last firing between the pre- and postsynaptic neurons is above 0 ($\Delta t > 0$). W is weakly potentiated with an amount of ΔW (derived from Fig. 2), if the pre synaptic (postsynaptic) neuron emits spikes with rate above T_+ whilst the postsynaptic (pre synaptic) neuron spike emission rate is below T_+ but above the low threshold, T_a . For depression of W , where $\Delta t < 0$ from Fig. 2, it is applied if the pre synaptic (postsynaptic) neuron emits spikes above T_+ and the post-synaptic (pre synaptic) neuron emits spikes below T_a . The synaptic plasticity rules are summarised in 1-3.

$$W_{ij}(t+1) = \begin{cases} w_{max}, & W_{ij}(t) = 0, (S_{pre} \geq T_+, S_{post} \geq T_+), \Delta t > 0 & (1) \\ \max(w_{min}, \min(w_{max}, W_{ij}(t) + \Delta W)), & [(S_{pre} \geq T_+, T_a < S_{post} < T_+); \\ & (T_a < S_{pre} < T_+, S_{post} \geq T_+)], \Delta t > 0 & (2) \\ \max(w_{min}, \min(w_{max}, W_{ij}(t) - |\Delta W|)), & [(S_{pre} \geq T_+, S_{post} \leq T_a); \\ & (S_{post} \geq T_+, S_{pre} \leq T_a)], \Delta t < 0 & (3) \end{cases}$$

4 Simulation Results

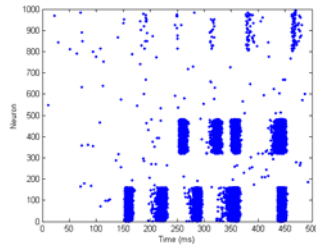
In our simulation, for every ms in each trial, each neuron receives background noisy external currents $\xi_i(t)$, where $\xi_i(t)$ is Gaussian noise with mean μ and stdev σ . Excitatory and inhibitory neurons receive external currents with standard deviations $\sigma_{Ne}=3$ and $\sigma_{Ni}=1$, respectively. During a learning trial, for $t > 150$ to $t \leq 350$ ms, the external current distribution to target stimulus subpopulation 1 is changed to a uniform one from range 0 to γ with $\gamma=30$. Then, for $t > 250$ to $t \leq 450$ ms, the target stimulus subpopulation 2 is stimulated with the same range of currents as its subpopulation to be associated. We ran two batches of simulations: 1) learning with familiar stimuli and 2) learning with novel stimuli. For (1), a stimulus is assumed to

have been learned prior to establishing relationship between two different stimuli, while for (2) only a small subset of synapses are initialised with some strength values.

4.1 Learning with Familiar Stimuli

For implementing associative learning with familiar stimulus, neurons in the same subpopulation are connected with a set of random W_i values in the range of 0 and 3. With such pre-initialised synaptic connections, neurons in the same subpopulation always fire synchronously. An example of associative learning results between two stimuli, P1 and P3 is depicted in Fig. 3.

A. Trial 1



B. Trial 2

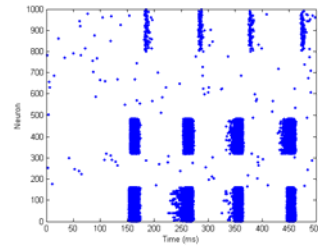


Fig. 3. Synchronisation of neuronal network activity after two learning trials for a pair of familiar stimuli $P1 \leftrightarrow P3$. Currents to excitatory subpopulation neurons of P1 (neurons: 1-160) are intensified for 200 ms ($t > 150$ to $t \leq 350$ ms), then P3 (neurons: 321-480) is stimulated for the same duration ($t > 250$ to $t \leq 450$ ms). Association of patterns is measured based on synchronous activity in each member of a stimuli learning pair subpopulation. A) Learning trial 1: activation of P1 is observed in between 350 to 450 ms, B) Learning trial 2: activation of P3 (in between $t > 150$ to $t \leq 250$ ms) and activation of P1 (in between $t > 350$ to $t \leq 450$ ms) as the results of pattern association.

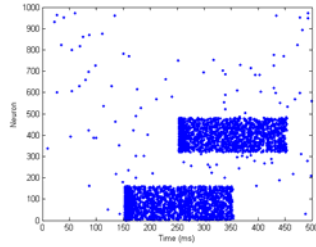
After two trials, association of $P1 \leftrightarrow P3$ could be established. Initially, intensified currents to subpopulations P1 and P3 activate their respective subpopulation only. Then, at times $350 < t \leq 450$ ms, there exists prolonged activity of P1 by activation through P3. In trial 2, prospective activity can be observed with activation of P3 within stimulation period of P1 (in $150 < t \leq 250$ ms) and activation of P1 within stimulation period of P3 (in $350 < t \leq 450$ ms).

4.2 Learning with Novel Stimuli

For learning with novel stimuli, only 20% of neurons within the same subpopulation are initialised with W_i values in the range of 0 and 1. The initial values of W_i represent some random connectivity assumed to result from any previous learning. Initially, in our simulation, the so initialised synaptic connections are not enough to have synchronous activity within a subpopulation compared to when learning with

familiar stimuli. Results of association learning with novel stimuli P1 and P3 are depicted in Fig. 4.

A. Trial 1



B. Trial 10

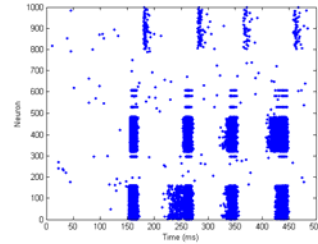


Fig. 4. Neuronal network activity after one and ten learning trials for stimuli pair $P1 \leftrightarrow P3$. Currents to excitatory subpopulation neurons of P1 is intensified for 200 ms ($t > 150$ to $t \leq 350$ ms), then P3 is stimulated for the same duration ($t > 250$ to $t \leq 450$ ms). A) In the early phase of learning, after one trial, neurons in subpopulations P1 and P3 fire 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 P3 (within $t > 150$ to $t \leq 250$ ms) and activation of P1 (within $t > 400$ to $t \leq 500$ ms) indicate association of $P1 \leftrightarrow P3$.

From Fig. 4, during the early phase of learning, after stimulations to P1 and P3, the neurons in both subpopulations only fire asynchronously caused by the injected current within $t > 150$ to $t \leq 350$ ms and $t > 250$ to $t \leq 450$ ms for P1 and P3, respectively. A spill-over of activity from P1 to P3 and vice-versa can only be observed after ten trials.

5 Conclusion

We have explored a simple associative learning scheme utilising Hebbian learning both for spike rates and timings (STDP) for synaptic plasticity, similar to [9]. Unlike other supervised approaches [5] where neuronal activity is forced to have relatively precise spike timing to match the desired target spike train, this scheme uses supervisory currents to establish an association between two stimuli. And unlike previous approaches [8], that only rely on sliding average spike rates, our approach has a plausibility advantage by incorporating spike timings, too [9]. However, it remains to examine whether a single branch (1), (2) or (3) of the weight update rule has a dominating affect on learning. The associations show in spill-over of activity between the two stimuli involved. This demonstrates once more that long-term associations between stimuli involving synaptic plasticity are triggered by overlapping short-term activity involving only short-term activity dynamics. We have run a series of simulation experiments for learning associations of familiar stimuli and

novel stimuli. For learning with familiar stimuli, associations between pair patterns are learned faster compared to novel stimuli.

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