NEURAL PROCESSING OF LONG LASTING SEQUENCES OF TEMPORAL CODES

Model of Artificial Neural Network based on a Spike Timing-dependant Learning Rule

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Abstract: It has been demonstrated, that spike-timing-dependent plasticity (STDP) learning rule can be applied to train neuron to become selective to a spatiotemporal spike pattern. In this paper, we propose a model of neural network that is capable of memorizing prolonged sequences of different spike patterns and learn aggregated data in a larger temporal window.

1 INTRODUCTION

There are strong experimental evidences that at least some living neural systems exchange information in almost binary fashion, in so called temporal spike codes (Prut et al., 1998; Gerstner and Kistler, 2002; Fellous et al., 2004; VanRullen et al., 2005, Kayser et al., 2009). Underlying concept of temporal coding states that precise spike timing encodes the information processed by neurons. It is an alternative to an established concept of rate coding, when count of spikes in certain time window encodes the information. There are known prominent models of neural networks based on rate coding, such as networks based on Bienenstock-Cooper-Munro (BCM) theory (Bienenstock et al., 1982). However, there are evidences that rate coding alone cannot account for the efficiency of information transmission in some biological neural systems (Gerstner et al., 1996; VanRullen and Thorpe, 2001).

Discovery of spike timing-dependant plasticity (STDP) learning rules strongly advocates in favor of temporal coding. Some researches refer to STDP as Hebbian learning, although STDP do not exactly fit Hebbian postulate. STDP learning rules are well established biological processes that guard amount of synaptic strength change depending on time difference between incoming (presynaptic) and outgoing (postsynaptic) spikes. There has been discovered a number of different STDP rules. STDP rules vary depending on synapse type or even on a position on a dendrite (Bi and Poo, 1998; Woodin et al., 2003; Abbott and Nelson, 2000; Caporale and Dan, 2008).

STDP learning rule that is common to the excitatory-to-excitatory synapses, in a certain range of parameters perfectly fits for training of neurons to respond to a repeated temporal code. There is an experimental evidence that pyramidal neurons of rat operates in this range (Feldman, 2000). In this case, the neuron trained with this STDP rule acts as a coincidence detector (Abbott & Nelson, 2000). Unsupervised learning of temporal codes by applying STDP training has been already explored by the number of authors (Masquelier et al., 2008, 2009; Song et al., 2000; Guyonneau et al., 2005; Gerstner and Kistler, 2002).

We focus our research on temporal coding and STDP learning rule.

In a recent paper Masquelier et al. (Masquelier et al., 2009) demonstrated winner-takes-all (WTA) artificial neural network that is capable of learning multiple spatiotemporal patterns in a noisy environment. However, such model is capable of learning only very short patterns in order of a few milliseconds. Although Masquelier experimented with 50ms length training sample patterns, neurons eventually learned only the very beginning of the pattern or a later part of it if the beginning was

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occupied by competing neuron. We executed a similar experiment and found that neurons became selective only for 1 or 2 milliseconds of the pattern. The rest of the pattern could be removed or replaced with a different pattern without any changes in a neuron selectivity.

It is evident that central neural systems of humans and many other advanced species are capable of learning long lasting patterns of sensory inputs, such as speech signals, observed motion patterns etc. If STDP training leads neurons to learning of coincidences of spikes in a window of a few milliseconds, then how is it possible for neurons to learn long lasting pattern of dynamic of sensory input? In other words, how would we train neurons to learn patterns of occurrences of different temporal codes?

In this paper we propose the model of unsupervised artificial neural network with STDP training rule that is capable of learning prolonged sequences of different short spatiotemporal patterns.

The model represents itself a combination of two WTA layers that are similar to the one demonstrated by Masquelier et al. (Masquelier et al., 2009) and inner layers for temporal memory and temporal modulation.

In the early stages of research we did not seek to achieve high biological realism, rather created a model that serves as a proof a concept that known STDP rules alone can lead to learning of long lasting combinations of spatiotemporal patterns.

2 UNDERLYING BIOLOGICAL MECHANISMS

2.1 Leaky Integrate-and-fire Neuron

In this section we provide mathematical model of leaky integrate-and-fire neuron that we used in the model.

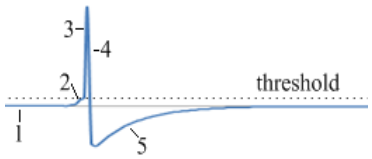

Figure 1: Neuron action potential as a function of time. Phases of action potential: 1 - resting potential, 2 - initial depolarization, 3- regenerative depolarization, 4 - repolarization, 5 - hyperpolarization. During phases 3 and 4 neuron is in period of complete refraction.

Underlying mechanism of neuron action potential (AP), in other words spike, was explained by Hodgkin and Huxley (Hodgkin and Huxley, 1952). For readers' convenience we added an illustration of different phases of action potential, since we refer to hyperpolarization phase later in this paper (Fig 1.)

Function of action potential:

$$AP(t) = W_{ap}\left(K_{dpl}\, e^{\frac{-\Delta t}{T_m}} - K_{hpl}\left(e^{\frac{-\Delta t}{T_m}} - e^{\frac{-\Delta t}{T_{ap}}}\right)\right) \quad (1)$$

where $\Delta t = t - t_{spike}$; constants $K_{dpl} = 3$, $K_{hpl} = 5$ and $W_{ap}=40$ define the amplitude of the function of action potential; $T_m=10$ms is the membrane time constant that defines the slope of the hyperpolarization phase, $T_{ap}=0.5$ms is the constant that defines the slope of the spike.

We executed our experiments in precision of one millisecond relative to the function of action potential, therefore we refer to single iteration of a simulation as one millisecond.

Synaptic strength w_j defines amplitude of postsynaptic potential (PSP) that would be raised in postsynaptic neuron membrane by presynaptic spike. Depending on synapse type, postsynaptic potential can be positive excitatory (EPSP), or negative inhibitory (IPSP). If the sum of PSP reached threshold, that would trigger neuron to produce action potential, in other words to fire a spike (see Fig. 1).

Function of postsynaptic potential raised by spike from individual synapse:

$$PSP_j(t) = \phi_j w_j \left(e^{\frac{-\Delta t}{T_m}}\left(1 + \kappa_{m_j}(t)\right) - e^{\frac{-\Delta t}{T_s}}\left(1 + \kappa_{s_j}(t)\right)\right) \quad (2)$$

where $\Delta t = t - t_{pre}$; $\phi = 1$ for excitatory synapses and $\phi = -1$ for inhibitory; time constant of the synapse $T_s=2.5$ms; membrane time constant $T_m=10$ms is the same as in equation 1. In simulation of the model, we optimized PSP calculations and instead of keeping PSP history for each synapse, we used accumulated exponential slopes in variables κ_m and κ_s. This is simple, but, to our knowledge, novel approach that helped to economize computing costs. κ_m and κ_s are updated at the moment of each presynaptic spike. See equations 3 and 4:

$$\kappa_{m_j}(t) = \begin{cases} \frac{w_{j_{(t-1)}}}{w_{j_{(t)}}} e^{\frac{-\Delta t}{T_m}} \left(1 + \kappa_{m_j}(t-1)\right) & if\ t = t_{pre} \\ \kappa_{m_j}(t-1) & otherwice \end{cases} \quad (3)$$

$$\kappa_{s_j}(t) = \begin{cases} \frac{w_{j_{(t-1)}}}{w_{j_{(t)}}} e^{\frac{-\Delta t}{T_s}} \left(1 + \kappa_{s_j}(t-1)\right) & if\ t = t_{pre} \\ \kappa_{s_j}(t-1) & otherwice \end{cases} \quad (4)$$

Here Δt is a time difference between times of current and previous presynaptic spikes, w_j prohibited to decay to 0. $w_{j(t-1)}$ and $w_{j(t)}$ denominates synaptic strength before and after STDP modification. Equations 3, 4 can be derived by solving trivial equation 5, assuming that at zero point $\kappa_0 = 0$ and t_1-t_0 = *const* and t_1-t_0 = t-t_0 when t_1 = t:

$$w_0 e^{-(t-t_0)}(1 + \kappa_0) + w_1 e^{-(t-t_1)} = w_1 e^{-(t-t_1)}(1 + \kappa_1) \quad (5)$$

Neuron membrane potential at any time:

$$P(t) = \begin{cases} AP(t) & if\ t = t_{spike} \\ AP(t) + \sum PSP(t) & otherwice \end{cases} \quad (6)$$

2.2 Spike Timing-dependant Plasticity

STDP rule is a function of time difference between presynaptic and postsynaptic spikes that guards the amount of change of synaptic strength. In our model we used single STDP rule, see Fig. 2. Long-lasting decrease of synaptic strength is called long term depression (LTD), lasting increase is called long term potentiation (LTP).

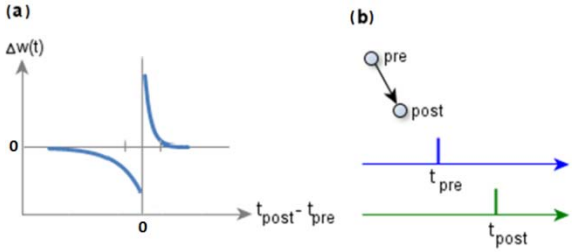

Figure 2: STDP rule of excitatory synapses (a) STDP as a function of spike timing difference. Based on Bi and Poo (Bi and Poo, 1998). (b) Schematic explanation of STDP, as a function of time difference between times of presynaptic and postsynaptic neuron spikes.

STDP function used in our model is expressed in equation 7. Synaptic strength change for excitatory synapses, where $\Delta t = t_{post} - t_{pre}$:

$$\Delta w_j = \begin{cases} A_{LTP} \cdot e^{\frac{\Delta t}{T_{LTP}}} & if\ \Delta t < 0 \\ -A_{LTD} \cdot e^{\frac{-\Delta t}{T_{LTD}}} & if\ \Delta t > 0 \\ 0 & if\ \Delta t = 0 \end{cases} \quad (7)$$

Synaptic strength values are limited between W_{min} and W_{max}, which in our model, vary depending on synapse type. To simplify the calculations of postsynaptic potentials, we prohibited synapses to decay less than $1*10^{-6}$. See equations 3 and 4. See section 0 for A_{LTP}, A_{LTD}, T_{LTP} and T_{LTD} constants.

In our model we used closest neighbor rule, that is only two closest spikes participate in modification of synaptic strength. Alternatively all-to-all rule could be used.

3 THE MODEL

Model diagram is displayed in Fig. 3. It consists of six main layers: L1 and L5 are competitive winner(s)-takes-all (WTA) layers (in our model we did not prohibited a few neurons to learn the same pattern, therefore we should say winners). L1 and L5 have corresponding inputs from L0 and L4. L3 is a layer of temporal memory; it is modulated by layer L2. In our model we did not attempt to match any layers in cortex or hippocampus, network structure and layer names are purely arbitrary.

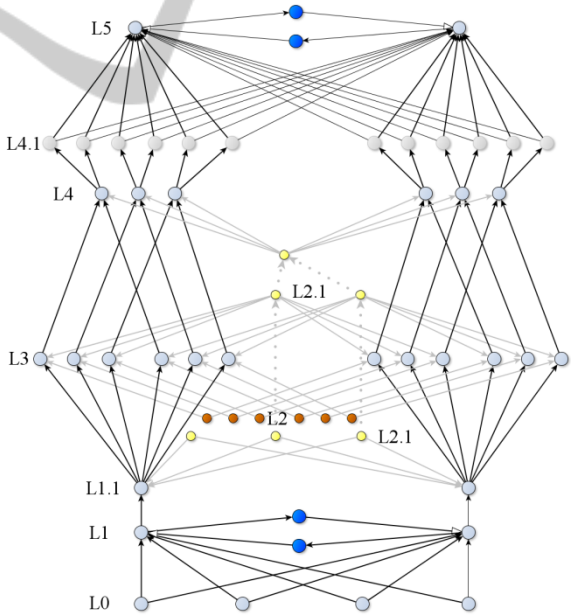

Figure 3: Diagram of the network model with temporal memory. Blue color denotes inhibitory interneurons. In real simulation inhibitory neurons are replaced by direct inhibitory synapses. Grayed lines denote synapses from L2, L2.1 subnetwork of a temporal modulation. Doted lines denote that it is the same neuron, split in a diagram for better visibility. Layer L4.1 added only for programming convenience and in our experiment it served as input multiplier for L5 WTA network.

Neurons in layer L0 periodically fire a sample pattern. L0 neurons also fire spontaneously with a probability P_{L0} in an each iteration of an experiment (each one millisecond). Spontaneous firing produces a Poisson noise. Noise increases probability of LTD in synapses L0 to L1 and is responsible for strength decay of synapses that do not participate in sample pattern. Though, we used a noisy input in our model, it is not mandatory, for neurons can successfully be trained without it, although noiseless patterns wouldn't be realistic. In that case, synapses that do not carry spikes from sample wouldn't be affected by STDP.

Neurons in layer L1 receive input from L0 and are interconnected with inhibitory synapses. Strengths of inhibitory L1 to L1 synapses are constant.

Layer L1 produces input for L1.1 interneurons via strong synapses with fixed weights. Strengths of L1 to L1.1 synapses are large enough to arouse a postsynaptic spike from resting potential with single presynaptic spike. Layer L1.1 is introduced for the reason that later memory read would not affect L0 to L1 synapses.

Layer L2, including neurons L2.1, is used for temporal modulation. Excitation of L2, L2.1 neurons imitates wave propagation in excitable media in single direction, only one neuron fires at the same time; it is looped. While L2 neurons produce a chain of spikes during excitation period, L2.1 produces only single spike. Weights of synapses outgoing from L2 and L2.1 do not change. See Fig. 4 for details.

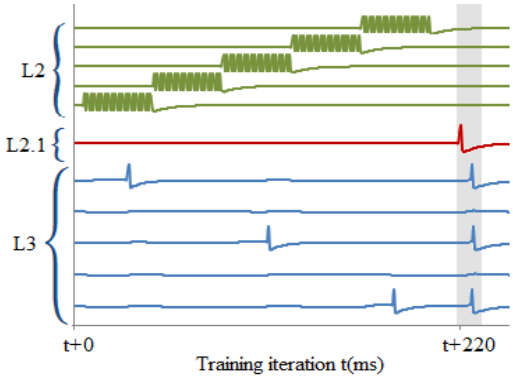

Figure 4: Temporal modulation of five neurons in layer L3 that receive input from a single neuron from layer L1.1. Layer L2 excites each neuron in L3 for approximately 40ms. If in that window L3 neuron receives EPSP from L1.1, it produces a spike and corresponding synapse updated by strong LTP. After 220ms L2.1 neuron raises additional spike in L1.1 and adds weak EPSP to the neuron groups in L3 and all L4 neurons. L3 that has a memory of previous spike, passes compressed pattern to L4. See network diagram in Fig 3. In particular case L1.1 neuron fired three times, as a result L3 produced a pattern 10101.

Each synapse from L1.1 neuron to L3 neuron represents a binary memory unit. It memorizes a fact of a spike from L1.1 relative to corresponding L2 neuron timing. L3 neurons are grouped by synapses from L1.1. Each L3 in a group receives a strong excitatory input from different L2 neuron. This input, however is not strong enough to produce a spike. Initially L1.1 to L3 synapses are weak and are prohibited from growing strong enough to raise a spike without additional excitation from L2 or L2.1. If L3 neuron is excited by spikes from L2 and during that period L1.1 fires, it would fire and synapse strength would grow by strong LTP.

During the experiment, strengths of synapses L2.1 to L3 do decay over time, so that memory slot could be reused on next L2, L2.1 loop iteration. It is known that LTP in living synapses lasts from a few hours to months or longer (Abraham, 2003) therefore synaptic strength decay in our model is consistent with biological features of synapses.

L2.1. neurons activate memory read. Each L2.1 has strong synapses to all L.1.1 neurons, weak synapses to all L4 neurons and weak synapses to subgroups in L3. L3 neurons grouped by L2.1 represent the memory window. Spike from L2.1 raises a spike in L1.1 by its own. Excitation from L2.1 to L3 is much weaker than from L2 and produced by a single spike, therefore only strong synapse from L1.1 to L3 can raise a spike in L3.

Layer L4 serves as an input to WTA layer L5. L4 has moderate fixed strength synapses from L3; therefore a spike from L3 can raise a spike in L4 only when L4 neuron is excited by L2.1.

We added layer L4.1 only for programming convenience. We found that multiplying inputs to WTA layer would make training process more robust in a wider range of parameters. Also it increases a chance of beneficial permutation of initial synaptic strengths. Since we experimented with relatively small network, we duplicated inputs to L5 to gain more stable training process. In case of a larger network this would not be necessary. Alternatively L4.1 layer can be replaced by multiplying synapses from L4 o L5, instead of adding the entire layer of interneurons. Analogically to layer L0, L4.1 produces Poisson noise.

Layer L5 is analogical to L1; however we tuned it with different STDP parameters. Additionally, we introduced stochastic threshold in L5 neurons, see section 0.

Layer L1 was trained during entire simulation, while training of layer L5 started only after first 100000 iterations of a simulation. We simply prohibited neurons in layer L4 from firing at the first stage of experiment.

3.1 Parameters of the Simulation

We used genetic algorithm to tune L1 and L5 WTA sub-networks, the rest of the parameters are completely arbitrary.

General parameters of the model simulation listed in tables 1, 2 and 3. For parameters of training sample data and for special case of layer L5 threshold see sections 0 and 0.

Table 1: STDP Parameters for synapse types.

Synapse type		Parameter				
From	To	W_{max}	A_{LTP}	A_{LTD}	T_{LTP}	T_{LTD}
L0	L1	0.56	0.064	0.037	9.01	55.71
L1.1	L3	21	30	0.03	24	34
L4.1	L5	0.75	0.32	0.076	8.37	459

Table 2: Initial synaptic strengths.

Synapse type		W_0	Synapse type		W_0
From	To		From	To	
L0	L1	0.44*	L2.1	L3	8
L1	L1	1.411	L2.1	L4	18
L1	L1.1	30	L3	L4	10.9
L1.1	L3	15	L4.1	L4	28
L2	L3	2.5	L4.1	L5	0.68*
L2.1	L1.1	40	L5	L5	5

*. Initial synaptic strengths randomly distributed around mean values W_0 in range +/- 2.5% of W_0.

Table 3: Sizes of layers and threshold values.

Layer	Number of neurons	Neuron threshold
L0	250	-
L1	20	6.89
L1.1	20	11.71
L2	125	-
L2.1	25	-
L3	250	11.71
L4	100	11.71
L4.1	200	11.71
L5	20	10.976

For evolutionary tuning we used multi-agent system with a population of 100 agents. Each agent represented itself a functional WTA network. Genome of each agent contained initial and maximal synaptic strengths: W_0 and W_{max}; parameters for STDP function: A_{LTP}, A_{LTD}, T_{LTP} and T_{LTD}. Initial genome values for each agent were normally distributed around arbitrary mean values. In each generation, each agent was trained 20 times with different training sample sets. Synaptic strengths were reset at the beginning of each training. Errors made by each agent were counted for all 20 trainings. When the training was completed, the worst performed agents (60% of the population) were replaced by the new mutants made from the best performed agents. Each gene mutated with probability 0.3; new value was random in a range +/- 5% from inherited value. We did not use any crossover. Multiple experiments with different initial values were executed for a few hundreds of generations each. Genome values of the best performed agent from final generation were used as parameters for the model.

3.2 Training Samples

Sample spike patterns produced from layer L0 represented itself five 4x250 matrixes of 0 and 1 values. One indicated spike time relative to the sample start time and column position. Spikes were distributed uniformly across all sample matrix with occurrence probability p=0.04. For convenience, we called L0 samples “letters" and denominated in minuscule letters a, b, c and d. "Letters" were displayed with 40ms intervals. During the gaps between letters and during letter display, L0 produced random spikes with the same probability p=0.04. During first 100000 iterations letters were displayed in random order.

After 100000 iterations, letters were combined into consistent "words", denominated by capital letters A, B, C and D. Each “word” was made from five non repeating letters, that is made from random permutations of a, b, c, d. Words were displayed in random order and aligned to start right after L2.1 scan time. During scan time L0 produced random letter. Internals between letters remained the same 40ms.

Additionally we injected Poisson noise into L0 and L4.1outputs. We generated Poisson noise by firing random spike with probability P_{L0}=0.04 for layer L0 and with probability P_{L41}=0.01 for layer L4.1. In our experiments spike density during display of samples was higher than in intervals between samples; however it has already been demonstrated that neurons can successfully learn

when density is the same (Masquelier et al., 2008, 2009). What are theoretical boundaries of noise to sample spike density ratio, when STDP learning would start to fail is a good question, it requires further theoretical research to answer.

3.3 Learning Conditions in Layer L5. Introducing Stochastic Threshold

Patterns of "words" produced by L4 layer are quite different from strictly fixed samples of "letters". Pattern represents itself only single "column" of incoming spikes, however these are not synchronous. Spikes fluctuate in 2-3 milliseconds range (See Fig. 5).

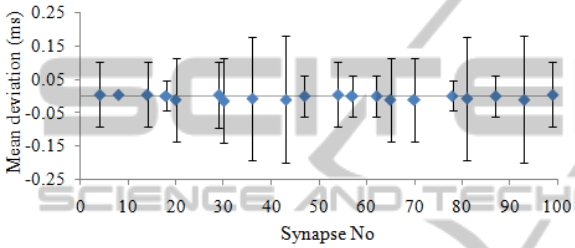

Figure 5: Mean deviation from pattern center in a single "word" in L4 output (L4 to L4.1 synapses). Error bars denominate standard deviations. Data retrieved from a single experiment, the pattern repeated 521 times, only consistent spikes that were repeated more than 80% of times taken into account.

Fluctuations in patterns of "words" are caused by variations of synaptic strength in L1.1 to L3 synapses and also depend on pre-existing value of postsynaptic potential in L4 and L3 neurons. We did not made analysis of which factor is dominant. Another important detail is that due to presence of errors in L1, not all spikes are equally consistent. In Fig. 5 displayed L4 to L4.1 synapses that produced consistent spikes at the range 83% to 100% of all occurrences of the "word". In the rest of the synapses spike occurred in less than 3% of the times.

Initially we failed to achieve L5 layer training in these conditions within acceptable error rate. Usually all neurons learned a single pattern or a few at once. We solved this problem by introducing stochastic threshold in L5 neurons: when neuron reached its firing threshold, it didn't fire immediately but with probability 0.8. This accelerated inhibition from "lucky" competing neurons.

It must be noted, that attempts to apply stochastic threshold in layer L1 only increased error rate.

4 RESULTS

We conducted a series of simulations of the entire model in continuous mode. Also, because of high computing cost of simulation of the entire network, in order to estimate performance we conducted experiments witch each of WTA sub networks separately. Each of the simulations took 700000 iterations; first 100000 iterations were dedicated to train L1 layer only with random “letters”. Typical output from layer L5 at the beginning and at the end of the training is displayed in Fig. 6.

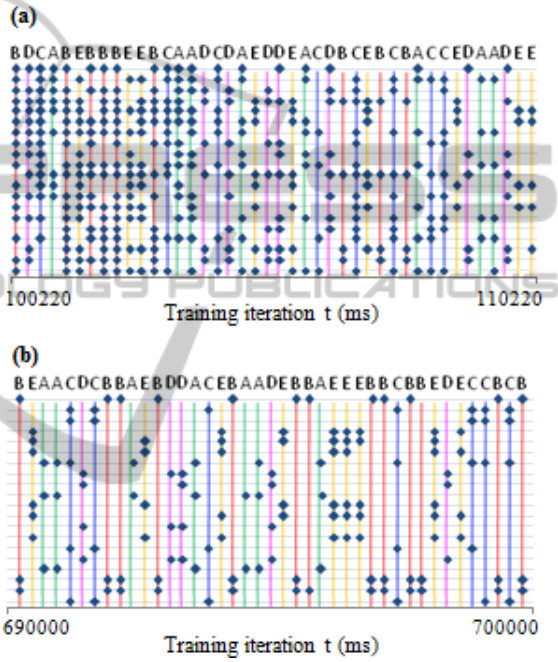

Figure 6: Spike output from layer L5 at the beginning and at the end of the training. Output from each of 20 L5 neurons aligned along vertical axis. Letters above pattern denominate one of 5 sample "words" displayed at the time. (a) Output at the beginning of the training. Even though WTA network exposed to a very few appearances of each sample of "word", consistent pattern started to emerge at the very beginning of the training. (b) Output at the end of the training.

4.1 Overall Performance of the Model

For estimation of the error rate, at the end of the experiment we counted responses of individual neurons relative to the sample occurrence times during last 5000 iterations. For layer L1 we used bias of 8 iterations latency for neuron response, and bias of 16 iterations for layer L5. The sample to which neuron was the most selective was assigned to the neuron as a learned one. If neuron response

count was less than a half of average sample, such neuron was treated as non selective to any sample. Each missed sample or neuron response out of the biased sample window was treated as error. We did not analyze the cases when neuron learned more than one sample, instead treated responses to other samples as errors.

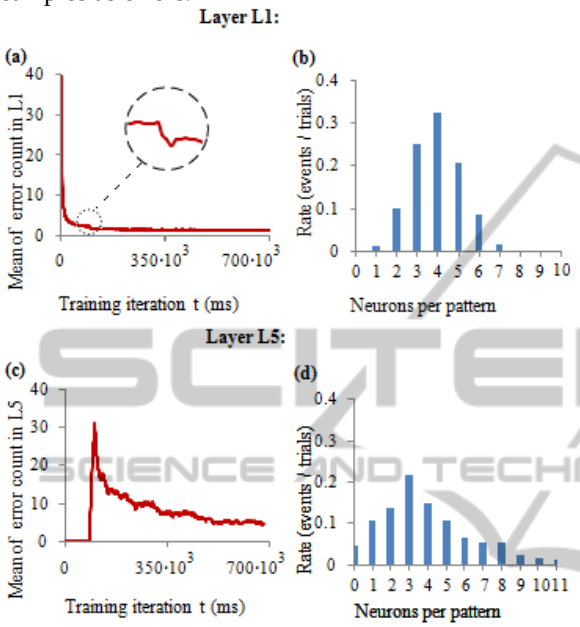

Figure 7: Mean error rate and selectivity distribution in WTA layers L1 and L5. Data obtained from 100 experiments, each experiment made for 700000 iterations. (a) Mean error rate in layer L1. Sample patterns were regenerated for each experiment. Zoomed part of the series indicates a drop of error rate after stochastic appearance of one of the five samples (random letters) were replaced by consistent sequences (random words). (b) Distribution of the number of neurons selective to one sample in layer L1. (c) Mean error rate in layer L5. Three pre-recorded sample patterns of “words” were used in 100 experiments, each in 1/3 of experiments. (d) Distribution of the number of neurons selective to one sample in layer L5.

We conduced 100 of experiments to estimate mean error rate for layers L1 and L5 separately. Initial synaptic strengths were reset at each experiment. Errors were counted in sliding 3000 iteration window for L1 and 18000 iterations for L5. Window sizes were proportional to rate of samples 1 to 6: each word consisted of 5 letters plus 1 letter for scant time. Window was moved by step of 1000 iterations (See Fig. 7). For layer L1 we generated sample “letters” at each experiment, for layer L5 we used recorded input from three different simulations of the entire model. Therefore, our estimation of L5 error rate contains larger bias.

Layer L5 produced significantly larger error rate, at the last 10000 iterations of experiments it reached mean value of 4.514, while L1 produced only 1.207.

There is an interesting observation in layer L1 error rate: at the moment when random “letters” are replaced by consistent sequences, we see modest but steep drop in error rate (Fig. 7(a)). Most likely this is caused by reduced rate of sequences of the same "letter", what makes a trained neuron harder to fire subsequently, because of previous hyperpolarization.

There were noticeable differences between layer L1 and L5 in distribution of neurons selective to the single pattern (See Fig. 7 (b) and (d)). Mean values of the number of neurons per single sample were quite close: 3.956 for L1 and 3.99 for L5, but significantly different standard deviations: 1.24 for L1 and 2.59 for L5. There were no any non-learned samples in L1, but in L5 non-learned samples occurred with the rate 0.046. Rate of neurons that did not learn any pattern was significantly larger for layer L1 and was 0.22, while in L5 it was 0.05. We cannot tell which factor had the biggest influence to this difference: different set of parameters, stochastic threshold in L5, difference of input patterns, or it was simply caused by biased measurements of layer L5. It requires detailed theoretical study of limiting and optimal parameters of STDP rules.

5 DISCUSSION

We demonstrated the model of an unsupervised neural network that is capable of learning prolonged combinations of spatiotemporal patterns of spikes in continuous mode. In this way we demonstrated that STDP learning rules alone can be applied to train neural network to learn long lasting sequences combined of short samples. Moreover, the model is capable of memorizing and reproducing sequences in which network input samples were displayed. Reproduction of sequences can be achieved by subsequently activating L2.1 neurons.

The fact, that memory of events in time can be reproduced, implies that such memory could be copied, transferred, compared etc. Also, it should be relatively easy to extend our model enabling it to learn combinations of "words", although that would require additional, more complex modulation in different time scales.

5.1 Biological Plausibility of the Model

The model itself and a range of parameters of simulation are arbitrary and cannot be used as a

reference to a simulation of true biological process. However, the model is based on known biological processes, and presence of temporal coding is supported by experimental evidence.

Since we designed our network to be as simple as possible, there are, probably, many ways to implement a neural network with similar or the same features that would be more realistic in biological sense or would have a better performance.

For an instance, for temporal modulation it would be more realistic to use inhibitory neurons instead of excitatory. There are experimental evidences that gamma rhythm oscillations are generated by inhibitory interneurons (Cardin et al., 2009).

We used only the simplest closest-neighbor approach to STDP learning rule. Other variations could be considered for future experiments. For an instance, a possible impact of triplet rule (Pfister and Gerstner, 2006) should be taken into account.

5.2 Limitations of the Model and Guidelines for Future Research

The model requires explicit timing for the occurrence of training samples. In order to use our model for real world data, timing of sensory input must be aligned to activation periods of layer L2. However, additional chains of modulation that synchronizes sensory input with L2 layer activation periods and/or vice versa should solve this problem.

Another obvious limitation of the model is a "blind spot" at each memory read, however this problem could be overcome by multiplying L1.1 to L4 layers, in that way creating overlapping or sliding memory window.

Simplistic structure of WTA networks used in our model is disputable as well. With increase of different sample count, intervals between the same repeated sample would increase as well, that would make learning harder and harder. Training individual or groups of neurons one-by-one with a limited number of samples would solve the problem and boost the performance. However, how we would implement this approach for a short temporal code in rapidly changing environment is a question that we cannot answer yet. Well known adaptive resonance theory (ART) (Carpenter and Grossberg, 2009) solves similar problem by introducing a self organized network and a resonant state between input and already learned data. However, the achievement of resonance necessary for ART requires a prolonged state of neural activity (rate code) that is not the case of our model. Although, various modifications of our model that would introduce additional rate code are possible. This is also a matter of future research.

The nonlinear nature of STDP and leaky integrate-and-fire neuron makes the tuning of the parameters of WTA networks a really challenging task. We used genetic algorithm for this matter, however, we cannot claim that we reached optimal point of the model parameters. There is little known of theoretical limits and optimal points of STDP rule. Our next step will be detailed theoretical research of STDP in the noisy environment from perspective of the probability theory and statistics.

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