The effects of external electrical field on a neural network with synaptic plasticity and conduction delays

Jia Zhao, Bin Deng, Xile Wei, Jiang Wang, Cong Men, Yingmei Qin, Jianbing Sun
School of Electrical Engineering and Automation, Tianjin University, Tianjin, 300072, China
E-mail: jiangwang@tju.edu.cn

Abstract: Effects of external DC stimuli to the evolution of a neural network model are studied. It's found that the external DC stimuli can enhance the neural network's average firing rate, induces the neural network to get to a balanced state that exhibits rhythmic activity with high rate. The activity of the neural network can be affected by the synaptic connection strength, the conduction delays, the external stimuli and so on. These parameters can serve as factors in modulating the evolution of the neural network with different firing activities. Furthermore, these results may also relate to the mechanism of memory and learning process.

Key Words: Spike-timing-dependent plasticity (STDP), Weak direct current stimuli, Rhythmic activity

1 INTRODUCTION

For human being, the brain performs many sophisticated computational tasks with a fast speed and high precision unparalleled by present computers, such as image and voice recognition. The mechanism of the brain achieves such a function is helpful both to better understand the brain and for the artificial applications induced by it[1]. Recent advances in brain research have generated renewed awareness and appreciation that the brain operates as a complex non-linear dynamic system, and synchronous and phase-locked oscillations may play a crucial role in information processing, such as feature grouping, saliency enhancing and phase-dependent coding of objects in short-term memory [2, 3]. Learning is central to understanding neuronal information processing, which has been studied from the molecular level up to the behavioral level. In more recent years, a new concept that temporal order instead of frequency is more important in cellular learning has been proposed. This new learning paradigm, which known as spike-timing-dependent plasticity(STDP), has rapidly get wide attention[1, 4]. Synaptic plasticity describes mechanisms that take place at the connection site between two neurons (synapse), when the synaptic weight related to the post-synaptic response to a single pulse is strengthened (potentiation) or weakened (depression)[5]. The STDP model is a departure from traditional Hebbian models of learning, which states that neurons that fire action potentials together will have their interconnections strengthened[4]. Perhaps because of STDP's combination of elegant simplicity, biological plausibility, and computational power, the research of STDP make a great progress in many aspects. Previous studies have shown how STDP can implement input selectivity according to the spike-time correlation structure of input spike trains for single neurons and feedforward networks [6, 7, 8]. STDP has also been supposed to play a role in the hippocampus theta phase precession phenomenon [9, 10]. Several more recent studies of STDP have focused on parameterizing STDP with respect to factors such as rate, higher-order spiking motifs, or dendritic location[11]. For example, Robert Froemke and Yang Dan reported that the first spike pairing in a train of triplet or quadruplet spike-pairings determines whether long-term potentiation(LTP) or long-term depression(LTD) ensues in layer-2/3 pyramidal cells[12]. Similar findings were acquired in hippocampal cell culture by Guo-qiangBis team[13]. Besides, the computational role and functional implications of STDP have been explored from many points of view [14, 15, 16].

Transcranial electrical stimulation (TES) with weak currents is actively investigated to treat a range of neurological and psychiatric disorders [17, 18]. Low-intensity TES is also used as a tool for cognitive research in healthy subjects because of its safety profile [19, 20]. These studies leverage the induction of lasting changes, but evidently these long-term effects must be mediated by immediate effects during stimulation. Indeed, there is evidence that low-intensity TES can affect ongoing brain activity [21, 22]. Animal studies indicate that electric fields will incrementally polarize somatic membranes at levels below synaptic background activity [23]. Yet, there is increasing evidence that very weak electric fields can acutely modulate coherent network activity, indicating that small incremental polarization of individual neurons can entrain ongoing network dynamics when operating coherently on a population of coupled neurons [24, 25]. Furthermore, Davide Reato et al. explicate and differentiate the specific aspects of network dynamics, especially endogenous oscillatory activity [26].

The synaptic connection presented in a neural network exerts significant impacts on its function. Traditionally, a predefined topological structure is adopted in neural net-

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work modeling, which may not reflect the true situation in real-world networks such as the brain network. In this paper we study a self-organized network (SON) model whose synaptic connections evolve according to the spike-timing dependent plasticity (STDP) mechanism. Specifically, we investigate how the external weak direct current(DC) stimuli will influence the dynamical evolution and the emergent topology of the network. We find that the external weak DC stimuli can significantly enhance the rhythmic activity of the entire neural network. This result is related to the TES which is now used to treat some neurological and psychiatric disorders. It may also have important implications on the study and memory of the brain network.

The rest of this paper is organized as follows. We first introduce the neural model, the neural network's constitution and the STDP rule used in this paper. Then we research the evolution process of the neural network. The effects of the external DC stimuli on the evolution of the network is also investigated. We further study the changes of the average firing rate, the effects of conduction delays and the percentage of excitatory neurons' synaptic strength during the evolution of the network. In the final section, the conclusions are given.

2 THE NEURAL NETWORK MODEL AND THE STDP RULE

Each neuron in the network is described by the simple spiking model [27]

$$\begin{aligned} \dot{v} &= 0.04v^2 + 5v + 140 - u + I \\ \dot{u} &= a(bv - u) \end{aligned} \tag{1}$$

with the auxiliary after-spike resetting

$$if\ v \geq 30mv, then \begin{cases} v \leftarrow c \\ u \leftarrow u + d \end{cases} \tag{2}$$

Here variable v and u are dimensionless variables. v represents the membrane potential of the neuron and u represents a membrane recovery variable, which accounts for the activation of K^+ ionic currents and inactivation of Na^+ ionic currents, and it provides negative feedback to v. Synaptic currents or injected currents are delivered via the variable I. a, b, c and d are dimensionless parameters. The parameter a describes the time scale of the recovery variable u, b describes the sensitivity of the recovery variable u to the subthreshold fluctuations of the membrane potential v, c describes the after-spike reset value of the membrane potential v caused by the fast high-threshold K^+ conductances and d describes after-spike reset of the recovery variable u caused by slow high-threshold Na^+ and K^+ conductances. According to equation (2), the membrane voltage and the recovery variable are reset when the spike reaches its apex at $+30mV$. Depending on the values of the parameters, the model can exhibit many properties of biological neurons[28]. The resting potential of the neuron model is between -70 and $-60mV$ according to the value of b. And the threshold potential can be as low as $-55mV$ or as high as $-40mV$, which depends on the history of the membrane potential prior to the spike. We use $(b, c) = (0.2, -65)$ for all neurons in the network. For excitatory neurons, we use the values $(a, d) = (0.02, 8)$ corresponding to neurons that can exhibit regular spiking firing patterns. For inhibitory neurons, we use the values $(a, d) = (0.1, 2)$ corresponding to neurons exhibiting fast spiking firing patterns.

In this paper, we simulated an anatomically realistic network model that consisting of 1,000 cortical spiking neurons with axonal conduction delays and synaptic plasticity[29]. The network is composed by excitatory (80%) and inhibitory (20%) neurons. The ratio of excitatory to inhibitory neurons is 4 to 1, as in the mammalian neocortex. Each excitatory neuron is connected to 100 random neurons, so that the probability of connection is 0.1, again as in the neocortex. And each inhibitory neuron is connected to random 100 excitatory neurons only. Each neuron is described by the simple spiking model (1). The synaptic connections among neurons have fixed conduction delays, which are random integers between 1 ms and 20 ms. We set $1ms$ delay to all inhibitory connections and 1 to 20 ms delay to all excitatory connections. The initial values of excitatory weights are $s_{ij} = 6$ and the inhibitory weights are $s_{ij} = -5$. In the network model, synaptic connections are modified according to the spike-timing-dependent plasticity (STDP) rule[15]. The function $F(\Delta t)$ determines the amount of synaptic modification arising from a single pair of pre- and postsynaptic spikes separated by a time Δt. The function

$$F(\Delta t) = \begin{cases} A_+ \exp(\Delta t/\tau_+) & if\ \Delta t < 0 \\ -A_- \exp(-\Delta t/\tau_-) & if\ \ \Delta t \geq 0 \end{cases} \tag{3}$$

provides a reasonable approximation of the dependence of synaptic modification on spike timing observed experimentally, where $\Delta t = t_{pre} - t_{post}$. The parameters τ_+ and τ_- determine the ranges of pre-to-postsynaptic interspike intervals over which synaptic strengthening and weakening occur. A_+ and A_-, which are both positive, determine the maximum amounts of synaptic modification, which occur when Δt is close to zero. Here, we set $\tau_+ = \tau_- = 20ms$, $A_+ = 0.1$ and $A_- = 0.12$. If a spike from an excitatory presynaptic neuron arrives at a postsynaptic neuron (possibly making the postsynaptic neuron fire), then the synapse is potentiated (strengthened). In contrast, if the spike arrives right after the postsynaptic neuron fired, the synapse is depressed (weakened). If pre- and postsynaptic neurons fire uncorrelated Poissonian spike trains, there are moments when the weight of the synaptic connection is potentiated or depressed. We choose the parameters of the STDP rule so that depression is stronger than potentiation. In contrast, if the presynaptic neuron often fires before the postsynaptic one, then the synaptic connection slowly potentiates. Indeed, such a connection causes the postsynaptic spikes and should be strengthened. In this way, STDP strengthens causal interactions in the network. The magnitude of potentiation or depression depends on the time interval between the spikes. For each fired neuron, we consider all its presynaptic neurons and determine the timings of the last excitatory spikes arrived from these neurons. Since these spikes made the neuron fire, the synaptic weights are po-

tentiated according to the value of STDP at the presynaptic neuron adjusted for the conduction delay. Notice that the largest increase occurs for the spikes that arrived right before the postsynaptic neuron firing, that is, the spikes that actually caused postsynaptic spike. Otherwise, when an excitatory spike arrives at a postsynaptic neuron, we depress the synapse according to the value of STDP at the postsynaptic neuron. Indeed, such a spike arrived after the postsynaptic neuron fired, and hence the synapse between the neurons should be weakened. We artificially keep the synaptic weights s_{ij} between 0 and the maximal synaptic strength sm, where sm is $10mV$ here. Other parameters used in this paper are given in each case.

3 THE EVOLUTION PROCESS OF THE NEURAL NETWORK

In this section, we focus on the evolution process of the neural network. This neural network can spontaneously self-organize into groups and generate patterns of stereotypical polychronous activity as is shown in Ref[29]. Here, we mainly investigate the evolution process of the network in two kinds of external environments. We choose the average firing rate of the neural network as the reference of the evolution process. And the evoluting percentage of the excitatory neurons' synaptic strength during the evolution process can also serve as a reference of the evolution process.

When the neural network is under no stimuli, it exhibits rhythmic activity with low rate at the beginning of the evolution and the 200 inhibitory neurons(upper) in the network fire more frequently than the excitatory neurons(below), which we can see clearly in Fig. 1. As the synaptic connections of the network evolve according to the STDP rule, the rhythmic activity disappears in the evolution process and the spiking activity of the neurons becomes more Poissonian and uncorrelated after an evolution time of 100s. The spike raster of the network is shown in Fig. 2. The neural network converges to a state with an approximate balance of excitation and inhibition, which we can confirm by the average firing rate of the network in Fig. 5. These phenomena arise from the competition of the pre- and postsynaptic neurons' spiking times, which regulates the synaptic connection strength between neurons.

Then we apply external weak DC stimuli to the neural network with the strength of 0.5. In order to compare the evolution processes of the network under the two environments, we draw the network's spike rasters at the same time during the evolution. Fig. 3 is the spiking activity of the network when the evolution time is 2s. It also exhibits rhythmic activity with low rate, which is similar with the spiking activity of the network that under no stimuli. This may be primarily related with the initial strengths of the synaptic connection and the structure of the network. After an evolution time of 100s, the neural network converges to an approximate balance state which still exhibits rhythmic activity with a higher rate as is shown in Fig. 4. This rhythmic activity of the neural network is evidently caused by the external weak DC stimuli. And more obviously rhythmic activity can be found when we enhance the strength of

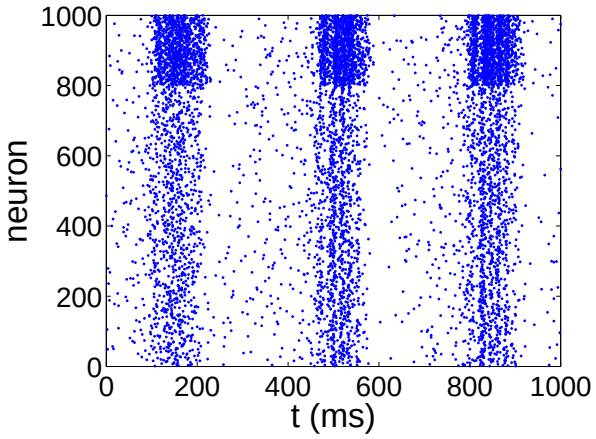

Figure 1: The spike raster of the network without external stimuli. The upper part that vertical axis exceeds 800 is the records of inhibitory neurons while the down part is the records of excitatory neurons. The network displays high-amplitude rhythmic activity. The evolution time of the network is 2s.

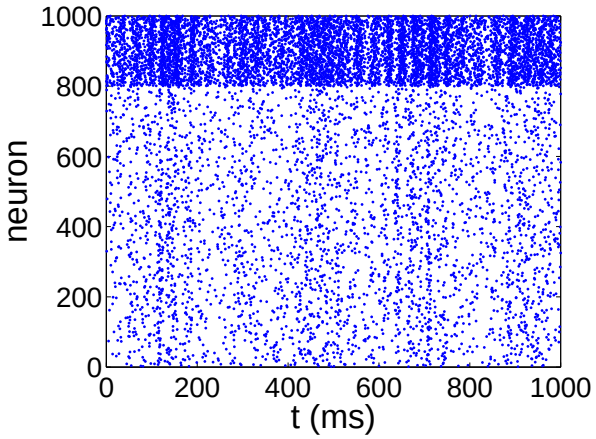

Figure 2: The spike raster of the network without external stimuli. The network displays uncorrelated Poissonian activity. The evolution time of the network is 100s.

the external DC stimuli.

The average firing rate of the whole neural network under different DC stimuli strength is also studied. As is shown in Fig. 5, the average firing rate of the neural network is high at the beginning of the evolution, then it quickly drops to a lower point. After an evolution time of 100s, the firing rate of the network gets to an approximately fixed value with small fluctuations. The four curves of the network's average firing rate in Fig. 5 have almost the same tendency during the evolution, no matter the network is under external stimuli or not. This may be caused by the structure of the network, such as the axonal conduction delays and the changes of the synaptic connection. As the external DC stimuli are strengthened, the average firing rate of the neural network is also enhanced(see Fig. 5). Thus the external weak DC stimuli can enhance the average firing rate of the neural network.

We also investigate the effects of the axonal conduction de-

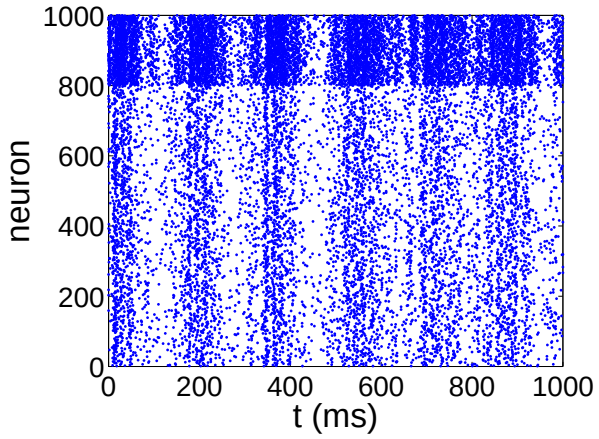

Figure 3: The spike raster of the network with external weak DC external stimuli. The network displays obvious rhythmic activity. The evolution time of the network is 2s and the strength of the DC stimuli is 0.5.

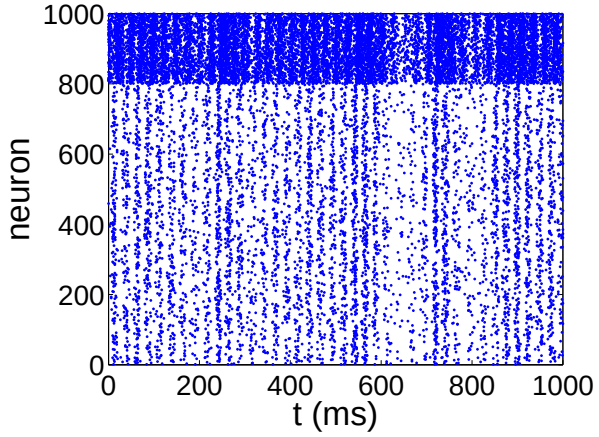

Figure 4: The spike raster of the network with external weak DC stimuli. It is evident that the network displays rhythmic activity. The evolution time of the network is 100s and the strength of the DC stimuli is 0.5.

lays on the evolution of the network. Here we just change the conduction delays of excitatory neurons, where the conduction delays of inhibitory neurons are fixed to $1ms$ as before. The firing rate of the neural network corresponding to different ranges of conduction delays is shown in Fig. 6. The maximal value of conduction delays D is increased gradually with a step of $1ms$ from $1ms$ to $20ms$. The firing rate here is the average of the whole evolution process when the range of conduction delays D is fixed. As is shown clearly in Fig. 6, the conduction delays can also influence the evolution of the network. The neural network under different stimuli has almost the same average firing rate when the conduction delay is $1ms$. And the average firing rates of the neural network enhanced generally when the maximal conduction delay is increased, though the changes of the average firing rate are not regular sometimes.

We now check how the synaptic connection of the neural

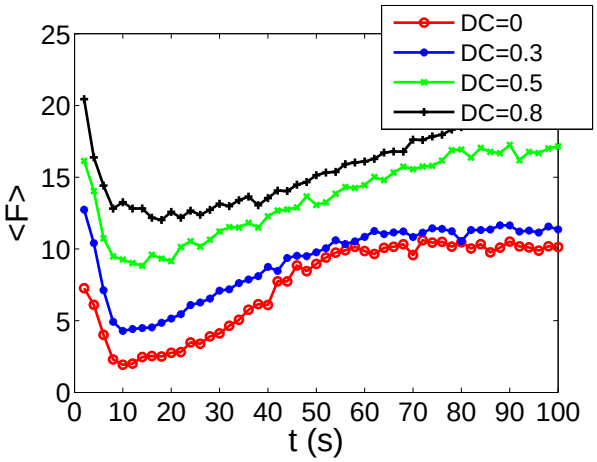

Figure 5: The average firing rate $< F >$ of the whole neural network corresponding to the evolution time. From bottom to top, the network is under different stimuli strengths which are enhanced gradually.

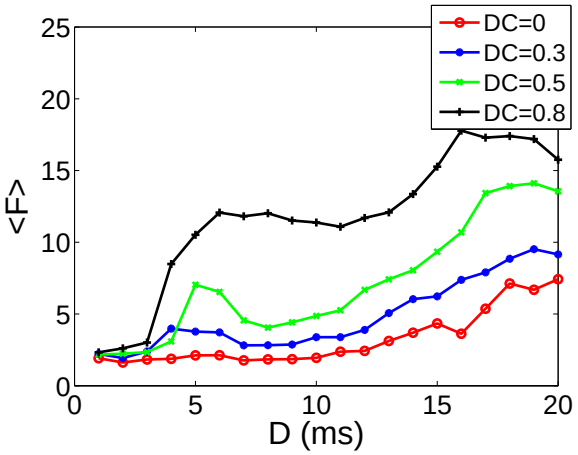

Figure 6: The average firing rate of the whole neural network corresponding to conduction delays for different stimuli.

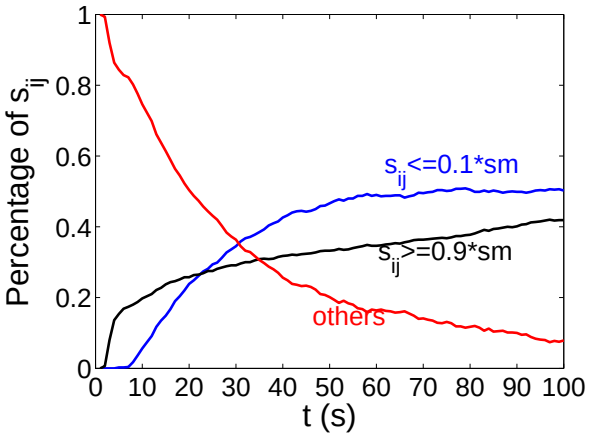

Figure 7: Percentages of the synaptic connection weights s_{ij} at three value levels during the evolution period of the network. sm is the maximal synaptic weight of the network.

network evolves when we apply weak DC stimuli to it. Because inhibitory synaptic weights are not plastic, whereas excitatory synaptic weights evolve according to the STDP modification function $F(\Delta t)$, we take the synapses of the 800 excitatory neurons into account, instead of the whole network. Fig. 7 is the evolution process of the synaptic connection when the strength of external DC stimuli is 0.8. The red line is the percentage of the synapses whose strength are less than $0.1 * sm$ during the evolution process, the blue line is the percentage of the synapses whose strength are more than $0.9 * sm$, and the black line is the percentage of the others. It shows clearly that most of the 800 excitatory neurons' synaptic connections converge to either 0 or the maximum sm from the initial values after the evolution time of 100s. So most of the connections between the neurons become either strong to sm or weak to 0, with only a few connections distribute sparsely between the two polarized weights. We also tested the initial excitatory synapses that are set to be other integers between 0 and sm, similarly polarized results can be obtained but need different evolution times.

4 DISCUSSION AND CONCLUSION

Effects of the external DC stimuli to the evolution of the network are investigated in details. As it is shown in the third part, the rhythmic firing activity is found at the end of the evolution time when the network is under external DC stimuli, which is different from the evolution consequence of the network without external stimuli. The synaptic plasticity, the axonal conduction delays and the external DC stimuli play important roles in the evolution of the network. The network structure determines the evolution tendency of the average firing rate, and the external DC stimuli changes the magnitude of the network's average firing rate. Furthermore, the percentage of excitatory synaptic strength in three ranges is also found to be polarized after the evolution.

Our findings in this paper may be significant for further studying the evolution of the neural network. Several properties of the neuronal network such as synaptic connection strength, axonal conduction delays and different external stimuli can serve as parameters in modulating the evolution of the neural network with different firing activities.

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