2017 International Symposium on Nonlinear Theory and Its Applications,
NOLTA2017, Cancun, Mexico, December 4-7, 2017

Effect of connectivity weights of inhibitory neurons in neuronal avalanches

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Abstract—In this paper, we investigated the effect of inhibitory synaptic connection strengths on neuronal activities in a neuronal avalanche reproducible network model. As a result, we found that the model network did not generate avalanches, if the strengths of inhibitory connections were too strong or too weak. This result suggests that an appropriate inhibition level exists for generation of avalanches and precise excitation/inhibition balance is realized when the neuronal avalanche phenomenon emerges.

1. Introduction

In the cortices, a number of neurons exist. By interacting with each other, the neurons organize huge complex networks. Then, these neurons exhibit diverse spontaneous neuronal activity. One of the major modes in the spontaneous neuronal activities is neuronal avalanches: synchronous neuronal firings continue to propagate for tens to hundreds milliseconds[1].

To characterize the neuronal avalanches, two measures are usually used: life time and size. The life time is defined as the duration of propagating synchronous activities and the size is defined as the number of firing neurons during the life time. Beggs and Plenz have experimentally shown that the distributions of the size and the life time obey power-laws whose slopes are -1.5 and -2.0, respectively[1].

Some theoretical studies show that the neuronal avalanches can be reproduced by spiking neural networks with spike-timing-dependent synaptic plasticity (STDP)[2, 3]. In Ref. [2], neurons are under limit cycle states. Then neuronal avalanches can be reproduced by STDP in a recurrent network composed of the Izhikevich neuron models[4]. In contrast, Kato and Ikeguchi have proposed a neuronal avalanche reproducible neural network model with excitable neurons by introducing pacemaker neurons. In Ref. [3], a critical strength of background input realizes the neuronal avalanches. However, many factors still exist to determine network activities and the other factors might also influence the behavior of the neuronal avalanches. One of the possible factors is strength of inhibitory connections. Therefore, in this paper, we investigated how the strength of inhibitory connections affect the slope of the size and the life time distributions.

2. Models

2.1. Izhikevich neuron model

We used the Izhikevich neuron model[4] as an element of our neural network. The neuronal dynamics is defined by 2-dimensional ordinary differential equations:

$$\begin{cases} \dot{v}_i = 0.04v_i^2 + 5v_i + 140 - u_i + I_i(t) + I_{\mathrm{ext}}(t), \\ \dot{u}_i = a_i(b_i v_i - u_i), \end{cases} \tag{1}$$

$$\text{if } v_i \geq 30[\mathrm{mV}],\ v_i \leftarrow c_i, u_i \leftarrow u_i + d_i.$$

In Eq. (1), v_i represents membrane potential of neuron i at time t, u_i represents a recovery variable of neuron i at time t, $I_i(t)$ represents synaptic current of neuron i at time t, and $I_{\mathrm{ext}}(t)$ represents external input of neuron i. Parameters a_i, b_i, c_i and d_i decide dynamics of the neuron. The synaptic current $I_i(t)$ is described by the following equation:

$$I_i(t) = \sum_{j,k} W_{ji}\delta(t - t_j^k). \tag{2}$$

In Eq. (2), W_{ji} represents synaptic weight between the neuron i and the neuron j, t_j^k represents the kth firing of the neuron j connected to the neuron i, and $\delta(\cdot)$ represents the delta function. Each neuron accepts noisy inputs that obey the Poissonian manner at f[Hz].

2.2. STDP learning

Synaptic weights between neurons in our neural network model change depending on relative spike timings between pre- and postsynaptic neurons[5]. The

amount of change in synaptic weight in the STDP is expressed by the following equations,

$$\Delta W_{ji} = \begin{cases} A_+ \exp(-\frac{t_i - t_j}{\tau}) \ (t_j < t_i), \\ -A_- \exp(-\frac{t_j - t_i}{\tau}) \ (t_j \geq t_i), \end{cases} \quad (3)$$

where t_i and t_j represent firing time of the postsynaptic neuron i and the presynaptic neuron j, A_+ and A_- represent a maximum value of long-term potentiation (LTP) and long-term depression (LTD), and τ represents a decay time constant of LTP and LTD.

3. Methods

We constructed a neural network of 1,250 neurons with excitatory neurons (80%) and inhibitory neurons (20%). In this paper, we used regular spiking neurons ($a_i = 0.02, b_i = 0.2, c_i = -65, d_i = 8$) as excitatory neurons and fast spiking neurons ($a_i = 0.02, b_i = 0.25, c_i = -65, d_i = 2$) as inhibitory neurons. They were randomly connected with probability 0.1.

Each neuron was stimulated by the noisy input at $f(= 170)$[Hz] and the strength of each pulse input was fixed during the simulation and set to 3.1 and 3.41 for excitatory and inhibitory neurons, respectively. In addition to the noisy input, pacemaker neurons also stimulated neurons in the network. The input for pacemaker neurons was constant and its strength was 5, which induced periodic firings. Each pacemaker neuron has 65 feedforward connections to neurons in the network. Synaptic strengths from pacemaker neurons were set to 20 at the initial condition.

The synaptic weights between excitatory neurons changed by the STDP learning and they were initially set to 0.01. On the other hand, synaptic weights between excitatory neurons and inhibitory neurons were fixed. We denoted W_{EI} as the synaptic weight from an excitatory neuron to an inhibitory neuron, W_{IE} as the synaptic weight from an inhibitory neuron to an excitatory neuron.

After the learning, the pacemaker neurons were removed and noisy input at $f_{\mathrm{aft}}(= 70)$[Hz] was applied to all neurons in the network. In addition, strong inputs, which was triggers of neuronal avalanches, were applied to three excitatory neurons every 200[ms]. The parameters A_+ and A_- were set to 0.1 and $1.05 \times A_+$ respectively, and τ was set to 20[ms].

Plastic synaptic strengths were constrained with hard bounds of $W_{ji} \in [0, 20]$ for connections from external neurons, and of $W_{ji} \in [0, 7]$ for the other plastic connections. Throughout this study, we fixed W_{EI} to 20. In our model, we avoided inhibitory neurons as targets of inhibitory neurons. We executed numerical simulations for 1,200[s]. The period of learning phase was 200[s] and the period of avalanche phase was 1,000[s]. Neuronal firing was checked every 1[ms] and we calculated the size and the life time.

4. Results

4.1. Influence of inhibitory connection weights on network activities

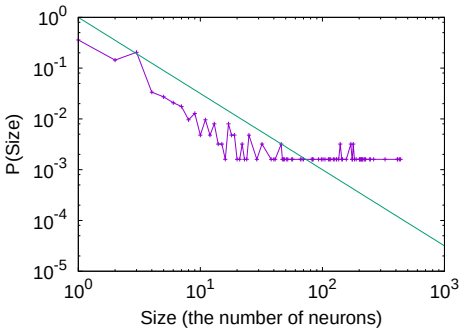
We investigated the influence of inhibitory connections weights on network activities. Then, we used W_{IE} as -0.15, -0.35 and -0.65. Figures 1 and 2 show the influence of the inhibitory connection strengths on the size distributions and the life time distributions. From Fig. 1, the slopes of the size distribution do not change significantly, but the cutoff ratios become smaller for the smaller W_{IE}.

On the other hand, Fig. 2 shows that the life time distributions change drastically. In Fig. 2, there is a large peak in the distribution, which indicates that two different distributions coexist. From these results, it can be considered that two assemblies are organized through STDP and coexist in the network. In the case of the stronger inhibition, the large peak disappears (Fig. 2(b)). Further stronger inhibitory connection weights make the slope of the distribution smaller (Fig. 2(c)). These results also indicate that the strong inhibition shunt the propagations of synchronization in the network and the long-lasting avalanches are more rarely generated. From these results, the moderate level of inhibition are necessarily to generate the neuronal avalanches.

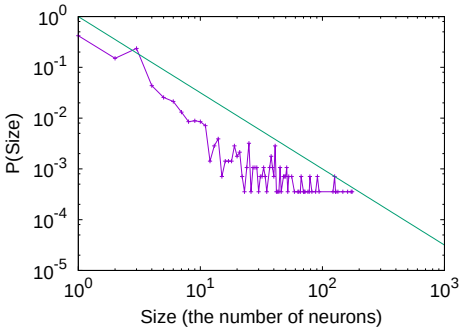
4.2. Influence of inhibitory connection weights on the distribution of plastic synapses

We denoted W_{EE} as the synaptic weight from an excitatory neuron to an excitatory neuron. We investigate the distribution of W_{EE} to understand the details of the influence of inhibitory connection strengths because the STDP learning changes W_{EE}. Figure 3 shows the synaptic weight distributions for different W_{IE}. For smaller W_{IE}, the ratio at the lower bound decreases and the ratio at the upper bound increases. This is due to the basic property of STDP[6].

For weak inhibition, the network exhibits high firing rates, then a large part of plastic connections dies out (Fig. 3(a)). Then, the network could be fragmented as mentioned before, because too many synapses reaches the lower bound. In fact, the maximum life time in Fig. 2(a) is smaller than that in Fig. 2(b). However, this effect is not apparent in the size distributions. Then, there might be a nonlinear relation between the size and the life time of the neuronal avalanches. On the other hand, in the case of strong inhibition, surviving synapses increase (Fig. 3(c)). However, even though the fraction of strong synapses increases, the excitability in the network is not enough to evoke the



(a)



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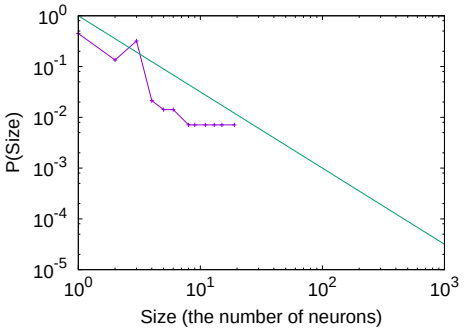
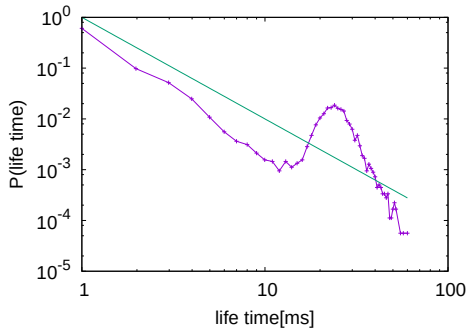
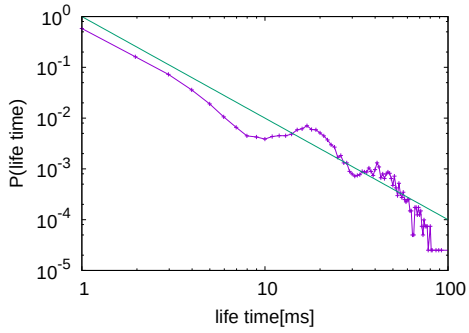


(c)

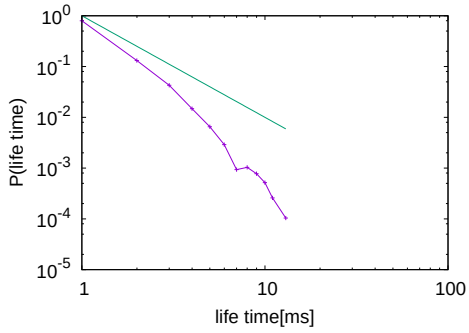
Figure 1: Influence of inhibitory connection weights on the size distributions. W_{IE} is fixed to (a) W_{IE}=−0.15, (b) −0.35 and (c) −0.65. Slopes of the green lines are −1.5.



(a)



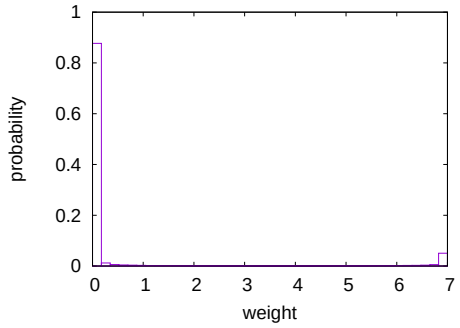
(b)



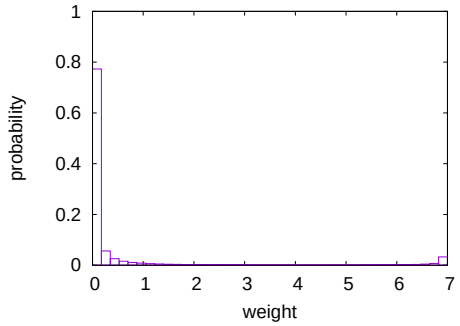
(c)

Figure 2: Influence of inhibitory connection weights on the life time distributions. W_{IE} is fixed to (a) W_{IE}=−0.15, (b) −0.35 and (c) −0.65. Slopes of the green lines are −2.0.

neuronal avalanches. Then, the appropriate strength of inhibition prevents the network from the fragmentation and sustains the appropriate excitability in the network.



(a)



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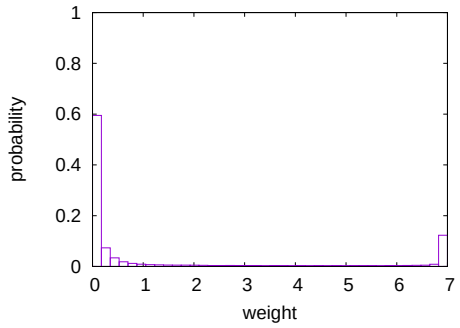

(c)

Figure 3: Synaptic weight distributions of W_{EE} for (a) $W_{\mathrm{IE}} = -0.15$, (b) -0.35 and (c) -0.65.

5. Conclusion

In this paper, we analyzed the influence of inhibitory connection weights on neuronal avalanches. As a result, the slope of the life time distribution changes when W_{IE} was changed from -0.15 to -0.65.

We found that the synaptic weight between neurons influenced the slope of the size and the life time distribution. Changing the value of W_{IE}, the slope of the life time distributions changed. In other words, the balance of excitatory neurons and inhibitory neurons affects the slope of the life time distribution. Moreover, in our simulations neuronal avalanches are realized in a network under the condition of $|W_{\mathrm{EI}}| \gg |W_{\mathrm{IE}}|$. Namely, to reproduce neuronal avalanches, the moderate level of inhibition may be inevitable even in actual neural networks. It is an important future problem to investigate the moderate level of inhibition quantitatively.

Acknowledgments

The research of H. K. was supported by JSPS KAKENHI Grant Number JP16K16127. The research of Y. S. was supported by JSPS KAKENHI Grant Number JP16K16126. The research of K. F. was supported by JSPS KAKENHI Grant Numbers JP16K16138 and JP15K12137. The research of T. I. was supported by JSPS KAKENHI Grant Numbers JP15TK0112 and JP17K00348.

References

[1] J. M. Beggs and D. Plenz, "Neuronal avalanches in neocortical circuits," The Journal of Neuroscience, **23**, 35 (2003): 11167-11177.

[2] X. Li and M. Small, "Neuronal avalanches of a self-organized neural network with active-neuron-dominant structure," Chaos, **22**, 2 (2012): 023104.

[3] H. Kato and T. Ikeguchi, "Spike timing-dependent plasticity in sparse recurrent neural networks," Proceedings of 2012 International Symposium on Nonlinear Theory and its Applications (2012): 485-488.

[4] E. M. Izhikevich, "Polychronization:computation with spikes," Neural Computation, **18**, 2 (2006): 245-282.

[5] G. Bi and M. Poo, "Synaptic modifications in cultured hippocampal neurons: dependence on spike timing, synaptic strength, and postsynaptic cell type," The Journal of Neuroscience, **18**, 24 (1998): 10464-10472.

[6] S. Song, K. D. Miller and L. F. Abbott, "Competitive Hebbian learning through spike-timing-dependent synaptic plasticity," Nature Neuroscience, **3**, 9 (2000): 919-926.