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Wasteful Inhibitions for Paradoxical Spike Propagation in a Recurrent Network

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Abstract—We have demonstrated that spikes propagate in a paradoxical direction in a recurrent network where the distance of inhibitory recurrent connections was anisotropic. In this study, we demonstrate that the paradoxical spike propagation also emerges in a recurrent network where the distance of excitatory recurrent connections is anisotropic. Spikes propagate in the direction where the density of excitatory connections is low and recurrent excitation to be weak. The common feature to the above two cases of directional spike propagation is that spikes propagate in a direction where inhibitory interneurons extend their axons to slightly more distant neurons than excitatory neurons regardless of the density of recurrent excitatory connections. In our network, the inhibitory spikes that arrive at excitatory neurons are too early to prevent the excitatory neurons from firing in the direction of extended inhibitory connections. This suggests that the network causes paradoxical spike propagation due to wasteful inhibitions rather than the anisotropic difference of the density of excitatory connections.

1. Introduction

Lubenov and Siapas have shown that theta oscillations propagate from the septal side to the temporal side in the hippocampal CA1 using animals that run in a track [1]. The CA1 neuron has almost no excitatory recurrent connection to propagate its activity to other neurons. It would be difficult to propagate its activity from the septal side to the temporal side by itself. One of the quite possible hypotheses is that the directional propagation of neuronal activity in the hippocampal CA3 is reflected in the CA1. The speed of propagation along the septotemporal axis observed in disinhibited longitudinal slices of CA3 is consistent with that of the propagation observed in CA1 [1].

Yoshida and Hayashi have demonstrated that a CA3 network model, in which neurons are locally connected, causes radial spike propagation from a stimulus site when the recurrent excitatory connections are subjected to a spike-timing dependent plasticity (STDP) rule [2]. However, the spike propagation emerging in the network was not directional. Spikes propagate radially from the stimulus site.

We have investigated whether the directional spike propagation emerges in a recurrent network by through the modification of recurrent connections and demonstrated that spikes propagate along a paradoxical direction when the distance of inhibitory recurrent connections is anisotropic [3]. Spikes propagated in the direction where axons of inhibitory interneurons extended and recurrent inhibitions were strong.

In this study, we show that the paradoxical spike propagation emerges also in a recurrent network where the distance of excitatory recurrent connections is anisotropic. When the inhibitions were properly strong, spikes propagated in the horizontal direction where recurrent excitations were weak due to the low density of excitatory connections.

The common feature to the above two cases of directional spike propagation is that spikes propagate in a direction where inhibitory interneurons extend their axons to slightly more distant neurons than excitatory neurons. It is expected that excitatory neurons are well inhibited in that direction because they receive inhibitory spikes in advance of excitatory spikes through the extended inhibitory connections, but spikes propagated in the direction. We investigated the mechanisms of the paradoxical spike propagation in terms of the timing of inhibitory spikes. An excitatory neuron received inhibitory spikes from inhibitory interneurons in the horizontal direction before the excitatory neuron fired, as expected. The excitatory neuron also received the inhibitory spikes from inhibitory interneurons in the vertical direction before the excitatory neuron fired. Although the inhibitions were always one step ahead of excitations in both directions, we found that inhibitory spikes arrived at excitatory neurons in the horizontal direction too early to prevent the excitatory neurons from firing. Consequently, the extended inhibitory connections were wasteful, but caused the paradoxical spike propagation regardless of the anisotropic difference of the density of excitatory connections. Finally we discuss conditions for causing directional spike propagation stably in a recurrent network.

2. Methods

2.1. Recurrent Network

A recurrent network consists of excitatory neurons and inhibitory interneurons. Both kinds of neurons are the Izhikevich's model [4]. Equations of ith neuron are as fol-

lows:

$$v_i' = 0.04v_i^2 + 5v_i + 140 - u_i + I_i(t), \quad (1)$$
$$u_i' = a(bv_i - u_i), \quad (2)$$

where v_i is the membrane potential and u_i is the membrane recovery variable. a is the rate of recovery, and b is the sensitivity of the recovery variable. $I_i(t)$ is the inputs from other neurons to ith neuron, and calculated by the following equation:

$$I_i(t) = \sum_j^{N_i} w_{ij} \sum_k^{N_j^{\text{fired}}} \delta(t - t_j^k - \tau_{ij}), \quad (3)$$

where N_i is the total number of presynaptic neurons of i th neuron and w_{ij} means the synaptic weight between ith and jth neurons. N_j^{fired} is the number of firing of jth neuron. $\delta(\cdot)$ is the Dirac delta function and t_j^k is kth firing timing of $j$the neuron. τ_{ij} is the synaptic delay between ith and jth neurons.

$$\text{if } v_i \geq 30, \text{ then} \begin{cases} v_i \leftarrow c \\ u_i \leftarrow u_i + d. \end{cases} \quad (4)$$

If v_i is larger than 30, the neuron fires a spike. Then v_i and u_i are reset to c and $u_i + d$, respectively. The excitatory neuron was modeled by the intrinsic bursting neuron, so that we set parameters as follows: $a = 0.02, b = 0.2, c = -55, d = 5$. We also modeled the inhibitory interneuron as a fast spiking neuron, so that we set parameters as follows: $a = 0.1, b = 0.2, c = -65, d = 2$.

Figure 1 shows a part of the network structure. 10,000 excitatory neurons were placed on 100×100 lattice points. 1,250 inhibitory interneurons were placed uniformly among excitatory neurons. Excitatory neurons were connected to surrounding 26 excitatory neurons and 1–7 inhibitory interneurons randomly selected within each connectable region. On the other hand, inhibitory interneurons were connected to 48 or 66 excitatory neurons randomly selected within each connectable region. The size of a connectable region was defined to allow a neuron to connect with the prescribed number of neurons. The connectable region of a neuron near the border was moved inside to prevent the region from getting out of the network. Each excitatory connection from an excitatory neuron to an excitatory neuron (E–E) or an inhibitory interneuron (E–I) had 1.4–1.7 ms delay, and E–E and E–I have respective synaptic weights of 40 and 30 initially. Each inhibitory connection from an inhibitory interneuron had 0.9–1.1 ms delay, and the synaptic weight was −60; however, the synaptic weight from inhibitory interneuron near the border was −180.

2.2. Learning Rules

We revised STDP functions proposed by Izhikevich [5]. A spike of ith neuron is paired with a arrival spike from jth neuron in the nearest neighbor manner. In each spike pair, the modification rate of a synaptic weight from jth neuron to ith neuron is calculated as follows:

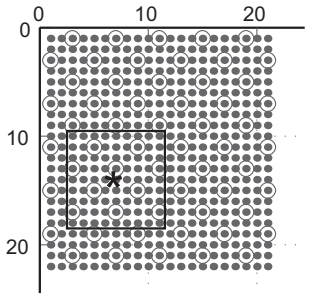


Figure 1: Structure of the recurrent network. Dots and open circles represent excitatory neurons and inhibitory interneurons, respectively. The solid box is a connectable region where a neuron (∗) connects to other surrounding neurons.

$$\Delta w_{ij} = \begin{cases} A_+ \, e^{-\Delta t_{ij}/\tau_{\text{STDP}}} & \text{if } \Delta\, t_{ij} > 0 \\ A_- \, e^{\Delta t_{ij}/\tau_{\text{STDP}}} & \text{if } \Delta\, t_{ij} \leq 0, \end{cases} \quad (5)$$

Δt_{ij} denotes an arrival timing of a spike from jth neuron relative to a spike timing of ith neuron. A_+, A_- are the maximal potentiation and depression rates respectively. τ_{STDP} is the time constant for STDP. We set these parameters as follows: $A_+ = 1.0$ $A_- = -1.2$, $\tau_{\text{STDP}} = 20$. Each synaptic weight was updated at each 1 sec as follows:

$$\Delta W_{ij} \leftarrow \sum \Delta w_{ij} + 0.5\Delta W_{ij}, \quad (6)$$
$$w_{ij} \leftarrow w_{ij} + \Delta W_{ij}, \quad (7)$$

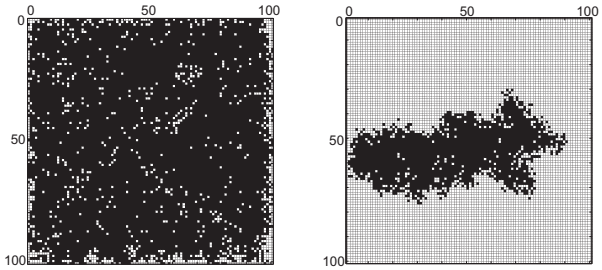
where $\Sigma\Delta w_{ij}$ is the sum of modification rate calculated by Eq.(5) in the last second. Synaptic weights were limited to the range of $10.0 \leq w \leq 60.0$.

3. Results

3.1. Directional Spike Propagation Emerged in the Network

We have demonstrated that the anisotropy of the connectable region of inhibitory interneurons causes the directional spike propagation in a recurrent network [3]. Here, we made the connectable region of an excitatory neuron anisotropic. We set the connectable regions of an excitatory neuron and an inhibitory interneuron as 9 (vertical) × 7 (horizontal) and 9×9, respectively. We examined this network in two conditions where inhibition was weak or strong. Weak and strong inhibitions were realized by setting the number of inhibitory connection as 48 and 66, respectively. In each condition, we observed spike propagation emerged in 30 trials. At the beginning of each trial, we initialized connections of all neurons and chose 35 neurons within the central 15×15 region of the network. The 35

neurons were fired by stimulation applied every 500 msec for 1, 000 sec.



(a) Weak inhibition. (b) Strong inhibition.

Figure 2: Neurons fired during spike propagation.(a) Inhibition is weak. (b) Inhibition is strong. Black squares (■) represent fired neurons.

Figure 2 shows a typical spike propagation emerged in two conditions. When the inhibition was weak, spikes propagated radially and most neurons fired in the network (Fig. 2(a)). Any directional propagation did not emerge in 30 trials. In contrast, when the inhibition was properly strong, directional spike propagation emerged in 14 out of 30 trials (Fig. 2(b)). In other 16 trials, spikes did not propagate from the central region where stimuli were applied.

3.2. Paradox of Directional Spike Propagation

We set the connectable region of an excitatory neuron and an inhibitory interneuron as 9×7 and 9×9, respectively. Thus, the density of excitatory connections was anisotropic. Figure 3 shows the average number of connections from excitatory and inhibitory neurons to an excitatory neuron in each of four directions in a certain trial when inhibition was strong. Excitatory neurons received

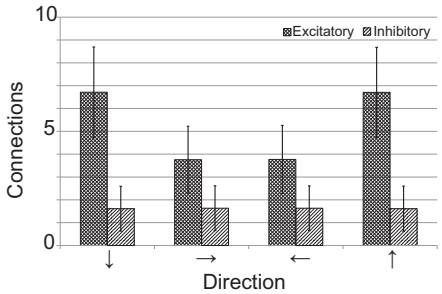


Figure 3: The number of received connections per an excitatory neuron in a network (mean ± SD). Excitatory neurons that are away from the edge of the network were included in the calculation.

relatively many excitatory connections in the vertical directions (↓↑) than the horizontal directions (⇄). On the other hands, the inhibitory connections was the same density in two directions. Thus, it seems to be difficult to propagate in the horizontal direction; however, spikes spread in the direction (Fig. 2(b)).

3.3. Wasteful Inhibitions for Directional Spike Propagation

When excitatory and inhibitory interneurons are the same position, inhibitory interneurons extend their axons to slightly more distant neurons than excitatory neurons in the horizontal direction. It is supposed that inhibitory spikes precede excitatory spikes in the horizontal direction because length of inhibitory connections is longer than that of excitatory connections; consequently, excitatory neurons would be well inhibited. Spikes, however, propagated in that direction (Fig. 2(b)). Therefore, we examined arrival timing of inhibitory spikes at excitatory neurons with respect to firing of those excitatory neurons.

In our network, spike propagation was inhibited only in the vertical direction. Inhibitory spikes seem to affect excitatory neurons anisotropically. Thus, we investigated the arrival timing of inhibitory spikes at excitatory neurons of spike propagation in each direction. We used the activities of neurons in radial spike propagation (Fig. 2(a)) because we could not obtain the activities of neurons in the vertical directions during the directional spike propagation (Fig. 2(b)). As shown in Fig. 4, we divided the network into four regions I–IV in order to investigate arrival timing of inhibitory spikes at excitatory neurons in each of horizontal and vertical directions.

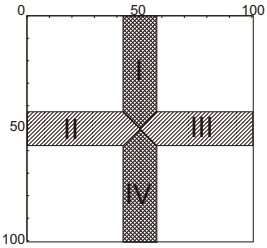
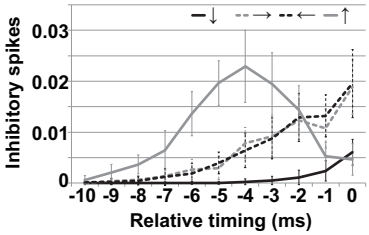


Figure 4: The definition of four regions I–IV in the network. White regions were not included in the calculation.

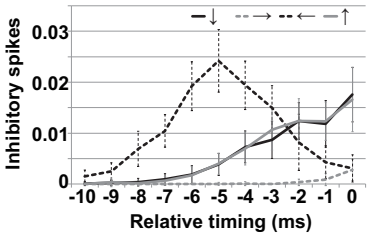
During the radial spike propagation, excitatory neurons received inhibitory spikes in each direction in a particular timing before they fired (Fig. 5). Excitatory neurons in regions I and IV were inhibited first by inhibitory interneurons in the vertical direction and then inhibited by inhibitory interneurons in the horizontal direction (Fig. 5(a), (d)). On the other hand, excitatory neurons in regions II and III were inhibited first by interneurons in the horizontal direction and then inhibited by interneurons in the vertical direction (Fig. 5(b), (c)).

Excitatory neurons in regions I and IV are inhibited by the inhibitory interneurons in the vertical direction around 4 msec before they fired. Excitatory neurons in regions II

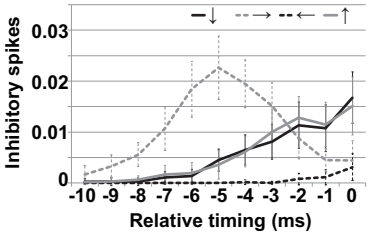
and III are inhibited by the inhibitory interneurons in the horizontal direction around 5 msec before they fired. The membrane potential of an excitatory neuron in our model decays in 5 msec. Hence, the firing of the neuron is affected by inputs especially arrived within 5 msec. The first inhibitions for each region tend to be wasteful especially in regions II and III because of its timing. On the other hand, there were no differences among the secondary inhibitions in each region. Therefore, the wasteful inhibitions allowed the network to propagate spikes only in the horizontal direction.



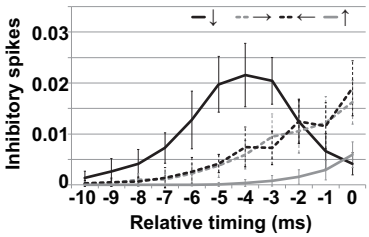
(a) Region I.



(b) Region II.



(c) Region III.



(d) Region IV.

Figure 5: The number of inhibitory spikes received by an excitatory neuron in each direction with respect to the firing of an excitatory neuron. (mean ± SE in 30 trials).

4. Discussions

In this study, we have demonstrated that the paradoxical spike propagation emerges also in a recurrent network where the distance of excitatory recurrent connections was anisotropic. Spikes propagated in the horizontal direction where inhibitory interneurons extend their axons to slightly more distant neurons than excitatory neurons. The inhibitory spikes arrived at excitatory neurons in the horizontal direction, but they were too early to prevent the excitatory neurons from firing. Consequently, the extended inhibitory connections were wasteful, and the wasteful inhibition was a cause of the paradoxical spike propagation.

Spikes propagated in the horizontal direction in the present paper, but the density of excitatory connections was low in the same direction. This suggests that inhibitory connections extending further in comparison with excitatory connections are crucial for determining the direction of spike propagation. If the direction in which inhibitory connections extend farther and the direction in which the density of excitatory connections is higher are in parallel, directional spike propagation may occur more stably.

It has been reported that pyramidal cells extend their axons along the septotemporal axis in CA3 [6]. O-LM cells, which are inhibitory interneurons in CA3, also extend their axons along the same axis [7]. This fact is consistent with the probable condition for causing directional spike propagation stably in a recurrent network. The condition for the directional spike propagation may be inherent in CA3.

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