



# Directional Spike Propagation by Anisotropic Inhibitory Connections Modulated through STDP in a Recurrent Network

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**Abstract**—It has been supposed that theta oscillations are synchronized across the hippocampus. However, it was reported last year that the theta oscillations propagate along the septotemporal axis in the hippocampal CA1. Although this requires that the hippocampal functions are reconsidered based on the propagation of the theta oscillations, it has not been well understood how the directional propagation is produced in the hippocampus. One of the reasonable mechanisms is that directional spike propagation in the hippocampal CA3 is reflected in the CA1 because the CA3 has rich excitatory recurrent connections. In this paper, we investigated whether the directional propagation emerges in a recurrent network, and found that it was produced certainly when the distance of inhibitory connections was anisotropic and weights of excitatory connections from excitatory neurons to inhibitory interneurons were modified through STDP.

## 1. Introduction

It has been supposed that theta oscillations, which play a crucial role in sequence learning of places, are synchronized across the hippocampus. However, Lubenov and Siapas have recently shown that theta oscillations propagate from the septal side to the temporal side in the hippocampal CA1 using animals that run on a track [1].

It would be difficult to understand the directional propagation of neuronal activity in the hippocampal CA1 because the CA1 has almost no excitatory recurrent connection. One of the quite possible hypotheses is that the directional propagation of neuronal activity in the hippocampal CA3 is reflected in the CA1 because the CA3 has rich excitatory recurrent connections. Moreover, 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 also demonstrated that a CA3 network model, in which neurons were locally connected, caused radial spike propagation from a stimulus site when the recurrent excitatory connections are subject to a spike-timing dependent plasticity (STDP) rule [2]. As the distance of local recurrent connections was isotropic in the CA3 network model, the propagation from the stimulus site would be caused radially.

In this paper, we investigated whether the directional spike propagation emerges in a recurrent network in which the distance of recurrent connections is anisotropic. At least excitatory connections between excitatory neurons were modified by a STDP rule in order to cause spike propagation as well as Yoshida and Hayashi model. When the distance of connections was anisotropic and excitatory synapses from excitatory neurons to inhibitory interneurons were not modified by a STDP rule, the present recurrent network caused directional propagation transiently, and then the directional propagation was getting non-directional with the passage of time. However, when the distance of inhibitory connections was anisotropic and excitatory connections from excitatory neurons to inhibitory interneurons were modified by a STDP rule, directional spike propagation was established stably whether the distance of excitatory connections from excitatory neurons is anisotropic or not.

## 2. Methods

### 2.1. Recurrent Network

The recurrent network consists of excitatory neurons and inhibitory neurons. Both kinds of neurons are the Izhikevich's simple model [4]. Equations of the neurons are as follows:

$$v' = 0.04v^2 + 5v + 140 - u + I, \quad (1)$$

$$u' = a(bv - u), \quad (2)$$

where  $v$  is the membrane potential and  $u$  is the membrane recovery variable.  $I$  is the sum of weighted inputs from other neurons.  $a$  is the rate of recovery, and  $b$  is the sensitivity of the recovery variable  $u$ .

$$\text{if } v \geq 30, \text{ then } \begin{cases} v \leftarrow c \\ u \leftarrow u + d. \end{cases} \quad (3)$$

If  $v$  is larger than 30, then the neuron fires a spike, and then  $v$  and  $u$  are reset to  $c$  and  $u + d$  respectively. The excitatory neuron was modeled by an intrinsic bursting neuron, so that we set parameters as follows:  $a = 0.02, b = 0.2, c = -55, d = 4$ . We also modeled the inhibitory interneuron as a fast spiking neuron, so that we set parameters as follows:  $a = 0.1, b = 0.2, c = -65, d = 2$ .

10,000 excitatory neurons were placed on  $100 \times 100$  lattice points. 1,250 inhibitory interneurons were placed uniformly among excitatory neurons. Figure 1 shows a part of the structure of the recurrent network. Each inhibitory neuron ( $\blacktriangledown$ ) was put on an excitatory neuron ( $\triangledown$ ). Each ex-

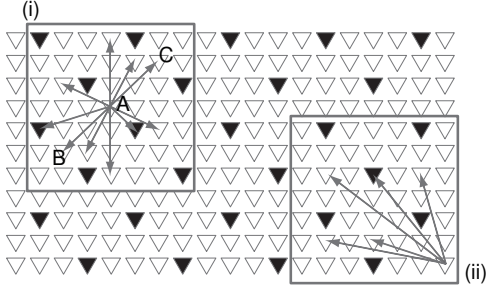


Figure 1: A part of the structure of the recurrent network. Open triangles ( $\triangledown$ ) are excitatory neurons. Filled triangles ( $\blacktriangledown$ ) are inhibitory interneurons, each of which are put on an excitatory neuron. Squares ((i) and (ii)) indicate axon projection fields of a neuron, and solid arrows indicate connections.

citatory neuron was connected to 26 excitatory neurons selected within its projection field (e.g. square (i) in Fig.1) in accordance with the following rule; when a neuron (e.g. A in Fig.1) was connected to another neuron randomly selected in the field (e.g. B in Fig.1), then neuron A was also connected to the neuron on the opposite side of the neuron B (e.g. C in Fig.1). Moreover, the excitatory neuron was connected to 1–7 inhibitory interneurons randomly selected within the projection field. Connections had a delay of 1.4–1.7 ms and the initial excitatory synaptic weights were 40. On the other hand, each inhibitory interneuron was connected to 64 excitatory neuron within its projection field in accordance with the above rule. Inhibitory connections had a delay of 0.9–1.1 ms, inhibitory synaptic weights were  $-55$ . Projection fields of neurons near the border were moved inside for keeping the number of neurons within the fields (e.g. square (ii) in Fig.1), and the inhibitory synaptic weights in the field were  $-82.5$ .

## 2.2. Learning Rules

We revised the STDP function proposed by Izhikevich [5]. Synaptic weights between excitatory neurons were updated by the following STDP rule. The magnitude of synaptic change  $\Delta w$  was calculated as follows:

$$\Delta w = \begin{cases} A_+ e^{-\Delta t/\tau} & \text{if } \Delta t > 0 \\ A_- e^{\Delta t/\tau} & \text{if } \Delta t \leq 0, \end{cases} \quad (4)$$

$\Delta t$  denotes the relative timing between the arrival time of a spike from the pre-neuron and the firing time of the post-neuron.  $A_+$  and  $A_-$  are the maximal potentiation and depression rates respectively.  $\tau$  is the time constant. We set these parameters as follows:  $A_+ = 1.0$ ,  $A_- = -1.2$ ,  $\tau = 20$ .

Each synaptic weight was updated at intervals of 1 sec as follows:

$$w \leftarrow w + w_{\text{now}} + w_{\text{past}}, \quad (5)$$

$$w_{\text{past}} \leftarrow 0.9 (w_{\text{now}} + w_{\text{past}}), \quad (6)$$

where  $w$  is the synaptic weight, and  $w_{\text{now}}$  is the sum of  $\Delta w$  over the last one second.  $w_{\text{past}}$  denotes the sum of past synaptic changes. After the update of synaptic weight by Eq. (5),  $w_{\text{past}}$  was updated by Eq. (6). The current synaptic change  $w_{\text{now}}$ , therefore influenced following updates of the synaptic weight. The synaptic weights are limited to the range of  $20.0 \leq w \leq 80.0$ .

## 3. Results

### 3.1. Comparison among spike propagations developed in three conditions (control, EA, and IA)

Here, we defined three conditions (control, EA, and IA). The first one is a non-anisotropic condition that is similar to those of Yoshida and Hayashi model as control. Excitatory and inhibitory neurons have connections within a square region in which the distance of connections is the same in vertical and horizontal directions. The second one is an excitatory anisotropic (EA) condition where an excitatory neuron has connections within a rectangle region in which the distance of connections is different in vertical and horizontal directions. The third one is an inhibitory anisotropic (IA) condition that an inhibitory neuron has connections within a rectangle region similarly to the EA condition. In fact, O-LM inhibitory interneurons in CA3 extend their axons to distant neurons in the septotemporal direction [3]. Table.1 shows the size of the projection field in three conditions.

Table 1: Size of the projection fields in three conditions

	Excitatory neuron	Inhibitory neuron
Control	$7 \times 7$	$9 \times 9$
EA	$9 \times 5$	$9 \times 9$
IA	$7 \times 7$	$11 \times 7$

To compare among the developing processes of spike propagation in three conditions, we observed spike propagation in 10 trials in each condition. In each trial, connections between neurons were initialized as shown in Table.1. Here, we applied stimuli ( $I = 200$ ) to 15 neurons within the central  $5 \times 5$  region of the network at intervals of 500 msec. The 15 neurons were randomly chosen every trial. Stimulation was started 5 sec after the beginning of the simulation, then the stimuli were applied to the same neurons to the end. The equations of neurons were updated by the fourth-order Runge-Kutta method every 0.1 msec for 1,000 sec. Synaptic weights between excitatory neurons were modified by the STDP rule, and the synaptic weights from exci-

tatory neurons to inhibitory interneurons were not modified here.

Figure 2 shows typical patterns of spike propagation obtained in each conditions. The patterns are remarkably different within initial 100 sec. In the control condition, spikes propagated radially in 8 trials out of 10 (Fig.2 (a), left). On the other hand, in the EA and IA conditions, the directional spike propagation was caused in a particular direction (Fig.2 (b) and (c), left panels). However, the directional spike propagation changed to the non-directional propagation with the passage of time in all conditions (Fig.2 (a)–(c), right panels). The directional spike propagation was just transient even when the distance of the recurrent connections was anisotropic.

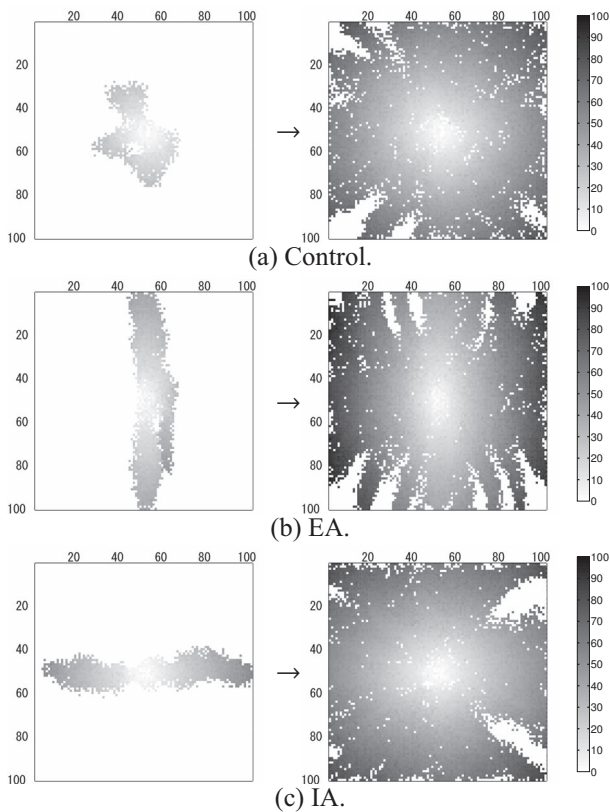


Figure 2: Typical patterns of spike propagation produced by local stimulation after 100 sec (left panels) or 1,000 sec (right panels) from the onset of the simulation. Gray scale of each dot indicates the firing timing of a neuron for 100 ms following each input.

### 3.2. Directional spike propagation in a network with strong recurrent inhibition

The directional propagation of neuronal activity was transiently produced, and it became non-directional with time, as shown in the previous section. Probably, when integrated EPSPs evoked by firing of nearby excitatory neurons happened to overcome integrated IPSPs evoked by fir-

ing of nearby inhibitory interneurons in a particular direction, spike propagated and the excitatory connections were strengthened in the direction through the STDP rule. Although the directional propagation grew transiently, other directional propagations would also grow little by little and the propagation became non-directional finally. Thus, we investigated whether the directional propagation was established stably by stronger inhibition that balanced with strengthened excitation. Here, inhibitory synaptic weights were set as  $-70$  ( $-105$  for inhibitory interneurons near the border).

In the control condition, the spike propagation was not produced in all trials. Figure 3 shows typical spike propagation patterns produced in the EA and IA conditions with the stronger inhibition. The directional spike propagation was produced in a particular direction in both conditions (Fig.3 (a), (b)). However, in the EA condition, the directional propagation was produced in only 5 trials out of 10 (Fig.3 (a), left) and propagation was non-directional in other 5 trials (Fig.3 (a), right). In the IA condition, the directional propagation was produced in only 2 trials out of 10 (Fig.3 (b), left) and propagation was not produced in other 8 trials (Fig.3 (b), right)

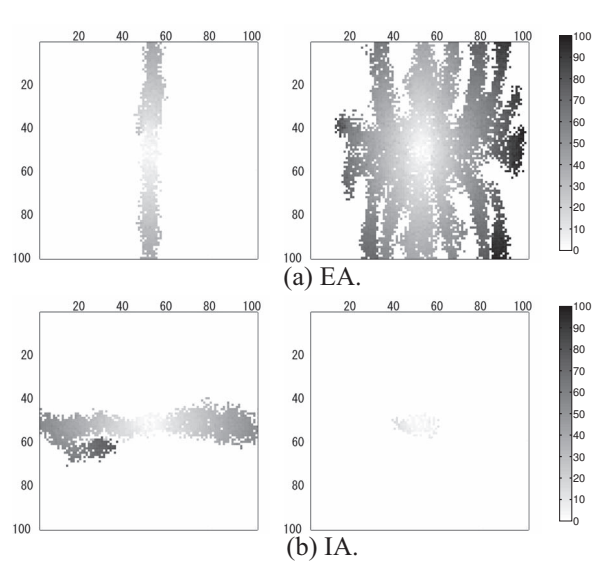


Figure 3: Typical patterns of spike propagation produced by local stimulation after 1,000 sec from the onset of the simulation. Gray scale of each dot indicates the firing timing of a neuron for 100 ms following each input.

### 3.3. Stable directional spike propagation by inhibition modified through a STDP rule

Although the strength of excitatory connections between excitatory neurons was dynamically changed by STDP, strength of inhibitory recurrent connections was fixed in the previous section. It is, therefore, supposed that recurrent inhibition could not properly counteract the strength-

ened recurrent excitation. In this section, excitatory connections from excitatory neurons to inhibitory interneurons were modified through STDP because the inhibitory connections are dynamically modified in the hippocampus [6]. Excitatory and inhibitory synaptic weights were set to 60 and  $-120$  ( $-180$  for inhibitory interneurons near the border) respectively. The weights of excitatory connections were updated within the range of  $10.0 \leq w \leq 80.0$ . Inhibitory interneurons were connected to 66 excitatory neurons within each projection field. Thirty neurons within the central  $15 \times 15$  region were chosen to be stimulated. The other settings were the same as in section 3.1.

Figure 4 shows the typical patterns of spike propagation produced in each condition. In the control and the EA conditions, spike propagation, which was not directional propagation, was produced in 8 trials and 2 trials out of 10 respectively (Fig.4 (a) and (b)). In the other trials, spike propagation was not produced. On the other hand, the directional spike propagation along the vertical axis was produced in the IA condition (Fig.4 (c)). Although, the width of the directional propagation was wider than that of the propagation in Fig.3 (b), the directional propagation shown in Fig.4 (c) was caused in 8 trials out of 10; spike propagation was incomplete in one trial, and no spike propagation was produced in another trial.

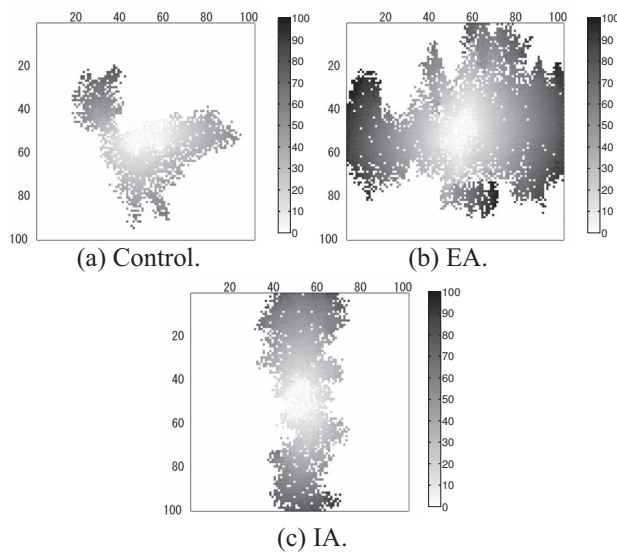


Figure 4: Typical patterns of spike propagation produced by local stimulation after 1,000 sec from the onset of the simulation. Gray scale of each dot indicates the firing timing of a neuron for 100 ms following each input.

#### 4. Discussions

We investigated whether directional propagation of neuronal activity was produced in a recurrent network, and found that the directional propagation was produced certainly when the distance of inhibitory connections was

anisotropic and weights of the excitatory connections from excitatory neurons to inhibitory interneurons were modified through a STDP rule, regardless of the anisotropy of the distance of the recurrent connections from excitatory neurons. It has been reported that inhibitory interneurons, O-LM cells, have axons, which extend along the septotemporal axis in CA3 [3]. This fact is consistent with the IA condition in the present model. It has also been reported that excitatory synaptic connections from excitatory neurons to inhibitory interneurons have synaptic plasticity [6]. Although it has not been elucidated whether the plasticity of the excitatory synaptic connections is dependent on spike-timing, it would be plausible that the synapses are modified through a STDP rule. The present results suggest that anisotropy of the distance of inhibitory connections determines the direction of spike propagation, and the synaptic change in excitatory connections from excitatory neurons to inhibitory neurons ensures that directional propagation is caused in a recurrent network. However, it seems to be paradoxical that spikes propagate in the vertical direction because connections of inhibitory interneurons extend along the vertical axis in the present model and recurrent inhibition is expected to be stronger in the vertical direction than in the horizontal direction. It is an interesting future problem to investigate how recurrent inhibition changes spatiotemporally during the development of the directional propagation and understand the mechanism of the propagation in the direction of connections of inhibitory interneurons.

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