Inhibition-mediated organization of cortical circuit
through the spike-timing-dependent plasticity

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Abstract—It was demonstrated in the paper that the spike-timing-dependent plasticity among excitatory neurons was modulated under the different connectivity of inhibitory neurons. As a result, it was shown that inhibitory circuit has not only simple activity regulation but also an active role on the cortical network formation.

1. Introduction

Cortical network shows various activity under the inhibition provided by interneurons. Such an inhibition has been considered as an activity suppression of principle neurons. Recent electrophysiological studies revealed that the interneurons played active role in cortical computation [1–4]. For example, the ocular dominance activity in primary visual cortex is significantly modulated by the inhibitory circuits that drastically change their dynamics depending on sensory experience during critical period [1]. However, it is still unclear how the inhibitory network behaves on synaptic plasticity in local circuit level.

Here, we computationally examined behavior and organization of the network consisted of excitatory and inhibitory neuronal groups. Synapses among excitatory neurons are mediated by the spike-timing-dependent plasticity (STDP) [5,6]. It was performed how network organization through STDP were modulated corresponding to various connectivity of interneurons. The variety of inhibitory connection was introduced such that the ratio of feedforward- and feedback- inhibition was systematically modified while synaptic weight distribution was constant. As a result, we demonstrate the functional role of inhibitory circuit in cortical network organization through the synaptic learning.

2. Methods

2.1. Neuron model

We employed the neuron model described by Izhikevich’s formulation that provides two-dimensional system of ordinary differential equations [7,8]. Dynamics of membrane potential ($v$) and a membrane recovery variable ($u$) are described as follows:

$$\frac{dv}{dt} = 0.04v^2 + 5v + 140 - u + I,$$  (1)

$$\frac{du}{dt} = a(bv - u).$$  (2)

The formulation can guarantee both of biologically plausible behavior like Hodgkin-Huxley model and computational efficiency like an integrate-and-fire neuron model.

The neuron model can provide several types of spiking activity experimentally observed in many brain regions just by modifying the four parameters, that is $(a, b, c, d)$. As excitatory and inhibitory neurons in our model network, the regular spiking model given as $(0.02, 0.2, -65, 8)$ and fast...
spiking model described as (0.1, 0.2, -65, 2), respectively [7, 8]. The $v$ and $u$ reset themselves after every spikes as follows:

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

The differential equation of $v$ also has a variable for input current, $I$, to be introduced in the following section.

2.2. External input

In the present study, individual neuron model receives four types of input currents which are DC ($I_{DC}$), pulse current ($I_{\text{pulse}}$), synaptic current via plastic or non-plastic synapses ($I_{\text{syna}}$), and background noise:

$$
I = I_{DC} + I_{\text{pulse}} + I_{\text{syna}} + 2 \sqrt{D} \zeta. \quad (4)
$$

In order to implement membrane fluctuation mimicking cortical neurons, DCs and noisy inputs described by Gaussian white with intensity $D$ were applied onto all neurons. Through the entire simulation, the amplitudes of DC were set to 5 mV/ms and $D$ was set to 10 (mV/ms)$^2$. Furthermore, the pulse currents as external sensory stimuli were repetitively applied onto excitatory neurons that were divided into two groups (Fig. 1A). Each group individually received repetitive stimulation for every 30 ms with 5 ms duration. To provide primitive sequential stimulation, the stimulation onto second group (E2) has 10 ms delay compared with the stimulation onto first group (E1) stimulation (Fig. 1B).

All types of synapses are described as the following equation (Eq. 5) with gating variable $s(t)$ obeying first order kinetics (Eq. 6).

$$
I_{\text{syna}} = -g_{\text{syna}} s(t)(v - E_{\text{syna}}) \quad (5)
$$

The equilibrium potential, $E_{\text{syna}}$, was set to 0 mV and -70 mV for excitatory and inhibitory synapses, respectively. All the synaptic conductance, namely, $g_{\text{syna}}$ of excitatory-to-excitatory (E-to-E), excitatory-to-inhibitory (E-to-I), inhibitory-to-excitatory (I-to-E), and inhibitory-to-inhibitory (I-to-I) synapses had 0.04 mS. The time varying variable, $s(t)$, is a gate function obeying this:

$$
\tau_s \frac{ds}{dt} = -s + \delta(t - t_{\text{spike},i}) \quad (6)
$$

where $\tau_s$ indicates a decay constant and was set to 5 ms. The delta function is the Dirac’s delta, which manages the occurrence of an $i$-th spike at $t_{\text{spike},i}$.

2.3. Network organization

Our network included 100 excitatory and 100 inhibitory neuronal population (Fig. 1A). In the network, the E-to-E had all-to-all connectivity at initial state. On the other hands, other connectivity, that is, E-to-I, I-to-E, and I-to-I, the connection probability is 0.5. The synaptic weights of E-to-E synapses are modified through STDP while other synapses are non-plastic.

It is examined how the E-to-E synapses are organized through STDP under the feedforward- or feedback-dominant inhibitory network (FFInet/FBInet). In present simulation, feedback inhibition is defined whether specific excitatory and inhibitory neuronal pair has reciprocal excitatory and inhibitory synaptic connection. In other word, whether an excitatory neuron has the direct and shortest inhibition from an inhibitory neuron as a corresponding partner.

To investigate the effect of the feedback inhibition, we introduced the rewiring variable for E-to-I and I-to-E that enables us to modulate the number of the neuron pairs having the feedback inhibition. The range of the variable is [-1.0, 1.0], the positive/negative value corresponds that inhibition is feedback-dominant/feedback-dominant network. The absolute value is rewiring probability. For example, if the variable is 1.0, synapses between excitatory and inhibitory neurons are rewired such that all the feedforward connection was disconnected and rewired to feedback inhibition while conserving the number and weight of entire synapses (Fig. 1C). On the other hand, if the variable is -1.0, feedback inhibition was disconnected and rewired to feedforward connection.

3. Results

We applied repetitive pulse stimulation to two different neuron groups, E1 and E2. E2 was stimulated right after stimulation of E1 with 10 ms delay. In the beginning of a simulation, the spiking activity of excitatory neuron seems uniform (Fig. 2, upper panel). The E-to-E synapses are persistently modified through the repetitive stimulation and
STDP learning. As a result, the network activity was gradually adjusted to external inputs. A raster plot showed the difference of the activities between E1 and E2.

One can see the time evolution of average synaptic weight and a distribution of synaptic weight in the middle and lower panel of figure 2. At the initial stage, synaptic weights were significantly modified through STDP. In the final stage, the shape of weight distribution became stable and the average synaptic weight converged to a stationary value. The additive STDP introduced in the present simulation typically leads to strong competition among synapses [6]. In other words, synaptic weight showed bimodal distribution with extremely strong and weak peaks.

We performed and compared feedback-dominant inhibitory network (FBInet) and feedforward-dominant inhibitory network (FFInet) (Fig. 3). In both cases, STDP provided the same shape of a weight distribution at the final stage after learning. However, a synapse matrix was clearly different. In the FBInet, the synapses from E1-to-E2 was relatively strengthened compared with E2-to-E1. On the other hand, the E2-to-E1 synapses were potentiated in FFInet.

Next, we explored whether the tendency is robust in multiple trials. Figure 4 summarizes asymmetricity of a weight matrix in final stage of a simulation. It was revealed that the E1-to-E2 synapses was consistently strengthened in the FBInet. In contrast, such a tendency was disappeared and much trial variability was shown in FFInet.

4. Discussion

We performed a simulation of neuronal population including excitatory and inhibitory neurons. And we investigated how the connectivity of inhibitory circuit affects population dynamics and network structure among excitatory neurons mediated by STDP learning.

We revealed that our network alternatively acquired asymmetric connectivity through STDP depending on the connectivity of inhibitory circuit, that is, FBI or FFI network. In particular, the network could straightforwardly strengthen the E1-to-E2 synapses for sequential timing of external stimulation in the FBInet while the opposite directional synapses are strengthened in the FFInet. It was revealed that the inhibitory circuit could modulate the memory formation of principle neurons for the external stimulation with temporal order through their various connectivity.

References


