Paper:

Synchrony-Induced Attractor Transition in Cortical Neural Networks Organized by Spike-Timing Dependent Plasticity

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Recent studies have shown that synchronous neural activity in the cortex area occurs related to behavior or recognition of animals, which suggests that such neural activity involves in information processing. Functions enabled by synchronous firing, however, are still unknown. Results reporting that a transition between recall states of associative memory is induced by external synchronous spikes in a neural network formed by spike-timing dependent plasticity indicate the possibility of a function of synchronous neural activity as a transition signal, requiring further examination using detailed cortical neuron models [1]. We introduced a mathematical model of pyramidal and fast-spiking cortical neurons based on Hodgkin-Huxley, and confirmed the transition between recall states through synchronous spike inputs in detailed neuron models.

Keywords: spike-timing dependent plasticity, neural synchrony, neural network, cortical neuron model

1. Introduction

Information processing by neural brain circuits, which is a typical example of an adaptive system, is realized by individual neurons mutually transmitting messages through synapses by generating electrical signals called spikes. This information exchange among neurons through spikes enables neural circuitry to conduct overall information processing in the brain. Therefore, the synaptic network connecting neurons – a neural network – is basic to information processing in the brain. Thus, the elementary process of adaptation in neural brain circuits arises in the modulation of synaptic connections, called synaptic plasticity. Synaptic plasticity changes the neural network by changing synaptic connection strength between neurons based on neuronal activity in response to external stimuli. Therefore, the neural brain circuit is a system that adapts its behavior to a new environment through synaptic plasticity.

Synaptic plasticity was first described by the Hebb rule [12], which states that synaptic connection changes depending on pre- and post-synaptic neuronal activity. Such activity has been described by the firing rate alone and was considered independent of spike time. It was found, however, that spike-timing dependent plasticity (STDP) indicates that plasticity is based on spike sequence and timing between pre- and post-synaptic neurons (**Fig. 1**) [4, 6, 15, 20, 24, 27]. This discovery of STDP provides a neural basis for information processing based on a temporal structure of spike sequences. Furthermore, reports have focused on firing correlation, especially on synchronous firing between neurons, mainly for those in the cortex [10, 11, 19]. This synchronous spike activity occurs selectively depending on external stimulus input or tasks such as behavior, implying the coding and transmission of information through synchronous spikes.

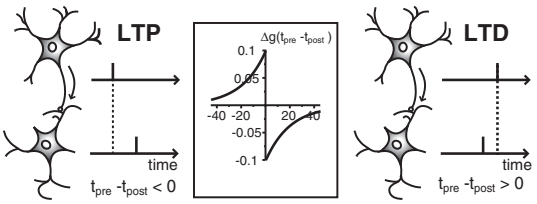

Fig. 1. Spike-timing dependent plasticity (STDP). In synaptic plasticity as an elementary process of learning, LTP or LTD is evoked depending on spike timing. When a post-synaptic neuron fires after pre-synaptic neuron firing, the synaptic connection strength increases. In contrast, if post-synaptic neuron firing proceeds pre-synaptic firing, the synaptic connection strength decreases.

These findings imply possible information processing by neural networks based on spike time [5, 7]. It remains unknown, however, just what information processing is realized by the temporal structure of the spike sequence. Many questions remain unanswered as to what the network structure generated by STDP is, what functions are provided, and what information synchronous spike activity represents, requiring further study [8, 9, 22, 23].

For this reason, we studied the function of the STDP-based network, focusing on the following points (**Fig. 2**): Does the STDP-based network memorize temporal spike patterns given from external stimuli? If so, how does memorized state of the network respond to correlated

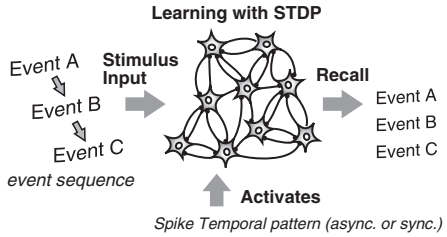

Fig. 2. Scheme of neural network model formed by STDP learning rules and problems posed. When spike patterns corresponding to external events are applied to the neural network as stimulus input, how is the neural network organized by STDP? Does the neural network memorizes given spike patterns? What effect does the correlation between the incoming spikes have on network behavior?

spikes from external input, especially to synchronized spikes?

In previous work, we found that STDP learning creates associative memory circuits to memorize spike patterns, and that the recalled state transits to the next induced by the synchronous activity of input spikes based on the sequence of given stimuli in the learning period [1]. This result indicates that synchronous neural activity may act as a transition signal to the next memorized state in associative memory, and is of interest related to higher functions. The model, however, is neurophysiologically unnatural because recurrent excitatory or inhibitory connections are formed by the same neuron group. The model uses the Leaky Integrate-and-Fire Model, as a single-neuron model, requiring additional experiments with more detailed neuron models. In this way, we improve on the previous model by introducing a more detailed cortical neuron model.

2. Methods

2.1. Single-Neuron Model

We take up two typical single neurons consisting of cortical neural networks, i.e., the excitatory pyramidal neuron and the inhibitory fast-spiking neuron.

A mathematical model of the excitatory pyramidal neuron is expressed in the following equation [2]:

$$C_{\mathrm{m}}\frac{dV}{dt} = -I_{\mathrm{Na}} - I_{\mathrm{K}} - I_{\mathrm{Ca}} - I_{\mathrm{SK}} - I_{\mathrm{L}} + I_{\mathrm{app}} \quad \ldots (1)$$

where membrane capacitance $C_{\mathrm{m}} = 1\ \mu\mathrm{F/cm^2}$, and $I_{\mathrm{Na}}, I_{\mathrm{K}}$ represent voltage-dependent ion channel currents described by Hodgkin-Huxley formalism.

$$I_{\mathrm{Na}} = g_{\mathrm{Na}} m^3 h (V - E_{\mathrm{Na}}) \quad \ldots (2)$$

$$I_{\mathrm{K}} = g_{\mathrm{K}} n^4 (V - E_{\mathrm{K}}) \quad \ldots (3)$$

$$\frac{dx}{dt} = \Phi_x[\alpha_x(V)(1-x) - \beta_x(V)x], \quad (x = m, h, n) \quad \ldots (4)$$

$$\alpha_m = \frac{-0.1(V+25)}{\exp(-0.1(V+25))-1}$$
$$\beta_m = 4\exp(-(V+50)/12) \quad \ldots (5)$$

$$\alpha_h = 0.07\exp(-(V+42)/10)$$
$$\beta_h = 1/\exp(-0.1(V+12)+1) \quad \ldots (6)$$

$$\alpha_n = \frac{-0.01(V+26)}{\exp(-0.1(V+26))-1}$$
$$\beta_n = 0.125\exp(-(V+36)/25) \quad \ldots (7)$$

where m, n and h represent gating variables of activation and inactivation, turning each ion channel on or off via voltage dependence. Parameters are follows: $g_{\mathrm{Na}} = 130$, $g_{\mathrm{K}} = 35\ [\mathrm{mS/cm^2}]$, $E_{\mathrm{Na}} = 55$, $E_{\mathrm{K}} = -97$ [mV]. I_{Ca} is similarly described as follows:

$$I_{\mathrm{Ca}} = m^2 I_{\mathrm{GHK}} \quad \ldots (8)$$

$$\frac{dm}{dt} = \Phi_m[\alpha_m(V)(1-m) - \beta_m(V)m] \quad \ldots (9)$$

$$\alpha_m = \frac{1.6}{1+\exp(-0.072(V-5))}$$
$$\beta_m = \frac{0.002(V+8.69)}{(\exp((V+8.69)/5.36)-1)} \quad \ldots (10)$$

$$I_{\mathrm{GHK}} = P_{\max} V([\mathrm{Ca^{2+}}]_{\mathrm{in}} - [\mathrm{Ca^{2+}}]_{\mathrm{out}}\xi)/(1-\xi)$$
$$\xi = \exp(-2FV/RT) \quad \ldots (11)$$

where $F = 96.5$ C/mol, $T = 293$ K, $R = 8.31$ J/(K·mol), $P_{\max} = 0.01\ \mu\mathrm{A/(\mu M{\cdot}mV{\cdot}cm^2)}$. The temperature factor is set constant: $\Phi_x = 10$. I_{SK} is expressed as,

$$I_{\mathrm{SK}} = g_{\mathrm{SK}} m (V - E_{\mathrm{SK}}) \quad \ldots (12)$$

$$\frac{dm}{dt} = \frac{m_\infty[\mathrm{Ca^{2+}}]_{\mathrm{in}} - m}{\tau_{\mathrm{SK}}[\mathrm{Ca^{2+}}]_{\mathrm{in}}} \quad \ldots (13)$$

$$m_\infty = \frac{[\mathrm{Ca^{2+}}]_{\mathrm{in}}}{[\mathrm{Ca^{2+}}]_{\mathrm{in}} + K_{d,\mathrm{SK}}}$$
$$\tau_{\mathrm{SK}} = \frac{\psi_{\mathrm{SK}}}{[\mathrm{Ca^{2+}}]_{\mathrm{in}} + K_{d,\mathrm{SK}}} \quad \ldots (14)$$

where $g_{\mathrm{SK}} = 0.85\ \mathrm{mS/cm^2}$, $E_{\mathrm{SK}} = -97$ mV, $K_{d,\mathrm{SK}} = 0.4\ \mu$M, $\psi_{\mathrm{SK}} = 2.8\ \mu$M·msec. Calcium concentration $[\mathrm{Ca}]^{2+}$ in I_{Ca} and I_{SK} equations are described by,

$$\frac{d[\mathrm{Ca^{2+}}]_{\mathrm{in}}}{dt} = -\eta I_{\mathrm{Ca}} + k_-[\mathrm{B}]O_c - k_+[\mathrm{Ca^{2+}}]_{\mathrm{in}}[\mathrm{B}](1-O_c) - g_{\mathrm{pump}}\frac{[\mathrm{Ca^{2+}}]_{\mathrm{in}}}{[\mathrm{Ca^{2+}}]_{\mathrm{in}} + K_{\mathrm{m,pump}}} \quad \ldots (15)$$

$$\frac{O_c}{dt} = -k_- O_c + K_+[\mathrm{Ca^{2+}}]_{\mathrm{in}}(1-O_c) \quad \ldots (16)$$

where [B] represents total Ca buffer in cells, O_c represents the binding ratio to the Ca buffer. $[\mathrm{Ca^{2+}}]_{\mathrm{in}}$ flows into cells by I_{Ca}, which is rapidly trapped by the Ca buffer and extruded slowly by the pump. Therefore, each parameter is set as follows: $k_- = 0.3\ \mathrm{msec^{-1}}$, $k_+ = 0.1\ \mathrm{msec^{-1}}\ \mu\mathrm{M^{-1}}$, $K_{\mathrm{m,pump}} = 0.75\ \mu$M, $g_{\mathrm{pump}} = 3.6\ \mu$M/msec, [B] = 30 μM, $\eta = 0.027$, $[\mathrm{Ca^{2+}}]_{\mathrm{out}} = 2100\ \mu$M. Leak current is given by $I_{\mathrm{L}} = g_{\mathrm{L}}(V - V_{\mathrm{L}})$, where $g_{\mathrm{L}} = 0.13\ \mathrm{mS/cm^2}$, $V_{\mathrm{L}} = -68.8$ mV. I_{app} represents an external input current by synaptic connections. A typical firing response when a constant current is applied to the single excitatory neuron