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Hebbian and Coincident Learning Dynamics at Proximal and Distal Dendrites in A Hippocampal Neuron

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Abstract— In hippocampal CA1 pyramidal neurons, action potentials are backpropagated into the dendrite, which affects the induction of synaptic plasticity. Recent studies report that these dendritic backpropagating action potentials are dichotomy and regulated depending on the distance from the soma. According to these studies, we assume that the dynamics of synaptic modification in a hippocampal CA1 pyramidal neuron has a difference between proximal dendrite (PD) and distal dendrite (DD): a rule of synaptic modification in PD depends on pre- and postsynaptic neuron's spike timing ("Hebbian learning rule"), and in DD depends on the coincidence between presynaptic neurons' spikes("coincident learning rule"). In computer simulations, we investigate characteristics of synaptic plasticity on these dynamics. The results showed that the only synapses on PD which receive synchronous spikes were strengthened. But on DD, not only synapses which receive synchronous spikes but also other synapses which receive asynchronous spikes are strengthened, in spite of low frequency. These results suggest that spatial information of higher frequency are mainly coded by the Hebbian rule on PD and temporal information of spike patterns are mainly coded by the coincident rule on DD.

1. Introduction

Hippocampus is one of the most important functional elements in the brain for learning and memory. Synaptic plasticity have been investigated about relationship of learning and memory, and learning rules of synaptic modification have been proposed by many researchers.

In physiological experiments, hippocampal CA1 neurons are highly sensitive to spatiotemporal patterns of input stimuli [8]. One series of experiments showed that LTP of hippocampal CA1 neurons were highly sensitive to the temporal pattern of stimuli: when the mean rate of the stimuli was held constant, and stimuli which had negative correlation in successive inter-stimulus intervals produced smaller LTP, whereas those that had positive correlation produced larger LTP than those that had no correlation [9]. As a rule of synaptic modification, Hebb postulated an idea that the synaptic transmissions is strengthened only if the pre- and post-synaptic elements are activated simultaneously. This rule has been assured widely in various cortical areas and modified variously. However, these Hebb rules are not sensitive to the temporal input spikes patterns. Thus, Tsukada et al. assume that synaptic efficacy of hippocampal CA1 pyramidal neurons are modified by "spatiotemporal learning rule" [8][10]. This rule indicates that synaptic weights are modified by local spikes among presynapic neurons and temporal synaptic weights.

In hippocampal CA1 pyramidal neurons, action potentials are induced in the axon and backpropagated into the dendrites, which affect the induction of synaptic plasticity [5]. Recent studies reported that these dendritic backpropagating action potentials are dichotomy and attenuated depending on distance from the soma [2][3]. According to these studies, we assume that synapses on proximal dendrites and on distal dendrites differ in the dynamics of synaptic plasticity. Thus we construct a model of hippocampal CA1 pyramidal neuron. This model has different synaptic modification dynamics that depend on distance from the soma. We assume that modification of synapses on proximal dendrites at distal dendrite depends on pre- and postsynaptic neuron's spike timing ("Hebbian learning rule"), on the other hand, on distal dendrites depends on the coincidence of presynaptic neuron's spikes ("coincident learning rule") because the backpropagating action potentials are vanishingly small. In this paper, we investigate the differences of synaptic distributions on each dynamics, when asynchronous spikes with different frequencies or local synchronous spikes are input.

2. The Model

In this study , we assume that the hippocampal neural network consists of one postsynaptic neuron (a pyramidal neuron) and N excitatory presynaptic neurons. Each synapse is modified by the dynamics which depend on the distance from soma. For simplicity, we divided its dendrites into two layers: proximal dendrite (PD) and distal dendrite (DD). Area of PD is *Layer* I and DD is *Layer* II, and each excitatory input neuron N project on both layer (See Fig.1).

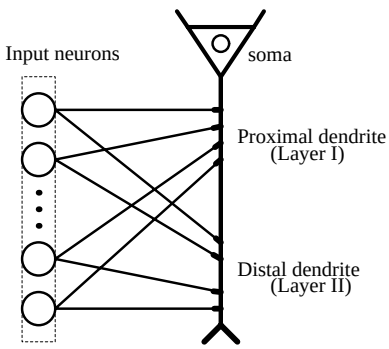


Figure 1: Structure of the hippocampal CA1 neural network.

2.1. The model of a postsynaptic neuron

We adopt an integrate-and-fire neuron model as a postsynaptic neuron, whose subthreshold membrane potential $V(t)$ obeys the following equation:

$$\frac{dV(t)}{dt} = -\frac{1}{\tau}V(t) + RI(t) , \tag{1}$$

where τ is the time constant of neuronal membrane decay. When $V(t)$ reaches the threshold θ, a spike is generated and the membrane potential is reset to V_{reset} where $0 < V_{reset} < \theta$. For simplicity, we set V_{reset} to 0. $RI(t)$ is the amount of synaptic inputs at time t, which is represented by the following equation:

$$RI(t) = \sum_{i=1}^{N} w_i^{\mathrm{I}}(t)x_i(t) + \sum_{i=1}^{N} w_i^{\mathrm{II}}(t)x_i(t) , \tag{2}$$

where $x_i(t)$ is an input from $i(= 1, ..., N)$th presynaptic neuron at time t, $w_i^{\mathrm{I}}(t)$ represents the weight of synaptic connection in *Layer* I from i neuron, and $w_i^{\mathrm{II}}(t)$ represents the weight of synaptic connection in *Layer* II from i neuron. The input pattern from i presynaptic neuron is the same but the synaptic modifications rules are different between in *Layer* I and in *Layer* II. The details of synaptic modification dynamics are described in the next subsection.

2.2. Dynamics of synaptic modification

We assume that amount of the glutamate binding to the postsynaptic NMDA channel trigger synaptic modification [6][8]. This hypothesis can be formulated mathematically as follows:

$$\frac{dw_i(t)}{dt} = \eta F(\,[\,\xi\,]_i\,) , \tag{3}$$

where $w_i(t)$ represents the weight of synaptic connection from i neuron, η is the learning rate, and $[\,\xi\,]_i$ is the amount of glutamate binding to the postsynaptic NMDA channel at synapse i. The details of $[\,\xi\,]_i$ dynamics are explained in the next subsection. F is a threshold function, when the amount of the glutamate binding is below the lower threshold θ_{d_1} or $\theta_{d_2} < [\,\xi\,]_i < \theta_p$, no modification occurs. The synaptic weight is depressed when $\theta_{d_1} < [\,\xi\,]_i < \theta_{d_2}$, and it is potentiated when $\theta_p < [\,\xi\,]_i$(Fig.2). $F([\,\xi\,])$ obeys the following equations:

$$\begin{aligned} F([\,\xi\,]) &= -a\mathrm{sig}(\xi - \theta_{d_1}, \beta_1) + b\mathrm{sig}(\xi - \theta_{d_2}, \beta_2) \\ &\quad + c\mathrm{sig}(\xi - \theta_p, \beta_3) , \end{aligned} \tag{4}$$

$$\mathrm{sig}(x, \beta) = \frac{1}{1 + e^{-\beta x}} , \tag{5}$$

where β is the decay time constants, and a, b, c are proportional constants that are characterising the sigmoid function.

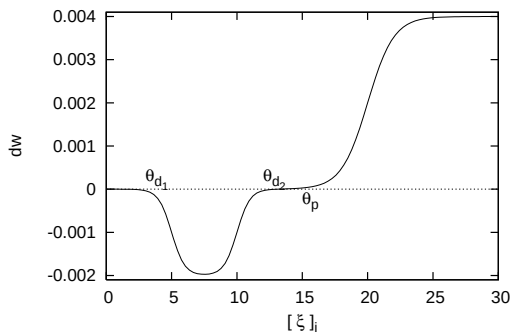


Figure 2: The diagram of the synaptic modification rule dependent on the amount of glutamate binding to the postsynaptic NMDA channel

2.3. Dynamics of the amount of glutamate binding

It has been reported that the LTP depends on the amount of glutamate binding to the postsynaptic NMDA channel, and that the binding decays exponentially in time [4]. We assume that this "decay" is essential to the sensitivity of LTP to the temporal spikes, and this property is expressed by the following equation:

$$\frac{d\,[\xi(t)]_i}{dt} = -\frac{1}{\lambda}\,[\xi(t)]_i + I_i(t) , \tag{6}$$

where $I_i(t)$ is an quantity of the glutamate binding caused by synaptic inputs, and λ is the time constant of exponential decay. We assume that dynamics of $I_i(t)$ are different between synapses in *Layer* I and in *Layer* II. In *Layer* I, the change of glutamate binding is triggered by dendritic backpropagating action potentials [5]. On the other hand, in *Layer* II, the backpropagation is attenuated because of the long distance from soma [2][3]. Thus, we assume that the change of glutamate binding is not trigger by the backpropagation but by the temporal pattern of coincidence, which is a extension of "spatiotemporal learning rule"[8].

The quantity of glutamate binding $I_i(t)$, in the *Layer* I, is written by the following equation:

$$I_i(t) = \mathrm{I_a} \iint_0^t x_i(l) w_i(l) K(l-m) y(m)\, dl dm\,, \qquad (7)$$

where $\mathrm{I_a}$ is a proportional constant of dendritic backpropagating action potential, $K(\Delta t)$ is a window function, and $y(t)$ is the output value of postsynaptic neuron. According to the physiological experiments in the hippocampus [9], we assume that the window function $K(\Delta t)$ depends on the spike timing of pre- and postsynaptic neurons. Fig.3 displays the window function that is used for our simulations. The window function $K(\Delta t)$ following the equation:

$$K(\Delta t) = \alpha(1-\beta\Delta t^2)\exp(-\gamma\Delta t^2)\,, \qquad (8)$$

where Δt is firing time difference between the pre- and postsynaptic neurons, and α, β, γ are the decay time constants that are characterising the window function.

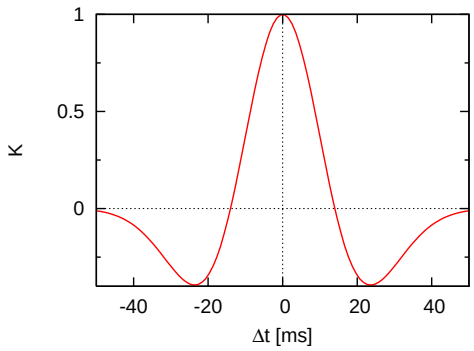


Figure 3: The diagram of the window function

On the other hand, the amount of glutamate binding $I_i(t)$ in *layer* II is written by the following equation:

$$I_i(t) = \mathrm{I_b} \iint_0^t x_i(l) w_i(l) K(l-m) \Big\{ \sum_{k \neq i}^{N} x_k(m) w_k(m) \Big\}\, dl dm\,, \qquad (9)$$

where $\mathrm{I_b}$ is a proportional constant of the glutamate binding caused by a single input spike, and $K(\Delta t)$ is the window function same as *Layer* I's, and Δt is the temporal difference among presynaptic neurons' spikes. In Eq. (9),

$$\sum_{k \neq i}^{N} x_k(m) w_k(m)$$

can be considered as the spatial coincidence factor among presynaptic inputs.

3. Simulation results

The network has $N = 100$ input neurons, and each neuron projects on both of *Layer* I and *Layer* II. Thus there are 200 synapses in this network. The membrane time constant τ was chosen to be 10ms, and the initial synaptic weights are symmetrical and uniformly distributed in the range [0.9, 1.1). The threshold θ was 20mV, and V_{reset} was 0mV. The input spikes at each synapse were Poisson trains with a constant mean frequency of $<x_i(t)>$. The decay time constant of glutamate binding is $\lambda = 200$ms [8]. On these conditions, we investigated differences of synaptic plasticity on each Layer when asynchronous spikes with different frequencies or local synchronous spikes are input.

3.1. Synaptic plasticity depending on the rate of presynaptic inputs

To clarify the characteristics of synaptic plasticity depending on the rate of synaptic inputs, we applied as an external input non correlated spatiotemporal patterns whose frequency is 2-40 Hz to the network. These inputs obey independent Poisson processes. Fig. 4 shows that the averages of synaptic weights in each layer. The synaptic plasticity of *Layer* II induces potentiation or depression when the input frequency is relatively lower than that of *Layer* I. This result implies that spatiotemporal learning rule is more sensitive to the input frequency than Hebb's learning rule.

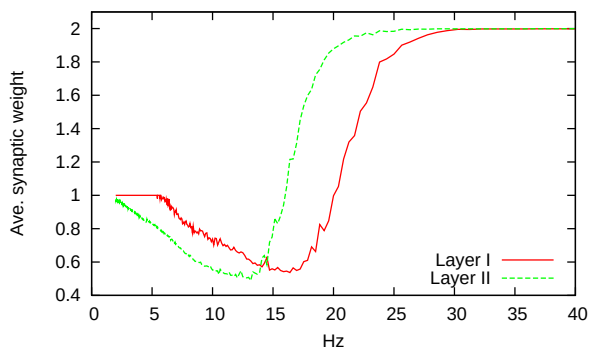


Figure 4: Frequency dependent synaptic plasticity.

3.2. Synaptic plasticity depending on locally correlative inputs

In this section, we investigate the case when a specific part of synaptic inputs are synchronous for 1000ms (Fig.5). We assume that average frequency of synaptic inputs is 10Hz, according to results of frequency dependent synaptic plasticity (Fig.4). We expected that it would be easy to distinguish the differences of responses for correlative inputs on both layers, because synapses weights on both layers are depressed at this frequency. The synapses from ♯41 to ♯60 in both layers receive synchronous inputs, and those from ♯1 to ♯40 and ♯61 to ♯100 receive independent Poisson process patterns (see Fig.5).

On *Layer* I, the only synapses which receive synchronous spikes are strengthened, and the rest synapses were weakened. This implies that synchronous inputs can

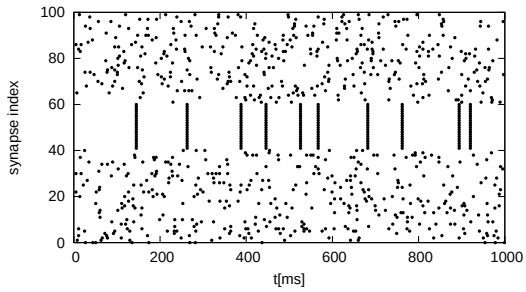
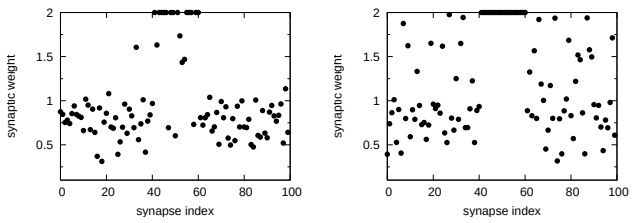


Figure 5: Raster plot of the input pattern with locally correlation



Layer I (PD) *Layer* II (DD)

Figure 6: Distribution of synaptic weights on *Layer* I (PD) and *Layer* II (DD)

well strengthen synaptic weights even if the input frequency is quite low. On the other hand, on *Layer* II, not only the synapses which receive synchronous spikes but also tens of synapses which do not receive these inputs are strengthened in spite of low frequency. The only synapses on DD, the input spikes of which correlate highly with synchronous spikes train, would be strengthened,

4. Discussion

In this paper, we assume that the hippocampal CA1 pyramidal model neuron of which the synaptic plasticity depends on the distance from the soma. We divided the dynamics of synaptic modification into two types for each dendrite (PD or DD), and investigate the characteristics of the synaptic plasticity. Froemke et al also report that the dynamics of Hebbian learning are different by the site of the synapses [1]. On the other hand, we assume additional dynamics of the coincident rule. This is because we want to focus on the dynamics induced by the temporal relation between presynaptic spikes. For simplicity, we assume that the dynamics of synaptic modification on PD obeys Hebbian learning rule, and on DD obeys only the coincident rule.

On this assumption, we find that the only synapses on PD which receive synchronous spikes are strengthened, and the rest synapses are weakened. On the other hand, on DD, not only synapses which receive synchronous spikes but also other synapses which receive asynchronous spikes are strengthened, in spite of low frequency. These synapses' inputs would have high correlation with synchronous inputs. According to these results, we can consider that the coincident rule have higher sensitivity for temporal spike patterns than the Hebbian rule. Therefore we can say that temporal information of spike patterns are mainly coded on DD.

5. Conclusion

We simulate Hebbian and coincident learning dynamics at proximal and distal dendrites in a hippocampal neuron. These results of computer simulation suggest that spatial information of higher frequency are mainly coded by the Hebbian rule on PD and temporal information of spike patterns are mainly coded by the coincident rule on DD.

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