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The STDP with Fluctuations Agrees with the Changes and the Distributions of the Synaptic Weights

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Abstract—The synaptic modifications are considered to play an important role in forming the neural network by following plasticity rules. According to electrophysiological measurements, synaptic modifications follow spike-timing dependent plasticity (STDP) and synaptic weights have a unimodal distribution with a nonzero mode and a long-tail. However, the shape of the biologically observable distribution is far from that of the distribution derived theoretically by the STDP model. This difference implies existence of other plasticity rules modifying the synaptic weights. Morphological observations can obtain the long-term time courses of the sizes of the individual dendritic spines, which are strongly related to the synaptic weights. According to the observations, synaptic weights are continuously shuffled by fluctuation. Is the fluctuation the source of the synaptic weight distribution? This paper quantitatively examines the STDP model combined with two fluctuations; the activity-dependent fluctuation and the intrinsic fluctuation. This paper demonstrates that the STDP with the fluctuations agree with various features of biological neural networks; the synaptic weight distributions, and the state dependence of the changes. This result is a key to revealing mechanism of development of neural networks.

1. Introduction

Synaptic connections between neurons are widely considered to play an important role in information processing in human brain [1–4]. The mechanism of the development of neural networks is key to revealing human intelligence. Some experiments found that the synaptic weights represented as excitatory post-synaptic currents (EPSCs) are widely distributed and have a nonzero mode and a long-tail [5–12]. Others suggest that the synaptic weight distribution is monotonically decreasing, or at least has a very small mode value [13]. On the other hand, the changes in the synaptic weights depend on the difference between the spike (action potential) timing of the pre- and the post-synaptic neurons, and on the temporal amplitude of the synaptic weight [6, 14, 15]. This plasticity rule is called *spike-timing dependent plasticity* (STDP) [12, 14–20]. Various formulations of the STDP models have been presented according to experimental results, biological limitations, and information theory. The *multiplicative STDP* agrees with the biological STDP on the spike-timing dependences and the state dependences [16]. However, unfortunately, the synaptic weight distribution derived theoretically from the multiplicative STDP is very narrow and is not consistent with biological synaptic weight distributions [20]. Another STDP model called *log STDP* derives a distribution similar to a biological one [20], but it does not agree with the biological STDP on state dependences [15, 16]. The conflict between the change and the distribution suggests the existence of another factor modifying the synaptic weights (see Fig. 1).

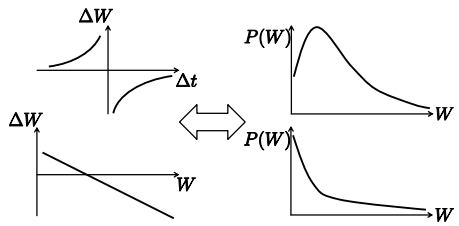


Figure 1: The concept of this paper. The short-term synaptic weight changes ΔW induced by the spike-timing dependent plasticity (STDP) depend on the timing difference Δt of the neurons' spikes and the temporal amplitudes W of the synaptic weights (see the left half). If the STDP mediates the synaptic weights W, it can derive the synaptic weight distribution $P(W)$ similar to those obtained from biological experiments (see the right half). Actually, the existing STDP models cannot [20].

Recently, morphological observations found that the sizes of dendritic spines change gradually and continuously [21–23]. This plasticity rule is called *spine volume plasticity* in this paper. The spine volume plasticity derives a unimodal distribution with a nonzero mode and a long-tail of the spine volumes. Remarkably, even after the action potentials are blocked by drug, the spine volumes are still varied and form a monotonically decreasing distribution. Since the spine size is strongly related to the amplitudes of the evoked mEPSCs, these results suggest the existence of the synaptic weight changes independent to the action potentials, i.e., the intrinsic plasticity, and the shape of the synaptic weight distribution depending on the intensity of the action potentials.

The existing STDP models do not take into account the intrinsic plasticity and the intensity dependences of the synaptic weight distribution. This paper presents a novel framework of synaptic plasticity called *fluctuation STDP*, and demonstrates that the fluctuation STDP is consistent with various datasets concerning the synaptic weights; the amounts of changes induced by the biological STDP [15, 16] and the shapes of synaptic weight distribution [21, 22].

2. Models and Methods

2.1. Fluctuation STDP

This section presents *fluctuation STDP*, but first introduces a common form of the *spike-timing dependent plasticity* (STDP). When the timing t_{pre} of a pre-synaptic action potential precedes the timing t_{post} of a post-synaptic action potential, i.e., $\Delta t = t_{\mathrm{pre}} - t_{\mathrm{post}} < 0$, the synaptic weight W is potentiated immediately at the time t_{post}. In other words, the long-term potentiation (LTP) is induced. In the case of $\Delta t > 0$, the synaptic weight W is depressed immediately at the time t_{pre}. In other words, the long-term depression (LTD) is induced. The synaptic plasticity described above is called STDP and is expressed as the following common form [6, 14, 15]:

$$W \leftarrow W + \Delta W(W, \Delta t),$$
$$\Delta W(W, \Delta t) = \begin{cases} +A_+(W)\exp\left(-\frac{|\Delta t|}{\tau_+}\right) & \text{if } \Delta t < 0 \\ -A_-(W)\exp\left(-\frac{|\Delta t|}{\tau_-}\right) & \text{if } \Delta t > 0, \end{cases} \quad (1)$$

where $\leftarrow$ implies "is updated to", and $A_+(W)$ and $A_-(W)$ are functions determining the amplitudes of the LTP and the LTD, respectively. The fluctuation STDP employs the following amplitude functions $A_+(W)$ and $A_-(W)$:

$$A_+(W) = c_+ + \nu_p W, \;\; A_-(W) = c_- W + \nu_p W, \quad (2)$$

where $\nu_p \sim \mathcal{N}(0, \sigma_p^2)$ and "$\sim$" implies "follows". These formulations are the same as the *multiplicative STDP* [16]. When the pre-synaptic neuron elicits an action potential, the synaptic weight W is updated as:

$$W \leftarrow W + \nu_f,$$

where $\nu_f \sim \mathcal{N}(0, \sigma_f^2)$. This phenomenon is called *activity-dependent fluctuation* in this paper. The activity-dependent fluctuation is assumed to be disrupted by NMDA receptor blockers just like the STDP. In addition, independently of pre- and post-synaptic activities, the synaptic weight W is updated as:

$$W \leftarrow W + (\tilde{S}W + \tilde{s})\nu_s,$$

where $\nu_s \sim \mathcal{N}(0, 1)$. This updating can be expressed alternatively as the stochastic differential equation (SDE). This phenomenon is called *intrinsic fluctuation* in this paper, and is the same as the *intrinsic plasticity* in the spine volume plasticity [21, 22]. This phenomenon is not disrupted by NMDA receptor blockers in contrast to the STDP and the activity-dependent fluctuation.

For comparisons, this section introduces other formulations of the STDP models. One of the most simple formulations is the *additive STDP* [16, 20], which is expressed by using the common formulation shown in Eq. (1) as

$$A_+(W) = c_+ + \nu_p, \;\; A_-(W) = c_- + \nu_p,$$

where $\nu_p \sim \mathcal{N}(0, \sigma_p)$. The *multiplicative STDP* [16] was designed according to the electrophysiological measurements [15], and is expressed as the formulation shown in Eq. (2). The *log STDP* [20] was designed to lead a synaptic weight distribution similar to a log-normal distribution [9]. The formulation is omitted.

Throughout this paper, the following parameter values for the temporal window are used: $\tau_+ = 17$ [ms], $\tau_- = 34$ [ms]. The parameter values shown in the original study [16] used for the multiplicative STDP are adjusted according to the electrophysiological measurement [15] in the simplified condition of $\tau_+ = \tau_- = 20$ [ms]. All the parameter values are adjusted once again under the condition of $\tau_+ = 17$ and $\tau_- = 34$ [ms]. The value of σ_n is set to the half of the original study because of the existence of the intrinsic fluctuation. The parameter values used for the intrinsic fluctuation are the same as the original study [21, 22].

The activity of the post-synaptic neuron is assumed to be almost uncorrelated to that of the pre-synaptic neuron. This assumption is reasonable when the post-synaptic neuron accepts numerous synapses. When the synapse weight changes are enough small, they can be expressed as a SDE [16, 18–20]. In addition, the synaptic weight distributions at the steady-state can be estimated by a Fokker-Planck Equation (FPE). The fitness of the theoretically derived synaptic weight distribution with parameter values θ to the histogram $\boldsymbol{m}$ obtained from the biological experiments can be evaluated as a log-likelihood function $L(\theta; \boldsymbol{m})$. The parameter values θ can be optimized by maximizing the log-likelihood function $L(\theta; \boldsymbol{m})$ by using a random optimization [24].

2.2. Datasets

The first dataset obtained from rat hippocampal slices by the morphological observations [21] contains two histograms of synaptic weights. One histogram is obtained from groups under a natural condition (control groups) and shows a unimodal distribution with a nonzero mode and a long-tail. The other histogram is obtained from rat hippocampal slices which have the NMDA receptors and/or Na^+ channels blocked by drug, and is monotonically decreasing, In this case, the synaptic weight changes dependent on the NMDA receptors and the action potentials, i.e., STDP and activity-dependent fluctuation, are disrupted. In contrast, the synaptic weight changes independent to them, i.e., intrinsic fluctuation, still occur. The synaptic weights are measured as spine volumes in cubic micrometer (μm^3). The conversion coefficient k from EPSC [pA] to spine volume [μm^3] is assumed to 8.0×10^{-4} in this paper.

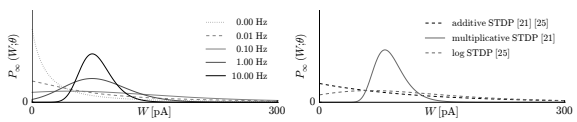


Figure 2: The synaptic weight distributions $P_\infty(W;\theta)$ are derived from the STDP models. (left) These shapes derived from the fluctuation STDP depend on the action potential probability f. (right) These shapes derived from the other STDP models do not depend on the action potential probability f.

The second dataset obtained from rat hippocampal slices by the electrophysiological measurements [15] contains the amounts ΔW of the changes in the synaptic weights W after the repeated STDPs when the initial synaptic weights are varied, i.e., the state dependence of the STDP. The synaptic weights are measured as PSCs in picoampere (pA).

3. Results

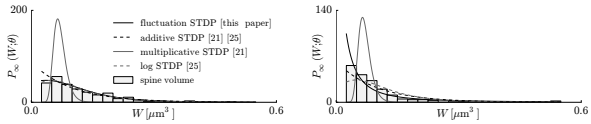
3.1. Activity-Dependence of Distribution

In case of the fluctuation STDP, the second moment of the SDE contains the terms of zeroth, first, and second order with respect to the action potential probability f, where they correspond to the intrinsic fluctuation, the activity-dependent fluctuation, and the STDP. In contrast, the first moment only contains the term of second order corresponding to the STDP. Thus, according to the FPE, the synaptic weight distribution $P_\infty(W;\theta)$ derived from the fluctuation STDP depends on the action potential probability f. When the action potential probability f is nearly zero, only the term of zeroth order, i.e., the intrinsic fluctuation, has effect, and the synaptic weight distribution $P_\infty(W;\theta)$ is monotonically decreasing with the increasing synaptic weight W. When the action potential probability f is small, the term of first order, i.e., the activity-dependent fluctuation, is dominant over the diffusion term, and the synaptic weight distribution $P_\infty(W;\theta)$ resembles a uniform distribution. When the action potential probability f is large, the term of second order, i.e., the STDP, is dominant over the diffusion term, and the drift term has effect. Hence, the synaptic weight distribution $P_\infty(W;\theta)$ becomes a unimodal distribution.

On the other hand, in the cases of the additive, multiplicative, and log STDPs, both the first and second jump moments contain only the terms of second order. According to the FPE, the synaptic weight distribution $P_\infty(W;\theta)$ derived from one of the three STDP models does not depend on the frequency f as depicted in Fig. 2.

3.2. Fitness to Histogram of STDP

This section compares the synaptic weight distributions $P_\infty(W;\theta)$ derived from the STDP models with the histograms of the synaptic weights obtained from the morphological observations [21]. Since the synaptic weight distribution $P_\infty(W;\theta)$ derived from the fluctuation STDP depends on the action potential probability f as mentioned



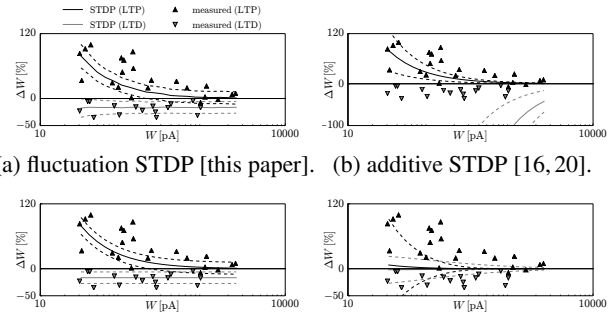
(a) control groups. (b) the NMDA receptors blocked.

Figure 3: The synaptic weight distributions $P_\infty(W;\theta)$. The histograms are obtained from the morphological observations [21]. The curves are derived from the STDP model. The black solid line, the black dotted line, the gray line, and the gray dotted line correspond to the fluctuation STDP, the additive STDP, the multiplicative STDP, and the log STDP. (a) The left figure shows the case of the control groups. (b) The right figure shows the case that the NMDA receptors are blocked, i.e., the action potential probability $f = 0$.

Table 1: Fitness to Histogram as Log-Likelihood Function $L(\theta; \boldsymbol{m})$.

	Histograms	
Models	(i) **control**	(ii) **blocked**
fluctuation STDP	-606	-381
additive STDP	-609	-386
multiplicative STDP	-1306	-685
log STDP	-606	-391

in the previous section, the action potential probability f is treated as a parameter of the fluctuation STDP hereafter. In the case of the control groups, the NMDA receptors and the Na^+ channels are not blocked, and the action potential probability f has a positive value. The parameter values are adjusted as described in the previous section. Figure 3 (a) shows the case of the control groups. The fluctuation STDP derives the synaptic weight distribution $P_\infty(W;\theta)$ which is a unimodal distribution with a nonzero mode and a long-tail and is very similar to the corresponding histogram. The fitness evaluated by the log-likelihood function $L(\theta; \boldsymbol{m})$ is summarized in Table 1. The additive STDP derives a monotonically decreasing distribution $P_\infty(W;\theta)$ and its shape is different from the histogram. The multiplicative STDP derives a unimodal distribution $P_\infty(W;\theta)$ with a nonzero mode and a long-tail but its shape is very narrow and almost different from the histogram. The log-likelihood functions $L(\theta; \boldsymbol{m})$ of both the STDP rules are worse than that of the fluctuation STDP. The log STDP derives the synaptic weight distribution $P_\infty(W;\theta)$ very similar to that $P_\infty(W;\theta)$ derived from the fluctuation STDP and the histogram. On the other hand, in the case that the NMDA receptors are blocked, the histogram of the measured spine volumes is monotonically decreasing as shown in Fig. 3 (b). This case corresponds to the action potential probability $f = 0$, and the fluctuation STDP derives a monotonically decreasing distribution $P_\infty(W;\theta)$ of the synaptic weights. When the synaptic weight distributions $P_\infty(W;\theta)$ are assumed to be derived only from the STDP models, the synaptic weight distributions $P_\infty(W;\theta)$ keep their shapes after the NMDA receptors are blocked. Hence, the multiplicative STDP and the log STDP derive a unimodal distribution with a nonzero mode and a long-tail as is the



(a) fluctuation STDP [this paper]. (b) additive STDP [16, 20].

(c) multiplicative STDP [16]. (d) log STDP [20].

Figure 4: The amounts ΔW of the changes in the synaptic weights W induced by the STDP. The black triangles ▲ (The gray triangles ▼) denote the amounts ΔW of the changes induced by the LTPs (the LTDs) obtained from the electrophysiological measurements [15]. The lines are obtained from the STDP models. The black solid lines denote the average of the changes ΔW of the LTPs induced by the STDP models, and the black dashed lines denote their standard deviation. The gray solid and dashed lines denote the case of the LTDs. The fluctuation STDP, the additive STDP, the multiplicative STDP, and the log STDP are summarized from (a) to (d).

case in the control groups. In the both cases, the synaptic weight distributions $P_\infty(W;\theta)$ derived from the fluctuation STDP are the most similar to the histograms obtained from the morphological observations according to the similarities evaluated by the log-likelihood function $L(\theta;\boldsymbol{m})$ and summarized in Table 1.

3.3. State Dependence of STDP

The amounts ΔW of the changes in the synaptic weights W induced by the STDP models are obtained in the same manner as the second dataset, and are summarized in Fig. 4. Since the initial synaptic weights are varied, the results show the state dependence of the STDP models. As shown in Fig. 4 (a), the amounts ΔW of the changes of both the LTP and the LTD induced by the fluctuation STDP are similar to those of the experimental results. The additive STDP can only agree with the average changes of the LTPs, but causes the excessively large LTDs and the different standard deviations. The multiplicative STDP agrees with the electrophysiological measurements [15] as with the fluctuation STDP. The log STDP causes the small averages and the large standard deviations in the weak synaptic weights, and causes the small standard deviations in the strong synaptic weights when compared to the electrophysiological measurements.

4. Discussions

The multiplicative STDP derives a narrow distribution but the histograms of the synaptic weights obtained from the morphological observations are widely distributed. If the STDP models derive a wider distribution, the STDP models should have a large diffusion term or a large negative drift term when compared to the multiplicative STDP. The additive and log STDPs try to realize such a distribution by tuning the state dependences. On the other hand, owing to the additional diffusion terms, i.e., the activity-dependent fluctuation and the intrinsic fluctuation, the fluctuation STDP can derive such a distribution despite not tuning the state dependences. In conclusion, the fluctuation STDP can agree with the experimental results of both the synaptic weight distributions and the state dependences in contrast to the existing STDP models; the additive STDP, the multiplicative STDP, and the log STDP.

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