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A neural circuit model of emotional learning using two pathways with different granularity and speed of information processing

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1. Introduction

Animals, including humans, learn on the basis of experience and memory, with emotions, and increase their survival probability through reactions involving emotional memory. Such learning is called emotional learning (Uwano and Ono, 1997; LeDoux, 1996). It was clarified that the primary region for emotional learning is the amygdala (LeDoux, 1996). LeDoux (1996) suggested on the basis of physiological and anatomic findings that two pathways, thalamo-amygdala (direct) and thalamo-cortico-amygdala (indirect) pathways, play important roles in emotional learning. The direct pathway can process faster than the indirect pathway, although the information is coarser. On the other hand, the indirect pathway processes information in more detail, but more slowly. The direct pathway may be particularly useful in situations requiring a rapid response; it is a rough processing system.

Armony et al. (1997) proposed a neural circuit model of emotional learning using two pathways with different granularity of information processing. However, the model cannot account for the time process of emotional learning because the model does not include a precise time process. Moreover, the model does not clarify the circuit problem whereby neurons in the memory consolidation site for emotion do not directly contact the central nucleus

ABSTRACT

We propose a neural circuit model of emotional learning using two pathways with different granularity and speed of information processing. In order to derive a precise time process, we utilized a spiking model neuron proposed by Izhikevich and spike-timing-dependent synaptic plasticity (STDP) of both excitatory and inhibitory synapses. We conducted computer simulations to evaluate the proposed model. We demonstrate some aspects of emotional learning from the perspective of the time process. The agreement of the results with the previous behavioral experiments suggests that the structure and learning process of the proposed model are appropriate.

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(CE) of the amygdala that provides the principal source of emotional outputs (Paré et al., 2004).

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The remainder of this article is organized as follows. In Section 2, we propose a neural circuit model of emotional learning using two pathways with granularity and different speed of information processing. Section 3 describes the results of a computer simulation to examine the proposed model. Section 4 presents our conclusions.

2. Proposed Model

Our proposed model network is shown in Fig. 1. The proposed model consists of sensory input, thalamus, cortex, and the amygdala. The route of the conditioned stimulus (CS) includes variable connections, whereas the route of the unconditioned stimulus (US) is fixed. In the thalamus, the neuronal groups directly connected to amygdala are partially received from the sensory input whereas the neuronal groups connected to cortex are fully received. This corresponds to a difference in the granularity of information processing. In the cortex, semantic conversion means that the primary features are converted to semantic features in the association cortex area through an excitatory and inhibitory network. The semantic





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Fig. 1. Proposed model. Solid and dotted connections indicate fixed and variable connections, respectively. Opened and filled arrowheads indicate excitatory and inhibitory connections, respectively.

conversion is supposed to be learned in a past. In the amygdala, the lower side is the medial sector of the central amygdala (CEm) that provides the principal source of emotional outputs (Paré et al., 2004), whereas the upper part is the lateral division of the central nucleus (CEI), including the lateral capsular division (CEc). Wilensky et al. (2006) showed that the CE is involved not only in the expression but also in the acquisition of emotional learning. The CEI receives input from both the cortex and the thalamus, whereas the CEm receives from only the thalamus. That is, the CEI is a full memory consolidation site whereas the CEm is a partial one. Thus, the inhibitory interneurons between the CEI and CEm are supposed to compensate for the partial memory consolidation of the CEm. Here, the left and right sides of the amygdala are supposed to be opposed to each other, for instance, for fear and calmness.

In order to derive a precise time process, we utilized a spiking model neuron proposed by Izhikevich (2007). The model neuron was reduced from a number of biophysically accurate Hodgkin–Huxley-type neuronal models to a two-dimensional system ordinary differential of the form:

$$\dot{\nu} = 0.04\nu^2 + 5\nu + 140 - u + I_{\text{ext}}(t) \tag{1}$$

$$\dot{u} = a(bv - u) \tag{2}$$

with the auxiliary after-spike resting

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

The external current $I_{ext}(t)$ is expressed as

$$I_{\text{ext}}(t) = -\sum_{i} g_{\text{syni}}(t) (V - V_{\text{rev}}).$$
⁽⁴⁾

$$g_{\text{syni}}(t) = \begin{cases} w_i A_i \frac{t - t_i^f}{\tau_i^2} \exp\left(-\frac{t - t_i^f}{\tau_i}\right) & \text{if } t \le t_i^f \\ 0 & \text{if } t < t_i^f \end{cases}$$
(5)

Here, *v* represents the membrane potential of the model neuron, *u* represents a membrane recovery variable, *a*, *b*, *c*, and *d* are dimensionless parameters, *t* is time, $g_{syni}(t)$ is the time-varying synaptic conductance for the *i*th spike, V_{rest} is the reversal potential, w_i is the weight ratio, A_i is the maximum amplitude, t_i^f is the arrival time, and τ_i is the time constant. The parameter set for regular spiking (RS), a = 0.02, b = 0.2, c = -65, and d = 8.0, is used for excitatory neurons, whereas the parameter set for fast spiking (FS), a = 0.10, b = 0.2, c = -65, and d = 2.0, is used for inhibitory neurons.

The solid connections shown in Fig. 1 are fixed, whereas the dotted connections are variable, that is, they are learned by STDP, which is a minute time resolution version of the well-known Hebb learning rule. The STDP profile of excitatory synapses has been observed electrophysiologically (Markram et al., 1997; Bi and Poo, 1998; Froemke and Dan, 2002). From the profile, postsynaptic potentials arriving after presynaptic potentials induce long-term potentiation, and postsynaptic potentials arriving before presynaptic potentials induce long-term depression. Froemke and Dan (2002) derived a numerical description of the increase and decrease rates of synaptic plasticity $w(\Delta t)$ from electrophysiological data as follows:

$$\Delta w(\Delta t) = \begin{cases} \frac{1.02}{n} \exp \frac{-|\Delta t|}{15.5} + 1 & \text{if } \Delta t > 0\\ -\frac{0.52}{n} \exp \frac{-|\Delta t|}{33.2} + 1 & \text{if } \Delta t < 0, \end{cases}$$
(6)

where Δt (ms) is the temporal difference from a postsynaptic spike to a presynaptic spike and *n* is the number for which the maximum rate is obtained. Download English Version:

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