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Simulation of dopamine modulation-based memory model

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ABSTRACT

Neurological electrophysiology has revealed that both dopamine and stimulation tend to affect the formation of memory. This study aimed to investigate the regulatory effect of dopamine and stimulation on long-term and short-term memories using an integrated striatal dopamine and neuronal network model. The robustness of the model was verified by applying interference stimulation. The results showed that the duration of network firing response was associated with the value of dopamine and the duration of stimulation, and the network firing peak was not affected by the interference stimulation, suggesting that the model was strongly robust.

1. Introduction

Memory is a psychological process of accumulating and preserving individual experiences in the brain. From the perspective of information processing, it is a process for the human brain to encode, store and retrieve the input information. Memory can explain a lot of problems, such as psychological problems of humans and patients with brain injury. And the capacity of memory plays an important role in the process of performing complex cognitive activities. People can use memory to store the information about the environment. This information is eventually used by human, so it is of great significance to study memory. According to the length of retention time, memory can be divided into transient, short-term and long-term. Transient memory refers to a memory in an extremely short time after the perception (such as one second or so), short-term memory refers to a memory in a relatively short time (less than one min) and long-term memory refers to a memory in a longer time (more than one min) [1–3]. Short-term and long-term memories are inter-related rather than mutually exclusive [4,5]. Transformation of short-term memory to long-term memory relies on the transcription of cAMP-response element binding protein (CREB), which is a key regulatory molecule in the formation of long-term memory [6,7]. Long-term potentiation (LTP) is also essential for the formation of memory. Formation and maintenance of LTP is a neural mechanism generated by pre-synaptic and post-synaptic combined effects, of which post-synaptic mechanism is dominant. The post-synaptic mechanism of LTP formation is closely related to the intracellular cascade after the activation of N-methyl-D-aspartic acid receptor (NMDA) [8,9].

Several models have been proposed to describe the short-term memory(working memory) from different perspectives. For example, Reilly et al. [10] designed a working memory calculation model, which showed that the prefrontal cortex and basal ganglion were able to interact to complete a complex time expanding task, which was meant to maintain and update information by implementing a flexible working memory system with adaptive gating mechanism. The C-W model combined the recurrent neural network and the bistability of cells [11]. Another example is the Ca^{2+}-based working memory model [12]. Some studies investigated the roles of basal ganglia and dopamine in the working memory [13]. However, very few models investigated the effects of dopamine on short-term and long-term memories as well as the interactions of these two types of memories during their formation process.

As is known to all, dopamine is a neurotransmitter, whose absence in the brain may cause tremor, rigidity, bradykinesia and other Parkinson's symptoms [14]. There have been many models that can be used to explain the electrophysiological data and the results associated with the dopamine modulation, such as computational models of schizophrenia and dopamine modulation in the prefrontal cortex [15], And the model which verified the importance of combined effects of dopamine in the basal ganglia and the prefrontal cortex [16]. And the study also showed that, dopamine was found to affect movements and also played an important role in memory [17–19]. Diego et al. [20] revealed that activation of dopamine D1 receptor promoted the formation of cortical LTP, which was conducive to the formation of long-term memory. Regulatory effect of dopamine on protein expression is essential for neuronal plasticity. Dudman et al. [21] demonstrated

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that the dopamine D1 receptors mediated CREB phosphonation through the phosphonation of NMDAs, indirectly indicating that these receptors affect the formation of long-term memory.

This study aimed to investigate the effects of dopamine on short-term and long-term memories by integrating striatal dopamine and neuronal network models as well as explore the effects of stimulation duration and interference stimulation (which refers to a second short stimulation during the delayed period) on the network firing.

2. Integrative model

2.1. Striatal dopamine model

Dopaminergic neurons are mainly distributed in the mesencephalon and diencephalon, and are divided into six cell populations. Dopaminergic neuron pathways can be summarized into two systems: long dopaminergic neuron system and short dopaminergic neuron system. Striatal dopaminergic system in the mesencephalon (also known as the substantia nigra-striatal system), which belongs to the long dopaminergic neuron system, is the most important [22].

The striatal dopamine model is based on the dopaminergic nigrostriatal system. The striatum is composed of caudate nucleus, putamen and globus pallidus, and is an important component of basal ganglia. This group of cranial nerve nuclei plays an important regulatory effect on the motor function of the organism, such as stability of voluntary movement, regulation of muscle tension, coordination of somatic motor, etc. Regulatory effect of the striatum on the motor function mainly relies on the abundant internal dopaminergic innervation, where the dopaminergic neurons mainly originate from the substantia nigra (SN) region and ventral tegmental area (VTA) [23]. In this model, the impact of dopamine concentration on striatal neurons was represented as neurotransmitter factor γ, and this impact was mainly regulated by the activation of D1 receptors. The potential V_s of the striatal neurons should abide by the following equation [24]:

$$-C\frac{d}{dt}V_s(\theta_i,t)=\gamma(I_{Kir2}+I_{LCa})+I_{Ksi}+I_L+I_T(\theta_i,t) \tag{1}$$

where I_{Kir2} is the internal rectified current, which can be strengthened by the activation of dopamine receptor D1, and is an important component of D1 inhibition [25]. Simultaneously, the activation of receptor D1 can also increase the L-type calcium current [26]. In the formula, I_{LCa} refers to the L-type calcium current, I_L refers to the leakage current, and $I_T(\theta_i,t)$ refers to the synaptic input current of the ith striatal neuron, which is given by the following equation:

$$I_T(\theta_i,t)=\left\{b+\sum_{j=1}^{N}\frac{1}{N}[W(\theta_i-\theta_j)+\Delta W]r(\theta_j,t)\right\}V_s(\theta_i,t) \tag{2}$$

where b refers to a constant current, N is the number of neurons, θ refers to memory region, and r refers to the firing rate of the neurons.

2.2. Neuronal network model

The firing rate of neurons (r) with respect to the memory region θ and time t is given by [11]:

$$\tau_r\frac{d}{dt}r(\theta_i,t)=-f[r(\theta_i,t)]+g[I(\theta_i,t)]$$
$$f(r)=r \tag{3}$$

where θ refers to the memory region, ranging from $-\pi$ to π, which indicates that the neurons can be found in all directions, τ_r refers to the cell constant, given that $\frac{dr}{dt}=0$, the firing rate of the neurons can be obtained by $r=g(I)$, where $g(I)$ is given by:

$$g(I)=\begin{cases}0 & I<0\\ 0.20 & \le I<1\\ 5I-4.81 & \le I<2\\ 0.8I+3.62 & \le I\end{cases} \tag{4}$$

The total current of each neuron I includes the external current I_{ext} and the synaptic current I_{syn}:

$$I(\theta_i,t)=I_{ext}(\theta_i,t)+I_{syn}(\theta_i,t) \tag{5}$$

The external current I_{ext} is given by:

$$I_{ext}(\theta_i,t)=I_0+I_{cue}\left(\frac{1+\cos(\theta_i-\theta_0)}{2}\right)^p \tag{6}$$

where p refers to the shape index of stimulation, I_0 is a constant, and I_{cue} refers to the magnitude of stimulation.

The synaptic current I_{syn} is given by:

$$I_{syn}(\theta_i,t)=\sum_{j=1}^{N}\frac{1}{N}[W(\theta_i-\theta_j)+\Delta W]\,[r(\theta_j,t)+\beta r_s(\theta_j,t)] \tag{7}$$

where ΔW refers to synaptic adaption, r_s refers to the firing rate of striatal neurons, β refers to the coupling coefficient, $r_s(\theta_j,t)$ is given by:

$$r_s(\theta_j,t)=\frac{1}{1+\exp([-55-V_s(\theta_j,t)]/2.5)},V_s(\theta_j,t)\ge -58\text{mV} \tag{8}$$

where V_s refers to the potential of the striatal neurons.

Connection weight between neurons W includes excitatory and inhibitory weights, and may be illustrated by the following equation, where q is the weighted index:

$$W(\theta)=-W_I+W_E\left(\frac{1+\cos\theta}{2}\right)^q \tag{9}$$

Here W_I refers to the excitatory weights, and W_E refers to the inhibitory weights.

Research has shown that one possible pathway by which dopamine affects memory is from the ventral striatum to the intermediate region of the thalamus or frontal cortex [27]. In this study, we considered the direct effect of cortex–striatum. the striatal dopamine and the neuronal network models were coupled, and the simulation was performed using Matlab (R2012a), with a time step of 0.005 s. The main parameters of the neuronal network are $\tau_r=0.1$, $p=1$, $q=1$, $I_0=0.2$, $W_I=2$, $W_E=2.8$ and $I_{cue}=1$, $\theta_0=0^\circ$, in which a short stimulation was applied at $\theta_0(0^\circ)$(duration of 0.5 s). Under the premise of stabilization of striatal neurons ($dV_s/dt=0$), the dopamine neurotransmitter factor γ was regulated to observe the changes in the firing rate of PFC neurons. When γchanges, the firing rate of PFC neurons with respect to memory region θ and time t are shown in Figs. 1–3.

As seen in Figs. 1–3, for $\gamma=4$, the neuronal network failed to generate continuous firing but presented a transient firing under a short stimulation (Fig. 1). For $\gamma=4.15$, the neuronal network had continuous firing for about 17 s (Fig. 2), whereas for $\gamma=4.2$, the continuous firing lasted for more than 60 s (Fig. 3), suggesting the transition from short-term to long-term memory. The duration of network firing might differ with different γ values, where a larger γ might lead to a longer firing duration. In the model, the network firing was applied to encode the stimulation, and the value of neuronal network firing peaked at the beginning of stimulation, indicating that memory of the initial stimulation information was accurately retained in the model. Whereas, a case of unstable neuronal network firing or failure to maintain its firing peak at the stimulation position suggest that the initial stimulation memory is inaccurately retained in the model. Figs. 4–6 illustrate the sectional views of neuronal network firings for γ values of 4, 4.15 and 4.2, respectively. As seen in Fig. 4, after applying a short