Multiple coherence resonances and synchronization transitions by time delay in adaptive scale-free neuronal networks with spike-timing-dependent plasticity

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A B S T R A C T

In this paper, we numerically study the effect of spike-timing-dependent plasticity (STDP) on multiple coherence resonances (MCR) and synchronization transitions (ST) induced by time delay in adaptive scale-free Hodgkin–Huxley neuronal networks. It is found that STDP has a big influence on MCR and ST induced by time delay and on the effect of network average degree on the MCR and ST. MCR is enhanced or suppressed as the adjusting rate A0 of STDP decreases or increases, and there is optimal A0 by which ST becomes strongest. As network average degree ⟨k⟩ increases, ST is enhanced and there is optimal ⟨k⟩ at which MCR becomes strongest. Moreover, for a larger A0 value, ST is enhanced more rapidly with increasing ⟨k⟩ and the optimal ⟨k⟩ for MCR increases. These results show that STDP can either enhance or suppress MCR, and there is optimal STDP that can most strongly enhance ST induced by time delay in the adaptive neuronal networks. These findings could find potential implication for the information processing and transmission in neural systems.

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1. Introduction

The temporal coherence and spatial synchronization of neuronal networks is of great importance to the information processing and transmission in neural systems. In particular, synchronization is correlated with many physiological mechanisms of normal and pathological brain functions including several neural diseases [1–5]. Over the past decades, stochastic resonance (SR), coherence resonance (CR) and synchronization of neuronal systems have been extensively studied [6–10]. In recent years, the roles of different noises and spike trains for information processing and transmission in neuronal systems have been further studied, such as the effect of white and colored noise in the dynamics of FitzHugh–Nagumo model [11,12], SR induced by Lévy noise in Lotka–Volterra system [13], and spike train statistics for consonant and dissonant musical accords and spike train regularity and harmony perception in an auditory sensory system [14,15]. In particular, many phenomena of SR and synchronization in neuronal networks have been found, such as SR and CR due to synaptic and channel noise [16–26], spatial CR due to noise [27–29], spatial decoherence induced by small-world connectivity [30], multiple SR (MSR) and multiple CR (MCR) due to time delay and coupling [31–33], as well as synchronization transitions (ST) induced by time delay [34–40], coupling strength [41,42], and noise [43,44]. In these studies, however, neuronal networks with static synaptic coupling are always employed.

In reality, neuronal networks are adaptive due to synaptic plasticity, and synaptic strength varies as a function of neuromodulation and time-dependent processes [45–47]. One representative of this biological effect is spike-timing-dependent plasticity (STDP), which modulates coupling strength adaptively based on the relative timing between pre- and post-synaptic action potentials [45,46]. A series of biological works have confirmed the existence of STDP in excitatory synapses onto hippocampal pyramidal [46] and neocortical neurons [47], excitatory neurons in auditory brainstem [48], parvalbumin-expressing fast-spiking striatal interneurons [49], etc. It is suggested that synaptic plasticity may account for learning and memory [50,51]. Recent study showed that synaptic plasticity can induce a transition of spike propagation in neuronal networks [52]. In recent years, increasing attention has been paid to the effects of STDP on structure and dynamics of neuronal populations. The synchronization via STDP has been intensively investigated [53–60], for example, Kube et al. found that STDP modifies the weights of synaptic connections in such a way that synchronization of neuronal activity is considerably weakened [58], Mikkelsen et al. found that STDP induces persistent irregular oscillations between strongly and weakly synchronized states [59];
Yu et al. showed that STDP can largely depress the temporal coherence and spatial synchrony induced by external noise and random shortcuts in neuronal networks [60]. Very recently, we have studied the effect of STDP on MCR and ST induced by time delay in Newman–Watts small-world neuronal networks, and we found that STDP has a big influence on MCR and ST as well as on the effect of network randomness on MCR and ST [61]. Studies have shown that the cerebral cortex is a highly interconnected network of neurons, and the structural and functional networks of the brain are of scale-free architectures [62,63]. Therefore, it is of significance to study MCR and ST in adaptive scale-free neuronal networks.

In this paper, using adaptive scale-free Hodgkin–Huxley neuronal networks with STDP, we numerically study the effect of STDP on MCR and ST induced by time delay in the neuronal networks. We first study how time delay induces MCR and ST when the adjusting rate $A_p$ of STDP is fixed, and then we focus on the effect of STDP on the MCR and ST. Subsequently, we study how STDP influences the effect of network average degree on the MCR and ST. Meanwhile, mechanisms are briefly discussed. Finally, conclusion is given.

2. Model and equations

We use Hodgkin–Huxley neuron model [64] and scale-free network topology developed by Barabási and Albert [65]. The present network comprising $N=100$ neurons starts with $m_0$ connected nodes, and subsequently every new node is attached to $m(t)\leq m_0$ different nodes already present in the network, whereby the probability $p$ that a new node will be connected to node $i$ depends on its degree $k_i$ in accordance with $p_i = k_i/\sum k_j$. This growth and preferential attachment scheme yields a network with an average degree $\langle k \rangle = \sum_k k_i/N = 2m$ and a power-law degree distribution with the slope of the line equaling $-2.9$ on a double logarithmic graph.

The membrane potential dynamics of coupled Hodgkin–Huxley neurons on the complex networks with time delay can be written as:

$$\frac{dV_i(t)}{dt} = -g_{Na}m_i^3h_i(V_i - V_{Na}) - g_kn_i^4(V_i - V_k) - G_i(V_i - V_L) + I_{in}^{syn} + \xi_i(t)$$

(1)

$$\frac{dx_i}{dt} = \alpha_x(V_i)(1 - x_i) - \beta_x(V_i)x_i, \quad (x = m, h, n)$$

(2)

with opening and closing rates:

$$\alpha_m(V_i) = \frac{0.1(V_i + 40)}{1 - \exp[-(V_i + 40)/10]}, \quad \beta_m(V_i) = 4\exp[-(V_i + 65)/18]$$

$$\alpha_h(V_i) = 0.07\exp[-(V_i + 65)/20], \quad \beta_h(V_i) = \{1 + \exp[-(V_i + 35)/10]\}^{-1}$$

$$\alpha_n(V_i) = \frac{0.125(V_i + 55)}{1 - \exp[-(V_i + 55)/10]}, \quad \beta_n(V_i) = 0.125\exp[-(V_i + 65)/80]$$

where the constants $g_{Na} = 36$, $g_k = 120$, and $C_m = 0.3$ mS cm$^{-2}$ are, respectively, the maximal conductance of potassium, sodium, and leakage currents; $C = 1 \mu$F cm$^{-2}$ is the membrane capacitance; $V_{Na} = -77$ mV, $V_{K} = 50$ mV, and $V_{L} = -54.4$ mV represent corresponding reversal potentials. $\xi_i(t)$ is Gaussian white noises with zero mean $\langle \xi_i(t) \rangle = 0$ and auto-correlation functions $\langle \xi_i(t)\xi_j(t') \rangle = D_{ij}\delta(t-t')$, and noise intensity $D = 2.0$.

Synaptic current $I_{in}^{syn}$ takes chemical coupling form [60]:

$$I_{in}^{syn} = -\sum_{j=1}^{N} \epsilon_{ij}C_{ij}\xi_j(V_j - V_{syn}).$$

(3)

$$\dot{s}_j = \alpha(V_j)(1 - s_j) - \beta s_j,$$

(4)

$$\alpha(V_j) = \alpha_0 \{1 + e^{-V_j/(k_0)}\}$$

(5)

where $C_{ij} = 1$ if neuron $j$ couples to neuron $i$, and $C_{ij} = 0$ otherwise. The reversal potential is chosen as $V_{syn} = 0$. The synaptic recovery function $F(V_j)$ can be taken as the Heaviside function. $V_j(t - \tau)$ is the action potential of neuron $i$ at earlier time $t - \tau$, $\tau$ (in unit of ms) is delayed time. $V_{shp} = 5.0$ determines the threshold above which the postsynaptic neuron is affected by the presynaptic one. Other parameters $\alpha_0$ and $\beta$ are chosen as $\alpha_0 = 2$ and $\beta = 1$. According to STDP mechanism, synaptic coupling strength $\epsilon_{ij}$ varies through STDP modification function $F$, which is defined as follows:

$$\epsilon_{ij}(t + \Delta t) = \epsilon_{ij}(t) + \Delta \epsilon_{ij},$$

(6)

$$\Delta \epsilon_{ij} = \epsilon_{ij}(\Delta t),$$

(7)

$$F(\Delta t) = \begin{cases} A_p \exp\left(-\frac{\Delta t}{\tau_p}\right) & \text{if } \Delta t > 0 \\ -A_m \exp\left(-\frac{\Delta t}{\tau_m}\right) & \text{if } \Delta t < 0 \end{cases}$$

(8)

where $\Delta t = t_j - t_i$ ($t_j$ is marked as the spiking time of the $i$th ($j$th) neuron). The amount of synaptic modification is limited by $A_p$ and $A_m$, the adjusting rate of STDP. $\tau_p$ and $\tau_m$ determine the temporal window for synaptic refinement. Experimental investigations suggest that the temporal window for synaptic weakening is roughly the same as that for synaptic strengthening [46,47]. Potentiation is consistently induced when the postsynaptic spike generates within a time window of 20 ms after presynaptic spike, and depression is induced conversely. STDP is usually viewed as dominant depression. Hence, the parameters are set to be $\tau_p = \tau_m = 20$ [66] and $A_m/A_p = 1.005$. Here $A_p$ can be chosen as an alterable parameter of STDP. All excitable synapses considered in this paper are initiated as $\epsilon_{ij} = \epsilon_{max}/2 = 0.1$, where $\epsilon_{max} = 0.2$ is the coupling upper limit.

To view the change of average coupling strength, we average $\epsilon_{ij}$ over the whole population and time by:

$$\epsilon_{ave} = \lim_{T \to \infty} \frac{1}{N^2T} \sum_{i=1}^{N} \sum_{j=1}^{N} \epsilon_{ij}(t).$$

(9)

As mentioned above, the effect of depression is more powerful than potentiation (i.e., $A_m$ is larger than $A_p$), under which the
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