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Spike-timing-dependent plasticity optimized coherence resonance and synchronization transitions by autaptic delay in adaptive scale-free neuronal networks

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ABSTRACT

In this paper, we numerically study the effect of spike-timing-dependent plasticity on multiple coherence resonance and synchronization transitions induced by autaptic time delay in adaptive scale-free Hodgkin–Huxley neuron networks. As the adjusting rate A_p of spike-timing-dependent plasticity increases, multiple coherence resonance and synchronization transitions enhance and become strongest at an intermediate A_p value, indicating that there is optimal spike-timing-dependent plasticity that can most strongly enhance the multiple coherence resonance and synchronization transitions. As A_p increases, increasing network average degree has a small effect on multiple coherence resonance, but its effect on synchronization transitions changes from suppressing to enhancing it. As network size is varied, multiple coherence resonance and synchronization transitions nearly do not change. These results show that spike-timing-dependent plasticity can simultaneously optimize multiple coherence resonance and synchronization transitions by autaptic delay in the adaptive scale-free neuronal networks. These findings provide a new insight into spike-timing-dependent plasticity and autaptic delay for the information processing and transmission in neural systems.

1. Introduction

Stochastic resonance (SR) and coherence resonance (CR), a counterintuitive phenomenon that a suitable level of noise amplifies a weak signal and enhances the response of the nonlinear system, has been found in various neuronal systems [1–3]. Synchronization phenomenon is correlated with many physiological mechanisms of normal and pathological brain functions including several neural diseases [4,5]. Synchronization in complex networks including neuronal networks have been extensively studied [6,7]. In recent decade, SR and CR in neuronal networks have been observed, such as SR and CR in neuronal networks [8–18], spatial CR in excitable media and neuronal networks [19–21], spatial decoherence by small-world connectivity in neuronal networks [22], as well as multiple SR (MSR) and multiple CR (MCR) induced by time delay and neuronal coupling in neuronal networks [23–25]. Meanwhile, many novel synchronization phenomena have been found, such as synchronization induced by time delay [26–29] and synchronization transitions (ST) induced by time delay, coupling, and noise in neuronal networks [30–40].

Autapse is a special synapse which occurs between dendrites and axon of the same neuron and connects a neuron to itself, and these self-connections could establish a time-delayed feedback mechanism at the cellular level [41]. Autapses serve as feedback circuits and exist in approximately 80% of cortical pyramidal neurons including neurons in the human brain [42–44]. The roles of autapses in the firing dynamics of neuronal systems have been intensively studied. It is shown that autaptic activity can enhance the time precision of spikes of neurons, SR in scale-free neuronal networks, and the firing regulation of interneurons [45–48]. It can engineer the synchronization of action potentials in cultured neurons [49], induce rich firing patterns in a Hindmarsh–Rose model neuron [50], and enhance the propagation of weak rhythmic activity across small-world neuronal networks [51]. Recently, MCR and ST induced by autaptic time delay in neuronal networks have also been observed [52–57].

As is known, neural networks are adaptive due to synaptic plasticity, and synaptic strength varies as a function of neuromodulation and time-dependent processes. One representative of this biological effect is spike-timing-dependent plasticity (STDP), which modulates coupling strength adaptively based on the relative

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timing between pre- and post-synaptic action potentials [58,59]. A series of biological works have confirmed the existence of STDP, which commonly occurs in excitatory synapses onto hippocampal pyramidal and neocortical neurons [59,60], excitatory neurons in auditory brainstem [61], parvalbumin-expressing fast-spiking striatal interneurons [62], etc. Recently, the effect of STDP on the firing dynamics of neuronal systems has been intensively studied [63–71]. For example, Li et al. found that self- organization of a neural network with STDP can enhance spiking temporal coherence and SR [66]; Kube et al. found that STDP modifies the weights of synaptic connections in such a way that synchronization of neuronal activity is considerably weakened [67]; Mikkellsen et al. found that STDP induces persistent irregular oscillations between strongly and weakly synchronized states [70]; Yu et al. showed that STDP can largely depress the temporal coherence and spatial synchronization induced by external noise and random shortcuts [71], and it can enhance or depress SR in the small-world neuronal networks depending on its adjusting rate [72]. Very recently, we have studied the effect of STDP on MCR and ST induced by synaptic and autaptic time delay in small-world neuronal networks. We found that MCR is suppressed (enhanced) as the adjusting rate of STDP increases (decreases), and there is optimal adjusting rate by which ST becomes strongest [73,74]. So far, however, there is no study on MCR and ST induced by autaptic time delay in adaptive scale-free neuronal networks with STDP.

In this paper, we study the effect of STDP on MCR and ST induced by autaptic time delay in adaptive scale-free Hodgkin–Huxley neuronal networks, and we aim to study if there is optimal STDP for both MCR and ST. We first present MCR and ST induced by autaptic time delay when the adjusting rate of STDP is fixed, and then focus on the effect of STDP on MCR and ST by investigating how MCR and ST vary when the adjusting rate of STDP is varied. We also study how STDP influences the effect of network average degree and network size on MCR and ST. Mechanism is briefly discussed. Finally, conclusion is given.

2. Model and equations

We use Hodgkin–Huxley neuron model [75] and Barabási–Albert scale-free networks [76]. The present network comprising $N = 100$ neurons starts with m_0 connected nodes, and subsequently every new node is attached to $m(\leq m_0)$ 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 $p = k_i/\sum_j k_j$. This growth and preferential attachment scheme yields a network with an average degree $\langle k\rangle = \sum_i k_i/N = 2m$ and a power-law degree distribution with the slope of the line equaling – 2.9 on a double logarithmic graph. Here we set $m = m_0$.

In the presence of autaptic current, the firing dynamics of adaptive scale-free Hodgkin– Huxley neuronal networks can be written as:

$$C\frac{dV_i}{dt} = -g_{Na}m_i^3h_i(V_i - V_{Na}) - g_Kn_i^4(V_i - V_K) - G_L(V_i - V_L) + I_{aut_i} + I_i^{syn} + \xi_i(t) \quad (1)$$

where $C = 1\,\mu\text{Fcm}^{-2}$ is the membrane capacitance; $g_K = 36$, $g_{Na} = 120$, and $G_L = 0.3$ mS cm^{-2} are the maximal conductance of potassium, sodium, and leakage currents, respectively; $V_K = -77$ mV, $V_{Na} = 50$ 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 ., noise intensity $D = 2.0$. Gating variables m, h and n governing the stochastic dynamics of sodium and potassium channels obey the following equations:

$$\frac{dx_i}{dt} = \alpha_{x_i}(V_i)(1 - x_i) - \beta_{x_i}(V_i)x_i, \qquad (x = m, h, n) \quad (2)$$

with opening and closing rates:

$$\alpha_{m_i}(V_i) = \frac{0.1(V_i + 40)}{1 - \exp[-(V_i + 40)/10]},$$
$$\beta_{m_i}(V_i) = 4\exp[-(V_i + 65)/18],$$

$$\alpha_{h_i}(V_i) = 0.07\exp[-(V_i + 65)/20],$$
$$\beta_{h_i}(V_i) = \{1 + \exp[-(V_i + 35)/10]\}^{-1},$$

$$\alpha_{n_i}(V_i) = \frac{0.01(V_i + 55)}{1 - \exp[-(V_i + 55)/10]},$$
$$\beta_{n_i}(V_i) = 0.125\exp[-(V_i + 65)/80],$$

I_{aut_i} is autaptic current and here takes chemical form [77]:

$$I_{aut_i} = -g_{aut}[V_i(t) - V_{syn}]S_i(t - \tau)$$
$$S_i(t - \tau) = 1/\{1 + \exp[-k(V_i(t - \tau) - \theta)]\}, \quad (3)$$

where g_{aut} is autaptic conductance, V_{syn} is autaptic reversal potential and here we choose $V_{syn} = 2$ mV for excitatory autapses. $V_i(t - \tau)$ is the action potential of neuron i at earlier time $t - \tau$, τ (in unit of ms) is autaptic delayed time. We assume all neurons have equal g_{aut} and equal τ. Other parameters are: $k = 8$, $\theta = -0.25$.

We use synaptic current I_i^{syn} in chemical form as [71]:

$$I_i^{syn} = -\sum_{j=1(j\neq i)}^{N} \varepsilon_{ij}C_{ij}\eta_j(V_i - V_{syn}), \quad (4)$$

$$\dot{\eta}_j = \alpha(V_j)(1 - \eta_j) - \beta\eta_j, \quad (5)$$

$$\alpha(V_j) = \alpha_0/\left(1 + e^{-V_j/V_{shp}}\right), \quad (6)$$

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 α (V_j) can be taken as the Heaviside function. $V_{shp} = 5.0$ determines the threshold above which the postsynaptic neuron is affected by the pre-synaptic one. Other parameters α_0 and β are chosen as $\alpha_0 = 2$ and $\beta = 1$. Synaptic coupling strength ε_{ij} varies through STDP modification function F, which is defined as follows:

$$\varepsilon_{ij}(t + \Delta t) = \varepsilon_{ij}(t) + \Delta\varepsilon_{ij}, \quad (7)$$

$$\Delta\varepsilon_{ij} = \varepsilon_{ij}F(\Delta t), \quad (8)$$

$$F(\Delta t) = \begin{cases} A_p\exp(-|\Delta t|/\tau_p) & \text{if } \Delta t > 0 \\ -A_m\exp(-|\Delta t|/\tau_m) & \text{if } \Delta t < 0, \end{cases} \quad (9)$$

where $\Delta t = t_i - t_j$, and t_i (or t_j) marks the spiking time of the ith (jth) neuron. The amount of synaptic modification is limited by A_p and A_m, called the adjusting rate of STDP. τ_p and τ_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 [59,60]. Potentiation is consistently induced when the postsynaptic spike generates within a time window of 20 ms after presynaptic spike, and depression is induced conversely. Thus, the parameters are set to be $\tau_p = \tau_m = 20$ [78]. Considering that STDP is usually viewed as dominant depression, we choose $A_m/A_p = 1.005$. In this study, A_p is chosen as a main variable of STDP, and all excitable synapses considered are initiated as $\varepsilon_{ij} = \varepsilon_{\max}/2 = 0.1$, where $\varepsilon_{\max} = 0.2$ is the upper limit of coupling.