Multiple coherence resonance and synchronization transitions induced by autaptic delay in Newman–Watts neuron networks

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A B S T R A C T

In this paper, we study the effect of delayed autaptic self-feedback activity on the spiking temporal coherence and spatial synchronization of Newman–Watts networks of stochastic Hodgkin–Huxley neurons. As autaptic delay is varied, the spiking behaviors of the neurons intermittently become synchronous and non-synchronous, and meanwhile they are ordered and disordered, exhibiting both synchronization transitions (ST) and multiple coherence resonance (MCR). Moreover, the autaptic delays for CR and synchronization are close. When coupling strength or network randomness increases, or when channel noise intensity decreases, MCR is enhanced, but ST becomes strongest at optimal coupling strength, network randomness, or channel noise intensity. These results show that autaptic activity can induce both MCR and ST in the neuronal networks. This implies that autaptic activity can simultaneously enhance or reduce the temporal coherence and synchronization of the neuronal network. These findings could find potential implications for the information processing and transmission in neural systems.

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1. Introduction

Information transmission delays are inherent to neural systems due to the finite propagation speeds and time lapses occurring in both dendritic and synaptic processing [1]. Physiological experiments have revealed that time delay introduced by chemical and electrical synapses synapses can be up to several tenths of milliseconds in length and comparably short about 0.05 ms, respectively [2,3]. Several decades ago, Van der Loos and Glaser found a special synapse, known as autapse which occurs between the 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 [4]. Autapses serve as feedback circuits, which are common in the nervous system and have been discovered in a variety of brain areas. Tamas et al. showed anatomically that inhibitory interneurons in visual cortex form approximately 10–30 autapses [5]. Lübke et al. presented that autaptic connections exist in approximately 80% of the cortical pyramidal neurons, including neurons of the human brain [6]. Bacci et al. reported that fast-spiking but not low-threshold spiking interneurons of layer V in neocortical slices exhibit inhibitory autaptic activity [7]. Over the past decade, the effects of autapse on the firing dynamics of neurons have been extensively studied [8–21], for example, Bacci and Huguenard experimentally found that autaptic transmission enhances the precision of spike times of neurons [10]; Popovych et al. showed that time-delayed self-feedback can desynchronize groups of model

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neurons [12]; Prager et al. reported a semi-analytical method to study noise induced oscillation with time-delayed feedback [13]; Saada et al. found that an autapse can mediate positive feedback, which maintains persistent activity [16]; Rusin et al. experimentally demonstrated that delayed self-feedback stimulation can engineer the synchronization of action potentials in cultured neurons [18]; Hashemi et al. showed that the spike rate of a neuron depended on the duration of the activity of autapse [19]. Recently, Wang et al. found that delayed autaptic activity switches the spiking activity among quiescent, periodic and chaotic firing patterns in a Hindmarsh–Rose neuron [20], and the firing frequencies and inter-spike interval distribution of the spike train of a neuron shows periodic behaviors as autaptic delay time is increased [21]. They have also reviewed the firing dynamics of an autaptic neuron [22]. Very recently, Yilmaz and Özer have found that autaptic activity can enhanced detection of weak periodic signals in a stochastic Hodgkin–Huxley neuron [23] and pacemaker induced stochastic resonance in a scale-free neuronal network [24], as well as pacemaker mediated propagation of weak rhythmic activity across small-world neuronal networks [25].

Stochastic resonance (SR) is a counterintuitive phenomenon that a suitable level of noise enhances the response of a nonlinear system to external signals, and coherence resonance (CR) is a phenomenon that a suitable level of noise amplifies the intrinsic oscillation signal of nonlinear systems. SR and CR have been extensively studied in various nonlinear systems including neuronal systems over the past decades [26–28]. Synchronization phenomenon often occurs in the spiking activity of coupled neurons, and it is particularly correlated with many physiological mechanisms of normal and pathological brain functions including several neural diseases [29–33]. In the past years, synchronization phenomenon has been widely studied in complex networks including neuronal networks [34,35]. In recent decade, people have found some novel SR and synchronization phenomena in neuronal systems, such as temporal and spatial SR and CR in excitable and neuronal systems [36–42], channel blocking enhanced spiking regularity in clustered neuronal networks [43], and synchronization due to time delay in neuronal networks [44–49]. In particular, multiple SR (MSR) and multiple CR (MCR) (i.e., SR or CR intermittently appear with a varying parameter) [50–52], as well as synchronization transitions (ST) due to time delay [53–57], coupling strength [56–60], and noise [61,62] have been found in various neuronal networks. Very recently, we have observed ST induced by channel noise in delayed Newman–Watts networks of stochastic Hodgkin–Huxley (HH) neurons [63], ST induced by autaptic delay in non-delayed Newman–Watts HH neuron networks [64] and in delayed Newman–Watts HH neuron networks [65]. So far, however, the effect of autaptic delay on the synchronization of stochastic HH neuron networks has not yet been studied. Particularly, it is not clear if autaptic delay can induce MCR or both MCR and ST in stochastic HH neuron networks.

In this paper, we study the effect of autaptic self-feedback activity on the temporal coherence and spatial synchronization of Newman–Watts stochastic HH neuron networks. We aim to investigate if autaptic activity can induce MCR and ST in the neuronal network. We first study how autaptic delay induces MCR and ST, and then explore the effects of coupling strength, network randomness and channel noise on the MCR and ST. We find that the neurons exhibit both MCR and ST as autaptic delay is varied, and CR and synchronization appear almost at the same autaptic delays. When coupling strength or network randomness increases, MCR is enhanced and ST becomes strongest at optimal coupling strength or network randomness. Similarly, when channel noise intensity decreases, MCR is enhanced and ST becomes strongest at optimal channel noise intensity. Finally, mechanisms are briefly discussed, and conclusion is given.

2. Model and equations

According to Hodgkin and Huxley's work [66], the dynamics of the membrane potentials of a HH neuron with a large number of ion channels can be described by deterministic equation:

$$C\frac{dV}{dt} = -g_{\mathrm{Na}}m^3h(V - V_{\mathrm{Na}}) - g_{\mathrm{K}}n^4(V - V_{\mathrm{K}}) - g_{\mathrm{L}}(V - V_{\mathrm{L}}) + I_{aut}, \tag{1}$$

where $C = 1\,\mu\mathrm{F\,cm^{-2}}$ is the membrane capacitance, and $V_{\mathrm{Na}} = 50\,\mathrm{mV}$, $V_{\mathrm{K}} = -77\,\mathrm{mV}$, $V_{\mathrm{L}} = -54.4\,\mathrm{mV}$ are the reversal potentials for the sodium, potassium, and leakage currents, respectively, and $g_{\mathrm{K}} = 36\,\mathrm{mS\,cm^{-2}}$ and $g_{\mathrm{K}} = 120\,\mathrm{mS\,cm^{-2}}$ $g_{\mathrm{L}} = 0.3\,\mathrm{mS\,cm^{-2}}$ are maximal conductances for potassium, sodium, and leakage currents, respectively. However, for a HH neuron with a small number of ion channels, there is fluctuation (noise) arising from stochastic opening–closing of the channel gates, and the dynamics of the neuron should be described by stochastic HH model. The stochastic gating variables m, h, and n obey the following Langevin equations [67,68]:

$$\frac{dm}{dt} = \alpha_m(V)(1 - m) - \beta_m(V)m + \xi_m(t), \tag{2a}$$

$$\frac{dh}{dt} = \alpha_h(V)(1 - h) - \beta_h(V)h + \xi_h(t), \tag{2b}$$

$$\frac{dn}{dt} = \alpha_n(V)(1 - n) - \beta_n(V)n + \xi_n(t), \tag{2c}$$

with the experimentally determined voltage-dependent opening and closing transition rates:

$$\alpha_m(V) = \frac{0.1(V + 40)}{1 - \exp[-(V + 40)/10]}, \tag{3a}$$