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Optimal autaptic and synaptic delays enhanced synchronization transitions induced by each other in Newman–Watts neuronal networks

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ABSTRACT

In this paper, we numerically study the effect of electrical autaptic and synaptic delays on synchronization transitions induced by each other in Newman–Watts Hodgkin–Huxley neuronal networks. It is found that the synchronization transitions induced by synaptic delay vary with varying autaptic delay and become strongest when autaptic delay is optimal. Similarly, the synchronization transitions induced by autaptic delay vary with varying synaptic delay and become strongest at optimal synaptic delay. Also, there is optimal coupling strength by which the synchronization transitions induced by either synaptic or autaptic delay become strongest. These results show that electrical autaptic and synaptic delays can enhance synchronization transitions induced by each other in the neuronal networks. This implies that electrical autaptic and synaptic delays can cooperate with each other and more efficiently regulate the synchrony state of the neuronal networks. These findings could find potential implications for the information transmission in neural systems.

1. Introduction

The cerebral cortex is a highly interconnected network of neurons, in which the activity in any neuron is necessarily related to the combined activity of collective neurons. Studies of structural and functional brain networks in humans and other animals have witnessed small- world architectures over a wide range of scales in both space and time [1–3]. By using functional magnetic resonance imaging, power-law distributions were obtained upon linking correlated fMRI voxels [4], and the robustness against simulated lesions of anatomic cortical networks has also been found to rely mostly on the scale-free structure [5]. In neural systems, information transmission between neurons occurs at electrical and chemical synapses, and information transmission delays are inherent due to the finite propagation speeds and due to time lapses occurring by both dendritic and synaptic processing [6]. Physiological experiments have revealed that transmission delay introduced by chemical synapses can be up to several tenths of milliseconds in length, and transmission delays introduced by electrical synapses are comparably short and about 0.05 ms [7,8]. Several decades ago, Van der Loos and Glaser found a special synapse, known as autapse which occurs between dendrites and axon of the same neuron and connect a neuron to itself, and these self-connections could establish a time-delayed feedback mechanism at the cellular level [9]. 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 [10]. Lübke et al. presented that autaptic connections exist in approximately 80% of the cortical pyramidal neurons, including neurons of the human brain [11]. Bacci et al. reported that fast-spiking but not low-threshold spiking interneurons of layer V in neocortical slices exhibit inhibitory autaptic activity [12]. Over the past decade, the effects of autapse on the firing dynamics of neurons have been extensively studied [13–26]. For example, Bacci and Huguenard experimentally found that autaptic transmission enhances the precision of spike times of neurons [15]; Popovych et al. showed that time-delayed self-feedback can desynchronize groups of model neurons [17]; Prager et al. reported a semi-analytical method to study noise induced oscillation with time-delayed feedback [18]; Saada et al. found that an autapse can mediate positive feedback, which maintains persistent activity [21]; Rusin et al. experimentally demonstrated that delayed self-feedback stimulation can engineer the synchronization of action potentials in cultured neurons [23]; Hashemi et al. showed that

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the spike rate of a single Hodgkin–Huxley neuron with a delayed autaptic connection depended on the duration of the activity of the autapse [24]. 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 [25] and the firing frequencies and interspike interval distribution of the output spike train of a Hodgkin–Huxley neuron shows periodic behaviors as autaptic delay time is increased [26].

Synchronization is an important phenomenon that occurs in many biological and physical systems. In biological systems, synchronization is correlated with many physiological mechanisms of normal and pathological brain functions [27–29], and it is related to but not desirable for several neurological diseases such as epilepsy and tremor in Parkinson's disease [30,31]. Synchronization phenomenon has been extensively studied in complex networks including neuronal networks [32,33]. In recent decade, many novel phenomena have been found, such as synchronization induced by noise [34–36], neuronal coupling [37–41], sub-threshold stimulus [42] and time delay [43–49] in coupled neurons and neuronal networks, as well as small-world connectivity enhanced spatial and temporal coherence in neural media [50] and induced spatial decoherence in excitable media [51] and neuronal networks [52]. More importantly, synchronization in networks of diffusively time-delay coupled systems are analyzed, and sufficient conditions for various types of synchronization are obtained [53,54]. In recent years, a novel phenomenon of synchronization transitions in neuronal networks has attracted growing attention. Studies have shown that synchronization transitions can be induced by time delay [55–62], coupling strength [62–64], and even noise [65,66]. Recently, we have studied the effect of autapses on the synchronization of neuronal networks. We found that autaptic delay can induce synchronization transitions in Newman–Watts neuronal networks [67], chemical autaptic delay and electrical synaptic delay can enhance synchronization transitions induced by each other in Newman–Watts neuronal networks [68], and autaptic and synaptic delay can intermittently enhance synchronization transitions induced by each other in scale-free thermo-sensitive neuronal networks [69]. However, it is not clear how electrical autaptic and synaptic delays affect synchronization transitions induced by each other in neuronal networks.

In this paper, we study the effect of electrical autaptic and synaptic delays on synchronization transitions induced by each other in Newman–Watts Hodgkin–Huxley neuron networks. We first study the effect of autaptic delay on synchronization transitions induced by synaptic delay, and then the effect of synaptic delay on synchronization transitions induced by autaptic delay. Thirdly, we explore the effect of coupling strength on synchronization transitions induced by synaptic and autaptic delay. Finally, mechanism is briefly discussed and conclusion is given.

2. Model and equations

According to Newman–Watts (NW) topology [70], the present NW network comprising of $N=60$ identical Hodgkin–Huxley neurons starts with a originally regular ring in which each unit is connected to its two nearest neighbors, and then links are randomly added with probability p (network randomness) between non-nearest vertices. When all neurons are coupled with each other, the network contains $N(N-1)/2$ edges. The number of added random shortcuts M satisfies $M = p \times N(N-1)/2$. If $p=0$, the network is a regular ring, and for $p=1$ the network is globally coupled. For $0 < p < 1$, the Newman–Watts small-world topology occurs. Note that for a given p there are a lot of network realizations.

In the presence of autaptic current, the dynamics of NW Hodgkin–Huxley neuronal networks with delayed electrical coupling can be written as:

$$C\frac{dV_i(t)}{dt} = -g_{Na}m_i^3h_i(V_i - V_{Na}) - g_K n_i^4(V_i - V_K) - G_L(V_i - V_L) + \sum_j \varepsilon_{ij}\left[V_j(t-\tau) - V_i(t)\right] + I_{aut_i} + \xi_i(t) \quad (1)$$

where capacity $C = 1\mu\mathrm{Fcm}^{-2}$, $V_i(t)$ is the membrane potential of neuron i at time t, $V_{Na} = 50\mathrm{mV}$, $V_K = -77\mathrm{mV}$, $V_L = -54.4\mathrm{mV}$ are the reversal potentials for the sodium, potassium, and leakage currents, respectively. $g_K = 36\,\mathrm{mS\,cm}^{-2}$ and $g_K = 120\,\mathrm{mS\,cm}^{-2}$ are maximal conductance for potassium and sodium ions, respectively, and $G_L = 0.3\,\mathrm{mS\,cm}^{-2}$ is leakage conductance. Gating variables m, h and n governing the stochastic dynamics of sodium and potassium channels obey the following equations:

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

with opening and closing rates:

$$\alpha_m(V) = \frac{0.1(V+40)}{1-\exp[-(V+40)/10]},\ \beta_m(V) = 4\exp[-(V+65)/18] \quad (3a)$$

$$\alpha_h(V) = 0.07\exp[-(V+65)/20],\ \beta_h(V) = \{1+\exp[-(V+35)/10]\}^{-1}, \quad (3b)$$

$$\alpha_n(V) = \frac{0.01(V+55)}{1-\exp[-(V+55)/10]},\ \beta_n(V) = 0.125\exp[-(V+65)/80]. \quad (3c)$$

The term $\sum_j \varepsilon_{ij}[V_j(t-\tau_1) - V_i(t)]$ is delayed electrical coupling, where $V_j(t-\tau_1)$ is the action potential of neurons j at earlier time $t-\tau_1$, τ_1 (in unit of ms) is time delay between neurons i and j, and the summation takes over all neurons. ε_{ij} is coupling strength between neurons i and j; $\varepsilon_{ij} = \varepsilon$ if there is coupling, and $\varepsilon_{ij} = 0$ otherwise. $\xi_i(t)$ is Gaussian white noises with zero mean $\langle\xi_i(t)\rangle = 0$ and auto-correlation functions $\langle\xi_i(t)\xi_j(t')\rangle = D\delta_{ij}\delta(t-t')$, noise intensity $D=0.02$. I_{aut_i} is autaptic current. Here, we employ electrical diffusive-type autapse [22,25]:

$$I_{aut_i} = g_{aut}[V_i(t-\tau_2) - V_i(t)], \quad (4)$$

where g_{aut} is autaptic self-feedback strength (conductance), $V_i(t-\tau_2)$ is the action potential of neuron i at earlier time $t-\tau_2$, τ_2 (in unit of ms) is autaptic delayed time. Here we assume all neurons have equal g_{aut} and equal τ_2.

The synchronization of the neuronal network can be quantified by standard deviation σ defined as [71]:

$$\sigma = [\langle\sigma(t)\rangle] \quad \text{with} \quad \sigma(t) = \sqrt{\frac{\frac{1}{N}\sum_{i=1}^{N}V_i(t)^2 - \left(\frac{1}{N}\sum_{i=1}^{N}V_i(t)\right)^2}{N-1}}, \quad (5)$$

where $\langle\cdot\rangle$ denotes the average over time and $[\cdot]$ the average over different network realizations for each set of parameter values. Larger σ represents bigger deviation between the neurons, and smaller σ shows higher synchronization.

We perform numerical integrations of eqs. (1) – (4) using explicit Euler algorithm with time step of 0.001 ms. Total calculation time length is 5000 ms including 1000 ms for transient. Periodic boundary conditions are used and the parameter values for all the neurons are identical except for noise terms $\xi_i(t)$ for each neuron.