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Transition and enhancement of synchronization by time delays in stochastic Hodgkin–Huxley neuron networks

Yinghang Hao, Yubing Gong *, Xiu Lin, Yanhang Xie, Xiaoguang Ma

School of Physics, Ludong University, Yantai, Shandong 264025, PR China

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ABSTRACT

In this paper, we study the effect of time delay on spiking synchronizations in Newman–Watts networks of stochastic Hodgkin–Huxley (HH) neurons. It is found that as τ is increased, the neurons exhibit transitions from spiking synchronization (SS) to clustering anti-phase synchronization (APS) and back to SS. Furthermore, the SS after the APS is enhanced with increasing time delay. For different patch sizes (channel noise strength), network randomness (fraction of random connections), and coupling strengths, the neurons exhibit similar synchronization transitions and the APS always occurs at around $\tau=4$, representing that the time delay-induced APS behavior is robust to the channel noise, the number of random connections, and the coupling strength. A simple explanation for this phenomenon was given in terms of the relation of spiking time-period and time delay values.

Since the information processing in the neurons are fulfilled by the spiking activity of the membrane potential and the spiking synchronization plays a crucial role in the spiking activity, our results may help us understand the effect of time delay as well as the interplay of channel noise and time delay on the spiking activity and hence the information processing in stochastic neuronal systems.

1. Introduction

In the last two decades, synchronizations on neuronal networks have received much attention [1–21]. Synchronous activity is considered to play important roles in information processing in the brain [1–3], and they are also relevant to several neurological diseases such as epilepsy and tremor in Parkinson's disease [4,5]. Due to its importance, people have performed extensive studies on the synchronizations in neuronal systems [6–21] and found many phenomena, such as noise-induced synchronization in modified Hodgkin–Huxley (MHH) neurons [9], chemical synapse-induced synchronization in a ring neuronal network [10], two coupled map-based neurons [11], a square lattice noisy neuronal network [12], and scale-free networks of Morris–Lecar neurons [13]; burst-enhanced synchronization in an array of noisy coupled MHH neurons [14]; and synchronization in a large ensemble of MHH neurons with gap junctions [15] and small-world networks of neurons [16–20]. Recently, synchronization transitions in neuronal systems have attracted growing attention. People have found the transition from spatiotemporal chaos to bursting synchronization (BS) with increasing coupling strength in Hindmarsh–Rose neuron networks [21] and the transitions between various synchronizations under different coupling strengths and external currents in two electrically coupled MHH neurons [22].

In neuronal systems, time delays are inherent because of both finite propagation velocities in the conduction of signals along neurites and the delays in synaptic transmission [23]. More recently, firing dynamics of neuronal systems with time delays have been intensively studied. People have found that time delays can facilitate and improve neuronal synchronization [24–26], destabilize synchronous states and induce near-regular wave states [27], induce various spatiotemporal patterns [28] and multiple stochastic resonances [29], and enhance the coherence of spiral waves [30]. Very recently, time delay-induced synchronization transitions on neuronal networks have attracted growing interests. It is found that time delays can induce the synchronization transitions in two coupled fast-spiking neurons [31] and small-world neuronal networks of Rulkov map [32], and can intermittently induce the synchronization transitions on scale-free neuronal networks [33].

All these works have studied the synchronization transitions on deterministic neuronal networks with time delays. However, neurons are noisy elements. External noise arises from environmental fluctuations and synapses, and internal noise comes from stochasticity of dynamical processes in ion channels. The roles of noise in the firing dynamics of neurons have been extensively studied [34–45]. Obviously, it is of significance to study the synchronization and transition in stochastic neurons with time delays.

* Corresponding author. Tel.: +86 535 6697550; fax: +86 535 6672870.
E-mail address: gongyubing09@hotmail.com (Y. Gong).

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In this paper, based on Newman–Watts networks of stochastic HH neurons, we study the effect of time delay τ on the spiking synchronization and how the channel noise affects the time delay-induced spiking behavior. It is found that, as τ is increased, the neurons exhibit transitions from SS to clustering APS and back to SS. And the SS after the APS becomes enhanced with increasing time delay τ. For different network randomnesses, patch sizes (channel noise), and coupling strengths, there are similar synchronization transitions and the APS almost always occurs at around time delay $\tau=4$, which represents that the channel noise, the number of random connections, and the coupling strength nearly take no effect on the time delay-induced synchronization transitions.

2. Model and equations

According to the HH neuron model, the dynamics of the membrane potential $V(t)$ can be described by

$$C\frac{dV}{dt}=-g_{Na}m^3h(V-V_{Na})-g_K n^4(V-V_K)-g_L(V-V_L)+I(t), \tag{1a}$$

where the constants $g_{Na}=120\ \mathrm{mS/cm^2}$, $g_K=36\ \mathrm{mS/cm^2}$, and $g_L=0.3\ \mathrm{mS/cm^2}$ are the maximal conductance of sodium, potassium, and leakage conductance, respectively. $C=1\ \mu\mathrm{F/cm^2}$ is the membrane capacitance; $V_{Na}=50$ mV, $V_K=-77$ mV, and $V_L=-54.4$ mV are the reversal potentials of sodium, potassium, and leakage currents, respectively. We employ a periodic stimulus $I=\sin(0.3t)$, which is a subthreshold stimulus and does not trigger action potentials if intrinsic channel noise is not taken into account. Following Hodgkin and Huxley's work, the gating variables m, h, and n describe the mean ratios of the open gates of the working sodium and potassium channels, and the factors n^4 and m^3h are the mean portions of the open potassium and sodium ion channels within the membrane patch. To take into account the channel noise, the stochastic gating variables m, h, and n obey the following Langevin equations:

$$\dot{m}=\alpha_m(V)(1-m)-\beta_m(V)m+\xi_m(t), \tag{1b}$$

$$\dot{h}=\alpha_h(V)(1-h)-\beta_h(V)h+\xi_n(t), \tag{1c}$$

$$\dot{n}=\alpha_n(V)(1-n)-\beta_n(V)n+\xi_n(t), \tag{1d}$$

with voltage-dependent opening–closing transition rates given by

$$\alpha_m(V)=\frac{0.1(V+40)}{1-\exp[-(V+40)/10]}, \tag{2a}$$

$$\beta_m(V)=4\exp[-(V+65)/18], \tag{2b}$$

$$\alpha_h(V)=0.07\exp[-(V+65)/20], \tag{2c}$$

$$\beta_h(V)=\frac{1}{1+\exp[-(V+35)/10]}, \tag{2d}$$

$$\alpha_n(V)=\frac{0.01(V+55)}{1-\exp[-(V+55)/10]}, \tag{2e}$$

$$\beta_n(V)=0.125\exp[-(V+65)/80], \tag{2f}$$

where $\xi_{i=m,h,n}(t)$ are Gaussian white noises with vanishing mean and auto-correlation function $\langle\xi(t)_i\xi(t')_i\rangle=D_i\delta(t-t')$. $D_{i=m,n,h}$ represent the effective channel noise strengths:

$$D_m=\frac{2}{N_{Na}}\frac{\alpha_m\beta_m}{\alpha_m+\beta_m}, \tag{3a}$$

$$D_h=\frac{2}{N_{Na}}\frac{\alpha_h\beta_h}{\alpha_h+\beta_h}, \tag{3b}$$

$$D_n=\frac{2}{N_K}\frac{\alpha_n\beta_n}{\alpha_n+\beta_n}. \tag{3c}$$

The overall numbers of involved potassium and sodium ion channel are rescaled by N_{Na} and N_K, respectively. With the assumption of homogeneous ion channels densities, $\rho_{Na}=60\ \mu\mathrm{m}^{-2}$ and $\rho_K=18\ \mu\mathrm{m}^{-2}$, the ion channel numbers are given by $N_{Na}=\rho_{Na}S$ and $N_K=\rho_K S$, where S is the membrane patch size.

Now the membrane potential dynamics of coupled HH neurons on the complex networks can be described by the following equations:

$$C\frac{dV_i}{dt}=-g_{Na}m_i^3h_i(V_i-V_{Na})-g_K n_i^4(V_i-V_K)-g_L(V_i-V_L)+\sum_j\varepsilon_{ij}(V_j(t-\tau)-V_i), \tag{4a}$$

$$\frac{dx_i}{dt}=\alpha_{x_i}(v_i)(1-x_i)-\beta_{x_i}(v_i)x_i+\xi_{x_i}(t), \tag{4b}$$

where $x=m, h, n$ and $1\le i\le N$. Here N is the number of neurons and τ is the delay time in the unit of ms. Eqs. (1)–(4) constitute the stochastic HH network model. In the coupling term $\sum_j\varepsilon_{ij}[V_j(t-\tau)-V_i]$, V_i is the membrane potential of the ith neuron at time t and $V_j(t-\tau)$ is the membrane potential of the jth neuron at earlier time $t-\tau$, where τ is the time delay; $1\le(i,j)\le N$, and the summation is over all neurons; ε_{ij} is a coupling constant between the two neurons i and j, which is determined by the coupling pattern of the system and is identical for any two neurons, i.e., $\varepsilon_{ij}=\varepsilon$. If neurons i and j are connected, they have a constant coupling strength $\varepsilon=0.1$; otherwise $\varepsilon=0$.

The neuronal network here is constructed as follows [17]. It starts with a regular ring involving $N=60$ identical HH neurons, each neuron having two nearest neighbors. Links are then randomly added between non-nearest vertices. In the limit case, all neurons are coupled to each other and the network contains $N(N-1)/2$ edges. Using M to denote the number of added shortcuts, the fraction of shortcuts is given by $p=M/[N(N-1)/2]$, which can be used to characterize the randomness of the network. The sketches of the network with $N=10$ when $p=0$, 0.11 are shown in Fig. 1. Note that for a given p there are a lot of realizations of networks.

We introduce the standard deviation σ to measure the spatial synchronization. Here σ is defined as

$$\sigma=[\langle\sigma(t)\rangle],$$

$$\sigma(t)=\sqrt{\left[(1/N)\sum_{i=1}^{N}V_i(t)^2-((1/N)\sum_{i=1}^{N}V_i(t))^2\right]/(N-1)}, \tag{5a}$$

where $\langle\cdot\rangle$ denotes the average over time and $[\cdot]$ the average over 50 different network realizations for each p. The value of $\sigma(t)$

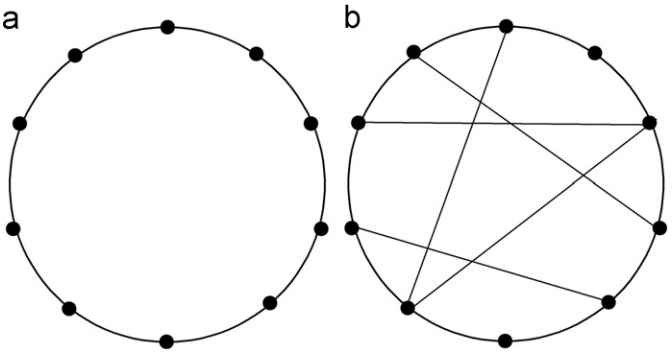

Fig. 1. (a) Regular ring network ($p=0$) and (b) random network ($p\approx0.11$).