BioSystems 106 (2011) 76–81

Contents lists available at ScienceDirect

BioSystems

journal homepage: www.elsevier.com/locate/biosystems

Influence of time delay and channel blocking on multiple coherence resonance in Hodgkin–Huxley neuron networks

Yubing Gong*, Yinghang Hao, Xiu Lin, Li Wang, Xiaoguang Ma

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

ARTICLE INFO

Article history:
Received 28 November 2010
Received in revised form 24 June 2011
Accepted 4 July 2011

PACS:
87.18.Tt
87.19.lj
87.19.ln

Keywords:
Neuron
Channel blocking
Noise
Newman–Watts network
Time delay
Coherence resonance

ABSTRACT

Toxins such as tetraethylammonium (TEA) and tetrodotoxin (TTX) may reduce the number of working potassium and sodium ion channels by poisoning and making them blocked, respectively. In this paper, we study how channel blocking (CB) affects the time delay-induced multiple coherence resonance (MCR), i.e., a phenomenon that the spiking of neuronal networks intermittently reaches the most ordered state, in stochastic Hodgkin–Huxley neuron networks. It is found that potassium and sodium CB have distinct effects. For potassium CB, the MCR occurs more frequently as the CB develops, but for sodium CB the MCR is badly impaired and only the first coherence resonance (CR) holds and, consequently, the MCR evolves into a single CR as sodium CB develops. We found for sodium CB the spiking becomes disordered at larger delay lengths, which may be the reason for the destruction of the MCR. The underlying mechanism is briefly discussed in terms of distinct effects of potassium and sodium CB on the spiking activity. These results show that potassium CB can increase the frequency of MCR with time delay, but sodium CB may suppress and even destroy the delay-induced MCR. These findings may help to understand the joint effects of CB and time delay on the spiking coherence of neuronal networks.

1. Introduction

Toxins such as tetraethylammonium (TEA) and tetrodotoxin (TTX) can make potassium and sodium ion channels poisoned and blocked, and thus allow reducing the number of working potassium and sodium ion channels, respectively (Hill, 2001). Since channel noise depends closely on the number of ion channels, it will change when channel blocking (CB) occurs. It is known that channel noise plays important roles in neuron firing, such as the threshold to spiking and the spiking rate itself (Skaugen and Walløe, 1979; Clay and DeFelice, 1983; Strassberg and DeFelice, 1993; DeFelice and Isaac, 1993; Fox and Lu, 1994; Chow and White, 1996; Schneidman et al., 1998), the anomalous noise-assisted enhancement of transmission of external signals, i.e., stochastic resonance (Collins et al., 1995, 1996; Schmid et al., 2001; Jung and Shuai, 2001; Gammaitoni et al., 1998; Hänggi, 2002), and the efficiency for synchronization (Schmid et al., 2003). Therefore, CB would exert significant influences on these firing activities. Studies have shown that the effect of CB has several different effects at the same time. On one hand, the reduction in the density of sodium channels results in an increase of the activation barrier towards excitation from the resting state and hence the reduction of neuronal activity. On the other hand, however, the corresponding channel noise component will also be increased due to reduction of absolute number of ion channels in the membrane patch. As a consequence, the increased channel noise will help to overcome the activation barrier and to initiate spontaneous spikes. A reduction in the density of potassium channels will in contrast generally result in lowering of the activation barrier and an increase of excitability and, simultaneously, an increase in the recovery time which should favor longer interspike time intervals. Moreover, the reduction in the total number of potassium ion channels will also increase the corresponding channel noise component that may lead to an increased variability of the refractory period (Goldman et al., 2001). Schmid et al. have shown that CB of sodium ion channels results in a reduction in the spontaneous action potentials and causes a less regular production of spikes. While the block of potassium channels causes a surprising increase in the spiking activity and yields in turn a more regular spontaneous spiking coherence (Schmid et al., 2004a,b). Recent studies have shown that appropriate potassium CB can enhance the spiking coherence of a chain of coupled neurons (Gong et al., 2008) and neuronal networks (Ozer et al., 2009; Gong et al., 2010).

It has been shown that the human brain has character of small-world networks (Watts and Strogatz, 1998; Volman et al., 2005; Bassett and Bullmore, 2006; Zhou et al., 2007). Hence, much attention has been devoted to studying the dynamics of small-world

* Corresponding author.
E-mail address: gongyubing09@hotmail.com (Y. Gong).

0303-2647/$ – see front matter © 2011 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.biosystems.2011.07.001

neuronal networks. In neuronal systems, information transmission delays are inherent because of both finite propagation velocities in the conduction of signals along neuritis, either an axon or dendrite, and in the synaptic transmission (Kandel et al., 1991). It has been found that time delays can facilitate and enhance synchronization (Dhamala et al., 2004; Rossoni et al., 2005; Burić et al., 2008), lead to a wide variety of spatio-temporal patterns (Roxin et al., 2005) and rather regular wave patterns (Ko and Ermentrout, 2007), enhance the coherence of spiral waves on diffusively coupled HH neuronal networks (Wang et al., 2008a), and induce multiple stochastic resonance on scale-free Rulkov neuron networks (Wang et al., 2009a). Furthermore, delays have a subtle effect on the synchronization transitions of neuronal networks. They can induce transitions between in-phase and anti-phase synchronizations in two coupled fast-spiking neurons (Wang et al., 2008b), map-based small-world neuronal networks (Wang et al., 2008c), intermittent synchronization transitions on scale-free neuronal networks (Wang et al., 2009b), and burst synchronization, anti-phase synchronization, as well as spiking synchronization in Newman–Watts networks of modified HH neurons (Xie et al., 2010).

Temporal coherence characterizes the regularity of a spike train of a neuron, and when coherence resonance (CR) occurs, the spikes will become the most ordered in time. Since spiking regularity is crucial to the time precision of information processing and signal transmission in neurons, it is of significance to study the effect of CB on the temporal coherence of the delay-induced spiking activity. The previous works have studied the effect of CB on the spiking coherence of a single neuron (Schmid et al., 2004a,b), a chain of coupled neurons (Gong et al., 2008), as well as neuronal networks (Gong et al., 2010). However, the effect of CB on the spiking coherence of delayed neuronal networks has not yet been studied.

In the present paper, we study the effect of CB on delay-induced multiple coherence resonance (MCR) in Newman–Watts networks of stochastic HH neurons, which is different from the previous works and extends the study of CB on the spiking activity of delayed neuronal networks. Firstly, we show that the spiking can intermittently exhibits the most ordered performance, i.e., multiple coherence resonance (MCR), when time delay is varied. Then we investigate how CB affects the delay-induced MCR behavior. The underlying mechanism is briefly discussed in terms of distinct effects of potassium and sodium CB on the spiking activity, and the effects of membrane patch size and network randomness on the delay-induced MCR.

2. Model and Equations

In the presence of CB, the dynamics of membrane potential of a stochastic HH neuron can be described by

$$C\frac{dV(t)}{dt} = -G_{\mathrm{Na}}m^3h(V-V_{\mathrm{Na}}) - G_{\mathrm{K}}n^4(V-V_{\mathrm{K}}) - G_{\mathrm{L}}(V-V_{\mathrm{L}}) \quad (1)$$

$$\frac{dz}{dt} = \alpha_z(V)(1-z) - \beta_z(V)z + \xi_z(t), \quad (z = m, h, n) \quad (2)$$

where $C = 1\ \mu\mathrm{F/cm^2}$; $V_{\mathrm{Na}} = 50$ mV, $V_{\mathrm{K}} = -77$ mV, and $V_{\mathrm{L}} = -54.4$ mV; $G_{\mathrm{K}} = g_{\mathrm{K}}^{\max}x_{\mathrm{K}}$, $G_{\mathrm{Na}} = g_{\mathrm{Na}}^{\max}x_{\mathrm{Na}}$, $G_{\mathrm{L}} = 0.3\ \mathrm{mS/cm^2}$; $g_{\mathrm{K}}^{\max} = 36\ \mathrm{mS/cm^2}$, $g_{\mathrm{Na}}^{\max} = 120\ \mathrm{mS/cm^2}$; x_{K} and x_{Na} are the fractions of working ion channels to the overall number N_{K} of potassium and N_{Na} of sodium ion channels, respectively. Smaller x_{K} and x_{Na} denotes higher level of CB for potassium and sodium ion channels. The voltage-dependent opening and closing rates $\alpha_z(V)$ and $\beta_z(V)$ are: $\alpha_m(V) = (0.1(V+40))/(1-\exp[-(V+40)/10])$, $\beta_m(V) = 4\exp((-(V+65))/18)$, $\alpha_h(V) = 0.07\exp((-(V+65))/20)$, $\beta_h(V) = 1/(1+\exp(((-(V+35))/10)))$, $\alpha_n(V) = 0.01(V+55)/(1-\exp(((-(V+55))/10)))$, $\beta_n(V) = 0.125\exp(((-(V+65))/80))$. $\xi_z(t)$ are Gaussian white noises, of which the noise strengths are: $\langle\xi_m(t)\xi_m(t')\rangle = (2/(N_{\mathrm{Na}}x_{\mathrm{Na}}))(\alpha_m(V)\beta_m(V)/(\alpha_m(V)+\beta_m(V)))\delta(t-t') = (2/(N_{\mathrm{Na}}x_{\mathrm{Na}}))((\alpha_m(V)\beta_m(V))/(\alpha_m(V)+\beta_m(V)))\delta(t-t')$, $\langle\xi_h(t)\xi_h(t')\rangle = (2/(N_{\mathrm{Na}}x_{\mathrm{Na}}))((\alpha_h(V)\beta_h(V))/(\alpha_h(V)+\beta_h(V)))\delta(t-t')$, $\langle\xi_n(t)\xi_n(t')\rangle = (2/(N_{\mathrm{K}}x_{\mathrm{K}}))((\alpha_n(V)\beta_n(V))/(\alpha_n(V)+\beta_n(V)))\delta(t-t')$; $N_{\mathrm{Na}} = \rho_{\mathrm{Na}}S$, $N_{\mathrm{K}} = \rho_{\mathrm{K}}S$, $\rho_{\mathrm{Na}} = 60\ \mu\mathrm{m^{-2}}$, $\rho_{\mathrm{K}} = 18\ \mu\mathrm{m^{-2}}$. S (in unit of $\mu\mathrm{m^2}$) is the membrane patch size. For more interpretations of these parameters, please see, e.g., the work by Schmid et al. (2004a,b).

The neuronal network under study is Newman–Watts network, which is one type of small-world networks (Newman, 2000). The network is constructed as follows. It starts with a regular ring comprising $N = 60$ identical HH neurons with each neuron having two nearest neighbors (i.e., connection degree $k = 2$), and links are then randomly added between non-adjacent vertices. In the limit case that all neurons are coupled to each other, the network contains $N(N-1)/2$ edges. Hence, the fraction of shortcuts (randomness) is given by $p = M/[N(N-1)/2]$, with M denoting the number of added shortcuts (Gong et al., 2006). Therefore, the dynamics of the delayed Newman–Watts networks of stochastic HH neurons with channel blocking can be described by

$$C\frac{dV_i}{dt} = -g_{\mathrm{Na}}^{\max}x_{\mathrm{Na}}m_i^3h_i(V_i-V_{\mathrm{Na}}) - g_{\mathrm{K}}^{\max}x_{\mathrm{K}}n_i^4(V_i-V_{\mathrm{K}}) - g_{\mathrm{L}}^{\max}(V_i-V_{\mathrm{L}}) + \sum_j \varepsilon_{ij}(V_j(t-\tau)-V_i) \quad (3a)$$

$$\dot{z}_i = \alpha_{z_i}(V_i)(1-z_i) - \beta_{z_i}(V_i)z_i + \xi_{z_i}(t), \quad (z = m, h, n) \quad (3b)$$

where $1 \le i \le N$, with N being the number of neurons; τ is the delay time (in unit of ms); ε_{ij} is the coupling strength between two neurons i and j, which is determined by the coupling pattern of the system. Let $\varepsilon_{ij} = 0.1$ if the neuron i and j are connected; $\varepsilon_{ij} = 0$ otherwise. Here, we assume that all the neurons have the same number of non-blocked sodium (same x_{Na}) or potassium ion channels (same x_{K}) in all cases.

The spiking regularity of any neuron i of the network is quantitatively characterized by the inverse of coefficient of variation (CV), which is defined as $\lambda_i = \langle T\rangle / \left(\sqrt{\langle T^2\rangle - \langle T\rangle^2}\right)$, and the network dynamics is characterized by the average of λ_i over all neurons:

$$\lambda_S = \frac{1}{N}\sum_{i=1}^{N}\lambda_i \quad (4)$$

where N is the number of neurons, $\langle T\rangle = (1/N')\sum_{i=1}^{N'}(t_{i+1}-t_i)$ and $\langle T^2\rangle = (1/N')\sum_{i=1}^{N'}(t_{i+1}-t_i)^2$ are the mean and mean-squared interspike intervals, respectively, and t_i is the time of the ith spike and N' is the number of spikes. Here we define that a spike occurs when V_i crosses the detection threshold $V_{det} = 0$ from below. The parameter λ_i characterizes the spiking coherence for a single neuron, and λ_S, the average of λ_i over all neurons, characterizes the averaged spiking coherence for a neuron. Larger λ_S value means more regular spiking behavior.

Numerical integration of Eq. (3) is carried out by using explicit Euler method with a time step of $\Delta t = 0.001$ ms. Periodic boundary conditions (i.e., the first neuron is coupled with the sixtieth one) are employed and the parameter values for all neurons are identical except for distinct initial values of the potential V_i and noise terms $\xi_i(t)$ for all neurons. Distinct initial values of potential V_i and noise $\xi_i(t)$ are obtained with a constant multiplied by 60 random numbers and 60 random numbers in Gaussian distribution, respectively.