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Influences of autapse and channel blockage on multiple coherence resonance in a single neuron

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ABSTRACT

We study how the spiking regularity of a single stochastic Hodgkin–Huxley neuron is effected in the presence of ion channel blocking and autaptic connection. In this study, we consider a chemical autapse expressed by its coupling strength and delay time. It is found that the neuron exhibits multiple coherence resonance (MCR) behavior induced by autaptic time delay at an appropriate level of ion channel blocking and autaptic coupling strength. This MCR behavior increases with the decrement of working potassium ion channels, whereas it decreases or completely disappears with the increment of a fraction of sodium ion channels blocking, regardless of autaptic coupling strength. Furthermore, this behavior is more explicit at intermediate autaptic coupling strength regardless of the ion channel blocking type. We briefly discuss the obtained results with the underlying reasons in terms of ion channel blocking type and autapse parameters. We also showed that ion channel noise, thus membrane patch size, should be at an optimal level to obtain MCR behavior otherwise, this behavior would be destroyed. The obtained results also showed that autaptic time delay is more operative on regularity than its coupling strength regardless of ion channel blocking. Considering the importance of spiking regularity on neuronal information processing, our results may help to understand the intersection of ion channel blocking and autaptic connections of a single neuron.

1. Introduction

Information processing in neuronal systems takes place with collaboration and interaction between diverse ion channels embedded in membrane. Experimentally, it has been shown that some neurotoxins such as tetraetylammonium (TEA), tetratoxin (TTX) and saxitoxin (STX) can be used for changing the properties of specific ion channels. By a fine-tuned addition of these toxins, a certain portion of relevant ion channels could be disabled or blocked and thus, the number of working relevant ion channels can be reduced [1]. Consequently, the noise level of relevant ion channels is increased, while its conductance decreases. Since the channel noise plays important roles in the spiking dynamics of neurons (e.g. spiking threshold, spiking rate etc.), the blockage of an ion channel would employ significant impacts on spiking dynamics [2].

In literature, there are numerous studies that investigate the effects of channel blocking on a single neuron or different neuronal networks [3–10]. For instance, Schmid et al. [3,4] showed that spiking regularity of a single neuron can be decreased or enhanced by reducing the working sodium or potassium ion channels. It is also reported that channel blocking has healing or destructive effects on the collective spiking regularity of an array of coupled neurons [5], small-world neuronal networks [6] and scale-free neural networks [7], depending on conditions. Novel studies have also revealed that

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the first-spike timing of a single neuron or neuronal networks can be changed via blocking some portion of ion channels [8–10].

On the other hand, information transmission within the nervous systems is achieved by synapses. Electrical and chemical synapses are well-known types of synapse. Several decades ago, it was found that some neurons have self-connections that constitute a feedback mechanism on a cellular level [11–15]. Van der Loss and Glaser [16] attributed this type of connection as autapse, which is found widely in different brain areas especially in cortical pyramidal neurons [13,17–20].

Experimental and computational studies show that the presence of autaptic connections impress spiking dynamics significantly [20–33]. In this context, Bacci and Huguenard [21] experimentally pointed out that the precision of spike times in neocortical fast spiking interneurons improves because of autaptic connections. Masoller et al. [22] demonstrated that there is a complex and non-trivial interplay between weak delayed feedback, noise and the subthreshold intrinsic dynamics of a thermoreceptor neuron. Hashemi et al. [24] reported that the dynamics of a single neuron closely related to time constants of delayed synaptic feedback which denote the duration of the synaptic activity. Wang et al. [25] investigated how the mode-locking pattern of a neuron changes with autapse in the presence of sinusoidal current input. They found that autaptic connections substantially readjust the neuron's firing patterns. In another study, it was revealed that the dynamics of the Hindmarsh–Rose (HR) neuron model can be altered between different firing patterns (quiescent, periodic and chaotic) by imposing a feedback term with a specific time-delay and autaptic intensity [26]. Yilmaz and Ozer [27] showed that the weak signal detection performance of a neuron is modified via autapse depending on its parameters. It is also demonstrated that the weak localized propagation of pacemaker activity can be improved by means of an autapse in a scale-free (SF) and small-world (SW) neuronal networks when the additional time-scales by the autapse, intrinsic dynamics and weak signal are locked-in each other [28,29]. Wang and Chen [20] revealed that autapses provide an opportunity to engineer the response of a HH model to subthreshold stimulus whose detection enhances via autaptic connections. Yilmaz et al. [31] showed that the firing regularity of a single neuron or SF neuronal networks exhibits coherence resonance (CR) or multiple coherence resonance (MCR) by the use of proper values of autaptic conductance whether the autapse is electrical or chemical. In a recent study, we have analyzed the impact of ion channel block on the spiking regularity of a neuronal network as a function of autaptic conductance [32]. In that study, the spiking dynamics are addressed at the network level, however, the analysis of dynamics at a single neuron would make contribution to that study. The effects of autapse and ion channel block on the spiking dynamics of a small-world neuronal network are analyzed by Uzun et al. and have been showed that these parameters have a significant role on the determining the neuronal dynamics [33].

In this paper, we build on these previous studies to determine the role of interaction both autapse and ion channel blocking, in particular the spiking regularity of a single neuron. To do this, we use a stochastic HH neuron model which relates the membrane area (that is the strength of channel noise) to the spiking characteristics in a manner that more closely mimics actual conditions. We focus on the relevance of a fraction of blocked sodium or potassium ion channels and autapse parameters. We thus deliver a comprehensive study that reveals under which condition the spiking regularity of the neuron might be optimal. The main results are presented in subsequent sections, but first we will describe the mathematical model in detail.

2. Mathematical model

The stochastic HH conductance-based neuron model is a type of scientific model to investigate the basic spiking dynamics of a neuron. Within this study, we will use the stochastic HH neuron model in which dynamical equations in the presence of both an autaptic connection and ion channel blocking are given as follows [31]:

$$C_m \frac{dV}{dt} + g_{Na}^{max} x_{Na} m^3 h\,(V - E_{Na}) + g_K^{max} x_K n^4 (V - E_K) + g_l (V - E_L) + I_{aut} = 0 \tag{1a}$$

$$\frac{dx}{dt} = \alpha_x(V)(1 - x) - \beta_x(V)x + \xi_x(t)\; x = (m, h, n) \tag{1b}$$

Here V is the membrane potential, $C = 1\mu\mathrm{Fcm}^{-2}$ is the membrane capacity and $E_{Na} = 50$ mV, $E_K = -77$ mV and $E_L = -54.4$ mV are the reversal potentials for the sodium, potassium and leakage channels, respectively. $g_{Na}^{max} = 120\mathrm{mScm}^{-2}$ and $g_K^{max} = 36\mathrm{mScm}^{-2}$ stand for the maximum sodium and potassium conductances, respectively. x_{Na} and x_K are two scaling factors which correspond to working (non-blocked) ion channels to the total number of sodium (N_{Na}) or potassium (N_K) ion channels, respectively. These scaling factors are confined to the unit interval [3,4,8,9]. m, h and n denotes activation and inactivation gating variables for the sodium ion channel and activation gating variable for potassium ion channel, respectively. The dynamics of these gating variables are controlled by voltage-dependent rate functions of α and β which is determined experimentally and can be found in Hodgkin and Huxley [34]. The probabilistic nature of the channels occurs as an independent zero mean Gaussian noise source, $\xi_x(t)$, whose autocorrelations are given below as [35]:

$$\left\langle \xi_m(t)\xi_m\left(t'\right)\right\rangle = \frac{2\alpha_m\beta_m}{N_{Na}x_{Na}(\alpha_m + \beta_m)}\delta\left(t - t'\right), \tag{2a}$$

$$\left\langle \xi_h(t)\;\xi_h\left(t'\right)\right\rangle = \frac{2\alpha_h\beta_h}{N_{Na}x_{Na}(\alpha_h + \beta_h)}\delta\left(t - t'\right), \tag{2b}$$