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Influence of autapse on mode-locking structure of a Hodgkin–Huxley neuron under sinusoidal stimulus



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HIGHLIGHTS

• Effect of autapse on mode-locking firing is studied by Hodgkin-Huxley neuron.

· Autaptic self-feedback can enhance or suppress the mode-locking firing.

• The mode-locking firing is sensitive to delay time.

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ABSTRACT

We investigated the mode-locking behaviors of a Hodgkin–Huxley neuron with an autapse under sinusoidal stimulus. A neuron without an autapse can exhibit rich *p*:*q* mode-locking (i.e. *p* output action potentials generated by *q* cycles stimulations) behaviors with periodic stimuli. In the presence of the autaptic connection, the *p*:*q* mode-locking behaviors are completely reset. The autapse extends the scope of mode-locking. The autapse can enhance or suppress the status of mode-locking. Even for some specified autaptic parameters, the neuron could be driven into the sub-threshold oscillation. Our results suggested that the autapse can serve as a potential control option for adjusting the mode-locking firing behaviors. We also found that changing the delay time is much more effectively operable to regulate the response behavior than the autaptic intensity.

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1. Introduction

Neurons transform the external stimulus into trains of actionpotentials or spikes, which are known as a crucial process in neural coding. In such process, there are always two competing frequencies within the neuronal dynamical system: the natural frequency of the neuron and the external forcing frequency. With the interplay between these two frequencies, neurons exhibit complex dynamical responses including mode-locking, quasi-periodicity, and chaos (Lee and Kim, 2006; Borkowski, 2009; Jensen and Krishna, 2012).

The mode-locking firing patterns have been studied in the context of neural information coding and is shown to be very important in neural signal processing (Pedemonte et al., 1996; Varela et al., 2001; David et al., 2009; Atencio and Schreiner, 2010).

It was observed the complected mode-locking firing patterns in the experiments of squid giant axon injected with sinusoidal inputs (Sato and Doi, 1992). In the case of a Hodgkin–Huxley (HH) neuron with periodic stimuli, the dynamical states have been investigated numerically and it is also revealed that a complex mode-locking firing dynamics involving various bifurcations, saddle-node, period-doubling, and inverse-flip bifurcations (Lee and Kim, 2006). Che et al. (2009) reported that a neuron response completed mode-locking behaviors in the periodic electrical field and phase lockings as well as chaotic dynamics depend on the parameter values of the amplitude and frequency of the electrical field. With different intrinsic dynamical properties, neurons also display different mode-locking firing patterns with various periodic inputs due to the different intrinsic dynamics (Wang et al., 2011, 2013).

On the other hand, the delayed feedback has a nontrivial effect on the dynamics of the nonlinear system as well as most of nature systems (Shi and Wang, 2012). Feedback is also extensively used to control coherence resonance and stabilize periodic orbits (Postlethwaite and Silber, 2007; Gaudreault and Drolet, 2012).

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Many biological systems operate under the influence of timedelayed feedback mechanisms (Reppert and Weaver, 2001; Kitano, 2002; Zhang et al., 2012).

In the nervous system, the so-called auto-synapse (autapse), a self-feedback connection, is common and plays an important role in brain function (Lübke et al., 1996; Ikeda and Bekkers, 2006). Masoller et al. (2008) investigated the interaction of time-delayed feedback and noise on the activities of neuron. They shown that the delayed feedback affects the patterns of neural activity in a determinant way. A stochastic HH neuron with delayed feedback can reduce the spontaneous spiking activity at characteristic frequencies with a special frequency-locking mechanism (Li et al., 2010). Those works reveal that the autaptic connection can significantly change the dynamical responses of a neuron. Bacci and Huguenard (2006) observed that the autaptic transmission enhances the precision spike time of neuron in vitro. It was also found that the dynamic behaviors of a Hindmarsh-Rose neuron with an autapse can switch among quiescent, periodic and chaotic firing patterns, as the autaptic parameters change (Wang et al., 2014). However, what the functional significance of autapse in the nerve systems is still not fully understood.

In this work, we address on the effect of the autapse on the mode-locking behaviors in a single neuron by numerical simulation on a classical HH neuron with a time-delayed self-feedback connection. The range of mode-locking is expanded by the autapse. The p:q mode-locking could be larger or smaller than that without an autapse depending on the autaptic parameters. These results suggest that the autapse serves as a potential control option in the neural information process.

This paper is organized as follows. In Section 2, we present the detail of the HH neuron with a delayed autaptic self-connection. In Section 3, we perform the simulation of mode-locking firing. Finally, the conclusion and the discussion are summarized in Section 4.

2. Model

The HH conductance-based neuron model is a type of classic scientific model to describe how the action potentials in neurons are initiated and propagated. This neuron model also produces the basic firing dynamics of a biological neuron. The HH neuron model is given as follows (Hodgkin and Huxley, 1952):

$$C_m dV/dt = -g_{Na} m^3 h(V - E_{Na}) - g_K n^4 (V - E_K), -g_L (V - E_L) - I_{aut} + I_{ext},$$
(1)

$$dm/dt = \alpha_m (1-m) - \beta_m m, \tag{2}$$

$$dh/dt = \alpha_h (1-h) - \beta_h h, \tag{3}$$

$$dn/dt = \alpha_n (1-n) - \beta_n n, \tag{4}$$

here *V* is the membrane potential. *m*, *h*, and *n* represent the activation and inactivation of the sodium current and the activation of the potassium current, respectively. $C_m = 1 \,\mu\text{F/cm}^2$ is the membrane capacitance. The constants $g_{Na} = 120 \,\text{mS/cm}^2$, $g_K = 36 \,\text{mS/cm}^2$, and $g_L = 0.3 \,\text{mS/cm}^2$ are the maximal conductances of the sodium, potassium, and leakage channels, respectively. $E_{Na} = 50 \,\text{mV}$, $E_K = -77 \,\text{mV}$ and $E_L = -54.5 \,\text{mV}$ stand for the corresponding reversal potentials. The voltage-dependent ion transition rates are

$$\begin{aligned} \alpha_m &= (0.1 - 0.01V) / [\exp(1 - 0.1V) - 1], \\ \beta_m &= 0.125 \exp(-V/80), \\ \alpha_h &= (2.5 - 0.1V) / [\exp(2.5 - 0.1V) - 1], \\ \beta_h &= 4 \exp(-V/18), \\ \alpha_n &= 0.07 \exp(-V/20). \end{aligned}$$

$$\alpha_n = 0.07 \exp(-V/20)$$

$$\beta_n = 1/[\exp(3 - 0.1V) + 1].$$

The external injected current I_{ext} is set as a sinusoidal form:

$$I_{ext} = A\sin\left(2\pi f_{in}t\right),\tag{5}$$

where A and f_{in} are the amplitude and the frequency of the external sinusoidal input, respectively.

The delayed self-feedback current I_{aut} is modeled as the following form (Li et al., 2010):

$$I_{aut} = g_{aut}(V(t) - V(t - t_{delay})).$$
(6)



Fig. 1. Arnold tongues for a HH neuron under sinusoidal stimuli without self-feedback. The tongues correspond to the stable phase-locking (p:q) regions are labeled by 3:1, 2:1, 1:1, 1:2, and 1:3.



Fig. 2. The evolution of the membrane potential (red-dashed/blue-solid line) of a neuron without/with self-feedback. The green solid lines denote the sinusoidal stimuli currents. The input and autaptic parameters are the following: (a) $f_{in} = 9 \text{ Hz}$, $g_{aut} = 0.1 \text{ mS/cm}^2$, $t_{delay} = 8.6 \text{ ms}$; (b) $f_{in} = 9 \text{ Hz}$, $g_{aut} = 0.3 \text{ mS/cm}^2$, $t_{delay} = 6.2 \text{ ms}$; (c) $f_{in} = 9 \text{ Hz}$, $g_{aut} = 0.1 \text{ mS/cm}^2$, $t_{delay} = 10.1 \text{ mS/cm}^2$, $t_{delay} = 37 \text{ ms}$; (d) $f_{in} = 16 \text{ Hz}$, $g_{aut} = 0.1 \text{ mS/cm}^2$, $t_{delay} = 12.2 \text{ ms}$; (e) $f_{in} = 38 \text{ Hz}$, $g_{aut} = 0.3 \text{ mS/cm}^2$, $t_{delay} = 9.4 \text{ ms}$; (f) $f_{in} = 168 \text{ Hz}$, $g_{aut} = 0.5 \text{ mS/cm}^2$, $t_{delay} = 2.2 \text{ ms}$. (For interpretation of the references to color in this figure caption, the reader is referred to the web version of this paper.)

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