Multiple firing coherence resonances in excitatory and inhibitory coupled neurons

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A B S T R A C T

The impact of inhibitory and excitatory synapses in delay-coupled Hodgkin–Huxley neurons that are driven by noise is studied. If both synaptic types are used for coupling, appropriately tuned delays in the inhibition feedback induce multiple firing coherence resonances at sufficiently strong coupling strengths, thus giving rise to tongues of coherency in the corresponding delay-strength parameter plane. If only inhibitory synapses are used, however, appropriately tuned delays also give rise to multiresonant responses, yet the successive delays warranting an optimal coherence of excitations obey different relations with regards to the inherent time scales of neuronal dynamics. This leads to denser coherence resonance patterns in the delay-strength parameter plane. The robustness of these findings to the introduction of delay in the excitatory feedback, to noise, and to the number of coupled neurons is examined. Mechanisms underlying our observations are revealed, and it is suggested that the regularity of spiking across neuronal networks can be optimized in an unexpectedly rich variety of ways, depending on the type of coupling and the duration of delays.

1. Introduction

Neurophysiological studies have revealed the existence of accurately timed patterns of spikes by a variety of cognitive and motoric tasks [1–6]. The timing of these spikes, or neuronal firings, is accurate to within the millisecond range, which poses great challenges with regards to the identification of mechanisms that would be able to ensure such precision. Following their initial observation in the cortex of monkeys [1,2], the precisely timed spikes have been reported and investigated for motor functions [3], the neuronal response of visual systems [4], and the complex spatial fingertip events [5], to name but a few examples. Not surprisingly, synchronized, precisely timed firings can be observed at virtually all neuronal processing levels, including the retina [7], the lateral geniculate nucleus [8], and the cortex [9,10].

Since it is well known that noise can play a constructive role in different types of nonlinear dynamical systems, which arguably describe also neuronal dynamics [11], this opens the possibility of exploiting such mechanisms for explaining, or at least supporting, the aforementioned precision of neuronal firings. Stochastic resonance [12–14] and coherence resonance [15–17] are amongst the most prominent examples by means of which noise of appropriate intensity is able either to enhance the detection of weak deterministic signals [18] or evoke coherent response in nonlinear dynamical systems in the absence of any deterministic inputs. The potential benefits of noise range from ice ages to crayfish and SQUIDs [19], to neural systems, as most recently reviewed in [20].

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Following initial advances on individual dynamical systems, the focus begun shifting to spatially extended systems [21], especially also to such with complex networks describing connections between the individual units [22,23]. For example, coherence resonance on a small world network was investigated in [24], while array-enhanced resonances were reported in [25]. Moreover, spatial coherence resonance was observed first near pattern-forming instabilities [26], and latter also in excitable media [27]. Excitable systems in general proved to be very susceptible to a multitude of noise-induced phenomena, as reviewed comprehensively in [28]. Adding spatial degrees of freedom, along with the possibilities for introducing other sources of heterogeneity, lead to the discovery of very interesting and quite exotic phenomena, such as the ghost resonance [29], and double as well as multiple stochastic [30–33] and coherence [34–37] resonances.

For neural systems, a wealth of interesting and new phenomena were made observable by integrating realistic features of neuronal dynamics into the studied models. Information transmission delays or synaptic delays, for example, are inherent to the nervous system because of the finite speed at which action potentials propagate across neuron axons, and due to time lapeses occurring at both dendritic and synaptic processing [38]. Following seminal works examining the impact of delays on excitatory and other dynamical systems [39–41], the stability and attainability of synchronous oscillations [42–44] and the role of delays in shaping spatiotemporal dynamics of neuronal activity [45] were investigated. Moreover, the role of delays in coupled Hodgkin–Huxley neurons was also investigated for the phenomenon of coherence resonance, and it was reported that properly tuned delays can lead to the occurrence of multiple resonances [46,47].

In this letter, we extend the scope of coherence resonance in models of neuronal dynamics by considering besides synaptic delays also different types of synaptic coupling. While the role of chemical synapses in coupled neurons with noise has been investigated in [48], and although the general dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons is known [49], our approach, joining these distinctive features of neuronal dynamics (synaptic delays, different types of synaptic coupling, and noise), allows for the identification of new ways by means of which the coherence, and particularly also the synchronization patterns in the corresponding delay-strength parameter plane when either inhibitory and excitatory or only inhibitory synapses are used for coupling. The details of these multiple firing coherence resonances, and in particular the conditions at which they occur, however, depend significantly on the type of coupling. Reported results suggest that characteristic time scales related to the information transmission and inhibition in neuronal networks may interplay in intricate ways, and by doing so give rise to new mechanisms for optimizing spiking regularity.

The remainder of this letter is organized as follows. In the next section we describe the model, then we present the main results separately for the two coupling scenarios, while lastly we summarize our findings and discuss their potential implications.

2. Model definition

For simplicity, we consider two Hodgkin–Huxley neurons [11] that are coupled by inhibitory and/or excitatory synapses. Equations describing the dynamics are:

\[
\frac{dV_i}{dt} = -g_{Na}m^3h(V_i - V_{Na}) - g_L(V_i - V_L) - g_Kn^4(V_i - V_K) + I + \sigma_i(t) + \sum_j I_{syn}^{ij},
\]

(1)

\[
\frac{dm_i}{dt} = \alpha_m(1 - m_i) - \beta_m m_i,
\]

(2)

\[
\frac{dh_i}{dt} = \alpha_h(1 - h_i) - \beta_h h_i,
\]

(3)

\[
\frac{dn_i}{dt} = \alpha_n(1 - n_i) - \beta_n n_i,
\]

(4)

where \(V_i\) is the transmembrane potential of the \(i\)th neuron, \(t\) is the time with its unit being millisecond. Moreover, \(m_i, h_i\) and \(n_i\) are the gating variables, where the voltage-dependent opening and closing rates are:

\[
\alpha_m = \frac{0.1(V_i + 10)}{1 - \exp\left(\frac{(V_i + 10)}{20}\right)},
\]

(5)

\[
\beta_m = 4 \exp\left[\frac{(V_i + 65)}{18}\right],
\]

(6)

\[
\alpha_h = 0.07 \exp\left[\frac{(V_i + 65)}{20}\right],
\]

(7)

\[
\beta_h = \left\{1 + \exp\left[\frac{(V_i + 35)}{10}\right]\right\}^{-1},
\]

(8)

\[
\alpha_n = \frac{0.01(V_i + 55)}{1 - \exp\left[\frac{(V_i + 55)}{10}\right]},
\]

(9)

\[
\beta_n = 0.125 \exp\left[-\frac{(V_i + 65)}{80}\right].
\]

(10)
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