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Enhancement of pacemaker induced stochastic resonance by an autapse in a scale-free neuronal network

YILMAZ Ergin[1*], BAYSAL Veli[1], PERC Matjaž[2,3] & OZER Mahmut[4]

[1] *Department of Biomedical Engineering, Engineering Faculty, Bülent Ecevit University, Zonguldak 67100, Turkey;*
[2] *Department of Physics, Faculty of Sciences, King Abdulaziz University, Jeddah 21431, Saudi Arabia;*
[3] *Faculty of Natural Sciences and Mathematics, University of Maribor, Koroška cesta 160, Maribor SI-2000, Slovenia;*
[4] *Department of Electrical and Electronics Engineering, Engineering Faculty, Bülent Ecevit University, Zonguldak 67100, Turkey*

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An autapse is an unusual synapse that occurs between the axon and the soma of the same neuron. Mathematically, it can be described as a self-delayed feedback loop that is defined by a specific time-delay and the so-called autaptic coupling strength. Recently, the role and function of autapses within the nervous system has been studied extensively. Here, we extend the scope of theoretical research by investigating the effects of an autapse on the transmission of a weak localized pacemaker activity in a scale-free neuronal network. Our results reveal that by mediating the spiking activity of the pacemaker neuron, an autapse increases the propagation of its rhythm across the whole network, if only the autaptic time delay and the autaptic coupling strength are properly adjusted. We show that the autapse-induced enhancement of the transmission of pacemaker activity occurs only when the autaptic time delay is close to an integer multiple of the intrinsic oscillation time of the neurons that form the network. In particular, we demonstrate the emergence of multiple resonances involving the weak signal, the intrinsic oscillations, and the time scale that is dictated by the autapse. Interestingly, we also show that the enhancement of the pacemaker rhythm across the network is the strongest if the degree of the pacemaker neuron is lowest. This is because the dissipation of the localized rhythm is contained to the few directly linked neurons, and only afterwards, through the secondary neurons, it propagates further. If the pacemaker neuron has a high degree, then its rhythm is simply too weak to excite all the neighboring neurons, and propagation therefore fails.

autapse, self-delayed feedback, neuron, channel noise, scale-free network

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1 Introduction

Neuronal dynamics exhibit important phenomena such as stochastic resonance (SR) [1–5], coherence resonance (CR) [6–9], and synchronization [10–13], providing a framework to understand how the neuronal system works. Much works have been done to investigate the underlying mechanism of the information encoding capabilities of the neuronal networks. In this context, the collective dynamics of neuronal ensemble are studied by observing the spatiotemporal pattern formation such us spiral waves and other wave forms [14–17]. On the appearance of these phenomena, noise is an inevitable part of the system, and contrary to intuition it has a therapeutic influence on the neuronal dynamics. In neuronal systems, channel noise, originated from random opening and closing of ion channel embedded in the neuronal membrane, is one of the noise sources within nervous systems [18–20], and its effects on the neuronal activity

*Corresponding author (email: erginyilmaz@yahoo.com)

have been extensively studied in a single neuron level and neuronal networks with different topologies [21–28].

Synaptic connections, which are responsible for neuronal communications, grow up between two neurons. There are two well-known categories of synapses within the nervous system, electrical and chemical synapses. Furthermore, there is also a type of synapse called *autapse,* a term reserved by Van der Loos and Glaser [29] for a kind of synaptic connection occurring between dendrite and axon of the same neuron. Autapses are morphologically similar normal synapses between neurons, and common in the nervous system [30–36]. They are modeled as a self-delayed feedback mechanisms due to the axonal time delay consuming in back travel of action potential from axon to dendrite, and common in many biological system [37,38]. The efficiency of the delayed-feedback mechanism on the controlling of chaos or turbulence in a chaotic attractor has been known [39]. By causing the transition between different firing patterns of Hopf oscillators, the delayed-feedback mechanism can control the coherence of these oscillators [40]. Besides, there are also some studies in which the effects of autapse on the dynamics of a single neuron and neuronal networks are investigated. It is experimentally showed that inhibitory autaptic feedback has an important role on the improvement of spike-timing precision of the inhibitory interneurons in neocortex [41]. At the same study, it is reported that adding of artificial inhibitory autapse by means of dynamical clamp to pyramidal neurons enhances spiking precision of them, distinctly [41]. Li et al. [42] investigated the effect of delayed-feedback on the firing activity of stochastic HH neuron and demonstrated that autapse (delayed-feedback) causes a burst type firing and a multimodal inter-spike interval by reducing the spontaneous firing activity at characteristic frequencies. By adding the dynamic short term synaptic plasticity properties, which not included in gamma oscillation models, to the Wang-Buzsáki model of gamma oscillations, Connelly [43] showed that autapse increases the synchrony of the membrane potentials across network during neocortical gamma oscillations. Wang et al. [44] theoretically showed that autapse can switch the Hindmarse-Rose (HR) neuron among different firing behaviors such as chaotic firing, periodic firing and quiescent for the proper values of the autaptic delay time and coupling strength. In another study, Wang et al. [45] demonstrated that autapse can serve as a potential control option in adjusting the mode-locking firing behavior of HH neurons under the effect of sinusoidal stimulus. Liu et al [46] reported that autapses do not show some synaptic plasticity properties such as short- term potentiation as compared to the neurons without autapse. Qin et al. [47] investigated autapse induced regular pattern formations in regular networks of HR neurons. They showed that autapse plays a critical role in exciting and regulating the collective activities of HR neurons by inducing target waves and spiral waves in the network [47]. Ma et al. [48] studied the effect of autapse in a chain type forward feedback neuronal network where the HR neuron model is used in modeling of local dynamics and assumed that only a single neuron acting as a pacemaker has an autapse. They showed that autapse has an important effect on the regulation of the collective activities of neurons [48]. Autapse induced synchronization was studied in a ring type network consisting of three identical neurons and reported that for finely tuned values of the autaptic coupling strength and the autaptic delay time all neurons in the network become synchronize and oscillate with the same rhythm [49]. In a recent study, we have demonstrated the effects of autapse on the weak signal detection capacity of a single HH neuron [50].

On the other hand, pacemakers are special cells dictating their own rhythms to whole networks. The most well-known organ including pacemaker cells is the human heart [51]. Thus, it deserves special attention and deeper investigations. In literature there are many studies reporting the effects of pacemaker activity on the complex neuronal networks [52–54]. The literature survey leaves us with the impression that there is no any study to investigate the propagation of the pacemaker rhythmicity throughout the neuronal network if the pacemaker neuron has an autapse. Therefore, in this study, we investigate this problem by using a biologically realistic model of HH neuron by which a scale-free neuronal network is constructed. We first study optimal conditions leading to the optimal propagation of the pacemaker rhythmicity in the absence of the autapse, and then we investigate how an autapse affects its propagation by using these optimal conditions.

2 Model and method

One of the utilized model to generate the spiking patterns of a real biological neuron is the conductance-based Hodgkin-Huxley neuron model [55]. The membrane potential dynamics of a HH neuron in a scale-free network can be given as

$$C_m \frac{\mathrm{d}V_i}{\mathrm{d}t} = -g_{Na} m_i^3 h_i (V_i - E_{Na}) - g_K n_i^4 (V_i - E_K) - g_L (V_i - E_L) + \sum_i^N \varepsilon_{ij} (V_j(t) - V_i(t)), \quad (1)$$

where $C_m = 1\,\mu\mathrm{F/cm}^2$ is the membrane capacity, $V_i(t)$ is the membrane potential of i at the time t, and $1 \leqslant i \leqslant N$. N is the total number of neurons in the network. $E_{Na} = 50$ mV, $E_K = -77\,\mathrm{mV}$ and $E_L = 54.4\,\mathrm{mV}$ are the reversal potential for sodium, potassium, and leakage currents, respectively. $g_L = 0.3\,\mathrm{mS/cm}^2$ is the leakage conductance. $g_{Na} = 36\,\mathrm{mS/cm}^2$ and $g_K = 120\,\mathrm{mS/cm}^2$ are the maximal conductance for sodium and potassium ion channels, respectively. ε_{ij} is the coupling strength between neuron i

and j. If neuron i and j are connected $\varepsilon_{ij}=\varepsilon$, otherwise $\varepsilon_{ij}=0$. m_i and h_i represent the activation and inactivation variables for sodium channels of neuron i, respectively. The activation variables for potassium channels of neuron of i is expressed with n_i. $I_{stim}=\sin(0.3t)$ is the subthreshold stimulus generating the localized rhythmic activity, which is introduced to the pacemaker neuron, which is chosen as a neuron having either the highest or the lowest number of neuronal connections. Since, we consider that only the pacemaker neuron has an autaptic connection or an autapse, the autaptic current given below is added only to the pacemaker neuron's membrane potential.

$$I_{aut}=\kappa[V_{pn}(t-\tau)-V_{pn}(t)], \tag{2}$$

where V_{pn} is the membrane potential of the pacemaker neuron, κ denotes the autaptic coupling strength and τ represents the autaptic time delay, which represents the elapsed time during axonal back transmission of membrane potential via the autapse. In HH neuron model, the dynamics of the gating variables change depending on membrane potential [54]. However, if the number of ion channel is finite, the stochastic effect caused by random open-close fluctuations of the ion channels may have significant effect on the neuronal dynamics. To take into account this effect, we use Fox's algorithm due to its computational efficiency. In Fox's algorithm, the gating dynamics is described by the stochastic Langevin generalization as follows [56].

$$\frac{\mathrm{d}x_i}{\mathrm{d}t}=\alpha_{x_i}(V_i)(1-x_i)-\beta_{x_i}(V_i)x_i+\zeta_{x_i}, \tag{3}$$

where $\alpha_{x_i}(V)$ and $\beta_{x_i}(V)$ [55] are voltage-dependent rate function for the gating parameter x_i and given as follows:

$$\alpha_m(V)=\frac{0.1(V+40)}{1-\exp[-(V+40)/10]}, \tag{4a}$$

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

$$\alpha_h(V)=0.07\exp[-(V+65)/20], \tag{4c}$$

$$\beta_h(V)=[(1+\exp[-(V+35)/10])]^{-1}, \tag{4d}$$

$$\alpha_n(V)=\frac{0.01(V+55)}{1-\exp[-(V+55)/10]}, \tag{4e}$$

$$\beta_n=0.125\exp[-(V+65)/10]. \tag{4f}$$

ζ_{x_i} represents the independent zero mean Gaussian white noise whose autocorrelation functions are given as follows [56]:

$$(\zeta_m(t)\zeta_m(t'))=\frac{2\alpha_m\beta_m}{N_{\mathrm{Na}}(\alpha_m+\beta_m)}\delta(t-t'), \tag{5a}$$

$$(\zeta_h(t)\zeta_h(t'))=\frac{2\alpha_h\beta_h}{N_{\mathrm{Na}}(\alpha_h+\beta_h)}\delta(t-t'), \tag{5b}$$

$$(\zeta_n(t)\zeta_n(t'))=\frac{2\alpha_n\beta_n}{N_{\mathrm{K}}(\alpha_n+\beta_n)}\delta(t-t'), \tag{5c}$$

where $N_{\mathrm{Na}}=\rho_{\mathrm{Na}}S$ and $N_{\mathrm{K}}=\rho_{\mathrm{K}}S$ represent the total numbers of sodium and potassium channels in a given cell size, respectively. S is the cell size or the membrane area used for the scaling of channel noise intensity. The number of channels per square micrometer of cell size is $\rho_{\mathrm{Na}}=60$ for sodium and $\rho_{\mathrm{K}}=18$ for potassium.

Following the procedure in [57], we construct the scale-free neuronal network, using $N=200$ neurons with different average degree of connectivity, k_{avg}. To quantitatively measure the level of transmission of the peacemaker activity we calculate Fourier coefficient Q from the average membrane potential, $V_{avg}(t)=\frac{1}{N}\sum_{i=1}^{N}V_i(t)$ during K=1000 periods of pacemaker. Before the calculations of Q, we consider enough transient time (t_r) which is bigger than the autaptic time delay. Fourier coefficients are calculated as follows [59]:

$$Q_{\sin}=\frac{2}{KT}\int_{t_r}^{KT+t_r}V_{avg}(t)\sin(\omega t)\mathrm{d}t, \tag{6a}$$

$$Q_{\cos}=\frac{2}{KT}\int_{t_r}^{KT+t_r}V_{avg}(t)\cos(\omega t)\mathrm{d}t, \tag{6b}$$

$$Q=\sqrt{Q_{\sin}+Q_{\cos}}, \tag{6c}$$

where $\omega=2\pi/T$ is the frequency of the pacemaker. To ensure statistical accuracy, Q and Q_i values in all figures below are obtained by averaging over 20 different network realizations for the given parameter sets. Here, Q represents a measure of the transmission of the localized pacemaker activity. Thus, the bigger Q means the higher transmission of the pacemaker rhythm in the network.

3 Results and discussion

In all previous studies, dealing with to the propagation of a weak, localized activity induced by pacemaker neuron in neuronal networks, the presence of an autaptic connection has been ignored. Thus, we intend to show the effects of an autapse on the transmission of a weak localized pacemaker activity in a scale free neuronal network. For this aim, firstly, we try to determine the optimal conditions, namely, an optimal channel noise, an optimal network average degree and an optimal coupling strength, leading to the best propagation of the weak localized pacemaker activity throughout

the network in the absence of an autapse. In Figure 1, we show the contour plot of the dependence of Q on the channel noise intensity (scaled by S) and network average degree k_{avg} for the coupling strength ε=0.05 in the left panel, ε=0.1 in the middle panel, and ε=0.2 in the right panel of Figure 1, when the weak periodic stimulus is applied to the neuron which has the highest connectivity in the lower tray of Figure 1, and one of the neurons which has the lowest connectivity in the upper tray of Figure 1.

Results reveal that in each case of coupling strength, regardless of the neuron acting as a pacemaker whether it can be the highest connectivity one or the lowest connectivity one within the network, we obtain similar red shaded resonance islands emerging at the same S–k_{avg} pair, which indicate the high level of the transmission of the localized pacemaker activity. These results show that the location of the pacemaker is not an important aspect considered in scale free networks for the propagation of the pacemaker activity. Similar findings are also reported in [54]. We also observe an increasing trend in both k_{avg} and channel noise intensity with increasing of coupling strength for the occurrence of a resonance. But, the best transmission of the weak localized pacemaker activity is obtained for the coupling strength of ε=0.05 at an optimal level of channel noise intensity S= 6 μm^2 and an optimal structure of the scale free network characterized by k_{avg}=12. The presence of an optimal level of channel noise intensity (S=6 μm^2) was also reported by Ozer et al. [22] for the best transmission of the pacemaker activity in small-world neuronal networks, and by Yilmaz and Ozer [26] for the best collective firing regularity in scale-free networks. In addition, the existence of the optimal network structure was reported in Yu et al. [58] for the best transmission of the weak signal, and in Yilmaz et al. [59] for the stochastic resonance in hybrid scale-free neuronal networks.

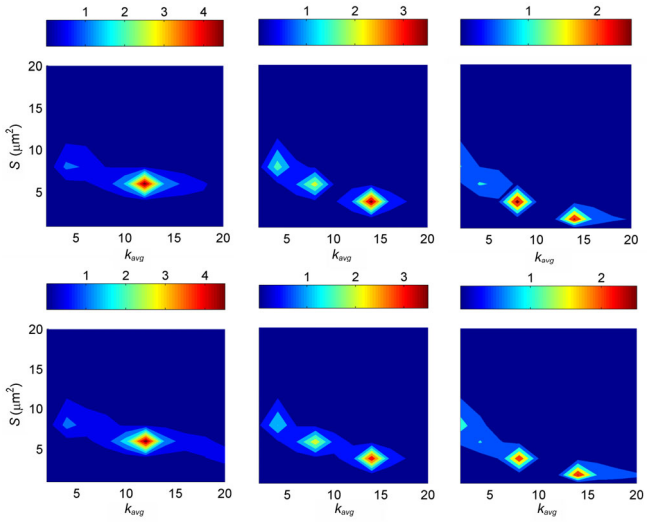

Figure 1 (Color online) The dependence of Q on S and k_{avg} for the coupling strength ε=0.05 in the left panel, ε=0.1 in the middle panel and ε=0.2 in the right panel, when the weak periodic stimulus is applied to the neuron which has the highest connectivity in the lower tray, and the neurons which has the lowest connectivity in the upper tray. Evidently seen that, irrespective of the location of the pacemaker neuron within the network we obtain similar resonance islands in S–k_{avg} parameters space for each ε, indicating the occurrence of the pacemaker induced stochastic resonance. These results emphasis the high capacity of scale-free networks in the transmission of weak signal.

After determining the optimal conditions satisfying the optimal transmission of the pacemaker activity across the whole network, we investigate the effects of an autapse on the propagation of the pacemaker activity. To this end, we suppose that only one neuron which acts as a pacemaker has an autapse modeled as an electrical synapse. In Figure 2, we demonstrate the dependence of Q on channel noise intensity for different values of the autapse parameters, i.e. the autaptic coupling strength (κ) and the autaptic time delay (τ), by considering that the pacemaker neuron has the lowest number of neuronal connections (ε=0.05, k_{avg}=12). In order to provide a comparison, we also show the case where the pacemaker neuron does not have any autaptic connections.

At first glance to Figure 2, we can see that an autapse either enhances or suppresses the transmission of weak, rhythmic pacemaker activity across the network. When τ=20 ms, an autapse significantly diminishes Q for κ=0.51, whereas it causes the two-fold increase in Q for κ=0.22. If we fix the autaptic coupling strength as κ=0.22 then change the autaptic delay time τ, we observe similar effect emerging in changing κ. For κ=0.22, the autapse prominently enhances the propagation of the pacemaker activity when τ=20 ms and τ=41 ms while it slightly reduces Q at τ=11 ms. From these results, we come to conclusion that if the autaptic delay time is approximately equal to the intrinsic,

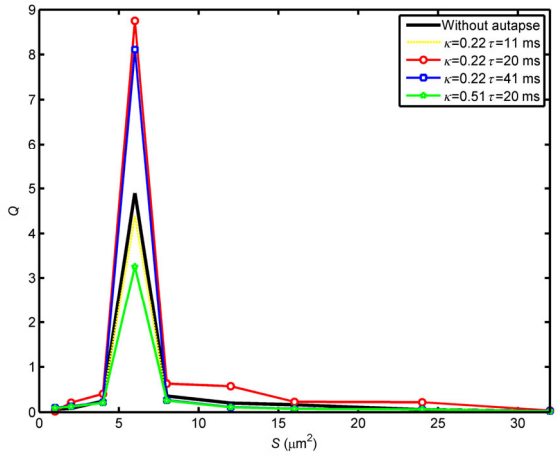

Figure 2 (Color online) The dependence of Q on S for k_{avg}=12 and ε= 0.05, where the weak periodic stimulus leading the pacemaker activity is applied to the one of the neurons which has the lowest connectivity. In figure, we consider the pacemaker neuron with and without autapse. For finely tuned values of the autaptic coupling strength κ and the autaptic delay time τ, the autapse prominently enhances the transmission of the pacemaker activity as compared to without an autapse.

subthreshold oscillation period of the HH neuron ($T_{asc}\approx 21$ ms) [60] or its integer multiples autapse improves the transmission of the pacemaker activity across the scale-free network for the proper values of autaptic coupling strength.

To demonstrate how the autapse effects the propagation of pacemaker activity when considered that the subthreshold periodic stimulus is applied to one of the neurons having the highest connectivity, we depict the dependence of Q on S for different autapse parameters in Figure 3.

It is seen in Figure 3 that an autapse either enhances or suppresses the propagation of pacemaker activity depending on its parameters. It prominently enhances Q when the autaptic delay time is roughly equal to T_{asc} or its integer multiples (κ=0.19). However, the enhancement is much more prominent provided that the pacemaker neuron has the lowest number of neuronal connections. This may be due to that synaptic current acting on the pacemaker neuron is greater for the neuron having the highest connectivity as compared to one which has the lowest connectivity. Because, this synaptic current may reduce to adopt the time scale dictated by autapse for the highest connectivity neuron, whereas it is so small for the lowest connectivity neuron and does not cause much inhibitive effect in adopting to the time scale dictated by autapse. Consequently, in the presence of an autapse the transmission of the rhythmic pacemaker activity is more intense when the pacemaker neuron is chosen as one of the neuron which has the lowest connectivity.

To present the above explanations more clearly, we give the individual Q values for each neuron in case the pacemaker neuron is chosen as the highest connectivity one (plus marker), and it is chosen the neurons having the lowest connectivity (cross mark) in Figure 4.

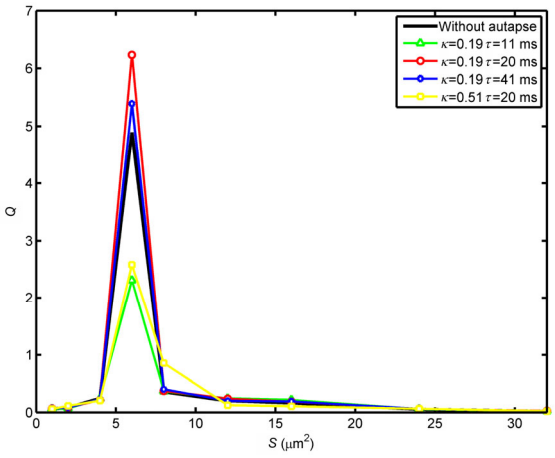

Figure 3 (Color online) The dependence of Q on S for k_{avg}=12 and ε=0.05, where the weak periodic stimulus leading the pacemaker activity is applied to the highest connectivity neuron (or main hub of network). In figure, we consider the pacemaker neuron with and without autapse. For finely tuned values of the autaptic coupling strength κ and the autaptic delay time τ, the autapse prominently enhances the transmission of the pacemaker activity as compared to without an autapse. But, increment in the transmission is limited by the synaptic current acting on the pacemaker neuron.

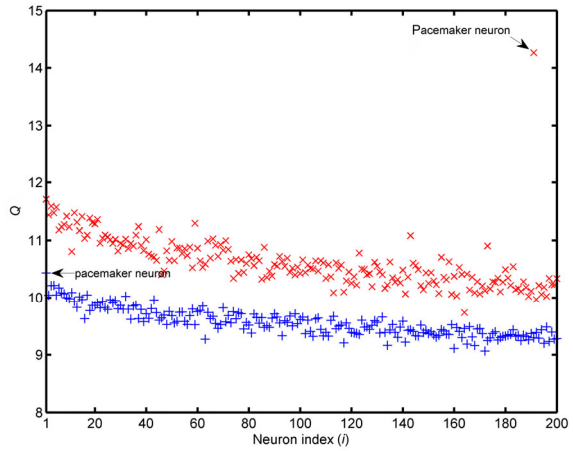

Figure 4 (Color online) The variation of individual Q_i values of neurons for the optimal cell size of S=6 μm^2 and k_{avg}=12, ε=0.05 in the presence of an autapse. Red cross markers (x) indicate Q_i values of neurons when the weak periodic stimulus is applied to the lowest connectivity neuron in the network with autapse parameters of κ=0.22, τ=20 ms. Blue plus markers (+) represent the individual Q_i values of neurons where the weak periodic stimulus is applied to the highest connectivity neuron in the network with the autapse parameters of κ=0.19, τ=20 ms. Autapse improves the transmission of the local pacemaker activity more powerfully in case the pacemaker neuron is chosen as the lowest connectivity one. Because, the autapse more easily dictates own time scale to the pacemaker neuron due to the less synaptic current coming to the pacemaker neuron.

It is obviously seen that the individual Q values of neurons are much higher when the subthreshold stimuli is applied to the pacemaker neuron having the lowest number of neuronal connections as shown in Figure 4. When the subthreshold stimulus is applied to the pacemaker neuron having lowest number of connections (in Figure 4, i=190), Q value of the pacemaker neuron is prominently higher than that of the pacemaker neuron having the highest number of connections (in Figure 4, i=1). This result explicitly reveals the effect of synaptic current on the pacemaker neuron.

To present a clear picture about the effects of an autapse on the pacemaker neuron, we show inter pike interval histogram (ISIH) of the pacemaker neuron when the subthreshold stimulus applied to one of the neuron which has the lowest connectivity in the upper tray of Figure 5 with (right panel) and without (left panel) an autapse, and similar graphs when the subthreshold stimulus is applied to the highest connectivity neuron in the lower tray of Figure 5. In the upper left panel of Figure 5, in the absence of an autapse, ISIH has a bimodal structure consisting of two clusters mainly occur around the intrinsic oscillation period of the HH neuron and its second harmonic. But, in the presence of an autapse (upper right panel of Figure 5) with κ=0.22 and τ=20 ms, ISIH has a distinct, sharp peak with a highest peak value occurring about the intrinsic oscillation period of HH neuron, which emphasis the presence of a dominant time scale added by the autapse. Such an ISIH was also observed by [50] in a single neuron where the effects of autapse on the weak signal detection performance of a HH

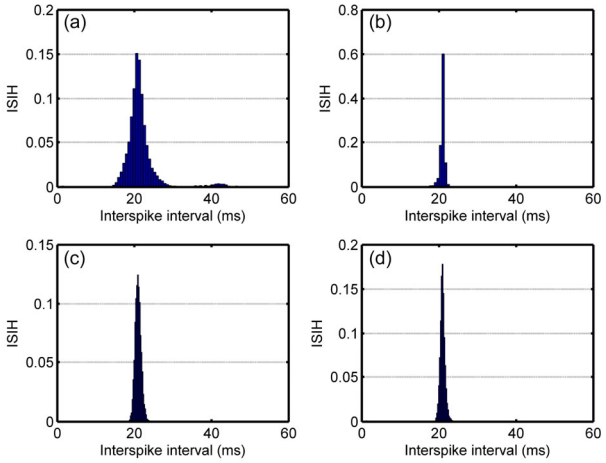

Figure 5 (Color online) ISIHs of the pacemaker neuron obtained from 10000 ISIs for k_{avg}=12, ε=0.05 in the presence (in right panels) and in the absence (in left panels) of an autapse, where the pacemaker neuron is the highest connectivity one ((c), (d)) and the lowest connectivity one ((a), (b)). In (b) κ=0.22, τ=20 ms and in the lower right panel κ=0.19, τ=20 ms. It is clearly seen that the pacemaker neuron exhibits a single dominant time scale, indicating an optimal phase locking between the spiking and the weak periodic signal for κ=0.22, τ=20 ms as compared to without an autapse (a). There is also a single dominant time scale in the lower trays, but the peak values of ISIH are less when compared to those in upper trays, which means relatively feebler phase locking between the weak periodic stimulus and the spiking of the pacemaker neuron.

neuron are investigated. However, in the lower tray of Figure 5, i.e, the pacemaker neuron is chosen as the highest connectivity one, ISIH of the pacemaker neuron with an autapse (lower right panel of Figure 5) is relatively more compact and has a higher peak value around T_{osc} as compared to that of without an autapse (lower left panel of Figure 5). Here, an autapse does not impose its own time scale to the pacemaker neuron, effectively. Thus, the increment in Q value in the presence of an autapse in Figure 3 remains limited.

The electrical activities of neuron and neuronal networks are deeply affected by autapses, and autapses can cause transitions (quiescent, periodic, and bursting) in the firing activities of the neuron and affect the fascinating phenomenon occurring in neuronal networks [61–63]. Thus, they have important effects on the neuronal dynamics, and merit special interests. Therefore, in the present study, we investigate the impact of an autapse on the propagation of the pacemaker activity within a scale-free neuronal network. We construct the network by using stochastic HH neurons, involving the noise from stochastic HH type ion channels where the cell size globally determines the intrinsic noise level. We first determine the optimal values of the coupling strength and the network average degree for an optimal propagation of the localized pacemaker activity in the absence of an autapse. We chose the pacemaker neuron as a neuron having either the highest or the lowest number of connections within the network. We found that the optimal propagation of the pacemaker rhythm requires an optimal average degree of k_{avg}=12, an optimal coupling strength of ε=0.05, and an optimal noise level or cell size of S=6 μm^2 in the absence of the autapse, irrespective of the connection level (degree) of the pacemaker.

Then, we consider that the pacemaker neuron has an electrical synapse between its axon and soma, and investigate its impact on the propagation of the pacemaker rhythm by using these optimal values. We find that the existence of an autapse as a part of the pacemaker significantly enhances the propagation of the pacemaker rhythm across the network if the autaptic delay time is equal to the period of the neuronal oscillations or its integer multiples irrespective of the connection level of the pacemaker neuron. However, we also find that this enhancement is much more prominent provided the pacemaker neuron has the lowest number of neuronal connections.

In future studies it remains of interest to test the robustness of these results also in other models of neuronal dynamics, for example near canard orbits [64], where subthreshold oscillations could contribute notably to higher values of Q, although this enhancement would not necessarily be linked to a better propagation of weak signals due to the absence of large-amplitude spikes.

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