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Autaptic pacemaker mediated propagation of weak rhythmic activity across small-world neuronal networks

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HIGHLIGHTS

- Effects of an autapse on the pacemaker neuron are studied.
- The autapse can either promote or enhance the transmission of the pacemaker rhythm.
- Optimal signal propagation requires a specific intensity of the intrinsic noise.
- Weakest signals propagate optimally only for specific autapse parameters.

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ABSTRACT

We study the effects of an autapse, which is mathematically described as a self-feedback loop, on the propagation of weak, localized pacemaker activity across a Newman–Watts small-world network consisting of stochastic Hodgkin–Huxley neurons. We consider that only the pacemaker neuron, which is stimulated by a subthreshold periodic signal, has an electrical autapse that is characterized by a coupling strength and a delay time. We focus on the impact of the coupling strength, the network structure, the properties of the weak periodic stimulus, and the properties of the autapse on the transmission of localized pacemaker activity. Obtained results indicate the existence of optimal channel noise intensity for the propagation of the localized rhythm. Under optimal conditions, the autapse can significantly improve the propagation of pacemaker activity, but only for a specific range of the autaptic coupling strength. Moreover, the autaptic delay time has to be equal to the intrinsic oscillation period of the Hodgkin–Huxley neuron or its integer multiples. We analyze the inter-spike interval histogram and show that the autapse enhances or suppresses the propagation of the localized rhythm by increasing or decreasing the phase locking between the spiking of the pacemaker neuron and the weak periodic signal. In particular, when the autaptic delay time is equal to the intrinsic period of oscillations an optimal phase locking takes place, resulting in a dominant time scale of the spiking activity. We also investigate the effects of the network structure and the coupling strength on the propagation of pacemaker activity. We find that there exist an optimal coupling strength and an optimal network structure that together warrant an optimal propagation of the localized rhythm.

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1. Introduction

Noise leads to different phenomena in nonlinear systems, a prominent example of which are neurons and neuronal networks. One of the most well-known phenomenon emerging in neuronal networks as well as other nonlinear systems [1], is the stochastic resonance (SR) [2–7], where the response of a nonlinear system to a weak stimulus exhibits a maximum in signal to noise ratio (SNR) in the presence of an optimal noise intensity. In neurons, channel noise stemming from random open–close fluctuations of ion channels is an important noise source [8–10]. Its effects on the weak signal detection, propagation and coding are studied in detail by using the stochastic Hodgkin–Huxley (H–H) model in a single neuron and in complex neuronal networks [5,11–13].

Information exchange between neurons takes place at special junctions called synapses. Synapses are mainly categorized as electrical synapses or chemical synapses with respect to signal transmission way. It is well-known that these special structures occur between one neuron's axon terminal and another neuron's dendrite. There is also a type of synapse, called autapse, a term introduced by Van der Loos and Glaser [14], which is morphologically similar to the normal synapses. Contrary to popular opinion, an autapse occurs between the dendrite and the axon of the same neuron. In nervous system, the possible existence of this type of synapse in different brain regions is reported in various experimental studies [14–17]. Inhibitory interneurons in the visual cortex form approximately 10–30 autapses [18]. Lübke et al. [19] demonstrated that the majority of the cortical pyramidal neurons including neurons in the human brain in the developing neocortex have autaptic connections. Bacci et al. [20] reported that fast-spiking interneurons in the neocortical slices of the layer V exhibit inhibitory autaptic activities.

In addition to the aforementioned studies presenting the possible existence of autapses, there are some studies investigating the effects of autapses on the neuronal dynamics. In theoretical works, it was shown that the response dynamics of a single neuron can be effectively modulated by a self-delayed feedback mechanism which can be modeled as an electrical or a chemical synapse [21,22]. Bacci and Huguenard [23] experimentally showed that inhibitory autaptic transmission plays a crucial role in enhancement of the spike-timing precision of the neocortical inhibitory interneurons. They also reported that although the pyramidal neurons in the layer V do not have any inhibitory autapses, adding of artificial inhibitory autapses via dynamic clamp enhances prominently the spike timing precision of them [23]. Li et al. [24] analyzed the effects of an electrical autapse on the response dynamics of a H–H neuron in the presence of the intrinsic channel noise by means of inter-spike interval histograms. They observed an autapse-induced degradation in the spontaneous spiking activity at the characteristic frequencies and a specific frequency-locking mechanism in the average inter-spike intervals. Masoller et al. [25] showed that the subthreshold activity of a thermo-sensitive H–H neuron affects significantly the response of the neuron to a delayed feedback and noise. In Ref. [26], it was shown that autapses enhance the synchrony of basket cell (a kind of interneuron) networks when they oscillate in the gamma range (40–80 Hz). Firing pattern transitions induced by autapses in a Hindmarsh–Rose (HR) neuron was studied theoretically by Wang et al. [27]. They showed that the autapse causes the switching of the HR neuron among quiescent, periodic and chaotic state depending on own parameters []. Wang et al. [28] showed that an autapse can enhance or suppress the mode-locking firing, and they suggested that the autapse can serve as a potential control option for adjusting the mode-locking firing behavior. Sainz-Trapaga et al. [29] demonstrated that a self-feedback causes spikes by increasing the amplitude of the subthreshold oscillations above the threshold. In a recently published study, we have shown that the weak signal detection capacity of a single H–H neuron can be prominently increased by an electrical autapse for appropriately tuned values of its parameters [30]. Moreover, in Ref. [31], it was demonstrated that the electrical activity of neurons is in general much more sensitive to electrical autapses than chemical autapses. In Ref. [32,33], it was shown that a negative feedback can suppress exciting neurons, and thus lead to defects in the network, while a positive feedback is likely to promote excitations and induce mode transitions by generating continuous signal sources that ultimately lead to pulse or target waves, or even spiral waves under an appropriate noise intensity.

On the other hand, pacemakers are special cells in tissue trying to impose their rhythms to whole neighbors. The most prominent organ having pacemaker cells is the human heart [34]. Thus, it merits special attention and deeper investigations. Therefore, in literature there are many studies investigating the effects of pacemaker activity on the complex neuronal networks [5,35–37]. But, there are very few studies investigating the effects of autapses on the phenomenon emerging in neuronal networks, especially pacemaker induced stochastic resonance. In the current study, our aim is to extend the study realized by Ozer et al. [5] in which they have investigated the transmission of the localized pacemaker activity across the small-world networks of stochastic H–H neurons. Different from their study, we consider that the single unit operating as a pacemaker has an autapse modeled as an electrical synapse. Thereby, we investigate the effects of autapse on the pacemaker induced stochastic resonance in small-world neuronal networks depending on ion channel noise. We separately investigate the effects of the coupling strength and the network structure on the transmission of pacemaker rhythm.

2. Model and methods

The membrane potential dynamics of coupled H–H neurons are governed by the following equations in a small-world network [5]:

$$C_m \frac{dV_i}{dt} = g_K^{\max} n_i^4 (E_K - V_i) + g_{Na}^{\max} m_i^3 h_i (E_{Na} - V_i) + g_L (E_L - V_i) + \sum_j \varepsilon_{ij} (V_j(t) - V_i(t)) \quad (1)$$

where $1 \leq i \leq N$. N is the total number of neurons in the network. V_i denotes the membrane potential of neuron i, and $C_m = 1\ \mu\text{F/cm}^2$ is the capacity of the cell membrane. $E_K = -77$ mV, $E_{Na} = 50$ mV, and $E_L = 54.4$ mV are the reversal potentials for the potassium, sodium and leakage current, respectively. $g_K^{max} = 36$ mS cm^{-2} and $g_{Na}^{max} = 120$ mS cm^{-2} respectively denote the maximal potassium and sodium conductance, when all ion channels are open. In the model, the leakage conductance is assumed to be constant, $g_L = 0.3$ mS cm^{-2}. ε_{ij} represents the coupling strength between two neurons i and j. m_i and h_i denote activation and inactivation variables for the sodium channel of neuron i, respectively. The potassium channel includes an activation variable, n_i. We assume that $\varepsilon_{ij} = \varepsilon$ if the neurons i and j are connected; otherwise $\varepsilon_{ij} = 0$. In the H–H model, the dynamics of gating variables change over time in response to membrane potential [38]. However, if the number of ion channel is finite, the stochastic effect originated by random open–close fluctuations of the ion channels may have remarkable effects on the neuronal dynamics. To take into account the channel stochasticity, we use Fox's algorithm [39] due to its widespread usage and computational efficiency. In Fox's algorithm, the gating dynamics is described by the Langevin generalization as follows [39]:

$$\frac{dx_i}{dt} = \alpha_{x_i}(V)(1 - x_i) - \beta_{x_i}(V)x_i + \zeta_{x_i}(t), \quad x_i = m_i, n_i, h_i \tag{2a}$$

where $\alpha_{x_i}(V)$ and $\beta_{x_i}(V)$ [38] are the voltage-dependent rate functions for the gating parameter x_i, and given as follows:

$$\alpha_m(V) = \frac{0.1(V + 10)}{1 - \exp[-(V + 40)/10]} \tag{2b}$$

$$\beta_m(V) = 4\exp[-(V + 65)/18] \tag{2c}$$

$$\alpha_h(V) = 0.07\exp[-(V + 65)/20] \tag{2d}$$

$$\beta_h(V) = \frac{1}{1 + \exp[-(V + 35)/10]} \tag{2e}$$

$$\alpha_n(V) = \frac{0.01(V + 55)}{1 - \exp[-(V + 55)/10]} \tag{2f}$$

$$\beta_n(V) = 0.125\exp[-(V + 65)/80] \tag{2g}$$

ζ_{x_i} denotes the independent zero mean Gaussian white noise whose autocorrelation functions are given as follows [39]:

$$\zeta_m(t)\zeta_m(t') = \frac{2\alpha_m\beta_m}{N_{Na}(\alpha_m + \beta_m)}\delta(t - t') \tag{3a}$$

$$\zeta_n(t)\zeta_n(t') = \frac{2\alpha_n\beta_n}{N_K(\alpha_n + \beta_n)}\delta(t - t') \tag{3b}$$

$$\zeta_h(t)\zeta_h(t') = \frac{2\alpha_h\beta_h}{N_{Na}(\alpha_h + \beta_h)}\delta(t - t') \tag{3c}$$

where N_{Na} and N_K represent the total numbers of sodium and potassium channels in a given cell size, respectively. The total number of ion channels are calculated as $N_{Na,K} = \rho_{Na,K}(CellSize)$. The number of channel per square micrometer of the cell size is $\rho_{Na} = 60$ for sodium and $\rho_K = 18$ for potassium, respectively. It is easily seen in Eq. (3) that when the cell size is large enough the stochastic effect added by the ion channels to the membrane potential is trivial, but when the cell size is small the stochastic effect due to the ion channels is very crucial [10].

The considered interaction network for H–H neurons is constructed by starting from a regular ring with periodic boundary conditions including $N = 60$ H–H neurons, each having $k = 2$ nearest neighbors. The probability of adding a link is denoted by p and can occupy any value from the unit interval, whereby $p = 0$ constitutes a regular graph (same with initial network structure) while $p = 1$ results in a random network. For $0 < p < 1$, the obtained network exhibits small-world properties. The probability p is given as follows

$$p = \frac{2M}{N(N - 1)} \tag{4}$$

where M denotes total number of links which added to regular ring by following the Newman–Watts model [39]. A regular ring network with $p = 0$ and a Newman–Watts small-word network with $p = 0.02$ is displayed schematically in Fig. 1.

The subthreshold stimulus generating the localized rhythmic activity is applied to the neuron $i = r = 30$ (henceforth called pacemaker neuron) with the mathematical form of $I_r(t) = A\sin(\omega t)$, where $A = 1\ \mu\text{A/cm}^2$ is the amplitude and $\omega = 0.3\ \text{ms}^{-1}$ is the frequency of the weak stimulus. Since we consider that only the pacemaker neuron has a delayed self-feedback or an electrical autapse, the autaptic current added to the pacemaker's membrane potential is given as follows [24]:

$$I_{aut} = \kappa[V_r(t - \tau) - V_r(t)]. \tag{5}$$

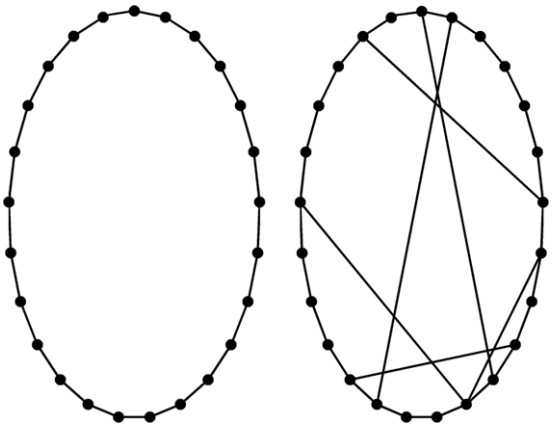

Fig. 1. Examples of considered network topologies. In the left panel, a regular ring having $p = 0$ with periodic boundary conditions and node degree $k_0 = 2$ is shown, whereas in the right panel a small-world network is depicted where $M = 6$ links were added randomly to every node with the probability of $p = 0.02$. For clarity regarding k and p only $N = 25$ nodes are displayed in each panel.

This autaptic current is proportional to the difference of the membrane potential of pacemaker neuron (V_r) at time t and $t - \tau$ [24]. κ denotes the autaptic coupling strength, and τ represents the autaptic time delay, which occurs because of the finite propagation speed during axonal transmission.

To quantitatively determine the correlation between the weak periodic signal and the collective activity of H–H neurons in the presence of both channel noise and an autapse, we first calculate the average membrane potential ($V_{avg}(t) = 1/N \sum_{i=1}^{N} V_i(t)$) during $T = 1000$ periods of the pacemaker. In calculations, we consider enough transient time (t_r) which is bigger than the autaptic delay time, before the calculations of Q. Then, we calculate the Fourier coefficients by using average membrane potential to measure the correlation between the weak signal frequency $\omega = 2\pi/t_s$ and the collective temporal activity of the network as follows:

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

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

$$Q = \sqrt{Q_{\sin}^2 + Q_{\cos}^2} \tag{6c}$$

where n is the number of periods t_s covered by the integration time. To ensure statistical accuracy, Q and Q_i values in all figures below are obtained by averaging over 50 different network realizations for the given parameter sets. Here, Q represents a measure of the transmission of the localized pacemaker activity. Thus, a bigger Q means a higher transmission of the pacemaker rhythm across the whole network. The numerical integration of the model is performed by using the standard Euler algorithm with a step size of 10 μs. Moreover, all neurons are initiated with the initial conditions $V_0 = -65$ mV, $m_0 = 0.0529$, $n_0 = 0.31768$, $h_0 = 0.59612$.

3. Results and discussion

Ozer et al. [5] have investigated the transmission of the localized rhythmic activity induced by a pacemaker neuron in a Newman–Watts small-world network of stochastic H–H neurons by means of Q and Q_i (see Model and methods), depending on ω, p, ε, *CellSize* (henceforth denoted by S in the study). In the current study, we assume that the pacemaker neuron has an electrical autapse modeled as a delayed self-feedback loop, which was not considered in Ozer et al. [5], and then we investigate its effects on the propagation of the pacemaker rhythm throughout the network by following their analysis steps. To do this, we firstly demonstrate the effects of different values of the autaptic coupling strength (κ) and the delay time (τ) on Q in Fig. 2(a) depending on channel noise intensity with $\varepsilon = 0.05$ and $p = 0.125$ for the angular frequency of pacemaker ($\omega = 0.3$ ms^{-1}). Also, we give the obtained Q values in Fig. 2(a), where the pacemaker neuron does not have an autapse.

In the absence of an autapse, the quantitative measure of the transmission of the localized rhythmic activity (Q) exhibits the stochastic resonance behavior with respect to channel noise intensity (scaled by S). This pure resonance also reveals the presence of an optimal channel noise intensity (or, an optimal cell size $S = 6\,\mu\text{m}^2$) for the optimal transmission of the local, rhythmic activity of the pacemaker neuron across the network [5]. However, in the presence of an autapse, we obtain not only a resonance-like dependence of Q on S but also the same optimal cell size of $S = 6\,\mu\text{m}^2$ for the optimal transmission of the pacemaker rhythm in each case of the autapse parameters. When $\tau = 8$ ms, the autapse slightly diminishes Q for a weak

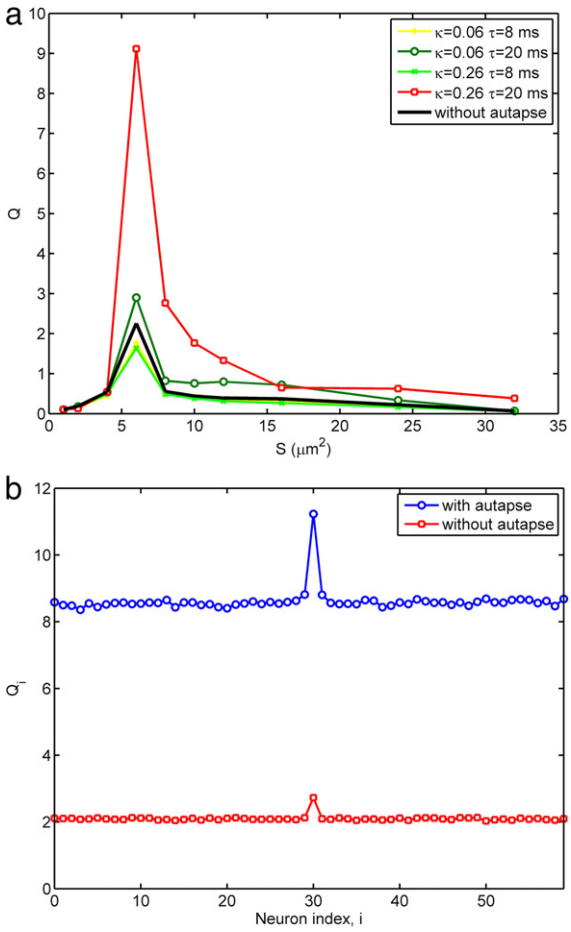

Fig. 2. An autapse on the pacemaker can significantly enhance the transmission of weak, localized rhythmic activity across the network. (a) The dependence of Q on S for various autapse parameters. For comparison, we also give the dependence of Q on S by assuming that the pacemaker neuron does not have an autapse. (b) The variation of individual Q_i values of neurons, as obtained for the optimal cell size of $S = 6\,\mu\text{m}^2$ in the presence ($\kappa = 0.26,\ \tau = 20$ ms and absence of an autapse ($p = 0.125$, $\omega = 0.3\ \text{ms}^{-1}$, $A = 1\ \mu\text{A/cm}^2$, $\varepsilon = 0.05$).

($\kappa = 0.06$) and an intermediate ($\kappa = 0.26$) autaptic coupling strengths. When $\tau = 20$ ms, the autapse feebly increases the propagation of pacemaker rhythm for a weak ($\kappa = 0.06$) autaptic coupling strength while it distinctly enhances the spread of the pacemaker rhythm across the network for $\kappa = 0.26$, as compared to without autapse. These results emphasize that autapse can either strongly promote or prominently suppress the spreading of localized pacemaker rhythm depending on the selection of its parameters. In Fig. 2(b), we give the individual Q_i values of each neuron at the optimal channel noise intensity ($S = 6\,\mu\text{m}^2$) by considering the presence and absence of an autapse in the pacemaker neuron. As seen in Fig. 2(b), Q_i values of each neuron are significantly improved by the autapse. Furthermore, in either case, the pacemaker neuron ($i = r = 30$) exhibits the most correlated response, corresponding to the highest Q_i value, with the subthreshold periodic stimulus due to its direct influence.

To present a clear picture about the dependence of Q on p in the presence of an autapse with $\kappa = 0.26$, $\tau = 20$ ms, $p\,\varepsilon$ we calculate Q depending on p for three different values of the coupling strength ε at the optimal cell size of $S = 6\,\mu\text{m}^2$ in Fig. 3. At each value of the coupling strength ε, Q exhibits a resonance-like behavior with respect to p, which reveals the presence of an optimal network structure for the propagation of the pacemaker rhythm. Moreover, the optimal p value, by p which Q reaches a maximum, decreases as the coupling strength is increased. This trend observed in the optimal p with the increasing of coupling strength has been reported in Refs. [5,40,41]; the latter have investigated the temporal coherence of the HR neurons in a small-world network. Meanwhile, the strength of the resonance (the maximum of Q) decreases as the coupling strength is increased. Importantly, these results reveal the existence of an optimal network topology characterized by $p = 0.125$, and an optimal coupling strength $\varepsilon = 0.05$ that together ensure an optimal transmission of the pacemaker rhythm at the optimal channel noise intensity ($S = 6\,\mu\text{m}^2$). The presence of an optimal probability of adding links has been also reported by Ozer et al. [5] for the best transmission of the localized pacemaker rhythm in the small-world neuronal networks of H–H neurons, where they have obtained $p = 0.1$ as an optimal, but in the present study we obtain $p = 0.125$ as an optimal. This difference in p might arise due to the interaction of the network structure and the autapse.

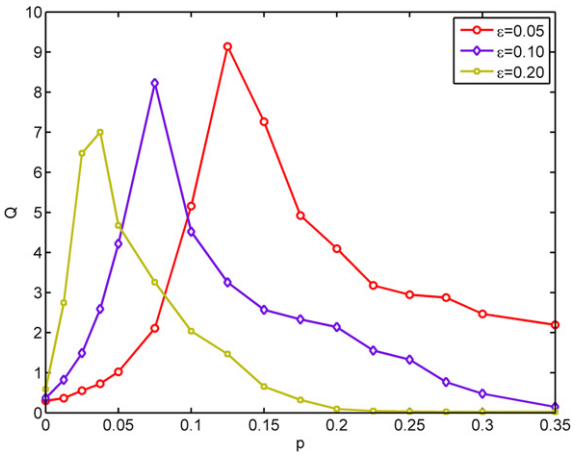

Fig. 3. An optimal network structure characterized by a proper value of p ensures the best propagation of the pacemaker rhythm. The dependence of Q on p for three different coupling strengths ε, as obtained for a fixed patch area $S = 6\,\mu m^2$ in the presence of an autapse ($\kappa = 0.26$, $\tau = 20$ ms) is presented.

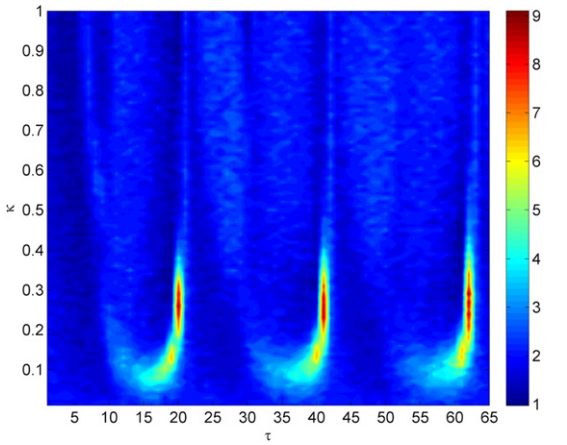

Fig. 4. An autapse can induce multiple stochastic resonances for specific parameters values. Presented are the effects of the autapse parameters on the values of Q for the optimal channel noise level $S = 6\,\mu m^2$. Multiple resonance regions, occurring approximately at integer multiples of the intrinsic oscillation period of the H–H neuron, can be observed for the specific range of the autaptic coupling strength ($p = 0.125$, $\omega = 0.3$ ms^{-1}, $A = 1\,\mu A/cm^2$, $\varepsilon = 0.05$, $S = 6\,\mu m^2$).

To present a global picture about the dependence of Q on the autapse parameters, namely, autaptic coupling strength κ and delay time τ, we measure Q values in $\kappa - \tau$ parameter space for the optimal network parameters equaling $p = 0.125$, $\varepsilon = 0.05$ and $S = 6\,\mu m^2$. Obtained results are given as a contour plot in Fig. 4. Evidently seen that Q exhibits multiple stochastic resonance phenomenon depending on the autaptic delay time τ in a limited span of κ. However, the enhancement effect of autapse on the propagation of the localized pacemaker rhythm occurs only for a specific range of autaptic coupling strength around $0.1 \leq \kappa \leq 0.35$ when the delay time τ is roughly equal to the intrinsic oscillation period ($T_{osc} \approx 21$ ms) of the H–H neuron [42] or its integer multiples. Similar range of the autaptic coupling strength has recently been reported by Yilmaz and Ozer [29], where they have analyzed the effect of the autapse on the weak signal detection capacity of a single stochastic H–H neuron. Interestingly, outside this range, autapse does not concretely improve the propagation of the pacemaker rhythm even though the autaptic delay time is equal to the intrinsic oscillation period or its integer multiples. This finding can be explained as follows; the delayed feedback or the autapse introduces some different time scales to the pacemaker neuron dynamics [24] depending on the value of the own parameters, and this time scales cause some different firing patterns with varying firing rates on the pacemaker neuron's firing behavior. When the entrained time scale by the autapse matches up with the intrinsic oscillation period and the period of the subthreshold stimulus, the enhancement effect of the autapse emerges. If the time scale stemming from the autapse does not agree with the period of the subthreshold oscillations and the stimulus the suppressing effect of the autapse on the transmission of the pacemaker rhythm may occur.

To provide a supporting evidence to above explanations, in Fig. 5, we present the inter-spike interval histograms (ISIHs) of the pacemaker neuron with and without autapse for the parameter set used in Fig. 3. In the absence of the autapse (in the upper panel of Fig. 5) the pacemaker neuron has an ISIH clustered around the period of the intrinsic oscillation with a

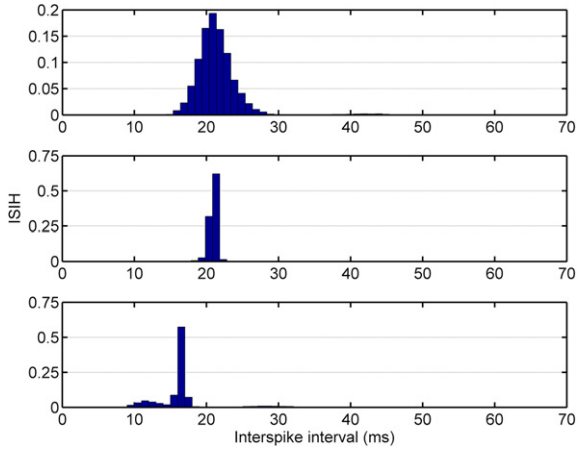

Fig. 5. An autapse can affect the dominant time scales of the firing activity of the pacemaker neuron. Presented are inter-spike interval histograms (ISIHs) of the pacemaker neuron obtained from 10000 inter-spike intervals at $S = 6\ \mu m^2$ (a) in the absence of the pacemaker autapse, (b) in the presence of the pacemaker autapse with $\kappa = 0.26,\ \tau = 20$ ms, and (c) in the presence of the pacemaker autapse with $\kappa = 0.26,\ \tau = 25$ ms.

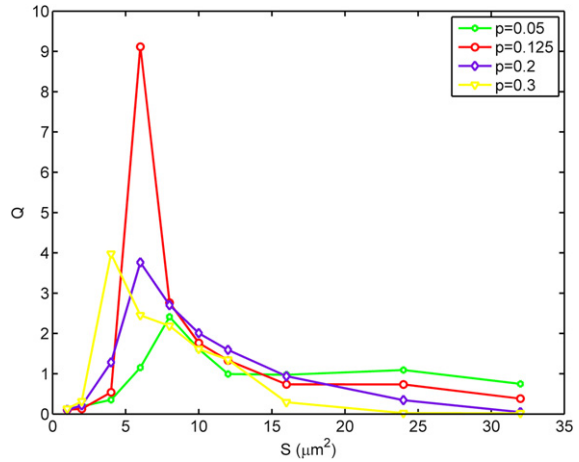

Fig. 6. An autapse can improve the transmission of the pacemaker activity only at specific channel noise intensities. Presented is the dependence of Q on S for various values of p obtained for fixed coupling strength $\varepsilon = 0.05$ in the presence of an autapse with $\kappa = 0.26,\ \tau = 20$ ms.

relatively wider peak. But, in the presence of the autapse with $\kappa = 0.26$ and $\tau = 20$ ms (in the middle panel of Fig. 5) ISIH of the pacemaker neuron has a single sharp, distinct peak with a higher peak value compacted around the intrinsic oscillation period of the H–H neuron, which indicates the existence of a dominant time scale introduced by autapse. On the other hand, when $\kappa = 0.26$ and $\tau = 25$ ms, another dominant time scale, which emerges in the firing activity of the pacemaker neuron, does not consistent with the period of the intrinsic oscillations and the stimulus (in the lower panel of Fig. 5). In this case, the rhythm imposed to neurons by the autapse is different from that of the stimulus, and thus the subthreshold stimulus is not transmitted across the whole network, effectively.

In the presence of an autapse with $\kappa = 0.26$ and $\tau = 20$ ms, the dependence of Q on the channel noise intensity (S) for various values of p is given in Fig. 6. Irrespective of the value of p, we obtain an optimal channel noise intensity range ($S = 4 - 6\ \mu m^2$), providing the pacemaker neuron to optimally impose own rhythm to the whole network. Besides, with increasing of p the optimal channel noise intensity at which Q reaches a maximum slightly moves the higher noise intensities (corresponding to smaller cell sizes). This optimal range of the intrinsic channel noise intensity has been reported in Ref. [40], where the collective firing regularity of the H–H neurons in small-world neuronal networks was investigated. In a recently published study [29], the best weak signal detection performance of a single H–H neuron has been obtained at $S = 16\ \mu m^2$ in the presence of an electrical autapse. The difference between optimal noise intensity may be originated from the noise scaling effect of the considered network structure. However, the optimal transmission of the localized pacemaker rhythm is ensured by $p = 0.125$ and $S = 6\ \mu m^2$.

Lastly, we investigate the dependence of Q on the coupling strength ε in the presence ($\kappa = 0.26, \tau = 20$ ms) and in the absence of an autapse with the optimal value of $p = 0.125$ and the optimal channel noise intensity $S = 6\ \mu m^2$. Obtained result are depicted in Fig. 7. The results in Fig. 7 reveal that, in either case, Q exhibits a resonance-like dependence

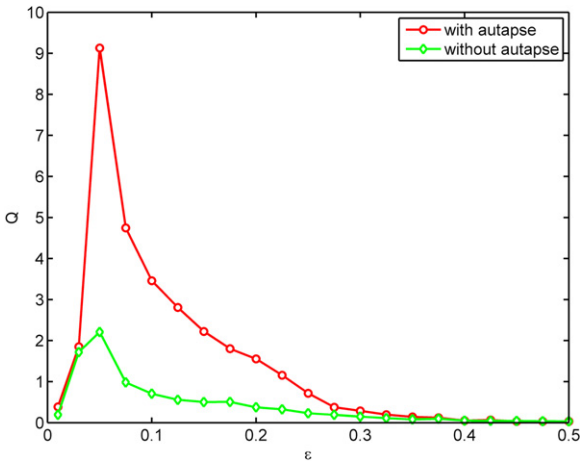

Fig. 7. Increasing the coupling strength decreases the positive effect of the autapse on the propagation of the pacemaker rhythm. Shown is the dependence of Q on the coupling strength ε for the optimal network structure characterized by $p = 0.125$ and with the optimal cell size $S = 6\ \mu m^2$, as obtained with the autapse ($\kappa = 0.26$, $\tau = 20$ ms) and without the autapse on the pacemaker.

on ε, indicating the existence of an optimal coupling strength value equaling $\varepsilon = 0.05$ for the optimal transmission of the localized pacemaker rhythm. In addition, these results not only emphasis that the autapse can distinctly enhance Q in a restricted range of the coupling strength around $0.05 < \varepsilon < 0.275$ but also that there is no significant effect of the coupling strength outside this range on the propagation of the pacemaker rhythm even though the pacemaker neuron has an autapse.

4. Conclusions

Autapses play an important biological role in regulating the electric activities of neuron by dictating different time scales to neuron. Thus, in the current study, using the stochastic H–H neuron model, we study the effects of an autapse on the transmission of the localized pacemaker rhythm across Newman–Watts small-world neuronal networks. We model the autapse as an excitatory electrical synapse, and consider that only the pacemaker neuron, which is under direct influence of the subthreshold periodic stimulus, has an autapse. Then, by systematically altering the parameters of the autapse (i.e., the autaptic coupling strength and delay time), we try to observe its effects on the transmission of the pacemaker rhythm in the whole network by means of Fourier coefficient Q and Q_i, depending on channel noise. We also separately investigate the effects of network parameters such as the coupling strength and the probability of adding links. Besides, obtained results in the presence and absence of an autapse are compared. The results reveal that the autapse can distinctly enhance or prominently suppress the transmission of the localized pacemaker rhythm depending on its parameters, when compared to that without an autapse. Moreover, we obtain an optimal channel noise intensity $S = 6\ \mu m^2$, an optimal coupling strength $\varepsilon = 0.05$ and an optimal network structure defined by $p = 0.125$ for the best transmission of the pacemaker rhythm in the presence of an autapse. For these optimal values, we find some different resonance islands, where the pacemaker rhythm is effectively transmitted, emerging in the range of $0.1 \leq \kappa \leq 0.35$ in $\kappa - \tau$ parameter space when the delay time (τ) is approximately equal to the intrinsic oscillation period of the H–H neuron or its integer multiples. We have also shown that autapse increases the phase locking between the spiking of the pacemaker and the weak stimulus by providing a single dominant time scale, which is coherent with the period of weak stimulus, in the spike trains of the pacemaker neuron. From the whole results, we conclude that when the time scale originated from the autapse resonates with the inherent oscillation period of the H–H neuron and the period of the subthreshold stimulus, the transmission of the localized pacemaker rhythm is promoted prominently as compared to without autapse.

In the current study, for simplicity, we consider that only a single neuron, called the pacemaker neuron, has an autapse that is modeled as an electrical synapse. But, in realistic circumstances, one neuron may have more autaptic connections [18], which could also be modeled as a chemical synapse. Therefore, in future studies, it is of interest to investigate the effects of chemical autapses on the fascinating nonlinear phenomena emerging in neuronal networks. In terms of real-life implications, we note that autapses have been shown to play a relevant role by epilepsy [43]. Theoretically, the role of time delays [44] and autapses on the detection of the first spike latency in stochastic neuronal networks might also merit further research.

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