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Effects of channel blocks on the spiking regularity in clustered neuronal networks

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In this paper, we discuss the influences of channel blocks on the spiking regularity in a clustered neuronal network by applying stochastic Hodgkin-Huxley neuronal models as the building blocks. With the aid of simulation results, we reveal that the spiking regularity of the clustered neuronal network could be resonantly enhanced via fine-tuning of the non-blocked potassium channel fraction x_K. While the non-blocked sodium channel fraction x_{Na} can enhance the spiking regularity of the clustered neuronal network in most cases. These results indicate that not only sodium channel blocks but also potassium channel blocks could have great influences on the regularity of spike timings in the clustered neuronal networks. Considering the importance of spike timings in neuronal information transforming processes, our results may give some implications for understanding the nonnegligible role of randomness in ion channels in neuronal systems.

channel blocks, spiking regularity, clustered neuronal networks

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1 Introduction

As we know, brain cortex contains about 10^{11} neurons with a single neuron connecting to more than 10^4 postsynaptic neurons through electrical or chemical synapses. Then, brain cortex becomes a very complex network [1]. The complex brain network is revealed to exhibit hierarchical and clustered (or modular) structures [2]. For a clustered network, it contains several small subnetworks with dense connections inside subnetwork and sparse connections between them. Compared to single neuron and single neuronal network, clustered neuronal network is at a mesoscopic level. In the past years, researchers mainly focus on investigating neuronal dynamics in single neuronal models or single neuronal networks. However, as we stated, brain cortex has complex network structures. In order to better understand the brain, it is not enough for just considering neuronal dynamics at a microscopic level (i.e., single neuron or single neuronal network). Thus, further studies of neuronal dynamics at a mesoscopic level becomes necessary.

In biological systems, noise is an unavoidable factor [3,4] and has been found to be related to some cognitive behaviors [5]. Understanding the influences of random fluctuations in neuronal systems is a central challenge in computational neuroscience. In neuronal systems, according to the originating sources of randomness, noise can be classified as synaptic noise and channel noise. For synaptic noise, it originates from neurotransmitters' quasi-random releasing by the synapses or presynaptic neurons' random inputs. For channel noise, it arises from the stochastic opening of ion channels. Researchers usually neglect channel noise [6–15]. However, it should be noted that channel noise could also have great effects on neuronal system when the membrane patch size is small [16]. Namely, channel noise could not be neglected when the membrane patch size is small.

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In recent years, the stochastic activity in ion channels has garnered increasing attentions as reviewed in ref. [17]. The importance of channel noise has been considered in several neuronal systems. For example, random fluctuations in ion channels have been included in establishing stochastic mathematical model of the auditory nerve for electrical stimulation [18]. And randomness from sodium ion channels has been found to be responsible for the theta rhythm observed in the entorhinal cortex [19]. Meanwhile, channel noise is also studied in the hippocampal CA1 pyramidal neurons [20]. In theoretical and simulating studies, it has been found that channel noise could have great influences on stochastic resonance [21], firing rhythms [22,23], phase synchronization [24], spiking regularity [25–27] and other neuronal dynamics [28–30].

Spike trains contain neuronal information and have been coded with different coding schemes [31–33]. Neuronal coding is divided into rate coding and temporal coding. For rate coding, neuronal information is just included in the neurons' firing rate sequences. While for temporal coding, relevant information is related to the timing of the spikes. Spiking regularity measures the regularity of spike timings. Thus, the variability of spiking regularity could have important effects on neuronal information transmission.

In neuronal dynamical studies, it has been found that channel noise could have great effects on the regularity of spontaneous spiking activities of neuronal systems [25–27, 34,35]. Numerical simulation results of these works showed that, potassium and sodium channel noises give different influences on spiking regularity of a single neuron[34,35] or array-coupled [25], Newman-Watts [26] and scale-free networks [27]. Potassium channel noise could enhance spiking regularity of a single neuron or a neuronal population, while sodium channel noise reduces it.

As mentioned above, it is necessary to further investigate channel noise's significance at a mesoscopic level. Up to now, the great influence of channel noise on the spiking regularity of a clustered neuronal network has not been studied yet. Therefore, here we devote ourselves to investigating how channel blocks affect the spiking regularity of a clustered neuronal network, with each subnetwork being regular and each neuron locally modeled by a stochastic hodgkin-huxley (HH) neuronal model. Our results could help us to make deeper understanding of the effects of channel noise on spiking regularity.

In the following section, we introduce a mathematical model of the system. Then, we present our numerical results to reveal the effects of channel blocks on the spiking regularity of the clustered neuronal network. Finally, we give a conclusion of our work and some discussions about our results.

2 Neuronal network model

In this paper, we consider a clustered neuronal network with M subnetworks arranged on a ring. And for each subnetwork, it has equal number of nodes, which are also arranged on a ring. For each node in a subnetwork, it connects to its $2k$ (k=2) nearest neighbors. Interaconnections between different subnetworks exist with the probability p. Therefore, the parameter p represents the fraction of total links in the network devoting to the connections between different subnetworks, and it is set as 0.05 in the whole paper. Meanwhile, M is taken as 2 if not declared specifically and N is taken as 120 in this paper.

We use a stochastic HH neuronal model as the local model. This stochastic HH neuronal model is extended from the traditional one [36] by considering sodium and potassium channel noises [37,38]. The mathematical equations of the studied system are expressed as follows:

$$C\frac{\mathrm{d}V_{I,i}}{\mathrm{d}t}=-G_{Na}(m_{I,i},h_{I,i})(V_{I,i}-V_{Na})-G_K(n_{I,i})(V_{I,i}-V_K)$$
$$-G_L(V_{I,i}-V_L)+\varepsilon_{\mathrm{int}\ ra}\sum_j A_I(i,j)(V_{I,j}-V_{I,i})$$
$$+\varepsilon_{\mathrm{int}\ er}\sum_J\sum_j B_{I,J}(i,j)(V_{J,j}-V_{I,i}), \quad (1)$$

$$\frac{\mathrm{d}m_{I,i}}{\mathrm{d}t}=\alpha_{m_{I,i}}(V_{I,i})(1-m_{I,i})-\beta_{m_{I,i}}(V_{I,i})m_{I,i}+\xi_{m_{I,i}}(t), \quad (2)$$

$$\frac{\mathrm{d}h_{I,i}}{\mathrm{d}t}=\alpha_{h_{I,i}}(V_{I,i})(1-h_{I,i})-\beta_{h_{I,i}}(V_{I,i})h_{I,i}+\xi_{h_{I,i}}(t), \quad (3)$$

$$\frac{\mathrm{d}n_{I,i}}{\mathrm{d}t}=\alpha_{n_{I,i}}(V_{I,i})(1-n_{I,i})-\beta_{n_{I,i}}(V_{I,i})n_{I,i}+\xi_{n_{I,i}}(t). \quad (4)$$

In eqs. (1)–(4), the subscript pairs (I, i) represent the i-th neuron of the I-th subnetwork. A_I=($A_I(i, j)$) and $B_{I,J}$=($B_{I,J}(i, j)$) ($I \neq J$) are two coupling matrixes. The former one is the coupling matrix for neurons of the I-th subnetwork. In the I-th subnetwork, if there is a connection between neuron (I, i) and neuron (I, j), then $A_I(i, j)$=1, otherwise, $A_I(i, j)$=0. The other matrix $B_{I,J}$ =($B_{I,J}$ (i, j)) ($I \neq J$) represents the connections of neurons which belong to different subnetworks. $B_{I,J}(i, j)$=1 if there is a link between neuron (I, i) and neuron (J, j), otherwise $B_{I,J}(i, j)$=0.

Here, we let the size of each subnetwork be n and the number of clusters in the considered network be M. In eq. (1), $\varepsilon_{\mathrm{int}\ ra}$ and $\varepsilon_{\mathrm{int}\ er}$ are the coupling strength of neurons inside a subnetwork and neurons between two nearest subnetworks, respectively.

In eqs. (1)–(4), $V_{I,i}$ is the membrane potential and $m_{I,i}$, $h_{I,i}$, $n_{I,i}$ are the gating variables. $m_{I,i}$ and $h_{I,i}$ represents the fractions of active and inactive sodium channel, respectively; and $n_{I,i}$ indicates the fraction of active potassium channel. In eqs.(2)–(4), $\alpha_{n_{I,i}}(V_{I,i})$ and $\beta_{n_{I,i}}(V_{I,i})$ ($y_{I,i}$ = $m_{I,i}$, $h_{I,i}$, $n_{I,i}$) are the transition rates which are dependent on voltage and expressed as [36]

$$\begin{aligned}
\alpha_{m_{I,i}}(V_{I,i}) &= \frac{0.1(V_{I,i}+40)}{1-\exp\left(-\dfrac{V_{I,i}+40}{10}\right)},\\
\beta_{m_{I,i}}(V_{I,i}) &= 4.0\exp\left[-\frac{(V_{I,i}+65)}{18}\right],\\
\alpha_{h_{I,i}}(V_{I,i}) &= 0.07\exp\left[-\frac{(V_{I,i}+65)}{20}\right],\\
\beta_{h_{I,i}}(V_{I,i}) &= \left\{1+\exp\left[-\frac{(V_{I,i}+35)}{10}\right]\right\}^{-1},\\
\alpha_{n_{I,i}}(V_{I,i}) &= \frac{0.01(V_{I,i}+55)}{1-\exp\left(-\dfrac{V_{I,i}+55}{10}\right)},\\
\beta_{n_{I,i}}(V_{I,i}) &= 0.125\exp\left[-\frac{(V_{I,i}+65)}{80}\right].
\end{aligned} \tag{5}$$

and $\xi_{m_{I,i}}(t)$, $\xi_{h_{I,i}}(t)$, $\xi_{n_{I,i}}(t)$ are the channel noises. They are assumed to be independent Gaussian white noise with the zero first-order moment. And their noise correlations are

$$\left\langle \xi_{m_{I,i}}(t)\xi_{m_{I,i}}(t')\right\rangle = \frac{2}{N_{Na}x_{Na}}\frac{\alpha_{m_{I,i}}(V_{I,i})\beta_{m_{I,i}}(V_{I,i})}{\alpha_{m_{I,i}}(V_{I,i})+\beta_{m_{I,i}}(V_{I,i})}\delta(t-t'), \tag{6}$$

$$\left\langle \xi_{h_{I,i}}(t)\xi_{h_{I,i}}(t')\right\rangle = \frac{2}{N_{Na}x_{Na}}\frac{\alpha_{h_{I,i}}(V_{I,i})\beta_{h_{I,i}}(V_{I,i})}{\alpha_{h_{I,i}}(V_{I,i})+\beta_{h_{I,i}}(V_{I,i})}\delta(t-t'), \tag{7}$$

$$\left\langle \xi_{n_{I,i}}(t)\xi_{n_{I,i}}(t')\right\rangle = \frac{2}{N_{K}x_{K}}\frac{\alpha_{n_{I,i}}(V_{I,i})\beta_{n_{I,i}}(V_{I,i})}{\alpha_{n_{I,i}}(V_{I,i})+\beta_{n_{I,i}}(V_{I,i})}\delta(t-t'). \tag{8}$$

Where $N_{Na}=\rho_{Na}S$ and $N_K=\rho_K S$ are the total number of sodium and potassium ion channels on a fixed excitable membrane patch. ρ_{Na}= 60 μm^{-2}, ρ_K =18 μm^{-2} are the ion channel densities of sodium and potassium ion channels. And S is the size of the membrane patch and taken as S =6 μm^2 in this paper. Some toxins like tetraethylammonium (TEA) and tetrodotoxin (TTX) could block some sodium or potassium ion channels [35]. If it happens, the numbers of working sodium and potassium ion channels will be reduced. Here, we induce two parameters x_{Na} and x_K ($0\leqslant x_{Na}, x_K\leqslant 1$) to express the fractions of working, *i.e.* non-blocked ion channels, to the total number of sodium and potassium ion channels [37]. Then, the numbers of working sodium and potassium ion channels take values of $x_{Na}N_{Na}$ and $x_K N_K$.

In eq. (1), C=1 μF cm^{-2} is the capacity of the cell membrane, and V_{Na}=50.0 mV, V_K =−77.0 mV and V_L= −54.4 mV are the reversal potentials for the sodium, potassium and leakage currents, respectively. While the leakage conductance is set as g_L=0.3 mS cm^{-2}, the potassium and sodium conductances are presented as follows:

$$G_{Na}(m_{I,i},h_{I,i}) = g_{Na}^{\max}x_{Na}m_{I,i}^3h_{I,i},\ G_K(n_{I,i}) = g_K^{\max}x_K n_{I,i}^4 \tag{9}$$

where $g_{Na}^{\max}=120$ mS cm^{-2} and $g_K^{\max}=36$ mS cm^{-2} denote the maximal conductance (when all the channels are at working state).

3 Simulation results

In this paper, we pay attention to the effects of channel blocks on spiking regularity of the clustered neuronal network. Measurement R is introduced to quantify it and expressed as follows:

$$R=\frac{1}{N}\sum_{i=1}^{N}R_i. \tag{10}$$

Here, N is the size of the clustered network, R_i is the inverse of the coefficient of variation and it can quantify the regularity of spike timings in a neuron. R_i is formulated as

$$R_i=\frac{\left\langle T_{i,k}\right\rangle}{\sqrt{\left\langle T_{i,k}^2\right\rangle-\left\langle T_{i,k}\right\rangle^2}}, \tag{11}$$

where $T_{i,k}=t_{i,k+1}-t_{i,k}$ represents the inter-spike interval with $t_{i,k}$ denoting the k-th spiking time of the i-th neuron; $\left\langle T_{i,k}\right\rangle$ and $\left\langle T_{i,k}^2\right\rangle$ represent the mean and the mean squared inter-spike intervals, respectively. Spiking times are identified as the moment at which the membrane potential V passes a certain value V_{th} upwarding (here V_{th} is taken as −20 mV). V_{th} can take values in a wide interval without altering the results.

Larger R means better spiking regularity of the whole neuronal network. In the followings, we take x_{Na} and x_K as control parameters to study the effects of sodium and potassium channel blocks on the spiking regularity of a clustered neuronal network. Without special statement, the inter-coupling and intra-coupling strength $\varepsilon_{\text{int}\,ra}$, $\varepsilon_{\text{int}\,er}$ are set equally as 0.1.

3.1 Effects of sodium channel blocks on the spiking regularity

At first, we investigate how R changes with the non-blocked sodium fraction x_{Na} for various x_K. The obtained results are presented in Figure 1.

The membrane potential of a neuron could reach a threshold value when Na^+ channels open and let enough Na^+ flow inward the neuron, i.e., generation of action potential needs enough Na^+ across the neuronal membrane. Smaller x_{Na} means fewer sodium channels at the working state, which leads to fewer Na^+ flowing inward. Then, membrane

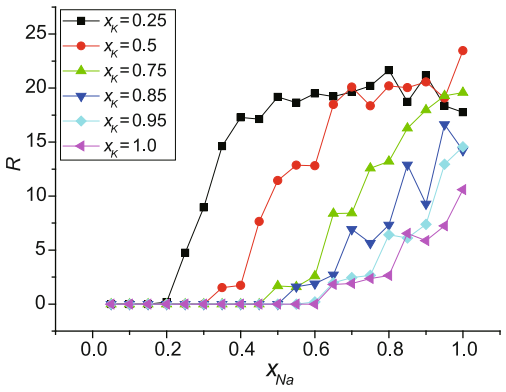

Figure 1 Variation of R as a function of x_{Na} for various x_K with M=2.

potential can not reach the threshold potential and neurons can not generate action potentials (or spikes). Thus, no matter what values x_K takes, R equals to zero when x_{Na} is small, as shown in Figure 1.

When x_{Na} becomes larger (e.g., when x_{Na} is larger than 0.6 for x_K =1.0, refer to Figure 1), R increases with x_{Na} for different values of x_K. Namely, spiking regularity of the neuronal network increases when x_{Na} becomes larger. In order to make it clear, we exhibit some spatiotemporal patterns at different values of x_{Na} by keeping x_K equal to one, shown in Figure 2. In this figure, each thick black line indicates that each neuron inside the neuronal network generates a spike (please refer to Figure 2(e), which shows the time series of a randomly chosen neuron corresponding to Figure 2(d). Figure 2(e) makes us better understand spatiotemporal patterns exhibited in Figure 2). Thus, the width between two thick black lines indicates the inter-spike intervals of each neuron. From Figure 2, we can see that changes of the inter-spike intervals for each neuron inside the neuronal network become more and more regular with x_{Na} increasing. Thus, the average spiking regularity of the

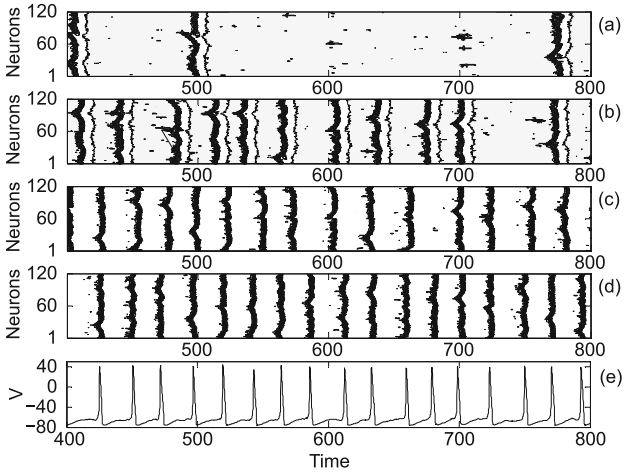

Figure 2 Spatiotemporal plots of membrane potentials of all neurons for various values of x_{Na} by fixing x_K = 1.0. x_{Na} is taken as 0.7,0.8,0.9,1.0 from (a) to (d). Time series of the membrane potential V of a randomly chosen neuron is shown in (e), which is corresponding to (d).

whole neuronal network could be enhanced by increasing the working sodium channel fraction x_{Na}. In other words, blocking sodium channels will reduce the spiking regularity of the neuronal network. Moreover, it is worth noting that the increasing of x_{Na} means the decreasing of sodium channel noise level. With the simulation results shown in Figures 1 and 2, we have found that when potassium channel noise level is fixed, reduction of sodium channel noise level could increase the spiking regularity of the clustered neuronal network.

3.2 Effects of potassium channel blocks on the spiking regularity

In the above subsection, we have found that sodium channel blocks could affect the spiking regularity of the clustered neuronal network. In what follows, we move to study the effects of potassium channel blocks accordingly. Similarly as above, we give the dependence of R on x_K for various x_{Na} (here x_{Na} is taken to be larger than 0.8 to ensure that the neurons could generate spikes), as shown in Figure 3.

Compared with Figure 1, we can clearly see that potassium channel blocks have different effects on the neuronal network's spiking regularity. The difference from the effects of sodium channel blocks is that there exists some intermediate working fraction of potassium channels x_K at which the spiking regularity of the neuronal network could reach a higher level. For x_{Na} = 1.0, the corresponding spatiotemporal patterns for various x_K are exhibited in Figure 4.

From this figure, we can see that the inter-spike intervals for each neuron inside the neuronal network for x_K = 0.4 are more regular than other cases. Thus, potassium non-blocked channel fraction x_K could induce coherence resonance behavior against spiking regularity in the clustered neuronal network, as shown by Figures 3 and 4.

4 Conclusions

In order to conclude the study, we further extend our results

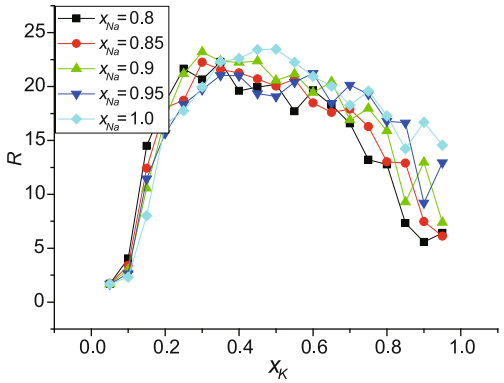

Figure 3 Variation of R as a function of x_K for various x_{Na} with M=2.

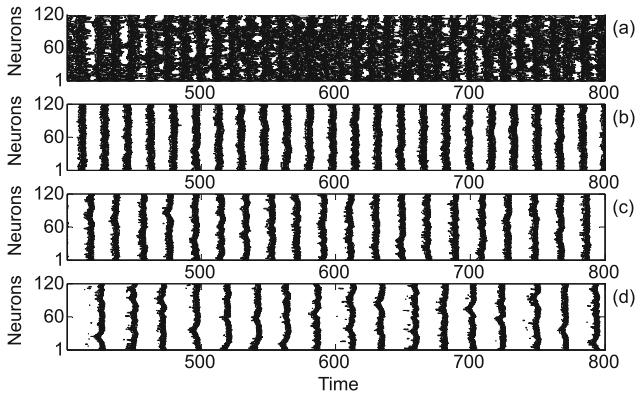

Figure 4 Spatiotemporal plots of membrane potentials of all neurons for different values of x_K by fixing x_{Na} = 1.0. x_K is taken as 0.1, 0.4, 0.7, 1.0 from (a) to (d).

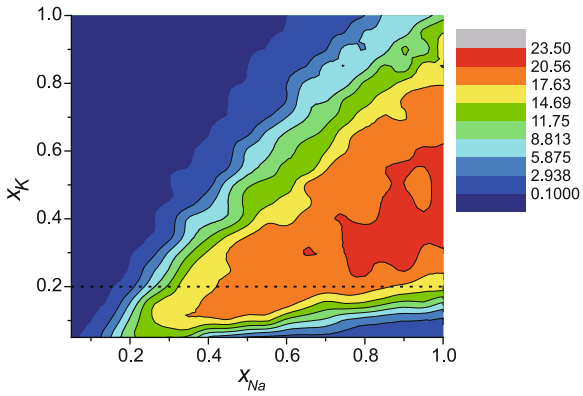

Figure 5 Contour plot of R as functions of non-blocked sodium channel fraction x_{Na} and non-blocked potassium channel fraction x_K for M=2. (colored online).

by simulating variations of R with respect to x_{Na} and x_K in a plan-plane for M = 2, as exhibited in Figure 5.

When we fixed the value of x_{Na} (x_{Na} >0.2), R increases and reaches to some maximum value and then decreases with x_K. Then, there exist some optimal values of x_K, at which the neuronal network exhibits higher spiking regularity (or R takes larger values) for various values of x_{Na}, as shown in Figure 5. Meanwhile, for most values of x_K (x_K is larger than 0.2, above the dashed line in Figure 5), the color changes from blue to red with increasing of x_{Na}, as exhibited in Figure 5. This indicates that the spiking regularity measurement R increases with x_{Na}. Then, we can draw the conclusion that non-blocked sodium channel fraction x_{Na} could enhance the spiking regularity when x_K is larger than 0.2. While for x_K < 0.2, there exists some optimal value of x_{Na}, where the spiking regularity of the neuronal network is relatively high. This means that, to some extent the effects of sodium channel blocks on spiking regularity are dependent on the number of blocking potassium channel blocks. With the further extension of the above obtained results to the multi-clustered neuronal network, we can observe the similar results as obtained when the neuronal network just has two clusters, as expressed in Figure 6.

In the former work, Schmid et al. [34,35] studied the spiking regularity of a stochastic HH model at a single neuron level, and showed that x_{Na} could enhance the regularity of spontaneous spike trains and x_K decrease it. After that, Gong et al. [25], Ozer et al. [26] and Yilmaz et al. [27] extended Schmid's work by discussing the effects of channel blocks on the regularity of spiking activities at a single neuronal network level, such as array-coupled, Newman-Watts and scale-free networks, respectively. They found that, similar as in single neuronal model, non-blocked sodium and potassium factions x_{Na} and x_K could increase or decrease the regularity of single neuronal networks. In our work, we further extended the former work to the clustered neuronal networks. With the obtained results, we can give a conclusion that the phenomenon-sodium and potassium channel blocks could increase or decrease the spiking regularity-can begenerally observed in neuronal systems.

In sum, the spiking regularity of the clustered neuronal network could be resonantly enhanced via fine-tuning of the non-blocked potassium channel fraction x_K. For the non-blocked sodium channel fraction x_{Na}, it could also enhance the spiking regularity resonantly when x_K takes smaller values (x_K <2.0 as shown in Figures 5 and 6). While in most cases, the non-blocked sodium channel fraction x_{Na} enhances the spiking regularity of the clustered neuronal network. Moreover, temporal coding as one of the coding rules is correlated with the spike timings. Thus, regularity of spike timings has close relationship with neuronal information coding. As reported in ref. [39], spiking regularity may also relate to transduction of information processing for neuronal activity. Therefore, the regularity of spike timings in a neuronal network may play an important role not only in information coding process but also in transmission process. Because ion channel blocks could affect the strength of ion channel noises, we hope that our results could give some implications for understanding the role of randomness in neuronal systems.

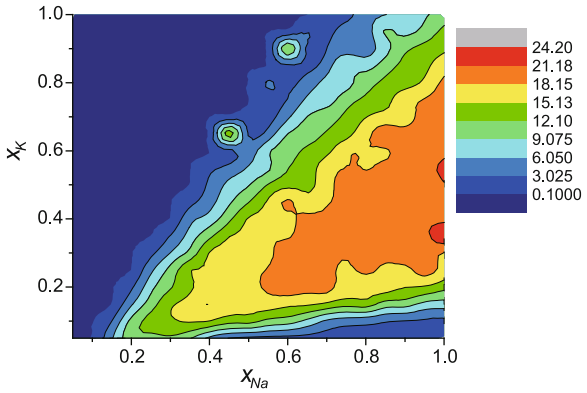

Figure 6 Contour plot of R as functions of non-blocked sodium channel fraction x_{Na} and non-blocked potassium channel fraction x_K for M=5. (colored online).

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1 Gerstner W, Kistler W M. Spiking Neuron Models. Cambridge: Cambridge Univ Press, 2002
2 Bullmore E, Sporns O. Complex brain networks: graph theoretical analysis of structural and functional systems. Nat Rec Neurosci, 2009, 10: 186–198
3 Lord G, Laing C. Stochastic Methods in Neuroscience. Oxford: Oxford Univ Press, 2010
4 Faisal A A, Selen L P J, Wolpert D M. Noise in nervous system. Nature Neurosci, 2008, 9: 292–303
5 Freeman W J. Deep analysis of perception through dynamic structures that emerge in cortical activity from self-regulated noise. Cognitive Neurodynamics, 2009, 3: 105–116
6 Lindner B, Garcia-Ojalvo J, Neiman A, et al. Effects of noise in excitable systems. Physics Reports, 2004, 392: 321–424
7 Lang X F, Lu Q S, Kurths J. Phase synchronization in noise-driven bursting neuron. Phys Rev E, 2010, 82: 021909
8 Richardson M J E, Swarbrick R. Fring-rate response of a neuron receiving excitatory and inhibitory synaptic shot noise. Phys Rev Letts, 2010, 105: 178102
9 Zhou C S, Kurths J. Noise-induced phase synchronization and synchronization transitions in chaotic oscillators. Phys Rev Letts, 2002, 88: 230602
10 Kang Y M, Xu J X, Xie Y. Signal-to-noise ratio gain of a noisy neuron that transmits subthreshold periodic spike trains. Phys Rev E, 2005, 72: 021902
11 Wu Y, Xu J X, He D H, et al. Generalized synchronization induced by noise and parameter mismatching in Hindmarsh-Rose neurons. Chaos, Solitons Fractals, 2005, 23: 1605–1611
12 Wang Q Y, Perc M, Duan Z S, et al. Dealy-induced multiple stochastic resonance on scale-free neuronal networks. Chaos, 2009, 19: 023112
13 Sun X J, Lu Q S, Perc M, et al. Effects of correlated Gaussian noise on the mean firing rate and correlations of an electrically coupled neuronal network. Chaos, 2010, 20: 033116
14 Sun X J, Perc M, Lu Q S, et al. Spatial coherence resonance on diffusive and small-world networks of hodgkin-huxley neurons. Chaos, 2008, 18: 023102
15 Guo D Q, Li C G. Population rate coding in recurrent neuronal networks with unreliable synapses. Cognitive Neurodynamics, 2012, 6: 75–87
16 White J A, Rubinstein J T, Kay A R. Channel noise in neurons. Trends Neurosci, 2000, 23: 131–137
17 Goldwyn J H, Shea-Brown E. The what and where of adding channel noise to the Hodgkin-Huxley equations. PLos Comput Biol, 2011, 7: e1002247
18 Imennov N S, Rubinstein J T. Stochastic population model for electrical stimulation of the auditory nerve. IEEE Trans Biomed Eng, 2009, 56: 2493–2501
19 White J A, Klink R, Alonso A, et al. Noise from voltage-gated ion channels may influence neuronal dynamics in the entorhinal cortex. J Neurophysiol, 1998, 80: 262–269
20 Cannon R C, O'Donnell C, Nolan M F. Stochastic ion channel gating in dendritic neurons: morphology dependence and probabilistic synaptic activation of dendritic spikes. PLos Comput Biol, 2010, 6: e1000886
21 Schmid G, Goychuk I, Hänggi. Stochastic resonance as a collective property of ion channel assemblies. Europhys Lett, 2001, 56: 22
22 Rowat P R, Elson R C. State-dependent of Na channel noise on neuronal burst generation. Comput Neurosci, 2004, 16: 87–112
23 Ginzburg S L, Pustovoit M A. Bursting dynamics of a model neuron induced by intrinsic channel noise. Fluct Noise Lett, 2003, 3: L265
24 Yu L C, Chen Y, Zhang P. Frequency and phase synchronization of two coupled neurons with channel noise. Eur Phys J B, 2007, 59: 249–257
25 Gong Y B, Xu B, Ma X G, et al, Effect of channel block on the collective spiking activity of coupled stochastic Hodgkin-Huxley neurons. Sci China Chem Sci, 2008, 51: 341–346
26 Ozer M, Perc M, Uzuntarla M. Controlling the spontaneous spiking regularity via channel blocking on Newman-Watts networks of Hodgkin-Huxley neurons. Europhys Lett, 2009, 86: 40008
27 Yilmaz E, Ozer M, Cavusoglu A. Effects of channel blocking on the spontaneous firing regularity of scale-free Hodgkin-Huxley neuronal network. International Symposium on Innovations in Intelligent Systems and Applications, 2011. 581–584
28 Sun X J, Lei J Z, Perc M, et al. Effects of channel noise on firing coherence of small-world Hodgkin-Huxley neuronal networks. Eur Phys J B, 2011, 79: 61–66
29 Ma J, Huang L, Tang J, et al. Spiral wave death, breakup induced by ion channel poisoning on regular Hodgkin-Huxley neuronal network. Commun. Nonlinear Sci Numer Simulat, 2012, 17: 4281–4293
30 Diba K, Koch C, Segev I. Spike propagation in dendrites with stochastic ion channels. J Comput Neurosci, 2006, 20: 77–84
31 Rieke F, Warland D, Van Steveninck R R, et al. Spikes: Exploring the Neural Code. Cambridge: MIT Press, 1997
32 Du Y, Lu Q S, Wang R B. Using interspike intervals to quantify noise effects on spike trains in temperature encoding neurons. Cognitive Neurodynamics, 2010, 4: 199–206
33 Gu H G, Jia B, Lu Q S. Exponential decay characteristics of the stochastic integer multiple neural firing patterns. Cognitive Neurodynamics, 2011, 5: 87–101
34 Schmid G, Goychuk I, Hänggi P. Effect of channel block on the spiking activity of excitable membranes in a stochastic Hodgkin-Huxley model. Phys Biol, 2004, 1: 61–66
35 Schmid G, Goychuk I, Hänggi P. Controlling the spiking activity in excitable membranes via poisoning. Phys A, 2004, 344: 665–670
36 Hodgkin A L, Huxley A F. A quantitative description of membrane current and its application to conduction and excitation in nerve. J Physiol, 1952, 117: 500–544
37 Fox R, Lu Y. Emergent collective behavior in large numbers of globally coupled independently stochastic ion channels. Phys Rev E, 1994, 49: 3421–3431
38 Fox R. Stochastic versions of the Hodgkin-Huxley equations. Biophys J, 1997, 72: 2068–2074
39 Zhou J, Qu Q J, Lan X. Impulsive pinning complex dynamical networks and applications to firing neuronal synchronization. Nonlinear Dyn, 2012, 69: 1393–1403