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Bursting synchronization in scale-free networks

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ABSTRACT

Neuronal networks in some areas of the brain cortex present the scale-free property, i.e., the neuron connectivity is distributed according to a power-law, such that neurons are more likely to couple with other already well-connected ones. Neuron activity presents two timescales, a fast one related to action-potential spiking, and a slow timescale in which bursting takes place. Some pathological conditions are related with the synchronization of the bursting activity in a weak sense, meaning the adjustment of the bursting phase due to coupling. Hence it has been proposed that an externally applied time-periodic signal be applied in order to control undesirable synchronized bursting rhythms. We investigated this kind of intervention using a two-dimensional map to describe neurons with spiking–bursting activity in a scale-free network.

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1. Introduction

Neuronal activity (i.e., the evolution of the action potential) in some areas of the brain cortex presents a fast time scale characterized by repetitive spiking and a slow timescale with bursting activity, such that neuron activity alternates between a quiescent state and spiking trains [1]. There are a number of mathematical models which emulate this spiking–bursting behavior, ranging from differential equations [3] to discrete-time maps [2,4]. Interacting bursting neurons can exhibit basically two types of common rhythmic bursting: synchronization of bursts, where the neurons burst at the same time, regardless of the further evolution of their spikes; and complete synchronization, which involves also the synchronization of spikes [1]. There follows that burst synchronization is weaker than complete synchronization and thus easier to achieve, in terms of the coupling strength needed. The existence of a slow timescale in coupled bursting neurons enables us to define a bursting phase and frequency (its time rate) for each of them, even though they may behave asynchronously in the spiking time scale [5].

Clinical evidences point out that the synchronization of individual neurons plays a key role in some pathological conditions like Parkinson's disease, essential tremor, and epilepsies [6]. Hence it has been proposed the use of an additive time-periodic external signal of small amplitude and given frequency in order to control undesirable neuronal rhythms [5,7]. This intervention is carried out experimentally by means of micro-electrodes inserted into the impaired region of the brain and carrying a suitable electric signal [8]. From the mathematical point of view this problem consists of the bursting synchronization of neurons with an external source, and one would like to know the parameter ranges for which phase synchronization occurs or does not occur. Such a study has been made in globally coupled maps [5] and also for power-law couplings [9].

Recent experiments show that some brain activities can be assigned to scale-free networks, as revealed by functional magnetic resonance imaging [10]. In scale-free networks the number of connections *per* neuron satisfies a power-law probability distribution, such that highly connected neurons are connected, on the average, with highly connected ones, a property also found in many social and computer networks [11,12]. This neuronal architecture is consistent with the fact that the

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brain network increases its size by the addition of new neurons, and the latter attach preferentially to already well-connected neurons [13]. There are many recent investigations of synchronization of several kinds in scale-free networks [14].

We have previously studied the onset of phase synchronization of bursting neurons modelled by a network of coupled two-dimensional maps exhibiting the scale-free property [15], with a power-law connectivity with an exponent compatible with the networks described by Chialvo and coworkers [10]. In this paper, we investigate the control of bursting synchronization by using a time-periodic external signal in a scale-free neuronal network. Our main goal is to derive conditions by which we can determine the parameter values for which this intervention is feasible. These parameters are the amplitude and frequency of the external signal and the number of connections for the neuron which receive such an excitation. The overall behavior resembles that exhibited by a chaotic oscillator subjected to a time-periodic forcing, for there are Arnold-tongue structures in the parameter space which represent phase-locking. These structures are parameter intervals such that neurons burst synchronously with the frequency of the driving signal.

This paper is organized as follows: in Section 2, we present our model for Rulkov neurons in a scale-free neural network. Section 3 deals with the control of synchronized bursting rhythms by application of an external time-periodic driving. Our conclusions are left to the last Section.

2. Bursting neurons in a scale-free network

We describe neurons possessing two timescales, for there is a slow bursting modulating the fast action-potential spiking [16]. A simple model which presents such behavior is the Rulkov map [4]

$$x_{n+1} = \frac{\alpha}{1+x_n^2} + y_n, \tag{1}$$

$$y_{n+1} = y_n - \sigma x_n - \beta, \tag{2}$$

where x_n is the fast and y_n is the slow dynamical variable. The first variable has a dynamical behavior emulating the spiking–bursting activity of a neuron, depending on the parameter α, whereas the latter variable undergoes a slow evolution because of the small values taken on by the parameters σ and β, which model the action of external dc bias current and synaptic inputs on a given isolated neuron [17]. Since there is always some diversity in the parameters describing each neuron, we choose α randomly within a certain range which yields chaotic behavior for the characteristic spiking of the fast variable x_n, and which turns out to be the interval [4.1,4.2].

We consider a given burst to begin when the slow variable y_n, which presents nearly regular saw-teeth oscillations, has a local maximum, in well-defined instants of time we call n_k. We can define a phase describing the time evolution within each burst and varying from 0 to 2π as n evolves from n_k to n_{k+1}:

$$\varphi(n) = 2\pi k + 2\pi \frac{n-n_k}{n_{k+1}-n_k}. \tag{3}$$

The duration of the chaotic burst, $n_{k+1}-n_k$, depends on the variable x_n and fluctuates in an irregular fashion when x_n undergoes chaotic evolution. There follows that the bursting phase rate also varies with time, such that we must look at the bursting frequency defined by

$$\Omega = \lim_{n\to\infty} \frac{\varphi(n)-\varphi(0)}{n}. \tag{4}$$

When the Euclidean distance between neurons does not play a significant role, the corresponding networks may be treated from the graph-theoretical point of view [18]. However, once we regard those neurons as embedded in a three-dimensional lattice (the brain, where they are connected by axons and dendrites), it is convenient to use a lattice embedded in a Euclidean space [19]. Considering an assembly of N neurons, each of them being described by the map (1)–(2), in a one-dimensional lattice such that $(x_n^{(i)}, y_n^{(i)})$ represent the fast and slow variables for the neuron i $(i = 1,2,\ldots N)$ at time n:

$$x_{n+1}^{(i)} = \frac{\alpha^{(i)}}{1+\left(x_n^{(i)}\right)^2} + y_n^{(i)} + \mathscr{C}^{(i)}(x_n^{(j)}, y_n^{(j)}), \quad (j \neq i), \tag{5}$$

$$y_{n+1}^{(i)} = y_n^{(i)} - \sigma^{(i)} x_n^{(i)} - \beta^{(i)}, \tag{6}$$

where we consider the case where all map parameters can be different for each site, and the coupling is performed only on the fast time scale by means of the term $\mathscr{C}^{(i)}$, the form of which depends on the network topology chosen to describe the neural network. We use free boundary conditions for the lattice and random initial conditions $x_0^{(i)}$.

We use the Barabási–Albert prescription to generate a scale-free lattice, for which the coupling term is

$$\mathscr{C}^{(i)}(x_n^{(i)}) = \frac{\epsilon}{k^{(i)}} \sum_{j\in I} x_n^{(j)}, \tag{7}$$

where $\epsilon > 0$ is the coupling strength and we assumed that each site i is coupled with a set I comprising $k^{(i)}$ other sites randomly chosen along the lattice. We obtained (7) from a sequence of steps $s = 0,1,2,\ldots s_{\max}$, starting from an initial lattice

with $N_0 = 11$ sites. At each step s a new site is inserted in the lattice of size N_s, such that it is connected to $\ell \geqslant 2$ randomly chosen sites. In a scale-free network the connections occur preferentially with the more connected sites, what can be accomplished by using a different probability for each site $P_s^{(i)} = k_s^{(i)}/N_s$, where $k_s^{(i)}$ is the number of connections *per* site at the step s. The process is repeated until we achieve a desired lattice size N, which we choose as $N = 230$ in the numerical simulations to be presented in this paper. After a number $s_{\max}$ of steps we have $k^{(i)}$ connections *per* site, corresponding to a probability $P^{(i)} = k^{(i)}/N$. Fig. 1 shows a histogram for the number of sites with connectivity k, obtained through this procedure for $N = 230$ sites, and yielding a power-law dependence $k^{-\varpi}$ with a slope $\varpi = 2.08$, which compares well with the experimental values reported in Ref. [10].

The effect of coupling in the bursting synchronization is illustrated by Fig. 2, where we plot time series of the fast variable for two distinct sites in the coupled map lattice. When the coupling is absent, neurons in the lattice burst in a non-coherent fashion [Fig. 2(a) and (c)]. For $\epsilon \neq 0$ the bursting phases approximately coincide, corresponding to bursting synchronization [Fig. 2(b) and (d)]. If the oscillator phases are synchronized, so are their time rates, hence we can characterize bursting synchronization by the existence of frequency plateaus. This can be done, by comparing the frequency of the coupled neuron $\Omega^{(i)}$, given by Eq. (4), with the unperturbed frequencies observed for zero coupling, $\Omega_0^{(i)}$, the latter being expected to fluctuate

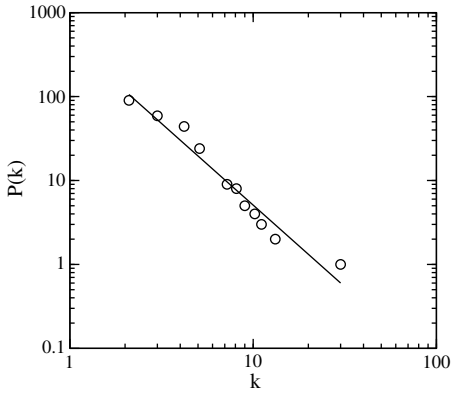

Fig. 1. Probability distribution for the connectivity of the final scale-free lattice with $N = 230$ sites. The solid line is a least-squares fit with slope -2.08.

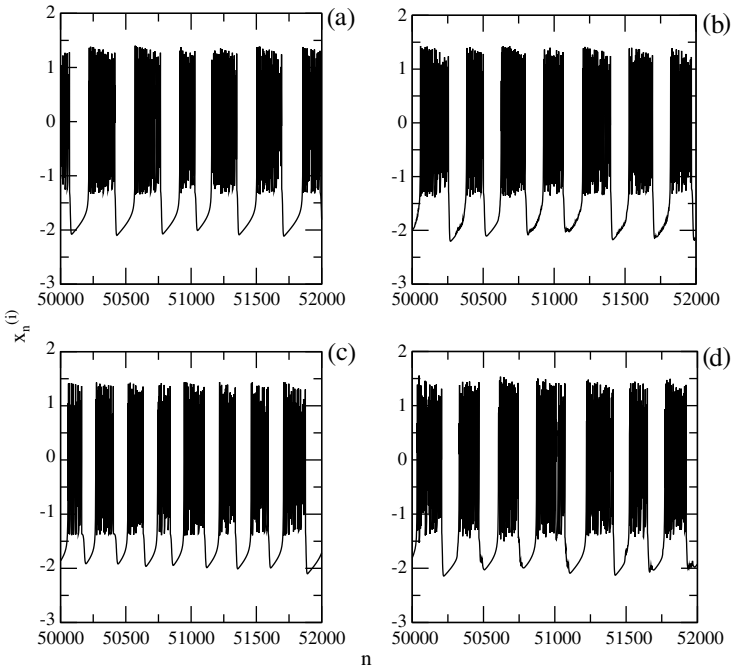

Fig. 2. Time evolution of the fast variable for two selected maps ($\alpha^{(i)} = 4.1$ and $\alpha^{(j)} = 4.2$) belonging to a scale-free lattice of Rulkov neurons with $\epsilon = 0$ [(a) and (c)] and $\epsilon = 0.04$ [(b) and (d)].

randomly. Fig. 3(a) shows such a plateau for large coupling strength. As the latter is decreased the frequency distribution approaches its zero-coupling limit $\Omega^{(i)} \approx \Omega_0^{(i)}$.

3. Control of synchronized bursting by an external signal

Given the existence of a synchronized bursting state we are interested in ways to suppress this state by application of a time-periodic external signal. Initially, we consider such intervention to occur at only one selected neuron $i = S$ (the remaining neurons remaining unchanged), to which a term $d\sin(\omega n)$ is applied, with amplitude and frequency given by d and ω, respectively. We have used coupling strength values for which the unperturbed lattice ($d = 0$) exhibits bursting synchronization, their corresponding frequencies $\Omega^{(i)}$ locking approximately at a common value, as depicted in Fig. 4(a), where the frequency mismatch $\Omega^{(i)} - \omega$ is plotted against ω for all neurons belonging to a given lattice. They exhibit a common locking region $[\omega_1, \omega_2]$ with width $\Delta\omega = \omega_2 - \omega_1$. The unperturbed frequency ω_0, however, is not located at the middle of the phase-locking interval, as it would seem at first. It is located nearer to the left end of the interval, so that we also consider the distance $\delta\omega = \omega_2 - \omega_0$.

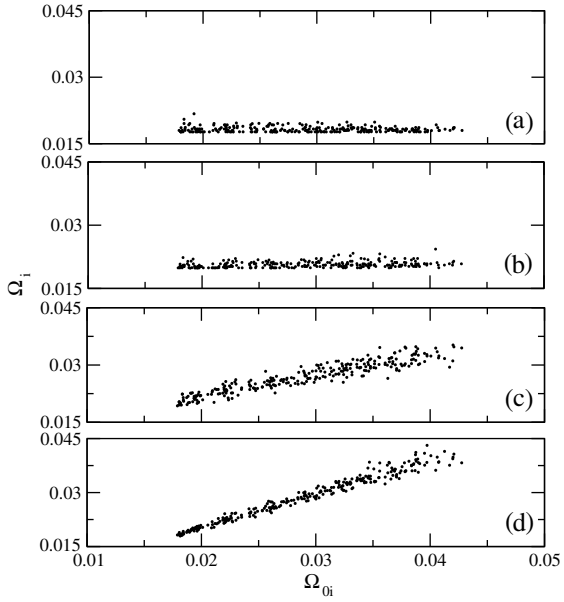

Fig. 3. Bursting frequency for the scale-free lattice *versus* the zero-coupling frequencies for (a) $\epsilon = 0.10$; (b) $\epsilon = 0.07$; (c) $\epsilon = 0.03$; and (d) $\epsilon = 0.01$.

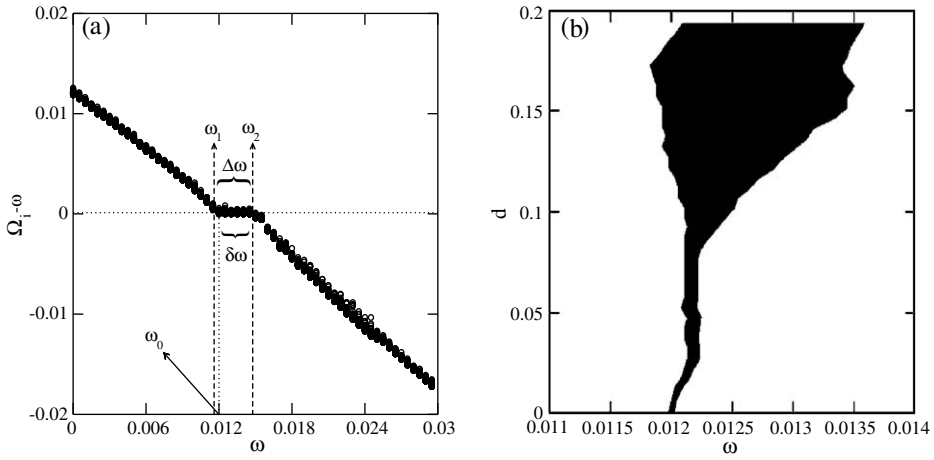

Fig. 4. (a) Frequency mismatch of bursting neurons *versus* the external driving frequency for a scale-free lattice. (b) Mode-locking tongue in the amplitude *versus* frequency of the driving signal applied at the site $S = 1$ with amplitude $d = 0.05$.

The frequency-locking interval shown in Fig. 4 is actually a cross-section of an Arnold-like tongue in the parameter plane amplitude *versus* frequency of the external driving signal [Fig. 4(b)]. This tongue-like structure characterizes a region where bursting synchronized is maintained, even with the external driving. Hence, the desirable parameter choices would be those outside this tongue. Notice that the latter is greatly asymmetric, specially for large signal amplitudes, as illustrated by Fig. 5, where we plot the distance $\delta\omega$ as a function of the driving amplitude. This is a beneficial effect since we would like to keep outside a locking region without increasing too much the amplitude of the signal, what would damage a biological neuron.

The asymmetry of the mode-locking tongue means that, if a frequency of the driving signal is smaller than of the unperturbed network, we would expect a minute effect in terms of synchronization. As a consequence, driving frequencies higher than the autonomous frequency of the lattice yield better results in terms of synchronization. A nice qualitative explanation for the asymmetry of the mode-locking tongues has been provided by Ivanchenko et al. [5]. An imposed positive signal precipitates a burst into a quiescent regime, and delays it when the signal is negative. If the driving frequency is higher than that of the mutually synchronized network, the periodic signal will fasten the oscillations of the neurons. On the other hand, when the driven neuron starts a burst, there is an abrupt change in the mean field perceived by all neurons in the lattice, pushing them to a quiescent state. As a result, higher frequencies would give better synchronization effects.

The wider the frequency-locking interval is, the more robust is the external driving with respect to imperfect parameter determination and noise, which is a question of considerable experimental importance. However, it may well happen that, if only one pinning (i.e., a driving applied on just one neuron) is considered, the corresponding locking interval would be too small so as to ensure robustness against imperfect parameter determination. For example, if the frequency of the pinning is known up to an uncertainty comparable to the island width, suppression of bursting would be non-reliable. On the other hand, simply increasing the amplitude of a single pinning would not work too, since this would lead to saturation [see Fig. 5].

The latter problem is particularly severe when the lattice size increases, as illustrated by Fig. 6(a), where we plot the locking interval width $\Delta\omega$ as a function of the inverse lattice size. Since the connectivity of the lattice sites in a scale-free network presents a power-law statistics, we expect that most of the sites are poorly connected, with very few well-connected ones. Accordingly, in Fig. 6(a), we show the results when the driving signal is applied to the most connected site in the lattice (circles), and to the two next most connected sites (squares and triangles). As expected, the former would give larger mode-locking tongues than the latter, which represent poorly connected neurons. Moreover, for all the cases the tongue width decreases with $1/N$, meaning that the locking interval becomes too small for large networks, what would turn the intervention practically unfeasible.

One possible way to circumvent this problem is to use more than one site to control at the same time (multiple pinnings) [5]. It was shown that increasing the number of pinnings augments the width of the mode-locking tongue, as expected. However, in practice we do not know in advance what sites are the most connected ones, since such an intervention experiment would be conducted in a macroscopic piece of tissue with scarce information about the existing connections. Hence it is more useful to compare the effect of one pinning ($N_p = 1$) applied to the most connected site and the effect of multiple pinnings ($N_p = 10$) applied to randomly chosen sites. Our results are depicted in Fig. 6(b). In both cases the tongues dilate as the number of pinnings is augmented with minor differences, showing that the use of multiple randomly chosen pinnings produces the same synchronization effect than a single pinning applied to the most connected site.

4. Conclusions

In conclusion, the use of external intervention for suppressing bursting synchronization in a coupled Rulkov map lattice has many similarities with the phase synchronization of a chaotic oscillator with an external periodic source. Tongue-like

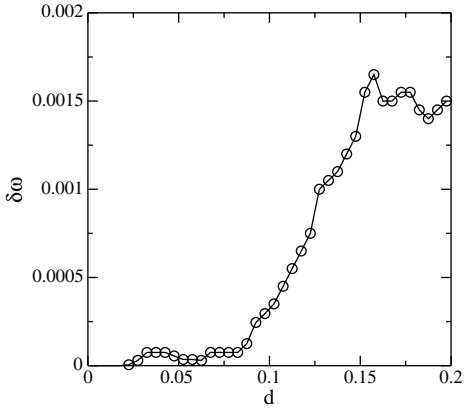

Fig. 5. Distance between the non-perturbed bursting frequency and the higher end of the mode-locking interval ω_2. The remaining parameters are the same as in Fig. 4.

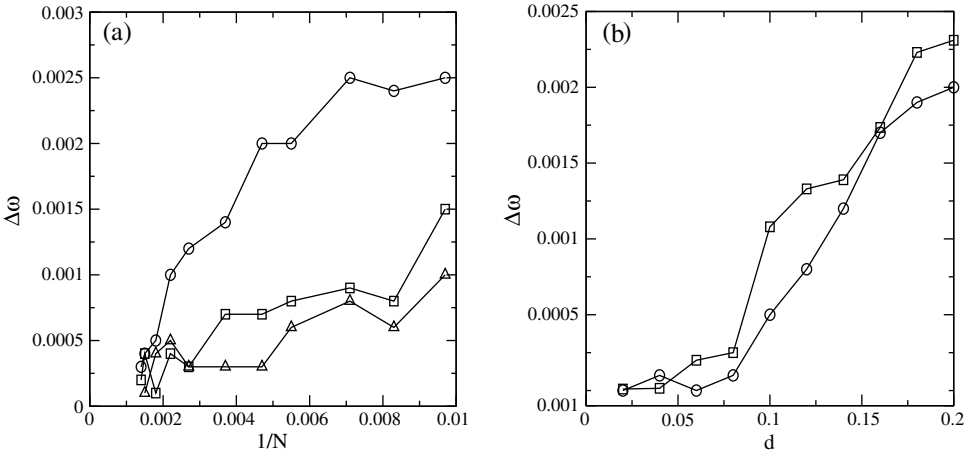

Fig. 6. (a) Width of the frequency-locking interval *versus* inverse lattice size for $N_P = 1$ driving signal, applied to the most connected site (circles), the second (squares) and the third (triangles) most connected sites, with amplitude $d = 0.20$. (b) Tongue width as a function of the driving amplitude for (a) $N_P = 1$ driving signal applied to the most connected site; (b) $N_P = 10$ driving signals applied to randomly chosen sites.

structures in the driving parameter space are obtained representing parameter values for which synchronization is maintained. Those tongues were shown to be greatly asymmetric but, as a general trend, their widths increase with the driving amplitude. In order to suppress bursting synchronization we must choose driving parameters outside these tongues. The tongues' widths decrease rapidly with the lattice size, such that larger widths would be obtained either by increasing the driving amplitude (a rather undesirable choice) or by using more than one external signal, or pinning. We numerically demonstrated that two factors should be taken into account in a practical intervention experiment: (i) the driving frequency must be higher than the autonomous bursting frequency of the lattice; and (ii) multiple driving signals (at least 10) should be used, since their average effect is similar to that of a single pinning applied to the most connected site.

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References

[1] Belykh I, de Lange E, Hasler M. Synchronization of bursting neurons: what matters in the network topology. Phys Rev Lett 2005;94:188101.
[2] Chialvo D. Generic excitable dynamics on a two-dimensional map. Chaos, Solitons & Fractals 1995;5:461.
[3] Hindmarsch JL, Rose RM. A model of neuronal bursting using three coupled first order differential equations. Philos Trans R Soc Lond B 1984;221:87.
[4] Rulkov NF. Regularization of synchronized chaotic bursts. Phys Rev Lett 2001;86:183.
[5] Ivanchenko MV, Osipov GV, Shalfeev VD, Kurths J. Phase synchronization in ensembles of bursting oscillators. Phys Rev Lett 2004;93:134101.
[6] Milton J, Jung P, editors. Epilepsy as a dynamic disease. Berlin: Springer-Verlag; 2003.
[7] Rosenblum M, Pikowsky A. Delayed feedback control of collective synchrony: an approach to suppression of pathological brain rhythms. Phys Rev E 2004;70:041904.
[8] Behabid A et al. Long-term suppression of tremor by chronic stimulation of the ventral intermediate thalamic nucleus. Lancet 1991;337:403.
[9] de Pontes JCA, Viana RL, Lopes SR, Batista CAS, Batista AM. Bursting synchronization in non-locally coupled networks. Phys A 2008;387:4417.
[10] Chialvo DR. Critical brain networks. Phys A 2004;340:756;
Sporns O, Chialvo DR, Kaiser M, Hilgetag CC. Organization, development and function of complex brain networks. Trends Cogn Sci 2004;8:418;
Eguiluz VM, Chialvo DR, Cecchi GA, Baliki M, Apkarian AV. Scale-free brain functional networks. Phys Rev Lett 2005;94:018102.
[11] Barabási A-L, Albert R. Emergence of scaling in random networks. Science 1999;286:509.
[12] Albert R, Barabási A-L. Statistical mechanics of complex networks. Rev Mod Phys 2002;74:47;
Dorogovtsev SN, Mendes JFF. Evolution of networks. Adv Phys 2002;51:1079.
[13] Bear MF, Connors BW, Paradiso MA. Neuroscience: exploring the brain. 2nd ed. Philadelphia: Lippincott Williams & Wilkins; 2002.
[14] He G, Yang J. Adaptive synchronization in nonlinearly coupled dynamical networks. Chaos, Solitons & Fractals 2008;38:1254;
Perc M. Effects of small-world connectivity on noise-induced temporal and spatial order in neural media. Chaos, Solitons & Fractals 2007;31:280;
Yu H, Peng J. Chaotic synchronization and control in nonlinear-coupled HindmarshRose neural systems. Chaos, Solitons & Fractals 2006;29:342;
Shiner JS, Davison M. Quantifying the connectivity of scale-free and biological networks. Chaos, Solitons & Fractals 2004;21:1;
Allegrini P, Grigolini P, Palatella L. Intermittency and scale-free networks: a dynamical model for human language complexity. Chaos, Solitons & Fractals 2004;20:95.
[15] Batista CAS, Batista AM, de Pontes JAC, Viana RL, Lopes SR. Chaotic phase synchronization in scale-free networks of bursting neurons. Phys Rev E 2007;76:16218.
[16] Dhamala M, Jirsa VK, Ding M. Transitions to synchrony in coupled bursting neurons. Phys Rev Lett 2004;92:028101.
[17] Rulkov NF. Modeling of spiking–bursting neural behavior using two-dimensional map. Phys Rev E 2002;65:041922;
Rulkov NF, Timofeev I, Bazhenov M. Oscillations in large-scale cortical networks: map-based model. J Comput Neurosci 2004;17:203.
[18] Dayan P, Abbott LF. Theoretical neuroscience: computational and mathematical modeling of neural systems. Cambridge: MIT Press; 2001.
[19] Rozenfeld AF, Cohen R, ben-Avraham D, Havlin S. Scale-free networks on lattices. Phys Rev Lett 2002;89:218701.