Bursting synchronization in clustered neuronal networks*

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(Received 4 February 2012; revised manuscript received 25 July 2012)

Neuronal networks in the brain exhibit the modular (clustered) property, i.e., they are composed of certain subnetworks with differential internal and external connectivity. We investigate bursting synchronization in a clustered neuronal network. A transition to mutual-phase synchronization takes place on the bursting time scale of coupled neurons, while on the spiking time scale, they behave asynchronously. This synchronization transition can be induced by the variations of inter- and intra-coupling strengths, as well as the probability of random links between different subnetworks. Considering that some pathological conditions are related with the synchronization of bursting neurons in the brain, we analyze the control of bursting synchronization by using a time-periodic external signal in the clustered neuronal network. Simulation results show a frequency locking tongue in the driving parameter plane, where bursting synchronization is maintained, even in the presence of external driving. Hence, effective synchronization suppression can be realized with the driving parameters outside the frequency locking region.

Keywords: bursting synchronization, neuronal network, cluster, external signal

PACS: 87.19.lj, 05.45.Xt **DOI:** 10.1088/1674-1056/22/1/018701

1. Introduction

The dynamics of complex networks has attracted much attention in recent years. Synchronization, as an emerging phenomenon of a population of dynamically interacting units, has been widely studied for its important role in many different fields such as biology, ecology, and sociology.[1–3] Recently, increasing evidences have been found to support that synchronization is especially relevant to a variety of neural phenomena.[4–7] Synchronization of bursting neurons includes the synchrony on the spiking time scale (spike synchronization) and synchrony on the bursting time scale (burst synchronization).[8] Previous works on the global coupling,[9] small-world,[10] and scale-free networks[11,12] showed that the transition to mutual-phase synchronization in bursting neurons occurs as the coupling strength is large enough. However, how the transition to synchronized bursting depends on the network properties such as the coupling topology and strength, especially for large-scale neuron populations with modular structure is an issue to be addressed.

Latest developments in the quantitative analysis of complex networks demonstrate that the brain's structural and functional systems share several important organizational principles in common, such as the property of modularity, and small-world networks.[13–16] A module, also called a cluster, is topologically defined as a subset of highly inter-connected nodes which are relatively sparsely connected to nodes in other modules.[14] Analysis of the connectivity between regions of the cerebral cortex in macaque monkeys and cats has revealed that cortical neuronal networks are modular,[17–19] i.e., are composed of certain subnetworks with differential internal and external connectivity. The organization of cortical areas into clusters permits the segregated processing of information of different modalities. Moreover, neuronal networks in some areas of the brain show a small-world property,[20] characterized by a relatively short minimum path length together with a high clustering coefficient.[21] Small-world networks turn out to be widespread in neural systems, often with important dynamical consequences. Recently, small-world architectures have been found in several empirical studies of structural and functional brain networks in humans and other animals.[22–24] Experimental studies also suggested that a small-world topology may be a general organization principle of the human brain.[25,26] Models of neural systems with small-world coupling display enhanced signal-propagation speed, computational power, and synchronizability.[27]

As is well known, the interplay between the intrinsic dynamics of individual neurons, the properties of the synaptic coupling, and their complex connection topology strongly affects the collective dynamics of neuronal networks.[28–35] Each property may play an important role in shaping the emergence of synchronous behavior. Therefore, modeling each node by a neuronal system and investigating bursting synchronization in clustered networks can extend the interest of the work on the understanding of intrinsic dynamics in biological neural systems.

In order to explore this, in this paper we study burst-

*Project supported by the National Natural Science Foundation of China (Grant Nos. 61072012, 61104032, and 61172009), the Natural Science Foundation of Tianjin Municipality, China (Grant No. 12JCZDJC21100), and the Young Scientists Fund of the National Natural Science Foundation of China (Grant Nos. 60901035 and 50907044).

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ing synchronization in a clustered neuronal network, and each cluster is modelled by a subnetwork of coupled map-based neurons[36] exhibiting small-world property. We will investigate its transition to phase synchronization with the variation of network parameters. Moreover, we will analyze the control of bursting synchronization in the clustered neuronal network by using a time-periodic external signal, which has been proposed as an electrical stimulation of the brain to suppress undesirable synchronous rhythms related to pathological conditions, such as epilepsy, essential tremor, and Parkinson's disease.[9,37,38] We aim to derive the parameter values for which this intervention is feasible, i.e., amplitude and frequency of the external signal.

The remainder of this paper is organized as follows. In Section 2, we describe the mathematical model of the clustered neuronal network. Section 3 is devoted to investigate the transition to bursting synchronization with the variation of coupling parameters. In Section 4, we analyze the control of bursting synchronization in a clustered neuronal network with a time-periodic external signal applied to one arbitrarily taken neuron. Finally, a brief conclusion is given in Section 5.

2. Model and measurement

2.1. Network model

A clustered neuronal network can be generated as follows. We assume that there are M subnetworks (clusters), each of which consists of N_I $(I = 1,2,\ldots,M)$ neurons. We randomly choose some pairs of neurons from the i-th and j-th subnetwork ($i = 1,2,\ldots,M$, $j = 1,2,\ldots,M$, and $i \neq j$) and then add links between them. By doing so, a clustered network is generated. For simplicity, we assume that each subnetwork contains equal number of neurons, i.e., $N_I = N$ is independent of the index I, and its topology structure exhibits the small-world property. The interconnections between any two subnetworks exist randomly with a probability P. An example of the considered network topology is shown in Fig. 1. This structure is quite different from previously studied hub-connected modular networks,[39] in which one subnetwork is taken as the hub module which directly controls all other subnetworks and there is no interconnection between non-hub modules.

To construct a small-world subnetwork, we follow the random rewiring procedure proposed by Watts and Strogatz.[21] Starting from a ring-like network with regular connectivity, where each node is connected to its K nearest neighbors, we rewire each edge at random with a probability of p. By increasing the probability p the architecture of the network is tuned between two extremes: regular ($p = 0$) and random ($p = 1$) networks. Small-world networks are characterized by intermediate value of the probability $0 < p < 1$.

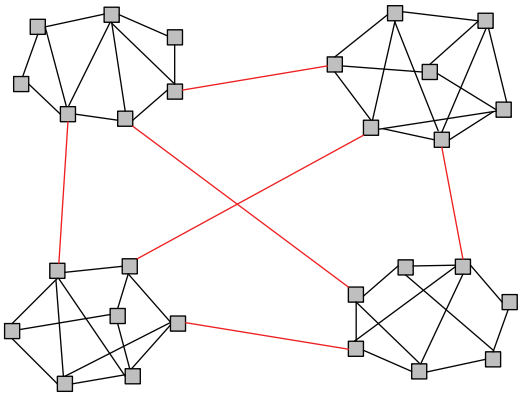


Fig. 1. (color online) Schematic presentation of the considered network architecture. The whole network consists of M= 4 subnetworks, each containing N= 7 neurons.

The map-based neuron model proposed by Rulkov *et al.*,[36,40,41] despite of its intrinsic simplicity and low dimensionality, can capture the main dynamical features of biological neurons,[42] but at essentially lower computational costs, which thus allows detailed analysis of the dynamics of large ensembles. In the present study, we use the two-dimensional map to simulate the dynamics of individual neuron in the network. Then, the temporal evolution of each unit can be described by the following set of discrete equations:

$$x_{I,i}(n+1) = \frac{\alpha_{I,i}}{1+x_{I,i}^2(n)} + y_{I,i}(n) + I_{I,i}^{\mathrm{syn}}(n), \tag{1}$$

$$y_{I,i}(n+1) = y_{I,i}(n) - \sigma x_{I,i}(n) - \beta, \tag{2}$$

where (I,i) represents the i-th neuron in the I-th subnetwork ($i = 1,2,\ldots,N$, and I= $1,2,\ldots,M$), and x and y are the fast and slow dynamical variables of the map, respectively. The first variable can emulate the spiking–bursting activity of a neuron, depending on the value of parameter α, whereas the latter variable undergoes a slow temporal evolution due to small value of the parameters σ and β. If the parameter α is selected within the range [4.1, 4.4], the fast variable x of the map-based neuron displays chaotic bursting behavior. The $I_{i,n}^{\mathrm{syn}}(n)$ is the coupling term, which has the following form:

$$\begin{aligned} I_{I,i}^{\mathrm{syn}}(n) \;=\; & \varepsilon_{\mathrm{intra}} \sum_j A_I(i,j)(x_{I,j}(n) - x_{I,i}(n)) \\ & + \varepsilon_{\mathrm{inter}} \sum_J \sum_j B_{I,J}(i,j)(x_{J,j}(n) - x_{I,i}(n)), \end{aligned} \tag{3}$$

where $\varepsilon_{\mathrm{intra}}$ is the coupling strength among neurons inside each subnetwork, and $\varepsilon_{\mathrm{inter}}$ is the coupling strength of neurons between different subnetworks. The matrix $\boldsymbol{A}_I = (A_I(i,j))$ is an intraconnectivity matrix for the I-th subnetwork: $A_I(i,j) = A_I(j,i) = 1$ if neuron i is connected to neuron j inside the I-th subnetwork, $A_I(i,j) = A_I(j,i) = 0$ otherwise, and $A_I(i,i) = 0$. The matrix $B_{I,J} = (B_{I,J}(i,j))$ is also a connectivity matrix, but it represents the interconnections between neurons belonging to different subnetworks: $B_{I,J}(i,j) = B_{J,I}(j,i) = 1$ if the i-th

neuron in the I-th subnetwork is connected to the j-th neuron in the J-th subnetwork, and $B_{I,J}(i,j) = B_{J,I}(j,i) = 0$ otherwise.

2.2. Measurement

In order to quantitatively characterize the synchronization degree of bursting neurons in the clustered network, we calculate the order parameter R which is defined as[9,11,43]

$$R = \frac{1}{N}\left|\sum_{j=1}^{N} \exp[\mathrm{i}\phi_j(n)]\right|, \tag{4}$$

where $\phi_j(n)$ is the burst phase for the j-th neuron at time n, and can be expressed as

$$\phi_j(n) = 2\pi k + 2\pi\frac{n - n_{j,k}}{n_{j,k+1} - n_{j,k}}, \quad (n_{j,k} < n < n_{j,k+1}), \tag{5}$$

where $n_{j,k}$ is the moment at which the k-th burst of the j-th neuron starts ($j = 1,2,\ldots,N$). The R is zero for weak correlation and tends to be unity for a full burst synchronization state. Larger R means higher degree of bursting synchronization in the neuronal network.

3. Bursting synchronization in clustered neuronal networks

In this section, we study bursting synchronization in a clustered network with $M = 4$ subnetworks, as shown in Fig. 1. Each subnetwork is composed of $N = 100$ map-based neurons and obtained from a regular ring ($K = 6$) with random rewiring probability p. We suppose $\alpha_{I,i}$ randomly and uniformly distributes in [4.1, 4.4] and $\sigma = \beta = 0.001$, so that each uncoupled neuron produces chaotic bursts. In the following, we will take the inter- and intra-coupling strength $\varepsilon_{\mathrm{inter}}$ and $\varepsilon_{\mathrm{intra}}$, as well as the probability P of random links between different subnetworks as control parameters, and investigate their different effects on the bursting synchronization of the clustered neuronal network.

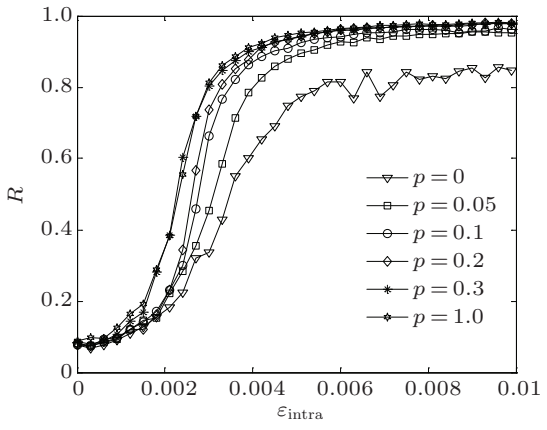


Fig. 2. Variations of the order parameter R with respect to $\varepsilon_{\mathrm{intra}}$ for different values of p. Other parameters: $\varepsilon_{\mathrm{inter}} = 0.005$ and $P = 0.05$.

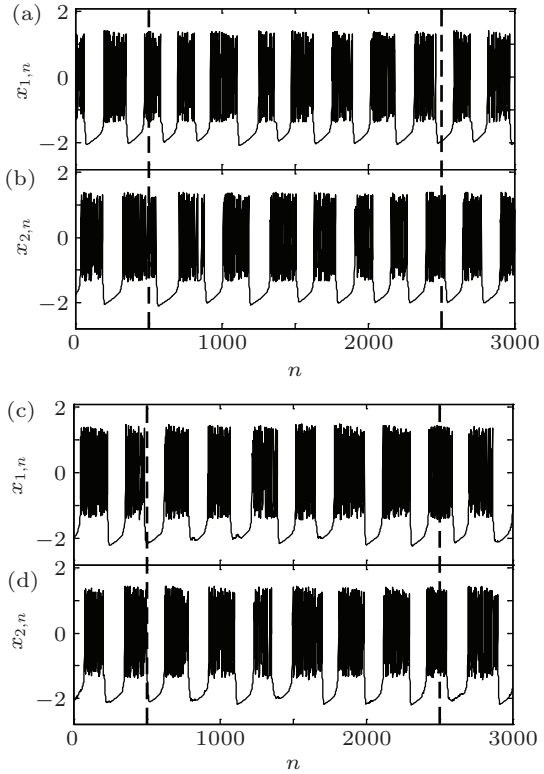


Fig. 3. Time evolutions of the fast variable for two randomly selected neurons in the clustered network, $p = 0.2$. (a) $n - x_{1,n}$ with $\varepsilon_{\mathrm{intra}} = 0.001$, (b) $n - x_{2,n}$ with $\varepsilon_{\mathrm{intra}} = 0.001$, (c) $n - x_{1,n}$ with $\varepsilon_{\mathrm{intra}} = 0.005$, (d) $n - x_{2,n}$ with $\varepsilon_{\mathrm{intra}} = 0.005$. Vertical lines correspond to the time n=500 and n=2500, respectively.

First, the effect of local coupling strength within each subnetwork on the bursting synchronization of the clustered neuronal network is investigated. We set $\varepsilon_{\mathrm{inter}} = 0.005$ and $P = 0.05$, such that the clustered network is not synchronized when $\varepsilon_{\mathrm{intra}} = 0$. We calculate the dependence of R on the intra-coupling strength $\varepsilon_{\mathrm{intra}}$ by different values of rewiring probability p. As shown in Fig. 2, the order parameter R undergoes a transition to coherence at a critical value $\varepsilon_{\mathrm{intra},c}$ for each particular value of p, indicating a transition to bursting synchronization. Obviously, the minimum of R is obtained for zero coupling. In this case, neurons burst at different times in a non-coherent fashion and the burst phases are spatially uncorrelated such that their contribution to the order parameter is small. On the other hand, in a completely phase synchronized state the order parameter magnitude R rapidly tends to unity, as a result of a coherent superposition of the phase vectors for all sites with the same amplitude at each time. Here, we take $\varepsilon_{\mathrm{intra,c}} = 0.002$ as an approximate value for the critical coupling strength. Above the transition threshold, bursting synchronization is achieved, as shown in Fig. 3(b); while below the threshold, the coupling is not strong enough to yield a synchronized burst, as shown in Fig. 3(a). It should be noticed that this synchronization occurs on the bursting time scale of coupled neurons, while on the spiking time scale, they behave asynchronously. That is to say, spikes inside each burst are not synchronized. Moreover, the transition is promoted by large

values of rewiring probability p. However, for $p > p_c \approx 0.3$, bursting synchronization, measured by R in Fig. 2, saturates and does not show significant difference. Similar results were obtained for our previous study on small-world networks of coupled map-based neurons.[10,39]

If the neuron's phases are synchronized, so are their bursting frequencies, which can be defined as an average speed of the phase increase

$$\Omega_j = \lim_{n\to\infty} \frac{\varphi_j(n) - \varphi_j(0)}{n}, \tag{6}$$

where $\varphi_j(n)$ is the burst phase for the j-th neuron at time n. In Fig. 4, we compare the bursting frequencies Ω_i of the neurons in the clustered network with those at zero-coupling $\Omega_{0,i}$, which fluctuate randomly among all sites. As one can see, when the intra-coupling strength is low ($\varepsilon_{\text{intra}} = 0.001$), a linear dependence between Ω_i and $\Omega_{0,i}$ is observed, indicating that there is no phase synchronization. However, when the coupling strength exceeds the synchronization threshold ($\varepsilon_{\text{intra}} = 0.005$), the frequencies of all coupled neurons are distributed around a mean value, implying that phase and frequency synchronization of the bursts is achieved.

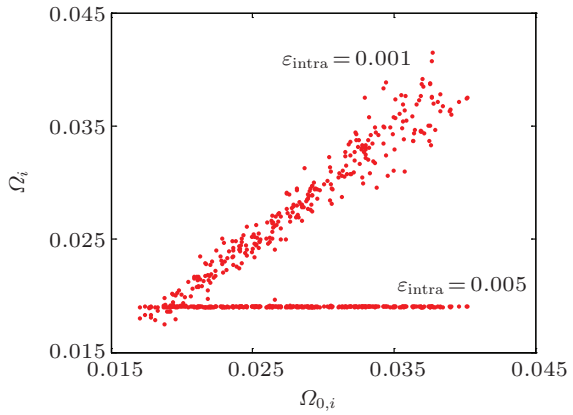


Fig. 4. (color online) Bursting frequency for the clustered neuronal network Ω_i versus the zero-coupling frequency $\Omega_{0,i}$ for different values of the intra-coupling strength $\varepsilon_{\text{intra}}$.

Next, we explore the dependence of bursting synchronization of the clustered network on the interconnectivity between subnetworks. Variation of the order parameter R with respect to inter-coupling strength $\varepsilon_{\text{inter}}$ and interconnection probability P between different subnetworks are shown in Figs. 5(a) and 5(b), respectively. It can be seen that for each particular value of P, R decreases to a minimal value at first, and then increases with the increase of inter-coupling strength $\varepsilon_{\text{inter}}$, characterizing a bursting synchronization transition. The transition is more pronounced for larger values of P, and the final synchronization degree of the clustered network is determined by the value of interconnection probability P (see Fig. 5(a)). Similar situations also occur when interconnection probability increases for different values of intra-coupling strength $\varepsilon_{\text{intra}}$ (see Fig. 5(b)). Thus, we can conclude that transitions of bursting synchronization can also be induced by the interconnection probability P between different subnetworks.

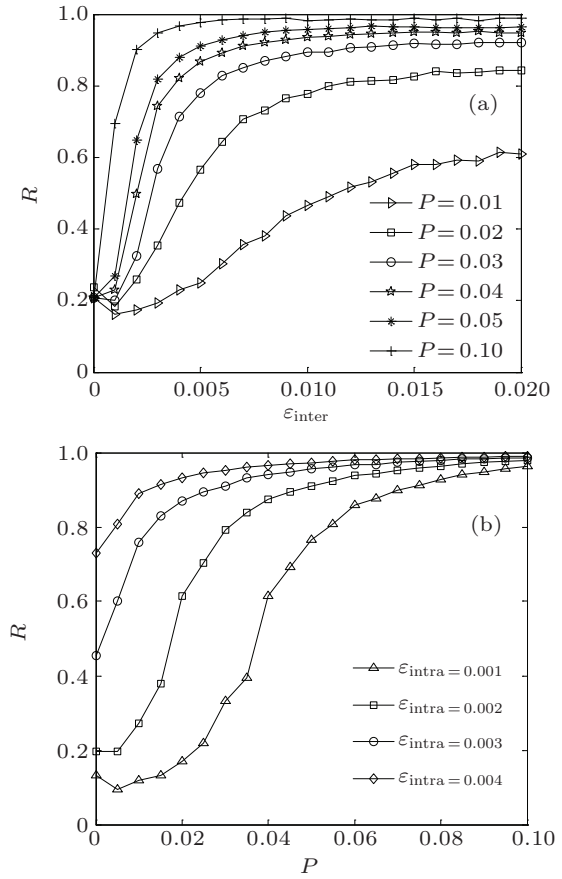


Fig. 5. (a) Variation of the order parameter R with respect to $\varepsilon_{\text{inter}}$ for different values of P. Other parameters: $\varepsilon_{\text{intra}} = 0.005$, $p = 0.2$. (b) Variation of the order parameter R with respect to P for different values of $\varepsilon_{\text{intra}}$. Other parameters: $\varepsilon_{\text{inter}} = 0.005$, $p = 0.2$.

In sum, the transition to bursting synchronization in a clustered neuronal network of small-world subnetworks can be induced by the variations of interconnection probability P between different subnetworks, inter- and intra-coupling strengths, $\varepsilon_{\text{inter}}$ and $\varepsilon_{\text{intra}}$. Similar results are also obtained for a network of regular subnetworks based on Hindmarsh–Rose (HR) neurons, in which two types of burst synchronization transitions are observed: one is due to spike-adding per burst and the other is caused by a change of bursting type from fold-homoclinic to fold-Hopf bifurcation.[43] However, in this work the clustered network model does not reveal such a property, since there is no change of the bursting type for coupled map-based neurons. On the other hand, compared with hub-connected small-world networks,[39] effects of interconnectivity between different subnetworks on the bursting synchronization of clustered neuronal networks are more prominent.

4. Synchronization control of the clustered neuronal network

In the previous section, we have demonstrated that neurons in a clustered network can gain bursting synchronization with the variation of coupling parameters. In this section, we consider the possibility of controlling such synchronized bursting rhythm by means of an externally applied time-periodic signal. In this intervention, rather than trying to synchronize the neurons with the external signal, one would wish to suppress those undesirable synchronous rhythms.

We consider the clustered neuronal network studied in Section 3, and apply a periodic signal $d\sin(\omega n)$ to one arbitrarily taken neuron i^* in the I-th subnetwork so that its fast variable x obeys the following equation:

$$x_{I,i^*}(n+1) = \frac{\alpha_{I,i^*}}{1+x_{I,i^*}^2(n)} + y_{I,i^*}(n) + I_{I,i^*}^{\rm syn}(n) + d\sin(\omega n). \quad (7)$$

The equation for the slow variable of the driven neuron and those for other neurons remain unchanged. Here, we set the value of network parameters: $\varepsilon_{\rm inter} = \varepsilon_{\rm intra} = 0.005$, and $P = 0.05$, such that the unperturbed ensemble ($d = 0$) exhibits bursting synchronization and their corresponding frequencies Ω_i lock approximately at a common value, as shown in Fig. 4.

In Fig. 6, we plot the the frequency difference of $\Omega_i - \omega$ for all neurons belonging to the network. With the variation of the driving frequency ω for different values of signal amplitudes d, the effect of phase locking of bursts is observed. For $d = 0.1$, a frequency locking region is obtained around $\omega = 0.016$, where $\Omega_i - \omega = 0$ is satisfied. The width of this frequency locking interval, $\Delta\omega$, increases with the signal amplitude in the direction of higher frequencies of the driving. However, a further increase the amplitude of driving force does not enlarge the frequency locking interval significantly (compare the synchronization plateau for $d = 0.2$ and 0.25). In fact, this widening trend is limited by the intensity of the driving signal, since for relatively large amplitudes of driving signal outside the synchronization region, the driven neuron switches to a frequency different from those of the driving signal and other mutually synchronized neurons.

To gain more insight into the effect of driving frequencies inside and outside the frequency locking region, in Fig. 7 we plot the time evolutions of phase difference between the bursting phase $\varphi(n)$ and the deriving phase $\varphi = \omega n$. For a frequency ω within the frequency locking interval, $\omega = 0.016$, the difference keeps constant and very small for all neurons. However, for a frequency ω outside the frequency locking interval, $\omega = 0.02$, there is an overall monotonic increase of the phase difference with the evolution of time, indicating that the corresponding frequencies are different from the external driving.

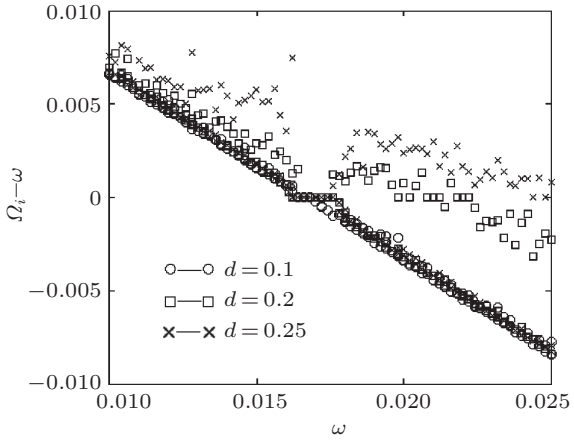


Fig. 6. Frequency difference between bursting neurons and external driving signal versus the external driving frequency for different values of the driving amplitude.

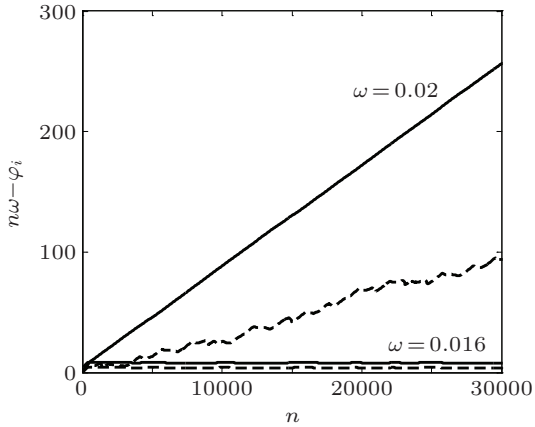


Fig. 7. Time evolutions of difference between bursting phase and driving phase for different values of driving frequency. The dotted line corresponds to the driven neuron, and the solid line corresponds to other neurons in the clustered network.

In order to generalize the above obtained results, we investigate the frequency locking region of the clustered neuronal network in a two-dimensional parameter space, where amplitude is plotted against frequency of the external driving signal. As shown in Fig. 8, an Arnold-like tongue is formed in the parameter plane (ω, d), characterizing a region where bursting synchronization is maintained, even with the external driving. Hence, the desirable choices for synchronization suppression would be those outside this tongue. Furthermore, the synchronization tongue illustrated in Fig. 8 is greatly asymmetric, since the unperturbed neuron frequencies are not located at the middle of the frequency locking interval, especially for large driving amplitudes. This is beneficial to experimental application, since we can keep outside the frequency locking region without increasing too much the amplitude of the signal. As we know, too strong stimulation may damage a biological neuron and change its intrinsic dynamics.

Finally, we investigate the influence of network topology on the synchronization control of the clustered neuronal network. In Fig. 9, we plot the width of frequency locking interval $\Delta\omega$ as a function of probability P of random links between

different subnetworks, whose value determines the synchronization degree of the clustered neuronal network, as shown in Fig. 5. Obviously, the width of synchronization region $\Delta\omega$ increases with interconnection probability P. That is to say, the synchronization control is more effective when there are more links between subnetworks. The reason for this effect may be that more random links among subnetworks allow the driving signal to spread across the whole network more quickly.

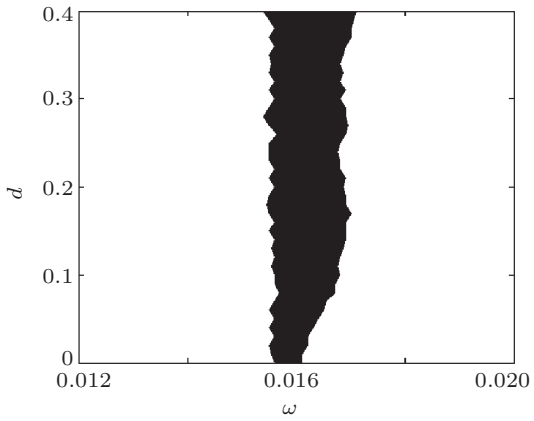


Fig. 8. The frequency locking tongue in the parameter plane: amplitude against frequency of the external driving signal.

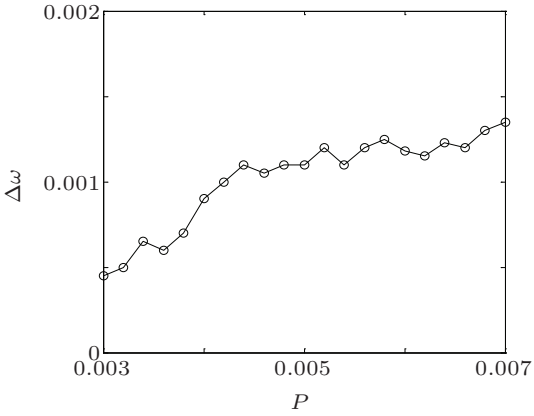


Fig. 9. Width of the frequency locking interval as a function of probability P of random links between different subnetworks, $d = 0.2$.

5. Conclusions

In this paper we have investigated bursting synchronization in a clustered neuronal network of small-world subnetworks. It is shown that this clustered neuronal network can get phase synchronization with the varization of several parameters, including the inter- and intra-coupling strength, as well as the probability of random links between different subnetworks. It is worth noting that this synchronization transition occurs on the bursting time scale, but not on the spiking time scale. Moreover, we have analyzed the control of bursting synchronization by using a time-periodic external signal in the clustered neuronal network. Simulation results show a frequency locking tongue in the driving parameter plane, representing parameters values for which bursting synchronization is maintained, even with the external driving. Hence, effective synchronization suppression can be realized with the driving parameters outside the frequency locking region.

In conclusion, we have investigated in detail the synchronization dynamics in a clustered neuronal network and the effect of several network parameters. As is well known, neuronal networks in the brain present the clustered property. Furthermore, plasticity is an important property of the brain, and change in the coupling strength is a main factor that affects this property. Therefore, the obtained results could facilitate our understanding not only of synchronization mechanisms of neurons within different brain areas but also of plasticity and its impact on synchronization in neuronal networks. On the other hand, the synchronization of bursting neurons is thought to play a key role in some pathological conditions like epilepsy, essential tremor, and Parkinson's disease. Hence, we have investigated the suppression of synchronized activity in an ensemble of bursting neurons from the viewpoint of a possible application in neuroscience. Particularly, we suggest that externally applied driving signal can be used for the purposes of deep brain stimulation, namely, for suppression of pathological brain rhythms.

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