Physica A 391 (2012) 2764–2770

Contents lists available at SciVerse ScienceDirect

Physica A

journal homepage: www.elsevier.com/locate/physa

The role of neural architecture and the speed of signal propagation in the process of synchronization of bursting neurons

Marko Gosak [*], Rene Markovič, Marko Marhl

Department of Physics, Faculty of Natural Sciences and Mathematics, University of Maribor, Koroška cesta 160, SI-2000 Maribor, Slovenia

ARTICLE INFO

Article history:
Received 16 September 2011
Received in revised form 24 November 2011
Available online 22 December 2011

Keywords:
Complex network
Chaotic oscillator
Bursting
Synchronization

ABSTRACT

Synchronized neuronal activity has been observed at all levels of human and any other nervous systems and was suggested as particularly relevant in information processing and coding. In the present paper we investigate the synchronization of bursting neuronal activity. Motivated by the fact that in neural systems the interplay between the network structure and the dynamics taking place on it is closely interrelated, we develop a spatial network representation of neural architecture in which we can tune the network organization between a scale-free network with dominating long-range connections and a homogeneous network with mostly adjacent neurons connected. Our results reveal that the most synchronized response is obtained for the intermediate regime where long- as well as short-range connections constitute the neural architecture. Moreover, the optimal response is additionally enhanced when the speed of signal propagation is optimized.

1. Introduction

Neural dynamics is a cooperative process of neurons and the existence of the synchronous rhythms is crucial for performing of the operational tasks in various specialized areas of the nervous system [1,2]. A particularly interesting type of a complex oscillatory rhythm of neurons is bursting, where the neural activity alternates, on a slow time scale, between a quiescent state and fast repetitive spiking [3]. Neural bursting has been identified in several processes and regions in the mammal brain [4] such as the olfactory system or the hippocampal region [5]. Remarkably, variations of rhythmic bursting activity are also believed to be involved in various clinical disorders and pathological conditions [6,7]. In order to explore the bursting synchronization mechanisms in ensembles of coupled neurons mathematical models have been utilized [8–10]. This is because the study of synchronization and desynchronization of neuronal bursting behaviours from biophysical models may help us to understand further the information processing and coding as well as disorders in the nervous system. Notably, in the last decade the focus of this interest has shifted to ensembles characterized with complex interaction topologies, as constituted by small-world or scale-free networks [11–16]. Motives are definitely related with the fact that such topological structures have been identified in the nervous system [17]. Moreover, Pontes et al. also took into account the spatial distance between coupled bursting neurons [18]. In their model the interaction strength decreased as a power law with increasing distance, thus enabling the emulation of the fractal nature of dendritic connections.

It is known that information flow in neural networks is not instantaneous. A finite signal transmission speed along the axons results in a space dependent time delay. Typical speed of signal conduction is on the order of m/s, leading to transmission times up to hundreds of milliseconds for information propagation through the cortical network [19]. Previous studies have already revealed that time delays can gradually affect the spatiotemporal dynamics in networks of coupled neurons [20]. Remarkably, Dhamala et al. [21] have shown that time-delayed coupling facilitates the existence of stable

---

[*] Corresponding author. Tel.: +386 2 2293893; fax: +386 2 2518180.
E-mail address: marko.gosak@uni-mb.si (M. Gosak).

0378-4371/$ – see front matter © 2011 Elsevier B.V. All rights reserved.
doi:10.1016/j.physa.2011.12.027

synchronized states of two chaotic neurons, whereas Burić et al. [22] extended the concept to a pair of noisy bursting neurons. Furthermore, many interesting phenomena such as zigzag fronts of excitations and antiphase synchronization [23] as well as multiple stochastic resonances [24] were found in neuronal networks with constant delays. Wang et al. [25] investigated delay-induced synchronization transitions on neuronal network and their study has revealed that an enhanced synchronized response is observed if the delay equals a multiple of the inherent oscillation frequency of neurons. Special attention has also been devoted to different types of coupling mechanisms and transmission delays. Along these lines Wang et al. [26] studied synchronization transition of bursting neurons with respect to attractive and repulsive coupling, whereas Hao et al. [27] focused on the difference between electrical and chemical coupling. In Ref. [28] burst synchronization via delayed inhibitory synapses has been analysed. Nevertheless, the use of constant fixed delays provides a good approximation for simple circuits consisting of a small number of cells. However, realistic neural networks are embedded in metric space and hence the network topology and the transmission delays are also a function of the Euclidean distance between the neurons. In view of that, several studies tackled synchronization of neural networks with distributed and time-varying delays [29–32]. It has been shown that space-dependent delays can destabilize the synchronous states and induce the formation of waves [33]. Later on, Ko et al. [34] and Ko and Ermentrout [35] widened the idea of distance-dependent delays to complex interaction topologies by studying the synchronous behaviour and wave formation in sparsely coupled neuronal oscillators.

In the present study we extend the scope of research of synchronization of bursting activity on complex networks with the presence of signal transmission delays. For this purpose we develop a network representation of neural architecture, where vertices are bursting neurons embedded in three-dimensional metric space and links model electrical coupling between them. The employed network model allows us to smoothly alter the topology from a scale-free network with dominating long-range connections to a network where principally only adjacent neurons are connected. In our previous studies a similar network model has been used in order to determinate the topology leading to optimal stochastic and coherence resonance responses in an ensemble of excitable neurons [36,37]. Here we widen this idea to identify the most favourable interaction topology for the synchronization of bursting neurons. Additionally, we consider transmission delays, which are a consequence of finite signal propagation speeds and they depend on the Euclidean distances between coupled neurons. Therefore, when the topology is altered, the distribution and the rate of delays are simultaneously modified as well. In contrast to previous studies on small-world and scale-free networks [11–16], the employed model thus enables the identification of topological as well as dynamical conditions which ensure the most synchronized bursting activity, without choosing any specific network configuration.

2. Mathematical model

To mimic the characteristic bursting dynamics of neurons, we make use of the iterated map proposed by Rulkov [8], which captures succinctly the main features of more complex time-continuous neuronal models, but is numerically much more efficient. The temporal evolution of the i-th neuron along with delayed coupling is defined as follows:

$$x_i(t+1) = \frac{\alpha_i}{1+x_i(t)^2} + y_i(t) + D\sum_{j=1}^{N} \varepsilon_{ij}[x_j(t-\tau_{ij}) - x_i(t)], \tag{1}$$

$$y_i(t+1) = y_i(t) - \gamma x_i(t) - \sigma, \tag{2}$$

where $y_i(t)$ and $x_i(t)$ are the slow and fast variable of the map, respectively, and are considered as dimensionless variables, t is the discrete time index and α, γ and σ are systems parameters. By choosing $\gamma = \sigma = 0.001 \ll 1$ we ensure that the dynamics of $x_i(t)$ is much faster than that of $y_i(t)$. Furthermore, we consider values of α_i above 4.0, where the Rulkov map produces chaotic oscillations. In this case the slow variable $y_i(t)$ exhibits saw-tooth oscillations, whereas the fast variable $x_i(t)$ emulates the spiking–bursting behaviour. When the slow variable reaches a local maximum the fast variable begins its firing interval. After the slow variable reaches a local minimum the firing interval switches then into a quasi-steady state. In order to take into account the diversity of neurons, values of α_i are assumed to follow a power-law $P(\alpha) \sim \alpha^{-\beta}$ with a scaling exponent $\beta = 2.5$. Values of α_i are then confined within the interval [4.1, 4.4], whereby the characteristic of the distribution remains intact. This arrangement of α_i implies a small number of highly active oscillators with small time intervals between the bursts whereas the majority of neurons are less active. Namely, higher the values of α_i correspond to higher frequencies of bursting patterns. In this manner we presume the existence of a small fraction of leader (precursor) neurons, which endeavour to trigger the response of its neighbours and thus act as global initiators of the bursts [38].

The sum in Eq. (1) stands for the coupling, where the coupling strength is symbolized by D and ε_{ij} is the connectivity matrix which has a value of 1 if the i-th and j-th neuron are electrically coupled and 0 otherwise. The calculation of the network connectivity is based on the spatially embedded vertex fitness network model [39]. First, N neurons are randomly distributed according to a uniform distribution inside a unit cube. Then, the i-th and the j-th neuron are connected if the following condition is fulfilled:

$$\frac{\alpha_i \alpha_j}{l_{ij}^{\delta}} > \Theta, \tag{3}$$

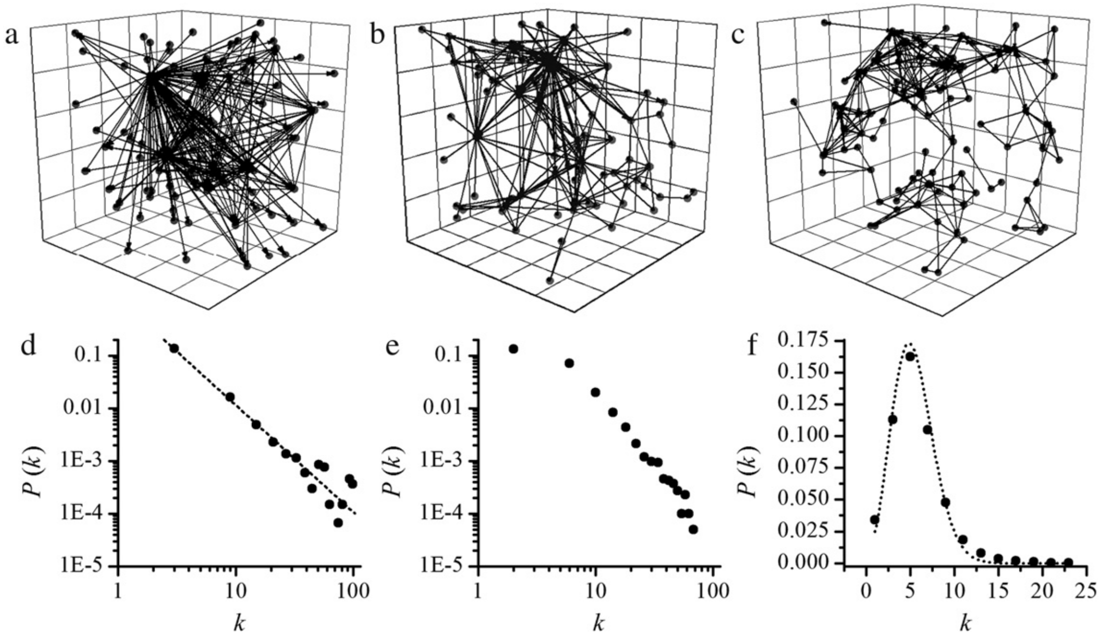

Fig. 1. Characteristic network structures and the corresponding degree distributions for $\delta = 0.1$ (a, d), $\delta = 1.7$ (b, e) and $\delta = 5.0$ (c, f). The number of neurons is $N = 100$ and mean vertex degree is $\langle k \rangle = 5$. The calculation of degree distributions is based on the average of ten independent network generations.

where Θ is a threshold that adjusts the mean vertex degree $\langle k \rangle$, I_{ij} is the Euclidean distance between the i-th and j-th neuron and δ is a control parameter that enables smooth alterations of the topology of the interaction network (see also our previous work [36,37]). The condition given in Eq. (3) entails that in general neurons with higher values of α_i, i.e. leader neurons, are more likely to be connected. The characteristic network configurations obtained for different values of δ along with the corresponding degree distributions are shown in Fig. 1.

It can be observed that for $\delta = 0.1$ (Fig. 1a) the network consist solely of long-range connections which originate mostly from a few leader neurons. In this case the creation of connections is not affected by the Euclidean distance and hence neurons with higher values of α_i have the highest vertex degree. The corresponding degree distribution (Fig. 1d) follows a power-law indicating a highly heterogeneous network structure. However, as δ is increased, the spatial distribution of neurons becomes more and more important and consequently short-range interactions emerge. For $\delta = 1.7$ (Fig. 1b) we have thus a mixture of long- and short-range link, whereas for $\delta = 5$ (Fig. 1c) only adjacent neurons are connected. It can also be noticed that for higher values of δ the degree distributions become more homogeneous, so that for $\delta \gg 1$ the degree distribution obeys a Poisson distribution (Fig. 1f), a typical feature of random geometric graphs.

The coupling term in Eq. (1) also involves a transmission delay τ_{ij}, which is, as announced in the Introduction, a consequence of finite speed of the action potential propagation. Accordingly, the transmission delay between the i-th and the j-th neuron is defined as follows:

$$\tau_{ij} = \frac{I_{ij}}{v}, \tag{4}$$

where v is the signal propagation speed. Since the model describing the bursting dynamics is discrete in time, the delays have to be converted into integer numbers. In particular, the value calculated in Eq. (4) is rounded down, i.e., only the integer part of the calculated value is considered. In our model the neurons are uniformly randomly distributed in a unit cube and hence the maximal possible distance between them is $\sqrt{3}$. Accordingly, $v = 2$ corresponds to instant communication with no delays, whereas v smaller then $\sqrt{3}$ give rise to finite delays τ_{ij}. It should be emphasized that the lengths of connections I_{ij} vary with δ and therefore the resulting delays are directly related to the topology of the network as well. To adjust the range of delays, v is used as a control parameter, whereby the resulting delays are always between 0 and the integer part of the fraction $\sqrt{3}/v$.

3. Results

We consider an ensemble of $N = 200$ neurons which form a network with a mean degree $\langle k \rangle = 5$ and are weakly coupled with a coupling strength $D = 0.003$. All presented results were averaged over at least 20 independent runs in order to assure the appropriate statistical accuracy. Our main interest is to examine the coherent behaviour in the system as a

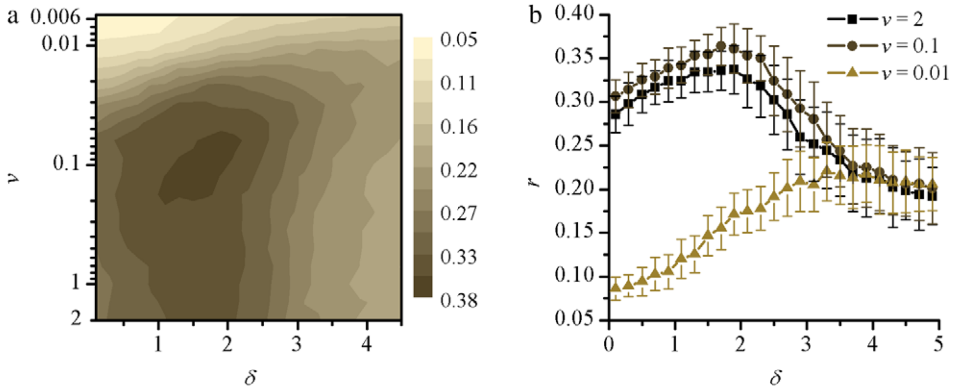

Fig. 2. The level of phase synchronization quantified via r as a function of network structure δ and transmission speed υ (a) and the cross sections of the colour map at different values of υ (b).

function of the network topology and the signal propagation speed. We focus mainly on the phase synchronization, which is a quite weak condition of synchronization, since it gives only an inside on how synchronous is the switching between the bursting and quasi-steady state phases of oscillators. For a given oscillator the phase increases for 2π at the beginning of each burst, i.e. each maximum of the slow variable $y_i(t)$. Between two bursts the phase increases linearly with the discrete time t. The phase $\varphi_j(t)$ for the j-th neuron is described by the following equation [12–14]:

$$\varphi_j(t) = 2\pi \left(n + \frac{t - t_j(n-1)}{t_j(n) - t_j(n-1)} \right), \quad t_j(n-1) \le t < t_j(n) \tag{5}$$

where the n-th burst of the j-th oscillator begins at time $t_j(n)$. Note that the duration of individual burst $t_j(n) - t_j(n-1)$ varies in an irregular fashion due to the chaotic dynamics. Furthermore, the neurons are heterogeneous and hence with the given coupling strength exact equality of bursting times cannot be realized. However, coupling gives rise to a more collective motion in terms of chaotic phase synchronization, where neurons try to harmonize their phases with each other, while the amplitude and spiking behaviour are in general poorly correlated. To capture the order of phase synchronization, we have to calculate the complex phase order parameter r_t at a given time t:

$$r_t = \frac{1}{N} \sum_{j=1}^{N} \mathrm{e}^{\mathrm{i}\varphi_j(t)}. \tag{6}$$

The phase order parameter in a system of completely synchronized oscillators equals one, whereas in the case of totally uncorrelated activity $r_t = 0$. In order to describe the average global collective motion we calculate the time averaged order parameter r:

$$r = \frac{1}{T} \sum_{t=1}^{T} r_t, \tag{7}$$

where $T = 10^5$ is the total number of iterations used in the calculations after $2 \cdot 10^4$ initial iterations were discarded as transients. In case of synchronization in a network of heterogeneous chaotic neurons, values of r are expected between 0 and 1, whereby higher values correspond to a greater extent of synchronization. Results showing r as a function of δ and υ are presented in Fig. 2. It can be observed that as long as $\upsilon > 0.02$ the optimal phase synchronization is achieved for intermediate values of δ, at which the network is constituted by long- as well as by short-range connections. For lower propagation speeds the most synchronized response is obtained at larger values of δ, where the lengths of connections are lower. Furthermore, the best synchronization is at $\upsilon \approx 0.1$, which shows that delayed coupling gives rise to the collective behaviour in a network of heterogeneous neurons. Thus the plot in Fig. 2 shows that the collective dynamics of weakly coupled chaotic neurons is strongly influenced by both the network configuration and the propagation speed of the signals.

To get some additional insights into the dynamical behaviour we examine the correlations between individual neurons by calculating the correlation matrix R, whose ij-th element is defined as follows:

$$R_{ij} = \frac{\sum [\bar{x}_i - x_i(t)][\bar{x}_j - x_j(t)]}{s_{x_i} s_{x_j}} \tag{8}$$

where $\bar{x}_i$ and $\bar{x}_j$ are the mean values of the fast variables during all iterations and s_{x_i} and s_{x_j} the standard deviations of the time series $x_i(t)$ and $x_j(t)$, respectively. If $R_{ij} = 0$ no correlation between the i-th and j-th oscillator exist, whilst $R_{ij} = 1$ signifies

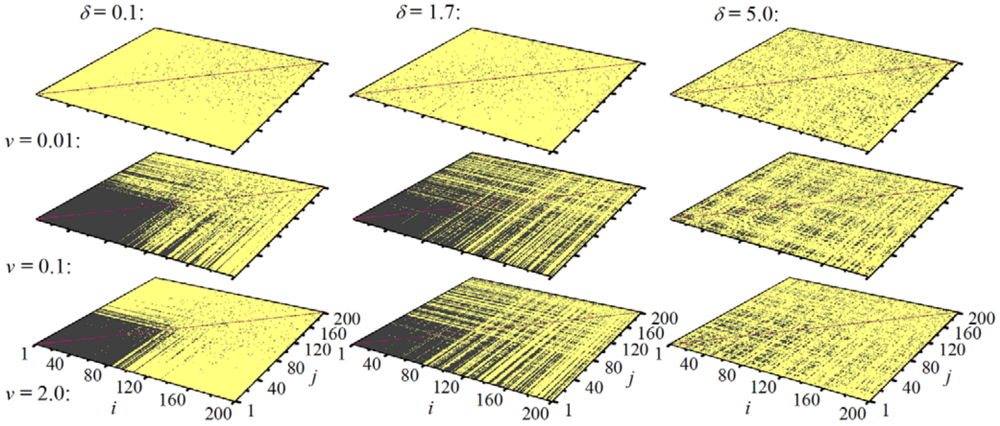

Fig. 3. Correlation matrices R_{ij} for different interaction topologies (columns) and different speeds of signal propagation (rows). If the correlation between the i-th and j-th neuron is more than 0.1, point (i, j) is marked black, otherwise it is yellow. Note that the neurons are indexed in descending order of α_i.

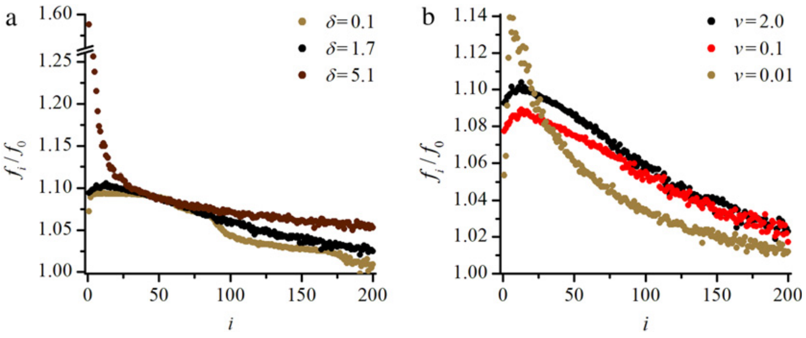

Fig. 4. Frequency of the i-th oscillator f_i at $v = 2$ (instantaneous communication) for different network structures (a) and for different propagation speeds at optimal network configuration, i.e. $\delta = 1.7$ (b). Note that the frequencies have been normalized to the minimal frequency $f_0 = 0.00291$.

completely synchronous motion. The correlation matrices for three characteristic network structures ($\delta = 0.1$, $\delta = 1.7$ and $\delta = 5$) and three different propagation speeds ($v = 2.0$, $v = 0.1$ and $v = 0.01$) are shown in Fig. 3. We can observe that in case of scale-free interaction topology ($\delta = 0.1$), a good correlation exists mainly between active neurons, which also have a high node degree, whereas the less active neurons are weakly correlated. On the other hand, in the network where short-range connections are dominative ($\delta = 5$), only some clusters of correlated neuronal activity exist, whose positions do not depend on the inherent dynamics of individual neurons. Notably, the biggest overall correlation is attained at intermediate values of δ, which once again confirms that the best synchronization is achieved if both long- and short-range interactions characterize the network. In this case the most active neurons function as "local" hubs that dictate the dynamics of the local interconnected neighbourhood and also communicate among themselves (Fig. 1b). Moreover, the optimal speed of signal transmission at $v = 0.1$ additionally improves the correlation among bursting neurons in comparison to $v = 2.0$, where no transmission delays are incorporated in the coupling.

To further investigate the impact of network topology and transmission speed on synchronized bursting, we examine the distribution of frequencies of individual neurons. The average bursting frequency of the i-th neuron f_i is defined as the total number of bursts n_i divided by the time interval in which they occur t_i:

$$f_i = \frac{n_i}{t_i}. \tag{9}$$

The system consists of a small number of very active oscillators with high intrinsic frequencies when they are uncoupled, and a large number of low-frequency oscillators. When they are coupled, the effect of different network configurations and different propagation speeds on the average frequency distribution are shown in Fig. 4. We use the standard deviation (SD) as a measure for dispersion of the frequencies. The results in Fig. 4a reveal that the frequency distribution is the most homogeneous for the optimal system constellation consisting of short- and long-range connections ($\delta = 1.7$). In particular, the SD's expressed as a percentage of the mean values for $\delta = 0.1$, $\delta = 1.7$ and $\delta = 5$ were 2.9%, 2.4% and 5.2%, respectively. Clearly, for intermediate network topology the coupled neurons maximally adopt their frequencies and try to unify their values. Moreover, in Fig. 4b we can observe that the optimal speed of signal transmission ($v = 0.1$) additionally unifies the frequencies. The SD's expressed as a percentage of the mean values for $v = 2.0$, $v = 0.1$ and $v = 0.01$ were 2.4%, 2.0% and

3.0%, respectively. It can be observed that the frequencies of more active neurons are considerably reduced, which reflects the fact that they have a higher node degree and hence have larger coupling fluxes. Remarkably, Yu et al. [14] who studied synchronization behaviour of bursting neurons in small-world networks also revealed that coupled neurons unify their bursting frequencies and that the more active neurons adopt the bursting frequencies of less active neurons. Furthermore, we found that this additional frequency lowering of leader neurons, or increasing their periods, corresponds to the average delay of the signal transduction. Of course, a threshold exist to which the oscillatory periods can be extended by these delays; when the threshold is exceeded, the oscillators cannot be delayed any further and the distribution of frequencies become again more heterogeneous (see the case for $v = 0.01$).

4. Discussion

We have studied synchronization of weakly coupled bursting neurons. Our results show that a highest level of synchronization is obtained when a proper ratio of long- and short-range connections exist in the network. Moreover, this optimal response is additionally enhanced when the speed of signal propagation is optimized. It is worth mentioning that the presented results are in agreement with our previous studies [36,37], where we studied the impact of the interaction topology on the regularity of noise induced oscillations in an ensemble of excitable neurons. The most coherent responses were also found in the intermediate regime between the scale-free network with dominating long-range connections and the strong geometric regime, thus indicating that this is the most favourable neural architecture for various types of dynamical behaviour. However, it should be noted that not in all dynamical regimes the intermediate network configuration ensures best synchronizability. We argue that the mechanism is similar and strongly related to the flexibility properties of the individual oscillators, which enables adopting of frequencies, as it has been discussed in Fig. 4 for the present case. Sensitivity and flexibility of oscillators and their relation to the frequency adaptation have been extensively discussed in our previous papers (for review see e.g. Ref. [40]). It has been shown that the oscillator's flexibility is in principle independent of its inherent dynamics, but in general oscillators are more adoptable in the proximity of bifurcation points (for example in an excitable steady state). For the model system under consideration, the system is more flexible in the region of chaotic bursting than in the region of periodic oscillations [41]. Therefore, reduction of the discrepancy of the frequencies leading to the most synchronized response (see Fig. 4), which is attained at the intermediate network configuration, can be achieved if the oscillators operate in the regime of chaotic bursting.

Our findings thus reveal that the both network structure as well as the speed of signal transmission are vital for assuring optimal conditions for synchronization of bursting neurons. Remarkably, alterations of exactly these factors have been found to be important and responsible for certain pathological conditions. Many neurodegenerative diseases such as multiple sclerosis are related to the process of demyelination in which the myelin shells that cover the neuron fibres vanishes and consequently the action potential propagation speed is significantly modified [42]. Moreover, a number of studies have established that several neuronal pathologies are associated with changes in efficiency of the intercellular coupling and neural network dysfunctions [7,43–45]. One kind of those dysfunctions is Parkinson's disease, which leads to movement disorders. A well established method to treat patients with Parkinson's disease is deep brain stimulation [46]. This treatment includes surgical implantation of electrodes in certain areas of the brain and connects them with an external current source of high frequency [47]. The synchronization/desynchronization of a neural network under external periodic forcing has already been studied from a theoretical point of view [16,18]. The authors investigated the ability of an external periodic signal to synchronize or desynchronize a neural network. They showed that the locking frequency interval width increases with the amplitude of the external signal. Hence to achieve desynchronization, the external frequency must be chosen outside the frequency looking interval. In the future it would be worth to investigate the impact of the joint effect of delayed transmissions between neurons, topology and external forcing.

We hope that further theoretical and experimental studies about the topology and dynamics of cellular networks will be conducted, and that the results will contribute to even better understanding of physiological and pathological conditions in biological tissues with direct applications in medicine. In particular, for diagnostic purposes it would be of much importance to detect and probably measure some relevant quantities in order to get indicators for (pathological) changes in the cellular network structure and the dynamical properties of signal transduction, where the speed of signal propagation is probably the most obvious element. Additional studies will be needed to understand the communicational links, the ways of cellular coupling, not only in a way of direct adjacent connections, like gap-junctions for example, but also long-range connections of cells at different locations in the tissue. This might have spectacular consequences in preventive diagnostic, like it has been indicated for chronic hypoxia in a very early study in this direction [48].

References

[1] F. Varela, J.P. Lachaux, E. Rodriguez, J. Martinerie, The brainweb: phase synchronization and large-scale integration, Nature Rev. Neurosci. 2 (2001) 229–239.
[2] T. Nowotny, R. Huerta, M.I. Rabinovich, Neuronal synchrony: peculiarity and generality, Chaos 18 (2008) 037119.
[3] R.C. Elson, A.I. Selverston, H.D. Abarbanel, M.I. Rabinovich, Inhibitory synchronization of bursting in biological neurons: dependence on synaptic time constant, J. Neurophysiol. 88 (2002) 1166–1176.
[4] D.A. McCormick, H.R. Feeser, Functional implications of bursting firing and single spike activity in lateral geniculate relay neurons, Neuroscience 39 (1990) 103–113.

[5] M.R. Mehta, A.K. Lee, M.A. Wilson, Role of experience and oscillations in transforming a rate code into a temporal code, Nature 417 (2002) 741–746.

[6] D.R. Weinberger, Implications of normal brain development for the pathogenesis of schizophrenia, Arch. Gen. Psychiatry. 44 (1987) 660–669.

[7] T.I. Netoff, R. Clewley, S. Arno, T. Keck, J.A. White, Epilepsy in small-world networks, J. Neurosci. 24 (2004) 8075–8083.

[8] N.F. Rulkov, Regularization of synchronized chaotic bursts, Phys. Rev. Lett. 86 (2001) 183–186.

[9] M. Dhamala, V.K. Jirsa, M. Ding, Transitions to synchrony in coupled bursting neurons, Phys. Rev. Lett. 92 (2004) 028101.

[10] T. Pereira, M.S. Baptista, J. Kurths, Multi-time-scale synchronization and information processing in bursting neuron networks, Eur. Phys. J. Special Topics 146 (2007) 155–168.

[11] I. Belykh, E. de Lange, M. Hasler, Synchronization of bursting neurons: what matters in the network topology, Phys. Rev. Lett. 94 (2005) 188101.

[12] C.A.S. Batista, A.M. Batista, J.A.C. de Pontes, R.L. Viana, S.R. Lopes, Chaotic phase synchronization in scale-free networks of bursting neurons, Phys. Rev. E 76 (2007) 016218.

[13] C.A.S. Batista, S.R. Lopes, R.L. Viana, A.M. Batista, Delayed feedback control of bursting synchronization in a scale-free neuronal network, Neural Netw. 23 (2010) 114–124.

[14] H. Yu, J. Wang, B. Deng, X. Wei, Y.K. Wong, W.L. Chan, K.M. Tsang, Z. Yu, Chaotic phase synchronization in small-world networks of bursting neurons, Chaos 21 (2011) 013127.

[15] E.J. Agnes, R. Erichsen Jr., L.G. Brunnet, Synchronization regimes in a map-based model neural network, Physica A 389 (2010) 651–658.

[16] C.A.S. Batista, A.M. Batista, J.C.A. De Pontes, S.R. Lopez, R.L. Viana, Bursting synchronization in scale-free networks, Chaos, Solition. Fract. 41 (2009) 2220–2225.

[17] E. Bullmore, O. Sporns, Complex brain networks: graph theoretical analysis of structural and functional systems, Nat. Rev. Neurosci. 10 (2009) 186–198.

[18] J.C.A. De Pontes, R.L. Viana, S.R. Lopez, C.A.S. Batista, A.M. Batista, Bursting synchronization in non-localy coupled maps, Physica A 387 (2008) 4417–4428.

[19] E.R. Kandel, J.H. Schwartz, T.M. Jessell, Principles of Neural Science, fourth ed., McGraw-Hill, New York, 2000.

[20] A. Roxin, N. Brunel, D. Hansel, Role of delays in shaping spatiotemporal dynamics of neuronal activity in large networks, Phys. Rev. Lett. 94 (2005) 238103.

[21] M. Dhamala, V.K. Jirsa, M. Ding, Enhancement of neural synchrony by time delay, Phys. Rev. Lett. 92 (2004) 074104.

[22] N. Burić, K. Todorović, N. Vasović, Exact synchronization of noisy bursting neurons with coupling delays, Chaos, Soliton. Fract. 40 (2009) 1127–1135.

[23] Q. Wang, Z. Duan, M. Perc, G. Chen, Synchronization transitions on small-world neuronal networks: effects of information transmission delay and rewiring probability, EPL 83 (2008) 50008.

[24] Q. Wang, M. Perc, Z. Duan, G. Chen, Delay-induced multiple stochastic resonances on scale-free neuronal networks, Chaos 19 (2009) 023112.

[25] Q. Wang, M. Perc, Z. Duan, G. Chen, Synchronization transitions on scale-free neuronal networks due to finite information transmission delays, Phys. Rev. E 80 (2009) 026206.

[26] Q. Wang, G. Chen, M. Perc, Synchronous bursts on scale-free neuronal networks with attractive and repulsive coupling, PLoS ONE 6 (2010) e15851.

[27] Y. Hao, Y. Gong, L. Wang, X. Ma, C. Yang, Single or multiple synchronization transitions in scale-free neuronal networks with electrical or chemical coupling, Chaos, Soliton. Fract. 44 (2011) 260–268.

[28] I. Franović, V. Miljković, Phase plane approach to cooperative rhythms in neuron motifs with delayed inhibitory synapses, EPL 92 (2010) 68007.

[29] A. Hutt, F.M. Atay, Effects of distributed transmission speeds on propagating activity in neural populations, Phys. Rev. E 73 (2006) 021906.

[30] T. Li, S. Fei, K. Zhang, Synchronization control of recurrent neural networks with distributed delays, Physica A 387 (2008) 982–996.

[31] Y. Sun, D. Zhao, J. Ruan, Consensus in noisy environments with switching topology and time-varying delays, Physica A 389 (2010) 4149–4161.

[32] K. Wang, Z. Teng, H. Jiang, Adaptive synchronization of neural networks with time-varying delay and distributed delay, Physica A 387 (2008) 631–642.

[33] P.C. Bressloff, S. Coombes, Synchrony in an array of integrate-and-fire neurons with dendritic structure, Phys. Rev. Lett. 78 (1997) 4665–4668.

[34] T.-W. Ko, S.-O. Jeong, H.-T. Moon, Wave formation by time delays in randomly coupled oscillators, Phys. Rev. E 69 (2004) 056106.

[35] T.-W. Ko, G.B. Ermentrout, Effects of axonal time delay on synchronization and wave formation in sparsely coupled neuronal oscillators, Phys. Rev. E 76 (2007) 056206.

[36] M. Gosak, D. Korošak, M. Marhl, Optimal network configuration for maximal coherence resonance in excitable systems, Phys. Rev. E 81 (2010) 056104.

[37] M. Gosak, D. Korošak, M. Marhl, Topologically determined optimal stochastic resonance responses of spatially embedded networks, New J. Phys. 13 (2011) 013012.

[38] J.-P. Eckmann, S. Jacobi, S. Marom, E. Moses, C. Zbinden, Leader neurons in population bursts of 2D living neural networks, New J. Phys. 10 (2008) 015011.

[39] S. Morita, Crossovers in scale-free networks on geographical space, Phys. Rev. E 73 (2006) 035104.

[40] M. Perc, M. Marhl, Local dissipation and coupling properties of cellular oscillators: A case study on calcium oscillations, Bioelectrochem. 62 (2004) 1–10.

[41] A.L. Shilnikov, N.F. Rulkov, The origin of chaos in a two-dimensional map modeling spiking-bursting neural activity, Int. J. Bifurcat. Chaos 13 (2003) 3325–3340.

[42] S. Reutskiy, E. Rossoni, B. Tirozzi, Conduction in bundles of demyelinated nerve fibers: computer simulation, Biol. Cybern. 89 (2003) 439–448.

[43] N. Rouach, et al., Gap junctions and connexin expression in the normal and pathological central nervous system, Biol. Cell. 94 (2002) 457–475.

[44] J.J. Palop, J. Chin, L. Mucke, A network dysfunction perspective on neurodegenerative diseases, Nature 443 (2006) 768–773.

[45] Y. He, et al., Impaired small-world efficiency in structural cortical networks in multiple sclerosis associated with white matter lesion load, Brain: J. Neurol. 132 (2009) 3366–3379.

[46] C.R. Buston, C.C. McIntyre, Current steering to control the volume of tissue activated during deep brain stimulation, Brain Stimul. 1 (2008) 7–15.

[47] W.M. Grill, A.N. Synder, Svjetlana Miocinovic, Deep brain stimulation creates an informational lesion of the stimulated nucleus, NeuroReport 15 (2004) 1137–1140.

[48] M. Gosak, M. Marhl, C. Guibert, M. Billaud, E. Roux, Pulmonary artery smooth muscle responses to KCl under normoxic and hypoxic conditions studied by means of a spatial network model, in: A. Zhang, M. Borodovsky, G. Özsoyoglu, A.R. Mikler (Eds.), Proceedings of the First ACM International Conference on Bioinformatics and Computational Biology, ACM New York, New York, 2010, pp. 475–477.