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Delayed feedback control of bursting synchronization in a scale-free neuronal network

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ABSTRACT

Several neurological diseases (e.g. essential tremor and Parkinson's disease) are related to pathologically enhanced synchronization of bursting neurons. Suppression of these synchronized rhythms has potential implications in electrical deep-brain stimulation research. We consider a simplified model of a neuronal network where the local dynamics presents a bursting timescale, and the connection architecture displays the scale-free property (power-law distribution of connectivity). The networks exhibit collective oscillations in the form of synchronized bursting rhythms, without affecting the fast timescale dynamics. We investigate the suppression of these synchronized oscillations using a feedback control in the form of a time-delayed signal. We located domains of bursting synchronization suppression in terms of perturbation strength and time delay, and present computational evidence that synchronization suppression is easier in scale-free networks than in the more commonly studied global (mean-field) networks.

1. Introduction

Complex networks are found in many scientific and technological applications, and a great deal of effort has been spent on studying such systems using tools derived from areas like statistical mechanics, graph theory, and nonlinear dynamics (Borhnoldt & Schuster, 2003). In these complex networks the nodes represent individuals or organizations, the links standing for their mutual interactions, according to a specified connection architecture (Albert & Barabási, 2002; Dorogovtsev & Mendes, 2002). A class of complex networks which has been intensively studied is the *scale-free* network, for which the connectivity – the number of connections for each node – presents a statistical power-law dependence (Barabási & Albert, 1999). If $P(k)dk$ denotes the probability of finding a node with connectivity between k and $k + dk$, for scale-free lattices one has $P(k) \sim k^{-\gamma}$ where $\gamma > 1$. As a consequence, in scale-free networks a few nodes are connected with a large number of other ones, whereas most of the nodes are connected with a small number of network units.

This power-law distribution of connectivities comes from two mechanisms (Barabási & Albert, 1999): (i) networks expand continuously by the addition of new nodes; (ii) new nodes attach preferentially to already well-connected nodes. As those mechanisms are common to many networks of physical, biological, and social interest, it is not surprising that a large number of networks have been found to exhibit a scale-free connectivity. Some examples are the World Wide Web (Barabási, Albert, & Jeong, 2000; Broder et al., 2000; Pastor-Satorras, Vázquez, & Vespignani, 2001), earthquakes (Baiesi & Paczuski, 2004), large computer programs (de Moura, Lai, & Motter, 2003), epidemic spreading (Pastor-Satorras & Vespignani, 2001), human sexual contacts (Lijeros, Edling, Amaral, Stanley, & Aberg, 2001), protein domain distributions (Wuchty, 2001), cellular metabolic chains (Barabási & Oltvai, 2004; Jeong, Tombor, Albert, Oltvai, & Barabási, 2000; Jeong, Mason, Barabási, & Oltvai, 2001), and human brain functional networks (Equiluz, Chialvo, Cecchi, Buliki, & Apkarian, 2005).

Scale-free neural networks have attracted a lot of attention, since the relative sparseness of their coupling architecture reduces the memory needed to store a given amount of information, as well as the computational effort needed to provide certain tasks (Perotti, Tamarit, & Cannas, 2006; Stauffer, Aharony, da Fontoura Costa, & Adler, 2003). Recently it has been found that, for stochastic neural networks the large-scale behavior admits a description in terms of a winner-take-all type dynamics, in such a way that the graph of charge transfers has scale-free properties with a power-law exponent $\gamma = 2.0$ (Piekniewski & Schreiber, 2008). Moreover, recent experimental evidence suggests that some brain activities can be assigned to scale-free networks, as revealed by functional magnetic resonance imaging, where the scaling exponent γ has

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been found to take on values between 2.0 and 2.2, with an average connectivity of $\langle k \rangle \approx 4$ (Chialvo, 2004; Equiluz et al., 2005; Sporns, Chialvo, Kaiser, & Hilgetag, 2004). The connection between the large-scale functional networks discussed in those works and a small-scale structural network of coupled neurons has been recently investigated by Hagmann et al. (2008) and Honey et al. (2009).

Moreover, a recent study by van der Heuvel, Stam, Boersma, and Hulshoff Pol (2008) using high-definition functional magnetic resonance imaging suggests that connectivity graphs formed out of all cortical and sub-cortical voxels have both small-world and scale-free properties, the latter having a scaling exponent around 2.0. On the other hand, Achard, Salvador, Whitcher, Suckling, and Bullmore (2006) have found that the human functional network is dominated by a neocortical core of highly connected hub-like neurons which do not obey properly a scale-free but rather have an exponentially truncated power-law degree distribution. Humphries, Gurney, and Prescott (2006), argue that the medial reticular formation (RF) of the brainstem is characterized by a neural network exhibiting small-world, but not scale-free properties.

One of the collective phenomena which arise from the network coupling is the synchronization of periodic, noisy, or even chaotic oscillations taking place at each network unit (Pikovsky, Rosenblum, & Kurths, 2003). Synchronization of oscillations are an important feature of network-coupled physical and biological systems, like arrays of coupled Josephson junctions (Wiesenfeld, Colet, & Strogatz, 1996) lasers (Roy & Thornbert Jt, 1994), and flashing fireflies (Mirollo & Strogatz, 1990). We shall be concerned particularly with neuronal networks where each unit receives excitatory inputs from a few thousands of other neurons (Bear, Connors, & Paradiso, 2002). The transition from inactive to active neural networks with scale-free architecture has been found to be a global bifurcation (López-Ruiz, Moreno, Pacheco, Boccaletti, & Hwang, 2007).

Neuronal activity (i.e., the evolution of the action potential) in cortical circuits often presents two distinct timescales: (i) a fast time scale characterized by repetitive spiking; and (ii) a slow timescale with bursting activity, where neuron activity alternates between a quiescent state and spiking trains (Belykh, de Lange, & Hasler, 2005). A characteristic feature of cortical circuits is that they produce synchronized bursting, while its individual neurons, when isolated, show irregular bursts, in such a way that synchronized bursting is a characteristic effect of the coupling neural architecture (Thomson, 2000).

The presence of synchronized rhythms has been experimentally observed in electroencephalograph recordings of electrical activity in the brain, in the form of an oscillatory behavior generated by the correlated discharge of populations of neurons across cerebral cortex. The behavioral state alters the amplitudes and frequencies of these oscillations, such that high frequency and low amplitude rhythms tend to occur during arousal and attention; whereas low frequency and high amplitude activity occurs during slow-wave sleep (Thomson, 2000).

Moreover, some types of synchronization of bursting neurons are thought to play a key role in Parkinson's disease, essential tremor, and epilepsies (Milton & Jung, 2003). As an example, the synchronous firing of neurons located in the thalamus and basal ganglia appears to cause resting tremor in Parkinson's disease, in such a way that the firing frequency is in the same range (3–6 Hz) of the tremor itself (Maistrenko, Popovych, & Tass, 2005). The peripheral shaking results from the activation of cortical areas due to the existence of a cluster of synchronously firing neurons that acts as a pacemaker (Nini, Feingold, Slovin, & Bergman, 1995). Hence a possible way to control pathological rhythms would be to suppress the synchronized behavior. This can be obtained through application of an external high frequency (>100 Hz) electrical signal, and it constitutes the main goal of the deep-brain stimulation technique (Benabid et al., 1991).

Deep-brain stimulation consists of the application of depth electrodes implanted in target areas of the brain like the thalamic ventralis intermedius nucleus or the subthalamic nucleus (Benabid et al., 1991). The overall effects of deep-brain stimulations are similar to those produced by tissue lesioning and have proved to be effective in suppression of the activity of the pacemaker-like cluster of synchronously firing neurons, so achieving a suppression of the peripheral tremor (Blond et al., 1992). While most progress in this field has come from empirical observations made during stereotaxic neurosurgery, methods of nonlinear dynamics are beginning to be applied to understand this suppression behavior. Rosenblum and Pikowsky have proposed a feedback procedure to control pathological brain rhythms through suppression of the synchronized behavior by a delayed feedback signal (Rosenblum & Pikowsky, 2004a). This strategy has been successfully applied to globally coupled networks, in which each unit interacts with all other neurons in a mean-field kind of coupling (Rosenblum & Pikowsky, 2004b).

In this letter we analyze the control of collective synchronized oscillations using a time-delayed feedback control signal in a scale-free network of bursting neurons, whose individual dynamics is governed by a two-dimensional dissipative map proposed by Rulkov (2001), see also Rulkov, Timofeev, and Bazhenov (2004). The latter describes the essentials of neuron bursting activity with some advantages over more sophisticated models like Hindmarsh–Rose equations (Dayan & Abbott, 2001), such as the use of less computer time, what makes it suitable for numerical simulations using a large number of neurons. We investigated the control parameter regimes for which there occurs bursting synchronization, and the effect of varying coupling parameters. We located domains of bursting synchronization suppression in terms of perturbation strength and time delay, and present computational evidence that synchronization suppression is easier in scale-free networks than in the more commonly studied global (mean-field) networks.

This paper is organized as follows: Section 2 deals with the model we use to describe neural networks, using a discrete map to simulate the local neuronal dynamics and a network architecture displaying the scale-free property. Section 3 discusses the ideas behind the stimulation technique using a delayed feedback signal, and how it is able bursting synchronization in the network. Our numerical results are shown in Section 4, as well as a discussion of some issues related to the influence of the particular aspects of the model we are using, as its parameters. Section 5 describes, in a semi-quantitative setting, the transition to the bursting synchronization, using the well-known Kuramoto model as a paradigm. The last Section is devoted to our conclusions.

2. Network model

2.1. Local dynamics

In the neuron models we consider in this paper, the time evolution of the action potential is supposed to exhibit two timescales. The fast timescale is related to the spiking neuron activity, whereas the slow timescale appears in the form of bursts characterized by the repetition of spikes (Dhamala, Jirsa, & Ding, 2004). Mathematical models of such bursting neurons may be built upon systems of three or more ordinary differential equations, like the models proposed by Hodgkin and Huxley (1952) or Hindmarch