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Effect of network architecture on burst and spike synchronization in a scale-free network of bursting neurons

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HIGHLIGHTS

- A directed scale-free network of bursting neurons is considered.
- Effect of network architecture on burst and spike synchronization is investigated.
- Average path length and betweenness centralization affect the synchronization.
- In-degree distribution also affects the synchronization.

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ABSTRACT

We investigate the effect of network architecture on burst and spike synchronization in a directed scale-free network (SFN) of bursting neurons, evolved via two independent α- and β-processes. The α-process corresponds to a directed version of the Barabási–Albert SFN model with growth and preferential attachment, while for the β-process only preferential attachments between pre-existing nodes are made without addition of new nodes. We first consider the "pure" α-process of symmetric preferential attachment (with the same in- and out-degrees), and study emergence of burst and spike synchronization by varying the coupling strength J and the noise intensity D for a fixed attachment degree. Characterizations of burst and spike synchronization are also made by employing realistic order parameters and statistical-mechanical measures. Next, we choose appropriate values of J and D where only burst synchronization occurs, and investigate the effect of the scale-free connectivity on the burst synchronization by varying (1) the symmetric attachment degree and (2) the asymmetry parameter (representing deviation from the symmetric case) in the α-process, and (3) the occurrence probability of the β-process. In all these three cases, changes in the type and the degree of population synchronization are studied in connection with the network topology such as the degree distribution, the average path length L_p, and the betweenness centralization B_c. It is thus found that just taking into consideration L_p and B_c (affecting global communication between nodes) is not sufficient to understand emergence of population synchronization in SFNs, but in addition to them, the in-degree distribution (affecting individual dynamics) must also be considered to fully understand for the effective population synchronization.

1. Introduction

We are concerned about population synchronization of bursting neurons. Bursting occurs when neuronal activity alternates, on a slow timescale, between a silent phase and an active (bursting) phase of fast repetitive spikings (Coombes & Bressloff, 2005; Izhikevich, 2000, 2006, 2007; Rinzel, 1985, 1987). This type of bursting activity occurs due to the interplay of the fast ionic currents leading to spiking activity and the slower currents modulating the spiking activity. Hence, the dynamics of bursting neurons have two timescales: slow bursting timescale and fast spiking timescale. Thanks to a repeated sequence of spikes in the bursting, there are many hypotheses on the importance of bursting activities in the neural information transmission (Izhikevich, 2004, 2006; Izhikevich, Desai, Walcott, & Hoppensteadt, 2003; Krahe & Gabbian, 2004; Lisman, 1997); for example, (a) bursts are necessary to overcome the synaptic transmission failure, (b) bursts are more reliable than single spikes in evoking responses

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in postsynaptic neurons, and (c) bursts can be used for selective communication between neurons. There are several representative examples of bursting neurons such as intrinsically bursting neurons and chattering neurons in the cortex (Connors & Gutnick, 1990; Gray & McCormick, 1996), thalamic relay neurons and thalamic reticular neurons in the thalamus (Lee, Govindaiah, & Cox, 2007; Llinás & Jahnsen, 1982; McCormick & Huguenard, 1992), hippocampal pyramidal neurons (Su, Alroy, Kirson, & Yaari, 2001), Purkinje cells in the cerebellum (Womack & Khodakhah, 2002), pancreatic β-cells (Chay & Keizer, 1983; Kinard, de Vries, Sherman, & Satin, 1999; Pernarowski, Miura, & Kevorkian, 1992), and respiratory neurons in pre-Botzinger complex (Butera, Rinzel, & Smith, 1999; Del Negro, Hsiao, Chandler, & Garfinkel, 1998).

These bursting neurons exhibit two different patterns of synchronization due to the slow and the fast timescales of bursting activity. Burst synchronization (synchrony on the slow bursting timescale) refers to a temporal coherence between the active phase (bursting) onset or offset times of bursting neurons, while spike synchronization (synchrony on the fast spike timescale) characterizes a temporal coherence between intraburst spikes fired by bursting neurons in their respective active phases (Omelchenko, Rosenblum, & Pikovsky, 2010; Rubin, 2007). For example, large-scale burst synchronization (called the sleep spindle oscillation of 7–14 Hz) has been found to occur via interaction between the excitatory thalamic relay cells and the inhibitory thalamic reticular neurons in the thalamus during the early stages of slow-wave sleep (Bazhenov & Timofeev, 2006; Steriade, McCormick, & Sejnowski, 1993). These sleep spindle oscillations are involved in memory consolidation (Gais, Plihal, Wagner, & Born, 2000; Sejnowski & Destexhe, 2000). In contrast, this kind of burst synchronization is also correlated with abnormal pathological rhythms associated with neural diseases such as movement disorder (Parkinson's disease and essential tremor) (Bevan, Magill, Terman, Bolam, & Wilson, 2002; Brown, 2007; Hammond, Bergman, & Brown, 2007; Park, Worth, & Rubchinsky, 2010; Uhlhaas & Singer, 2006) and epileptic seizure (Fisher et al., 2005; Uhlhaas & Singer, 2006). Particularly, for the case of the Parkinson's disease hypokinetic motor symptoms (i.e., slowness and rigidity of voluntary movement) are closely related to burst synchronization occurring in the beta band of 10–30 Hz range in the basal ganglia, while hyperkinetic motor symptom (i.e., resting tremor) is associated with burst synchronization of $\sim$5 Hz.

In this paper, we study burst and spike synchronization of bursting neurons, associated with neural information processes in health and disease, in complex networks. Synaptic connectivity in brain networks has been found to have complex topology which is neither regular nor completely random (Bassett & Bullmore, 2006; Bullmore & Sporns, 2009; Buzsáki, Geisler, Henze, & Wang, 2004; Chklovskii, Mel, & Svoboda, 2004; Larimer & Strowbridge, 2008; Song, Sjöström, Reigl, Nelson, & Chklovskii, 2005; Sporns, 2011; Sporns & Honey, 2006; Sporns, Tononi, & Edelman, 2000). Particularly, brain networks have been found to exhibit power-law degree distributions (i.e., scale-free property) in the rat hippocampal networks (Bonifazi et al., 2009; Li, Ouyang, Usami, Ikegaya, & Sik, 2010; Morgan & Soltesz, 2008; Wiedemann, 2010) and the human cortical functional network (Eguíluz, Chialvo, Cecchi, Baliki, & Apkarian, 2005). Furthermore, robustness against simulated lesions of mammalian cortical anatomical networks (Felleman & Van Essen, 1991; Scannell, Blakemore, & Young, 1995; Scannell, Burns, Hilgetag, O'Neill, & Young, 1999; Sporns, Chialvo, Kaiser, & Hilgetag, 2004; Young, 1993; Young, Scannell, Burns, & Blakemore, 1994) has also been found to be most similar to that of a scale-free network (SFN) (Kaiser, Martin, Andras, & Young, 2007). These kinds of SFNs are inhomogeneous with a few "hubs" (superconnected nodes), in contrast to statistically homogeneous networks such as random graphs and small-world networks (Albert & Barabási, 2002; Barabási & Albert, 1999). Many recent works on various subjects of neurodynamics (e.g., coupling-induced burst synchronization, delay-induced burst synchronization, and suppression of burst synchronization) have been done in SFNs with a few percent of hub neurons with an exceptionally large number of connections (Batista, Batista, de Pontes, Viana, & Lopes, 2007; Batisa, Batisa, de Pontes, Lopes, & Viana, 2009; Batista, Lopes, Viana, & Batisa, 2010; Ferrari, Viana, Lopes, & Stoop, 2015; Wang, Chen, & Perc, 2011; Wang, Perc, Duan, & Chen, 2009).

The main purpose of our study is to investigate the effect of scale-free connectivity on emergence of burst and spike synchronization in a directed SFN of bursting neurons, evolved via two independent local α- and β-processes which occur with probabilities α and β ($\alpha + \beta = 1$), respectively. The α-process corresponds to a directed version of the standard Barabási–Albert SFN model (i.e., growth and preferential directed attachment) (Albert & Barabási, 2002; Barabási & Albert, 1999). On the other hand, for the β-process only preferential attachments between pre-existing nodes are made without addition of new nodes (i.e., no growth) (Albert & Barabási, 2000, 2002; Bollobás, Borgs, Chayes, & Riordan, 2003; Dorogovtsev & Mendes, 2000). Consequently, degrees of pre-existing nodes are intensified via the β-process. These α- and β-processes occur naturally in the evolution of communication networks (e.g., world-wide web) and social networks (e.g., collaboration graph of actors or authors) (Albert & Barabási, 2000, 2002; Barabási et al., 2002; Bollobás et al., 2003; Dorogovtsev & Mendes, 2000). We expect that in addition to the growing α-process, incorporation of the β-process (intensifying the internal connections between pre-existing nodes) may be regarded as a natural extension in typical SFNs, independently of their specific nature. For details on the extended models of network evolution, refer to Refs. Albert and Barabási (2002), Albert and Barabási (2000), Bollobás et al. (2003) and Dorogovtsev and Mendes (2000) where local processes, incorporating addition of new nodes and addition or removal of connections between pre-existing nodes, are discussed. Following this line, as our brain network of bursting neurons we employ the SFN model evolved via the α- and the β-processes, as in our recent work on sparse synchronization of spiking neurons (Kim & Lim, 2015b). We also expect that generation of SFNs by preferential attachment via α- and the β-processes might be related to brain plasticity which refers to the brain's ability to change its structure and function by modifying structure and strength of synaptic connections during the development in humans (Pascual-Leone et al., 2011) and rats (Song et al., 2005). Our SFN is composed of suprathreshold Hindmarsh–Rose (HR) neurons. The HR neurons are representative bursting neurons (Hindmarsh & Rose, 1982, 1984; Rose & Hindmarsh, 1985), and they interact through inhibitory GABAergic synapses (involving the $GABA_A$ receptors). Population synchronization in the network of HR neurons with inhibitory synapses was much studied in many aspects (Che et al., 2011; Liang, Tang, Dhamala, & Liu, 2009; Pereira, Baptista, Kurths, & Reyes, 2007; Wu, Xu, & He, 2005). Following this line, we restrict our attention to only the inhibitory HR bursting neurons, although there are also many other excitatory bursting neurons (e.g., cortical chattering cells Connors & Gutnick, 1990; Gray & McCormick, 1996 and thalamic relay cells Lee et al., 2007; Llinás & Jahnsen, 1982; McCormick & Huguenard, 1992). Particularly, the sleep spindle rhythm in the reticularis thalami nucleus was studied in a population of inhibitory bursting neurons (Wang & Rinzel, 1993). We also expect that by providing a synchronous oscillatory output to the excitatory bursting cells, networks of inhibitory bursting neurons play the role of the backbones of bursting rhythms (Buzsáki, 2006; Buzsáki et al., 2004).

We first consider the case of "pure" α-process (i.e., $\alpha = 1$) with symmetric preferential attachment with the same in- and