

# Synchronized bursts following instability of synchronous spiking in chaotic neuronal networks

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We report on the origin of synchronized bursting dynamics in various networks of neural spiking oscillators, when a certain threshold in coupling strength is exceeded. These ensembles synchronize at relatively low coupling strength and lose synchronization at stronger coupling via spatio-temporal intermittency. The latter transition triggers multiple-timescale dynamics, which results in synchronized bursting with a fractal-like spatio-temporal pattern of spiking. Implementation of an appropriate technique of separating oscillations on different time-scales allows for quantitative analysis of this phenomenon. We show, that this phenomenon is generic for various network topologies from regular to small-world and scale-free ones and for different types of coupling.

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Achievements of the recent decade have given strong evidence that synchronous activity [1] plays an important role in the functioning of the nervous system and brain [2]. These examples range from coordinating movements in the motor system to information processing (recognition and perceptual binding) in the visual cortex and olfactory system. On the other hand, synchronization may play a destructive role, causing neural disorders like epileptic seizures or Parkinson's disease [3]. Moreover, the importance of desynchronization in cognitive processing is increasingly being recognized [4].

The need for the theoretical explanation of these findings stimulated extensive research in the field of nonlinear dynamics. The most simple and liable to analysis models of integrate-and-fire neurons, which mimic periodic subthreshold approach to the spiking state, were extensively studied to elucidate perfect synchronization between identical units [5] and frequency synchronization between non-identical ones [6]. The recently developed theoretical framework for chaotic synchronization paved the way to analyzing cooperative dynamics of more realistic models of chaotically spiking and bursting neurons [7]. Complete chaotic synchronization in small and large ensembles of *identical* neurons was found and methods of its prediction were developed [8].

However, in nature neurons are *not identical*. Therefore, the functional interdependence between momentary states of synchronized neurons, if any, becomes extremely complex and difficult to identify, especially in large ensembles [1]. At the same time, as chaotic synchronization has been observed in a variety of small groups of non-identical neurons [9], it gives strong grounds to expect it to appear in large ensembles too.

A promising way to make an advance here is the concept of *chaotic phase synchronization* (CPS) [10]. It implies the adjustment of *characteristic time-scales* of non-identical oscillators in course of interaction. Given an appropriate definition of phase and frequency one obtains

an efficacious tool for detecting this process. Neurons, known as multiple-time-scale systems, can generate either single spikes mediated by long intervals of silence, or trains of spikes, coined bursts. Remarkably, as we have recently shown, it is possible to identify phase synchronization on the bursting time-scale, while oscillations on the spiking time-scale are unsynchronized [11].

In this Letter we study the pathway to the formation of synchronized bursting in networks of intrinsically spiking neurons. We show that it is observed with increase of interneuronal coupling, as the networks achieve synchronous spiking, undergo its instability towards generation of bursting, which finally synchronizes. We analyze this phenomenon basing on the CPS concept and develop a proper technique to separate oscillations on spiking and bursting time-scales. We demonstrate, that, when the CPS regime gets unstable, spatio-temporal intermittency excites oscillations on the FTS, and eventually leads to the regime of synchronized bursts with a fractal-like spatio-temporal structure of the spikes. Simulations of scale-free, random, and small-world topologies give evidence of ubiquity of this phenomenon in complex neuronal networks.

We consider networks of non-identical neuronal model maps, proposed in [12]:

$$\begin{cases} x_j^{k+1} = f(x_j^k, x_j^{k-1}, y_j^k) + \varepsilon \sum_i G_{j,i}^k / K_i, \\ y_j^{k+1} = y_j^k + \mu(-x_j^k - 1 + \sigma_j + \varepsilon \sum_i G_{j,i}^k / K_i), \end{cases} \quad (1)$$

$$f(x, \tilde{x}, y) = \begin{cases} \alpha / (1 - x) + y, & \text{if } x \leq 0, \\ \alpha + y, & \text{if } 0 < x < \alpha + y \text{ and } \\ & \tilde{x} \leq 0, \\ -1, & \text{if } x \geq \alpha + y \text{ or } \tilde{x} > 0, \end{cases} \quad (2)$$

where  $x_j$  and  $y_j$  are the fast and slow variables respectively,  $j = \overline{1, N}$ . In all simulations we use  $\mu = 10^{-3}$ ,  $\alpha = 3.5$ ,  $\sigma_i \in [0.15, 0.16]$  (a uniform random distribution) that provides chaotic spiking in an isolated map;

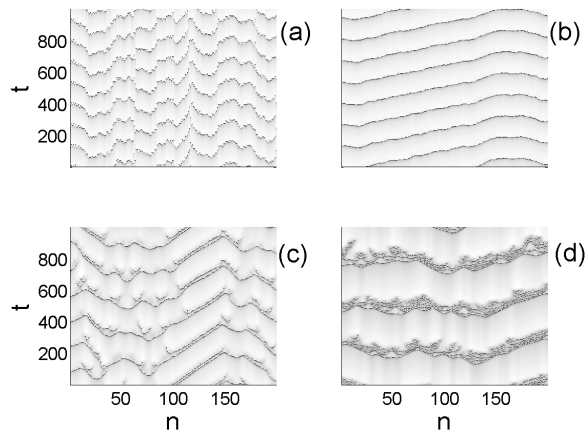


FIG. 1: Space-time plots illustrate different regimes that occur in the chain (1).  $x_j$  values are represented by colour, white corresponds to minimal values, black to maximal ones. Shown are (a) unsynchronized spiking,  $\varepsilon = 0.01$ , (b) synchronized spiking,  $\varepsilon = 0.05$ , (c) desynchronized state: synchronization is occasionally broken by fast repetitive spikes,  $\varepsilon = 0.1$ , (d) synchronized bursts with a fractal-like spatio-temporal structure of spikes,  $\varepsilon = 0.2$ .

$\varepsilon$  is the coupling strength,  $K_i$  is the number of entries in the  $i$ -th neuron. The sum is taken over all neighbours of a neuron in the network; all connections are reciprocal. The coupling function corresponds either to electrical  $G_{j,i}^k = x_j^k - x_i^k$ , or synaptical excitatory coupling  $G_{j,i}^k = (x_{rp} - x_i)\chi(x_j)$ , here the reversal potential  $x_{rp} = 1$ ,  $\chi(x) = 1$  if  $x > 0$ , and  $\chi(x) = 0$  otherwise.

To analyze the collective dynamics of this neural ensemble in terms of CPS one has to introduce frequency and phase characteristics of oscillations. For spiking dynamics we determine the average spiking frequency in neuron  $j$  by:

$$\omega_j = \lim_{k \rightarrow \infty} n_j^k / k, \quad (3)$$

where  $n_j^k$  is the number of spikes fired from the beginning up to the discrete time  $k$ . The phase of spiking reads:

$$\varphi_j^k = 2\pi \frac{k - k_{j,m}}{k_{j,m+1} - k_{j,m}} + 2\pi m_j, k_{j,m} \leq k < k_{j,m+1}, \quad (4)$$

$k_{j,m}$  being the moment of the  $m$ -th spike in neuron  $j$ .

Further on, we need the other characteristics, which correctly describe the slow time-scale (STS) oscillations in the bursting regime. The STS frequencies  $\Omega_j$  and phases  $\Phi_j$  are defined similar to their spiking time-scale counterparts  $\omega_j$  (3) and  $\varphi_j$  (4), except that not each spiking event contributes to the  $2\pi$  growth, but only the first one in a burst (in simulations, the one coming after at least 80 iterations in silent state). Note, that while neurons generate STS chaotic spiking (like for  $\varepsilon = 0$ ), both definitions are equivalent. If fast repetitive spikes form trains of bursts,  $\Omega_j$  will characterize the bursting frequency and  $\omega_j$  will characterize the average spiking fre-

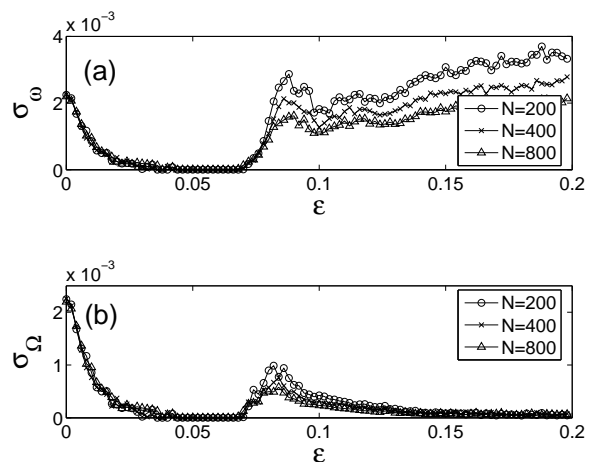


FIG. 2: Variances of slow time-scale (STS) and spiking frequencies  $\Omega_j$  and  $\omega_j$  over the chain (1) vs. coupling strength  $\varepsilon$  for different chain sizes  $N$ .

quency. This technique allows for a correct separation of the FTS and STS.

Now we summarize the regimes that occur for different coupling strengths  $\varepsilon$  in a regular *chain of electrically* coupled neurons [13]. At low coupling neuronal firings are unsynchronized (Fig.1(a)), at moderate coupling they get synchronized (Fig.1(b)). Suddenly, as we increase  $\varepsilon$ , the CPS regime becomes unstable and neurons start firing fast repetitive spikes occasionally (Fig.1(c)). Further increase of  $\varepsilon$  results in the regime of synchronized bursts with a fractal-like spatio-temporal structure of spikes (Fig.1(d)).

To quantify these transitions we have computed the variances of the STS and spiking time-scale oscillations frequencies vs. the strength of the electrical coupling for the chain lengths  $N = 200, 400, 800$  (Fig.2). We find three size-independent critical coupling strengths:  $\varepsilon_1 \approx 0.035$ ,  $\varepsilon_2 \approx 0.07$ , and  $\varepsilon_3 \approx 0.15$ , which define four intervals: (i) for  $\varepsilon \in [0, \varepsilon_1]$  oscillations on the single existing time-scale – the slow one – are unsynchronized, (ii) for  $\varepsilon \in [\varepsilon_1, \varepsilon_2]$  oscillations on the STS are synchronized, (iii) for  $\varepsilon \in [\varepsilon_2, \varepsilon_3]$  oscillations on the second time-scale – the fast one – are initiated, both time-scales demonstrate unsynchronized oscillations, (iv) and for large coupling  $\varepsilon > \varepsilon_3$  oscillations on the STS get synchronized again, oscillations on the FTS are still unsynchronized.

The transition to CPS at  $\varepsilon_1$  is what one would have intuitively expected *a priori*, as long as this is a conventional way how arrays of non-identical oscillators behave [1], but the instability of CPS at  $\varepsilon_2$  and the generation of the FTS by repetitive spikes further on demand a detailed study. To uncover the nature of this transition, we record the interspike intervals  $T_s$  in each neuron and plot their evolution for different coupling strengths (Fig.3). For  $\varepsilon < \varepsilon_2$  we observe that chaotic spikes construct only the STS ( $T_s > 100$ ), be it unsynchronized (Fig.3(a)) or

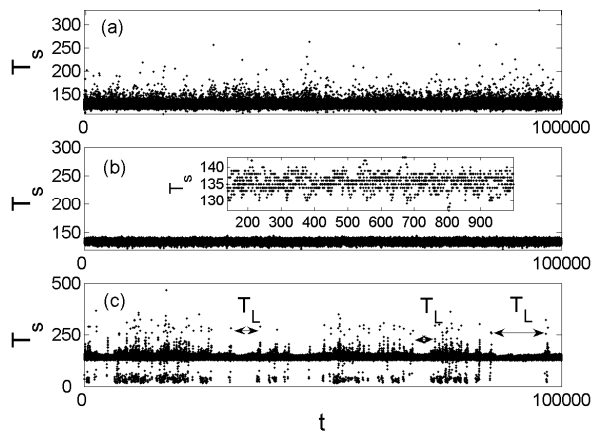


FIG. 3: Evolution of interspike intervals for (a)  $\varepsilon = 0.01$ , (b)  $\varepsilon = 0.05$ , (c)  $\varepsilon = 0.073$ . In (c) we denote several intervals of stability windows  $T_L$ .

synchronized (Fig.3(b)) dynamics. Note that in the synchronization regime the relative spike timing in neurons is locked but not tightly fixed. It varies from one spiking front to another one exhibiting flexibility of phase, typical of genuine CPS (see the inset in Fig.3(b)).  $T_s$  sequences (Fig.3(c)) demonstrate the intermittent nature of the developing instability. The time intervals, during which fast repetitive spikes are generated, are interrupted by windows of synchronized STS spiking (in Fig.3(c) one of them lasts as long as  $T_L \approx 10,000$  iterations, which is of the order of 100 STS interspike intervals). The closer  $\varepsilon$  is to  $\varepsilon_2$ , the larger become stable CPS windows  $T_L$ . In Fig.4(a),(b) we show statistical properties of interspike intervals  $T_s$  and time durations of stable windows  $T_L$ , respectively. Remarkably, the probability distributions of  $T_L$  demonstrate a power-law dependence over five decades in a *finite* interval of the coupling strength with  $\varepsilon$ -dependent exponents.

The shown complexity arises on the micro-scale, when a spike in an adjacent neuron makes a just fired neuron fire again (Fig.4(c)) should the coupling be strong enough. It is important to underline the principal role of the individual dynamics of oscillators in synchronization and desynchronization processes. In the classical case of coupled quasiharmonic oscillators the variables change smoothly and the coupling tries to synchronize the systems all the time. In case of spiking neurons, the coupling synchronizes them until the faster neuron fires. Its firing is also a synchronizing event, as it pushes the slower oscillator up. On the opposite, firing of the slower neuron desynchronizes them, as it pushes the faster one up towards the next firing. Varying  $\varepsilon$  we change the balance between synchronization and desynchronization and observe the instability of synchronization when short desynchronizing intervals prevail.

In addition, we implemented *synaptical excitatory* coupling instead of the *electrical* one and found no qualitative

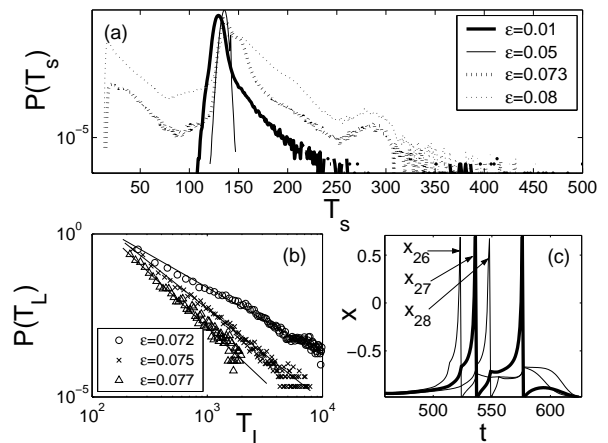


FIG. 4: Probability distributions of (a) interspike intervals  $T_s$  and (b) duration of stable CPS windows  $T_L$  for different values of the coupling strength (solid lines are guides for the eye). (c) Dynamics of fast variables  $x_j$  in three neighbored neurons. It is shown, how a spike in the 28-th neuron makes the 27-th neuron (bold line) fire a repetitive spike.

difference with respect to the shown effects. We would like to stress that the observed instability of the synchronous regime is not an artefact of a time-discrete system. We have observed it in ensembles of non-identical Hindmarsh-Rose neuronal oscillators too [14].

Next we study whether these results are valid in complex neuronal networks with a long-range synaptic connectivity [15], that is typical of biological ensembles. In the following we implement two types of complex topologies: scale-free and small-world ones [16]. Our interest in such networks has been additionally stimulated by the recent study [17], that reported scale-free properties of functional brain networks (with the exponent varying from 2.0 to 2.2). The scale-free network, we simulate, is characterized by the node degree distribution  $P(K) \propto K^{-\gamma}$ ,  $\gamma = 2.2$ , and the mean  $\langle K \rangle \approx 4.2$ . The small-world network has on the average 10 links per neuron and the probability of rewiring a short-range regular link is  $p = 0.1$ . The results of the simulations (Fig.5(a,b)) demonstrate the same scenario of the onset of bursting via instability of synchronized chaotic spiking. This similarity becomes quite natural, as one takes into account, that fast repetitive spiking is the result of interaction of two neighbours, which fire with a mismatch in time, as discussed above. Thus, the neighbour-to-neighbour interactions, and not the global architecture, are important.

What for topology does matter, is the global coherence. Having in mind, that precise timing of synchronized oscillations in biological ensembles is considered to be a functional means in cognitive tasks [2], we calculate the order parameter (i.e. coherence) for the STS oscillations:  $\rho = |\sum e^{i\Phi_j}|$ , Fig.5(c). In complex networks with long-range connectivity STS firing is tightly locked within 10 time durations of a single spike, while in lo-

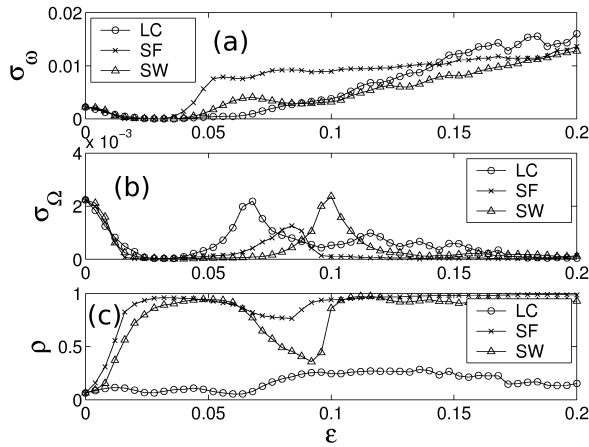


FIG. 5: Variances of (a) STS and (b) spiking frequencies  $\Omega_j$  and  $\omega_j$  and (c) the order parameter of the STS oscillations  $\rho$  vs. excitatory coupling strength for local coupling (LC), scale-free (SF), and small-world (SW) network topologies. Here  $N = 200$  and all data are averaged over 100 realizations of network topologies and random parameters of individual neurons.

cally coupled ensembles the global coherence is absent (Fig.5(c),1(b)). Thus the long-range coupling strength, subjected to synaptic plasticity, appears to be a plausible way for dynamical altering between coherent and non-coherent performance, suggesting, in turn, a mechanism for information processing in biological networks.

In summary, we have shown that ensembles of non-identical neurons generate an instability of synchronous chaotic spiking, as the coupling strength is increased. Arising spatio-temporal intermittency gives birth to bursting dynamics, which at stronger coupling becomes synchronous. This phenomenon has proved to be generic with respect to the type of coupling and network architecture. Beside general interest from the viewpoint of theoretical nonlinear dynamics, these findings may directly apply to neurobiological systems, indicating, that (i) excessive coupling does not necessarily improve synchrony of spiking, and (ii) it is also the population dynamics, not only individual parameters of neurons, that is responsible for forming spiking or bursting regimes. And we strongly expect the reported effects to be observed in biological experiments.

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- [1] A. S. Pikovsky, M.G. Rosenblum, and J. Kurths, Synchronization-A Universal Concept in Nonlinear Sciences (Cambridge University Press, 2001); S. Boccaletti, J. Kurths, G. Osipov, D. L. Valladares, and C. S. Zhou, Phys.Rep., **366**, 1 (2002).
- [2] A.K.Engel, P.Fries, and W.Singer, Nat. Rev. Neurosci. **2**, 704 (2001); L. M. Ward, Trends Cogn. Sci. **7**, 553 (2003); A. Schnitzler and J. Gross, Nature Rev. Neurosci. **6**, 285 (2005); M. I. Rabinovich et al., Rev. Mod. Phys. (to appear).
- [3] W. D. Hutchison *et al.*, J. Neurosci. **24**, 9240 (2004); I. Timofeev and M. Steriade, Neuroscience **123**, 299 (2004); P. Tass *et al.*, Phys. Rev. Lett. **81**, 3291 (1998).
- [4] E. Rodriguez *et al.* Nature **397**, 430 (1999); M. Breakspear *et al.*, J. Comput. Neurosci. **16**, 49 (2004).
- [5] L. F. Abbott, J. Phys. A **23**, 3835 (1990); R. E. Mirollo and S. H. Strogatz, SIAM J. Appl. Math. **50**, 1645 (1990); Y. Kuramoto, Physica D **50**, 15 (1991).
- [6] M. Tsodyks, I.Mitkov, and H. Sompolinsky, Phys. Rev. Lett. **71**, 1280 (1993); S. Bottani, Phys. Rev. Lett. **74**, 4189 (1995); C. C. Chow, Physica D **118**, 343 (1998); P. S. Bressloff and S. Coombes, Phys. Rev. Lett. **81**, 2168 (1998);
- [7] A. L. Hodgkin and A. F. Huxley, J. Physiol. **117**, 500 (1952); J. L. Hindmarsh and M. Rose, Proc. R. Soc. B **221**, 87 (1984).
- [8] L. M. Pecora and T. L. Carroll, Phys. Rev. Lett. **80**, 2109 (1998); L. M. Pecora, Phys. Rev. E **58**, 347 (1998); M. Dhamala, V. K. Jirsa, and M. Ding, Phys. Rev. Lett. **92**, 074104 (2004); I. Belykh, E. de Lange, and M. Hasler, Phys. Rev. Lett. **94**, 188101 (2005).
- [9] H. D. Abarbanel *et al.*, Neural Comput. **8**, 1567 (1996); R. C. Elson *et al.*, Phys. Rev. Lett. **81**, 5692 (1998); N.F.Rulkov, Phys. Rev. Lett. **86**, 183 (2001).
- [10] M. Rosenblum, A. Pikovsky, and J. Kurths, Phys. Rev. Lett. **76**, 1804 (1996).
- [11] M. V. Ivanchenko *et al.* Phys. Rev. Lett. **93**, 134101 (2004).
- [12] N.F. Rulkov, Phys. Rev. E **65**, 041922 (2002); N.F. Rulkov, I. Timofeev, M. Bazhenov, J. Comp. Neuroscience, **17** (2004) 203.
- [13] G. V. Osipov *et al.*, Phys. Rev. E **71**, 056209 (2005).
- [14] An extended paper comprising these results will follow.
- [15] Note, that electrical coupling between biological neurons employs gap junctions, i.e. electrical connections are necessarily short range ones, if not local.
- [16] S. H. Strogatz, Nature **410**, 268 (2001); R. Albert and A. L. Barabási, Rev. Mod. Phys. **74**, 47 (2002).
- [17] V.M. Eguiluz *et al.*, Phys. Rev. Lett., **94**, 018102 (2005).