PHYSICAL REVIEW E **76**, 015101(R) (2007)

Emergence of self-sustained patterns in small-world excitable media

Sitabhra Sinha,[1] Jari Saramäki,[2] and Kimmo Kaski[2]
[1]*The Institute of Mathematical Sciences, CIT Campus, Taramani, Chennai 600 113, India*
[2]*Laboratory of Computational Engineering, Helsinki University of Technology, P. O. Box 9203, Helsinki, FIN-02015 HUT, Finland*

(Received 25 July 2006; revised manuscript received 14 June 2007; published 9 July 2007)

Motivated by recent observations that long-range connections (LRCs) play a role in various brain phenomena, we have observed two distinct dynamical transitions in the activity of excitable media where waves propagate both between neighboring regions and through LRCs. When the LRC density p is low, single or multiple spiral waves are seen to emerge and cover the entire system. This state is self-sustaining and robust against perturbations. At $p=p_c^l$, the spirals are suppressed, and there is a transition to a spatially homogeneous, temporally periodic state. Finally, above $p=p_c^u$, activity ceases after a brief transient.

DOI: 10.1103/PhysRevE.76.015101 PACS number(s): 82.40.Ck, 05.65.+b, 87.18.Bb, 89.75.Hc

Pattern formation in excitable media has recently been a very exciting area of research, not least because it is observed in a wide variety of natural systems, ranging from spiral waves in the mammalian brain [1] to chemical systems such as the Belousov-Zhabotinsky (BZ) reaction [2]. The functional role of such patterns in biological systems makes it imperative to understand better the conditions in which they can spontaneously emerge. For example, these patterns have been implicated in the genesis of life-threatening arrhythmias in the heart [3], while in the brain they have been thought to provide a spatial framework for cortical oscillations [1]. Until now, work has mostly focused on generating patterns in excitable media by using stochastic stimulation [4]. In this case, however, the activity is not really self-sustained as the noise is akin to external intervention necessary for the initiation and persistence of spiral waves [5]. If such patterns are to be seen as spontaneously emerging from arbitrary initial conditions, then the pattern formation should be an outcome of the internal structure of the system alone. Furthermore, small variations in this structure may result in transitions between different dynamical regimes characterized by distinct spatiotemporal patterns.

Here we have considered excitable systems which have a regular topology with cells communicating only with nearest neighbors, but where there are a few random long-range connections, linking spatially distant cells. Such "small-world" topologies have been observed in a large number of real-world systems [6], and have also been associated with self-sustained activity in a chain of model neurons [7], as well as periodic epidemic patterns in disease spreading [8]. However, while it has been shown that such long-range connections do play a role in maintaining spiral waves that already exist [9], so far there have been very few attempts at showing the emergence of, and transitions between, different types of self-sustained patterns as a result of such topology. The observation of *spontaneous* pattern formation in natural systems where sparse long-range connections coexist with fairly regular underlying connection topology, as in the brain [10], points toward intriguing possibilities for the functional role of these connections. In this Rapid Communication, we have investigated a generic model of excitable media with increasing density of random long-range connections, and we report the existence of two qualitatively different regimes of self-sustained pattern formation. The correspondence of the observed patterns with those observed in nature, e.g., epileptic bursts and seizures [11], as well as their dependence on the topological structure of connections, makes these results highly relevant in our view.

The model we consider here consists of a two-dimensional array of $N\times N$ excitable cells coupled diffusively,

$$x_{t+1}^{i,j}=(1-D)f(x_t^{i,j},y_t^{i,j})+\frac{D}{4}\sum_{q=\pm1}f(x_t^{i+q,j+q},y_t^{i+q,j+q}).$$

Here, D is the diffusion coefficient and the dynamics of individual cells are described by a pair of variables x_t, y_t, evolving according to a discrete-time model of generic excitable media [12]:

$$x_{t+1}=f(x_t,y_t)=x_t^2e^{(y_t-x_t)}+k,$$

$$y_{t+1}=g(x_t,y_t)=ay_t-bx_t+c,$$

where we have fixed the parameters as $a=0.89$, $b=0.6$, $c=0.28$, and $k=0.02$ (i.e., within the range of values for which the model shows excitable behavior). When this system is excited with a suprathreshold stimulation, the fast variable x shows an abrupt increase. This triggers changes in the slow variable y, such that the state of the cell is gradually brought down to that of the resting state. Once excited, the cell remains impervious to stimulation up to a refractory period, the duration of which is governed by the parameter a. Neighboring cells communicate excitation to each other with a strength proportional to D, chosen as $D=0.2$ for most of our simulations. In addition to the diffusive coupling, we introduce long-range connections such that each cell receives a connection from a randomly chosen cell with probability p. The strength of this connection is chosen to be the same as that of the nearest neighbors, i.e., $D/4$. These random long-range links can be either quenched (i.e., chosen initially and kept fixed for the duration of the simulation), or annealed (i.e., randomly created at each time step). While the results reported below are for annealed random links, we could observe no qualitative difference between these two cases. Note that we have used both periodic and absorbing boundary conditions for the system, and observed no significant differ-

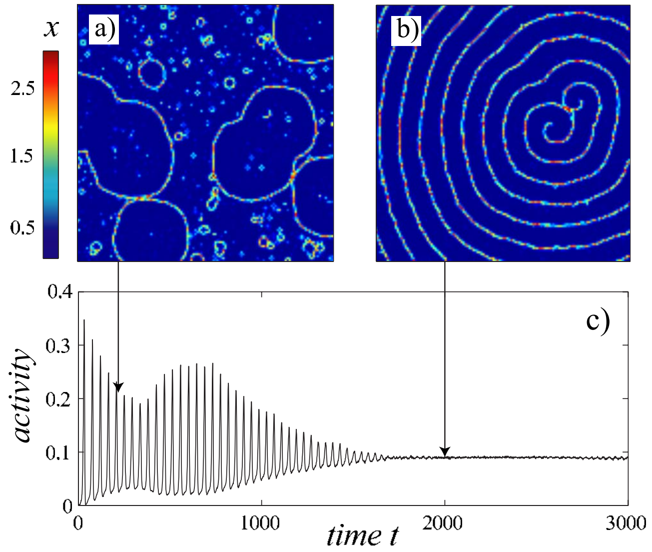

FIG. 1. (Color online) Emergence of spiral waves, recorded in a simulation of a $N^2=128\times128$ system, with shortcut probability $p=0.25$. After initialization, the dynamics of the system can be characterized by circular waves. At around $t\sim1500$ time steps, spiral wave are spontaneously created and subsequently takes over the dynamics. (a) and (b) show the state of the system at different times in terms of the fast variable x. Colors indicate excitation level of the cells. (c) Time series of the average activity, i.e., the fraction of cells with $x>0.9$.

ence in the results. All the following results were obtained for absorbing boundary conditions.

In the simulations, the observed patterns formed in the system for varying values of the shortcut density p can, generally speaking, be divided into three categories. First, below the lower critical probability, i.e., $0<p<p_c^l$, the state of the system after an initial transient period is characterized by self-sustaining single or multiple spiral waves covering the entire system. Second, at $p=p_c^l$, the spiral wave mode is suppressed and the system undergoes a transition to a regime in which a large fraction of the system gets simultaneously active, and then refractory, in a periodic manner. Third, when the value of p is increased above a system-size-dependent upper critical probability p_c^u, the self-sustained activity ceases and the system falls into the absorbing state where $x^{i,j}=0, \forall\ i,j$.

In the beginning of each simulation run, the system was initialized such that $x=1$ for a small number of cells, and $x=0$ for the rest. For small shortcut probabilities $p\ll p_c^l$, upon starting the simulation, multiple coexisting circular excitation waves were seen to emerge [see Fig. 1(a)], to be later taken over by spiral waves [Fig. 1(b)]. The activity time series in Fig. 1(c), displaying the fraction of cells where $x>0.9$, shows a high-frequency periodicity corresponding to the refractory period; then the periodicity disappears as the system settles into the spiral wave mode. For the time series preceding the onset of the spiral wave, we typically observe slow modulations of the envelope of the periodic oscillations, which arise from the interaction between the waves as well as from the shortcut-induced excitations. Power spectral densities of such series were observed to show a power-law-like decay, indicating the presence of $1/f$ noise.

The spiral waves were observed to be primarily created by a shortcut-induced excitation occurring in the refractory "shadow" of a circular wave front, sparking a semicircular wave whose transmission is partially hindered by the shadow. We verified that, indeed, spiral waves could be triggered in this fashion by using an externally applied signal to stimulate an appropriate point in this region and then observing the resultant patterns. Once a spiral wave is created, it will eventually take over the dynamics of the system, as the successive excitation wave fronts occur with the highest frequency compared to all other excitations, which will then be swept away. Evidently, the probability of a spiral wave creation per unit time increases with the system size N^2 and the shortcut probability p. Thus, for long times and large system sizes, excitation via random shortcuts will eventually always result in the formation of spiral waves. This is corroborated by Fig. 2, where the fraction of spiral wave configurations in 400 simulation runs is shown as function of time t, for varying system sizes at $p=0.05$ [Fig. 2(a)] and for fixed system size but increasing values of p [Fig. 2(b)]. The spatial structure of the spiral waves, i.e., an excitation front followed by a refractory shadow, makes them very robust against perturbations. For example, if the state of the system is frozen, and the state of large areas (say, up to a quarter of the total area) or every second cell are set to $x=0$, the spiral wave mode is quickly recovered once the simulation is restarted.

However, at high enough values of p, the shortcut-induced excitations become too numerous for sustaining the spiral wave dynamics. As almost every point is liable to be excited with a frequency proportional to its refractory period, the spirals become unstable and we see a transition to a new regime at $p=p_c^l\approx0.553$ (Fig. 3). This value of p_c was found to be independent of the system size N^2. Here, the spatial pattern becomes more homogeneous, as a large fraction of the system becomes simultaneously active and subsequently

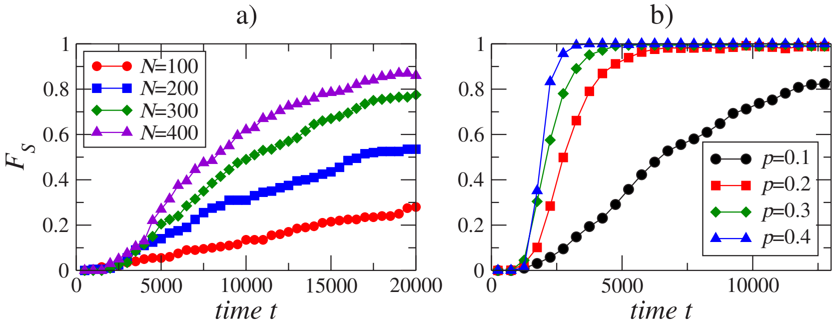

FIG. 2. (Color online) Fraction F_S of systems with spiral waves (averaged over 400 different random realizations of shortcuts) as function of time t: (a) Fixed shortcut probability $p=0.05$ with varying system size; (b) fixed system size $N^2=300\times300$ with varying shortcut probability p.

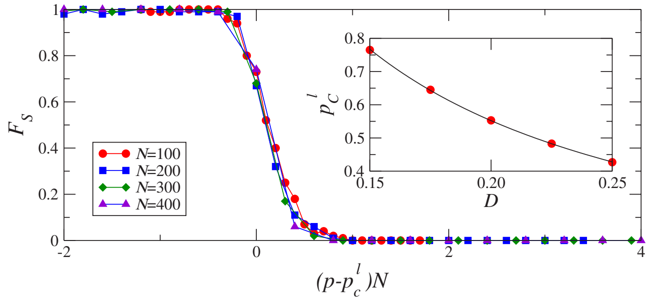

FIG. 3. (Color online) Fraction of spiral configurations F_S as a function of $(p-p_c^l)$ normalized by system size N, for shortcut probabilities p around $p_c^l \approx 0.553$. F_S is calculated at $t=20\,000$ time steps, averaged over 400 runs. Inset: Dependence of the critical value p_c^l on diffusion constant D. The circles are simulation results for $N=200$ while the curve shows fitting with $p_c^l \sim D^{-1.14}$.

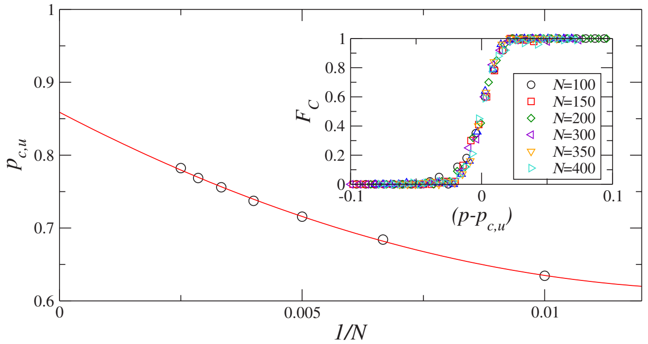

FIG. 5. (Color online) Inset: Fraction of configurations F_C where activity has ceased at $t=20\,000$ time steps in 100 simulation runs as function of $(p-p_c^u)$. Mainpanel: The upper critical shortcut probability p_c^u as a function of inverse system dimension $1/N$ (∘). The solid line displays a fitted quadratic function, where $p_c^u(N \to \infty) \approx 0.86$.

decays to a refractory state. However, some cells not participating in this wave of excitation carry on the activity to the next cycle, where it again spreads through almost the entire system. This results in a remarkably periodic behavior of the system in time, with a large fraction of cells being recurrently active with a period close to the refractory period of the cells [see Figs. 4(e) and 4(f)]. Often, small spiral-like waves were also observed [Figs. 4(a)–4(d)], but these were short lived and spatial correlations could not be maintained.

Finally, when p is increased still further, the very large number of shortcut connections guarantees almost simultaneous spread of excitation to nearly all cells. As a result, the dynamics of the system tends to "burn out," such that, after a transient, almost all cells become refractory and not enough susceptible cells are left to sustain the excitation. We observed that once a system-size-dependent value $p=p_c^u(N)$ is approached, the probability of reaching the absorbing state $(x^{i,j}=0, \forall\ i,j)$ grows rapidly (see Fig. 5) such that at high enough values of p, the system is never seen to sustain its activity. The limiting behavior for $N \to \infty$ was investigated by plotting p_c^u against $1/N$ and extrapolating its value for $1/N \to 0$. Fitting a quadratic function yielded $p_c^u \to 0.86$; hence it appears that the regime where activity is sustained has for all system sizes an upper limit for the shortcut density p. Note that this can be viewed as an approximation only, because there is no *a priori* reason to assume any particular form for the dependence of p_c^u on N.

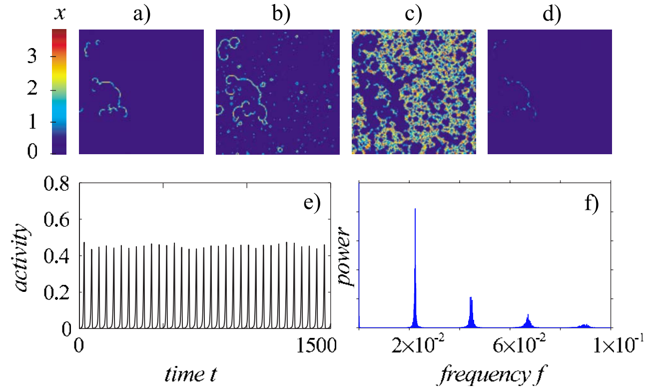

FIG. 4. (Color online) Periodic regime, recorded in a simulation of a $N^2=128\times 128$ system, with shortcut probability $p=0.6$. (a)–(d) Snapshots of the state of the system taken at intervals of $\Delta t=10$ steps. (e) Time series of the activity (see Fig. 1) and (f) the corresponding power spectrum. The main peak is at $f_0 \approx 0.022$, corresponding to a wavelength of ≈43 time steps; other peaks are harmonics at integer multiples of f_0.

We have also carried out simulations with disorder in the parameters describing the properties of individual cells. For instance, we have made the parameter a that controls the refractory period a quenched random variable ranging over a small interval. We find that there is a tendency for greater fragmentation of waves with disorder, and corresponding increase in the number of coexisting spiral waves, but otherwise no remarkable changes. The robustness of our results in the presence of disorder in the individual cellular properties underlines the relevance of this study to real-world systems, where cells are unlikely to have uniform properties. In addition, we have looked at the effect of the diffusion constant D. For higher D, the wave propagates much faster, so that, in a fixed amount of time, the system will initiate excitation through many more long-range connections than the system with lower D. Therefore, within the limits we have considered, the higher-D system will be equivalent to a lower-D system with a larger number of long-range connections, i.e., higher p (Fig. 3, inset).

The above results carry potential relevance for all natural systems that are excitable and have sparse, long-range connections. Experiments carried out in the excitable BZ reaction system with nonlocal coupling [13] have exhibited some of the features described above. For example, the coexistence of transient activity and sustained synchronized oscillations that we observe close to p_c^u has been reported in the above system, and can now be understood in terms of the model introduced here. Recently, there has also been a lot of research activity on the role of nontrivial network topology in brain function (e.g., Ref. [14]). In particular, studies show a connection between the existence of small-world topology and epilepsy [11,15]. This is one of the areas where our results can have a possible explanatory role. In the brain, glial cells form a matrix of regular topology with cells communicating between their nearest neighbors through calcium waves. Neurons are embedded on this regular structure and are capable of creating long-range links between spatially distant regions. It is now known that neurons and glial cells can communicate with each other through calcium waves [16]. Therefore, the aggregate system of neurons and glial cells can be seen as a small-world network of excitable cells. Here the neurons, which are outnumbered by glial cells approximately by one order of magnitude, form the sparse, long-range connections. Recently, the role of neural-glial communication in epilepsy has been investigated (e.g., Ref. [17]), but the observation of spiral intercellular calcium waves in the hippocampus [10], and the similarity of the patterns seen in our model with the observed features of epileptic seizures and bursts, makes it especially appealing to postulate the role of small-world topology in the generation of epilepsy. Experimental verification of this suggested scenario can be performed through calcium imaging in glial-neuronal coculture systems.

The relationship between the fraction of long-range connections and normal brain function is indicated by experimental evidence not just for epilepsy but for various other mental disorders as well. A recent study has found that the brains of patients with clinically diagnosed schizophrenia, depression, or bipolar disease have lower glia to neuron ratio compared to normal subjects [18]. Therefore, understanding the dynamical ramifications of increasing shortcuts in an excitable medium can motivate experiments that have the potential significance of aiding clinical breakthroughs in treating a whole class of mental disorders. This is also connected with the question of the evolutionary significance for increasing glia to neuron ratio with brain size [19]. It is known that excitable media of larger dimensions are more likely to exhibit spiral waves [20]. Therefore, decreasing the fraction of neurons (and therefore, long-range shortcuts) could be Nature's way of ensuring dynamical stability for neural activity.

To conclude, we have investigated spontaneous pattern formation in excitable media with small-world connections. Our results show the creation of nontrivial spatiotemporal patterns similar to those seen in many real-life systems. These patterns are formed through dynamics that is driven by the system's own internal architecture. Most important of all, the system exhibits a nontrivial transition point at which the pattern goes from the spatial domain, with multiple coexisting spiral waves where the global activity level remains more or less uniform, to the temporal domain, where the global activity level shows large oscillations as a large fraction of cells becomes simultaneously active and then refractory, with a strict periodicity. The connection to biological phenomena, most importantly, to calcium waves in the brain, and the possibility of the functional role of small-world connections in epileptic seizures and bursts, are expected to make our study of special relevance.

---

[1] X. Huang, W. C. Troy, Q. Yang, H. Ma, C. R. Laing, S. J. Schiff, and J.-Y. Wu, J. Neurosci. **24**, 9897 (2004).

[2] A. N. Zaikin and A. M. Zhabotinsky, Nature (London) **225**, 535 (1970).

[3] R. A. Gray, A. M. Pertsov, and J. Jalife, Nature (London) **392**, 75 (1998); F. X. Witkowski, L. J. Leon, P. A. Penkoske, W. R. Giles, M. L. Spano, W. L. Ditto, and A. T. Winfree, *ibid.* **392**, 78 (1998).

[4] J. García-Ojalvo and L. Schimansky-Geier, Europhys. Lett. **47**, 298 (1999); C. B. Muratov, E. Vanden-Eijnden, and E. Weinan, Proc. Natl. Acad. Sci. U.S.A. **104**, 702 (2007).

[5] P. Jung and G. Mayer-Kress, Chaos **5**, 458 (1995); Z. Hou and H. Xin, Phys. Rev. Lett. **89**, 280601 (2002).

[6] D. J. Watts and S. H. Strogatz, Nature (London) **393**, 440 (1998).

[7] A. Roxin, H. Riecke, and S. A. Solla, Phys. Rev. Lett. **92**, 198101 (2004); see also S. Yonker and R. Wackerbauer, Phys. Rev. E **73**, 026218 (2006).

[8] M. Kuperman and G. Abramson, Phys. Rev. Lett. **86**, 2909 (2001).

[9] D. He, G. Hu, M. Zhan, W. Ren, and Z. Gao, Phys. Rev. E **65**, 055204(R) (2002).

[10] M. E. Harris-White, S. A. Zanotti, S. A. Frautschy, and A. C. Charles, J. Neurophysiol. **79**, 1045 (1998).

[11] T. I. Netoff, R. Clewley, S. Arno, T. Keck, and J. H. White, J. Neurosci. **24**, 8075 (2004).

[12] D. R. Chialvo, Chaos, Solitons Fractals **5**, 461 (1995).

[13] M. Tinsley, J. Cui, F. V. Chirila, A. Taylor, S. Zhong, and K. Showalter, Phys. Rev. Lett. **95**, 038306 (2005); A. J. Steele, M. Tinsley and K. Showalter, Chaos **16**, 015110 (2006).

[14] V. M. Eguíluz, D. R. Chialvo, G. A. Cecchi, M. Baliki, and A. V. Apkarian, Phys. Rev. Lett. **94**, 018102 (2005).

[15] B. Percha, R. Dzakpasu, M. Žochoswski, and J. Parent, Phys. Rev. E **72**, 031909 (2005).

[16] A. C. Charles, Dev. Neurosci. (Basel, Switz.) **16**, 196 (1994).

[17] S. Nadkarni and P. Jung, Phys. Rev. Lett. **91**, 268101 (2003).

[18] R. A. Brauch, M. A. El-Masri, J. C. Parker, and R. S. El-Mallakh, JFMA **91**, 87 (2006).

[19] A. Reichenbach, Glia **2**, 71 (1989).

[20] S. Sinha, A. Pande, and R. Pandit, Phys. Rev. Lett. **86**, 3678 (2001).