Physics Letters A 378 (2014) 1579–1583

Contents lists available at ScienceDirect

Physics Letters A

www.elsevier.com/locate/pla

A reduced gradient description of stochastic-resonant spatiotemporal patterns in a FitzHugh–Nagumo ring with electric inhibitory coupling

A.D. Sánchez, G.G. Izús, M.G. dell'Erba, R.R. Deza *

Physics Department—FCEyN, National University of Mar del Plata (UNMdP) and IFIMAR (Mar del Plata Institute for Physical Research, UNMdP & CONICET), Deán Funes 3350, B7602AYL Mar del Plata, Argentina

ARTICLE INFO

Article history:
Received 3 January 2014
Received in revised form 25 March 2014
Accepted 28 March 2014
Available online 16 April 2014
Communicated by C.R. Doering

Keywords:
Synchronization and coupled oscillators
Neural networks and synaptic communication
Noise in biological systems

ABSTRACT

We study the synchronization (sync) properties of a ring of N units with *excitable* FitzHugh–Nagumo dynamics, when the *inhibitor* fields of nearest-neighbor units are coupled *diffusively* (electric coupling). The system is submitted to a common *subthreshold* adiabatic signal $S(t)$, and independent Gaussian white noises with common variance η. By running numerical integrations with increasing η, we observe the excitation activity to become spatiotemporally self-organized, until η is so strong that spoils sync. By means of a two-cell model *and projecting the dynamics along the slow manifolds*, we obtain a (signal-dependent) potential landscape which explains qualitatively the sync regime, and whose barrier heights give a good estimate of the optimal noise intensity.

1. Introduction

This work enrolls in the study of constructive effects of noise on the dynamics of complex systems [1–3], a field of ever increasing interest and activity during almost half a century which has uncovered phenomena like stochastic resonance [4–7], coherence resonance [8], and noise-sustained synchronization [9,10] in nonlinear dynamical systems. Our focus is the study of synchronization processes in populations of interacting nonlinear oscillators, a topic of particular relevance to understand key issues in neuroscience, where single neurons are often described as relaxation oscillators [11] displaying *excitable* behavior. This means that suprathreshold perturbations to their quiescent state (the stable stationary state of the cross-membrane potential) elicit "action potentials", in which the relaxation to the rest state proceeds through large excursions in the space of the model variables.

Complex systems (e.g. networks) made of excitable units usually display nontrivial behavior. Take for instance the FitzHugh–Nagumo (FHN) model [12,13], a two-component reduction of the Hodgkin–Huxley one and an archetypal model of activator–inhibitor systems, capable of displaying periodic oscillations, stable fixed points, and excitability [14]. This model has been largely used as battle horse to shed light on the dynamics of neural [15] and cardiac tissues [16], but also to discover a host of noise-induced phenomena. A known example is *coherence resonance* [8], appearing as a nonlinear response to a purely noisy excitation. In neuronal dynamics it is well established [17–19] that noise leads to various key effects, like stochastic resonance [4,17,20–22] and noise-assisted synchronization [11,23–26].

In previous study [27–30] we have been addressing the noise-sustained synchronization of a ring of autonomous units with excitable FHN dynamics, coupled through their activator fields but in phase-repulsive way. In all the cases, we found noise (and coupling)-induced nonequilibrium structures called by us *antiphase state* (APS), in which the neurons on the ring alternate regularly (except for noise-induced defects) their excitation states. Antiphase coupling plays an important role in circadian oscillation in the brain [31], synthetic genetic oscillators [32], the dynamics of astrocyte cultures [33], and has been used to investigate several aspects in the dynamics of neuronal and FHN coupled models [33–37] as well as Hodgkin–Huxley neurons [38,39]. In particular, we have made theoretical estimations of the noise thresholds for activation and synchronization of the APS in FHN system. This analysis was facilitated by the knowledge for this system [40,41] of a *nonequilibrium potential* (NEP) [42]. The NEP is the non-equilibrium analog of a free energy, and provides deep insight on the dynamical mechanisms leading to pattern formation and other phenomena where fluctuations play a constructive role [2,43]. Here we show that the same kind of stochastic-resonant spatiotemporal self-organization—featured with both APS and enhancement of the system's output—can be generated through *electric inhibitory*

* Corresponding author.
E-mail addresses: sanchez@mdp.edu.ar (A.D. Sánchez), izus@mdp.edu.ar (G.G. Izús), matiasdellerba@gmail.com (M.G. dell'Erba), deza@mdp.edu.ar (R.R. Deza).

http://dx.doi.org/10.1016/j.physleta.2014.03.048
0375-9601/

couplings, and that the resonant dynamics can be explained in terms of noise-sustained transitions between NEP attractors, as they exchange adiabatically their relative stability obeying the slow subthreshold signal. These are the fundamental ingredients driving the dynamics and determining the relevant noise scales. In the present case, the full NEP is not known. Hence, in order to obtain a NEP structure enabling to calculate barrier heights, we first resort to a projecting on the slow manifolds.

In Section 2 we briefly review the dynamic equations of the model; in Section 3 we provide numerical evidence of noise-sustained synchronization, and characterize the constructive role of noise in the process. Then we elucidate the observed dynamics in terms of the NEP of a reduced description in Section 4, and collect our conclusions in Section 5.

2. The model

Inhibitory coupling is a basic ingredient in the dynamics of neocortical pyramidal neurons [44] and cortical networks [45]. It also plays a major role in the dynamics of synchronous neural firing [46]. On the other hand, gap-junction coupling between inhibitory interneurons is substantially more abundant than that between excitatory ones [47] and the diffusive coupling of inhibitor fields comes out to be a crucial ingredient to explain the spontaneous emergence of low-frequency oscillations with spatially and temporally chaotic dynamics, in the transition from wake to anesthetic coma [48]. For the purpose of studying the effects of electric inhibitory coupling on the physical behavior of a network of *excitable* elements, the simplest activator–inhibitor dynamics can be assumed, and this is provided by the FHN model. The fast variable in this model (the *activator* u) mimics the action potential of the neuron and the slow one (the *inhibitor* or recovery variable v) is related to the time-dependent conductance of the K^+ channels in the axon membrane [49].

We consider a ring of N identical excitable FHN cells, with their *inhibitor* fields *electrically* coupled to those of their nearest-neighbor. The system is submitted to a *common* subthreshold harmonic signal $S(t)$ and independent additive Gaussian white noises in each component and each site. The equations for the model are

$$\begin{aligned}\dot{u}_i &= bu_i\big(1-u_i^2\big) - v_i + S(t) + r_1\xi_i^{(u)}(t) + r_2\xi_i^{(v)}(t),\\ \dot{v}_i &= \epsilon(\beta u_i - v_i + C) + E\big[(v_{i+1}-v_i)+(v_{i-1}-v_i)\big]\\ &\quad + r_3\xi_i^{(u)}(t) + r_4\xi_i^{(v)}(t),\end{aligned} \tag{1}$$

with $i = 1,\ldots,N$, $u_{N+1}\equiv u_1$, and $u_0\equiv u_N$. The activator–inhibitor timescale ratio ϵ is an important parameter in the model, since the single-cell NEP's integrability condition [40] relates β and the r's to it. $E>0$ is the electric inhibitory *coupling strength*, and the slow subthreshold external signal has the form $S(t) = A_0\sin\Omega t$. The $\xi_i^{(p)}(t)$, $p\in[u,v]$, are Gaussian random variables with $\langle\xi_i^{(p)}(t)\rangle = 0$ and cross-correlation $\langle\xi_i^{(p)}(t)\xi_j^{(q)}(t')\rangle = \eta\delta_{i,j}\delta_{p,q}\delta(t-t')$.

Tables 1 and 2 display most of the parameter values adopted throughout the work. Moreover, $r_3 = \epsilon r_1$, $r_4 = \epsilon r_2$ and $N = 256$. The parameters fulfill the above mentioned NEP's integrability condition and the requirement for the signal to be regarded as a subthreshold adiabatic perturbation (in particular, Ω must remain below the typical inverse deterministic time, i.e. the turnaround time of a single spike). Eqs. (1) imply direct electric connection (gap junction) between cells. The coupling term is *inhibitory*: when neuron i fires, neurons $i\pm1$ are less likely to fire.

3. Noise induced synchronization

Numerical integration of the stochastic system (1) with increasing η for appropriate values of E allows to visualize the noise-induced phenomena taking place: synchronization with the external signal of the ring's activity and (imperfect) spatiotemporal self-organization of the cells.

Table 1
Cell parameters.

ϵ	β	b	C
0.01	0.01	0.035	0.02

Table 2
Signal and noise parameters.

A_0	Ω	r_3	r_4
0.011	0.002	$\cos 0.05$	$\sin 0.05$

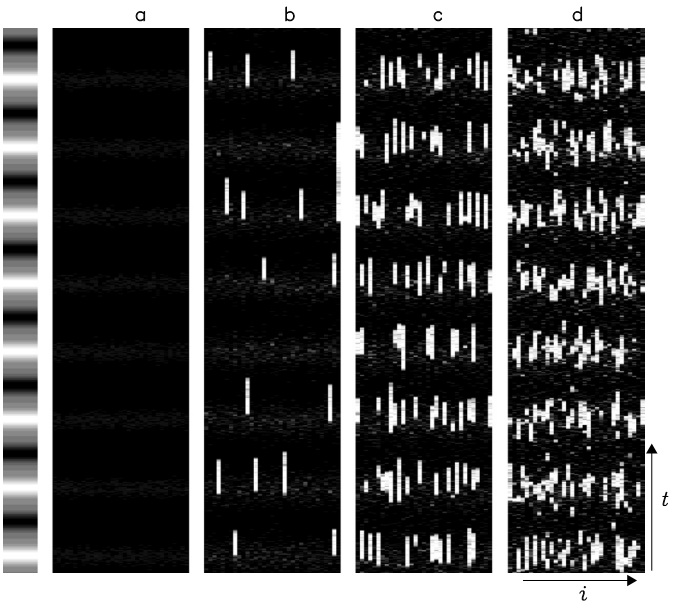

Fig. 1. Record of a set of 32 cells (white: 'activated', black: 'inhibited') with $E = 0.5$. a) $\eta = 2\times10^{-8}$ (subthreshold homogeneous oscillation), b) $\eta = 2\times10^{-7}$ (partially synchronized state), c) $\eta = 5\times10^{-7}$ (noise-sustained sync), and d) $\eta = 10^{-6}$ (Partially synchronized state). Left thin column refers to signal phase (white maximum, black minimum).

Fig. 1 collects—for η values increasing up to the one that maximizes the coherence of its collective behavior—the time evolution of the u variable for a ring's subset (32 neurons). Although the u values are displayed in grayscale (no thresholding whatsoever, otherwise the faint bands in the leftmost frames could not be seen), the picture looks quite pixeled because of excitability's own nature. For the purpose of quantifying the phenomenon we do introduce some threshold $u_{\rm th}$ (and call *active* those cells for which $u > u_{\rm th}$, see below), but its precise value is irrelevant as far as it exceeds the u-threshold for spike generation.

For η too low, only small-amplitude *highly homogeneous* [$u_i(t)\approx u_j(t)$] subthreshold oscillations (induced by the adiabatic signal) occur around the $S=0$ rest state (leftmost frame). As η increases, so does the number of cells that become noise-activated during roughly half a cycle of the external signal. For η even higher, the cells' activity enhances its coherence with the external signal as a consequence of its coupling-mediated self-organization: as one neuron activates, it usually inhibits its nearest-neighbor. The outcome of this phenomenon is the APS, which partially arises along the ring during the stage of noise-activation. In this scenario, noise (together with coupling and signal) plays a *constructive* role. Nonetheless for η too large, frame (d) in Fig. 1, the sync becomes eventually degraded.

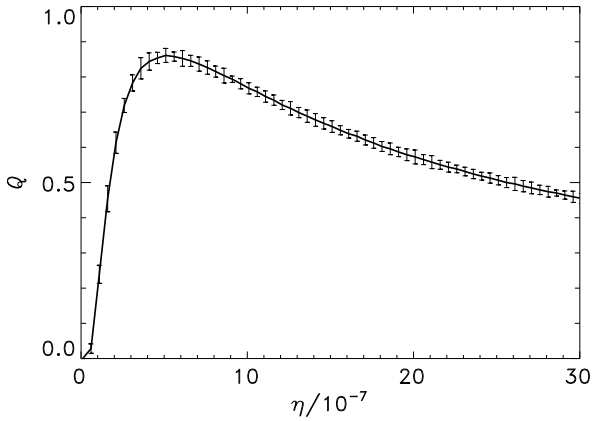

Fig. 2. Q-factor of the activity for $E = 0.5$ (average over 20 realizations). $Q_{\max}$ occurs at $\eta \approx 5 \times 10^{-7}$.

This behavior can be quantified by introducing the (time-dependent) normalized global activation or *activity*

$$Ac(t) = \frac{1}{N}\sum_{i=1}^{N}\theta\big[u_i(t) - u_{\rm th}\big] \tag{2}$$

(θ is the Heaviside step function). Since (as already stated) for $u_{\rm th}$ values in the useful range, $Ac(t)$ is quite insensitive to threshold, we take hereafter $u_{\rm th} = 0.4$. By normalization, $Ac = 1/2$ means states where half of the neuron population is active. Whereas an APS is ideally such a state, Ac does not reach the value $1/2$ because alternance may fail because of the local noise, a necessary ingredient for activation. The failure takes the form of defects where pairs of neighbor neurons remain inhibited or excited.[1]

We get an even more useful quantifier by selecting the Ω-component of $Ac(t)$

$$Q_{\sin} = \frac{1}{nT}\int_0^{nT} 2Ac(t)\sin(\Omega t)dt,$$

$$Q_{\cos} = \frac{1}{nT}\int_0^{nT} 2Ac(t)\cos(\Omega t)dt,$$

where n is the number of periods $T = 2\pi/\Omega$ covered by the integration time. This is the Q-factor

$$Q = \sqrt{Q_{\sin}^2 + Q_{\cos}^2}, \tag{3}$$

plotted in Fig. 2 as a function of η, for $E = 0.5$. Q goes through a maximum at $\eta \sim 5 \times 10^{-7}$, indicating optimal neuronal coherence and signal enhancement. At that noise intensity, the system reaches the best possible synchronization.

4. Approximate analytical treatment

We now undertake a theoretical study of the dynamics by exploiting the properties of the NEP during the time-evolution [27–30]. We consider the minimal description of an idealized case where all the even nodes on one hand, and all the odd ones on the other, have the *same* stochastic phase-space trajectory. That is, a reduced *two-neuron* system with variables (u_1, u_2, v_1, v_2), which allows the formation of an APS (besides homogeneous one). Model equations turn up from Eq. (1) specifying $N = 2$. Now it turns out that (at variance with the antiphase-coupled activator case) a NEP

[1] Presence of defects grants results being independent of N's parity.

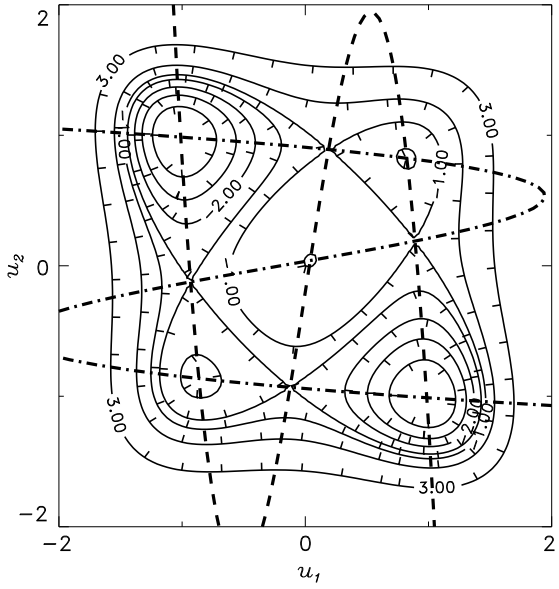

Fig. 3. Φ-landscape along the slow manifolds for maximum signal amplitude and $E = 0.5$. The system has two uniform attractors (both cells inhibited, both cells activated), two APS (with one cell activated and one inhibited), four saddles and one maximum. Nullclines are indicated in dashed and dashed dotted line.

cannot be easily found for this system. So we decided to further reduce this description by projecting the dynamics along the corresponding *slow manifolds*. Since in the single-neuron case [40,41] the equivalence between doing so at the NEP level or in the dynamical equations has been granted, we now proceed along the second way and get for the slow manifolds

$$\beta u_{1,2} - v_{1,2} + C + \frac{2E}{\epsilon}(v_2 + v_1 - 2v_{1,2}) = 0. \tag{4}$$

The projection—which confines the neurons' dynamics to their respective slow manifolds—is used *only* to calculate *heights* of barriers. The resulting system

$$\begin{aligned}
\dot{u}_1 &= \left(b - \frac{2E+\epsilon}{4E+\epsilon}\beta\right)u_1 - bu_1^3 - \frac{2E\beta}{4E+\epsilon}u_2 + S - C \\
&\quad + r_1\xi_1^{(u)}(t) + r_2\xi_1^{(v)}(t), \\
\dot{u}_2 &= \left(b - \frac{2E+\epsilon}{4E+\epsilon}\beta\right)u_2 - bu_2^3 - \frac{2E\beta}{4E+\epsilon}u_1 + S - C \\
&\quad + r_1\xi_2^{(u)}(t) + r_2\xi_2^{(v)}(t)
\end{aligned} \tag{5}$$

is gradient with a NEP

$$\begin{aligned}
\Phi(u_1,u_2) = -\frac{1}{\lambda_1}\Bigg[&-\frac{b}{2}\big(u_1^4 + u_2^4\big) - \frac{4E\beta}{4E+\epsilon}u_1u_2 \\
&+ \left(b - \frac{2E+\epsilon}{4E+\epsilon}\beta\right)\big(u_1^2 + u_2^2\big) \\
&+ 2(S-C)(u_1+u_2)\Bigg],
\end{aligned} \tag{6}$$

where $\lambda_1 = r_1^2 + r_2^2$.

Fig. 3 displays the Φ-level curves in the (u_1, u_2) plane, for maximal signal amplitude, $S(t) = A_0$. Inheriting the u_1–u_2 permutation invariance of the dynamical equations, the NEP (in the space spanned by the slow manifolds) and hence the distribution of its fixed points, are symmetric with respect to the $u_1 = u_2$ line. The fixed points can be obtained either from the intersection of the nullclines or by extremizing $\Phi(u_1, u_2)$. The ones lying along the $u_1 = u_2$ line are uniform states: the *rest–rest* and *active–active* attractors (recall that the latter is a point-like attractor in the

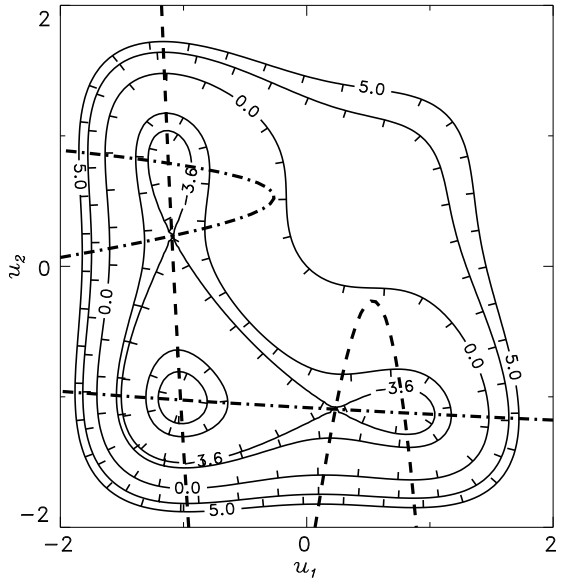

Fig. 4. Same as Fig. 3, for $S = 0$. The system has one uniform attractor (both cells inhibited), two APS and two saddles.

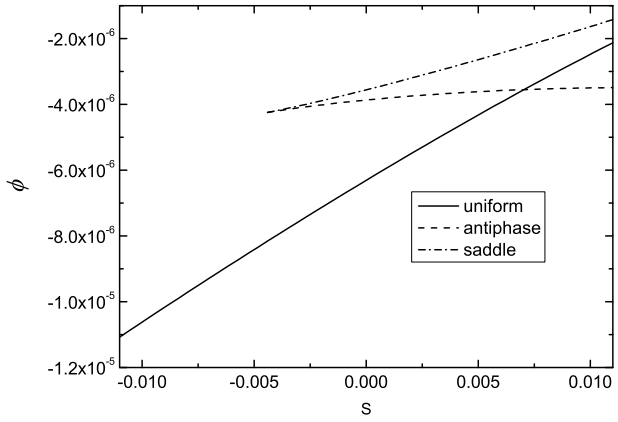

Fig. 5. Φ *vs* S for the uniform (rest–rest), the saddle and the APS, when $E = 0.5$. The last two fixed points disappear for S below $\sim -5 \times 10^{-3}$ (deterministic decay).

space of the projections, which does not rule out excitable behavior for the individual neurons) and a repeller in between. Besides, there are two mirror APS attractors and four saddle points. The complete equivalence between mirror fixed points in the reduced model indicates a degeneracy of the APS (u_1 activated, u_2 inhibited or u_2 activated, u_1 inhibited) which is also present in the full ring.

The number of attractors changes with the signal amplitude, as can be appreciated in Fig. 4—the counterpart of Fig. 3 for $S(t) = 0$—where the repeller, the *active–active* attractor, and two saddle points have vanished. For minimal signal amplitude, $S(t) = -A_0$ (figure not shown), only the uniform rest-state attractor survives.

The global stability of each attractor (depending on S) is given by its well's depth with respect to the saddle level. In Fig. 5, the Φ values of the uniform (solid line), either APS (dashed line) and either low-u saddle (dot-dashed line) are plotted as functions of S, between $\pm A_0$ (the repeller, the *active–active* attractor, and both high-u saddle points involve higher Φ-barriers and are not shown, since they are irrelevant to the discussed dynamics). Fig. 5 tells us that above $S \approx 0.07$, the uniform state yields its global stability to both APS.

For $S(t) = A_0$, the difference in Φ between the saddle and the uniform state is $\Delta\Phi = 7 \times 10^{-7}$. For a noise level of this order, the two-cell system would climb that potential barrier and transit to the APS, which has a lower Φ value. The APS can then *deterministically* return to the uniform state because the saddles lying in between collapse. $\eta \sim \Delta\Phi$ is the expected order of magnitude of noise for full sync, in good agreement with the numerical results of Fig. 2.

The two-cell approximation does not take into account the eventual formation of defects, which break the background activation's alternance of extended APS. We find it remarkable that these approximate theoretical results help elucidate the route to synchronization, and estimate the order of magnitude of the optimal noise intensity.

5. Conclusions

We have investigated the stochastic dynamics of a ring of FHN cells, with nearest-neighbor *electric* (diffusive) coupling between their *inhibitor* fields. The system is externally, adiabatically forced by a common subthreshold harmonic signal, and submitted to additive and independent Gaussian white noises of the same intensity η. Numerical integration for appropriate parameter values shows that local additive noise sustains extended APS (where the cells alternate their activation state), which moreover synchronize with the external subthreshold signal.

In addition to being the noise (together with coupling and signal) an essential ingredient in this process, the Q-factor exhibits a maximum as a function of η. The role of the noise is thus twofold: on one hand it is an essential ingredient for the phenomenon to occur and on the other, there is an optimal noise intensity for maximal coherence, as in the phenomenon of double stochastic resonance [50].

The numerical results can be better interpreted and quantitatively accounted for with reasonable accuracy by considering the NEP of a two-cell reduced model. At variance with the antiphase-coupled activator case, a NEP for the full four-dimensional space cannot be easily found. In the single neuron case, reduction to the slow manifold at NEP and dynamical equation levels turn out to be equivalent. Reassured by this result, we confined our description to the two-dimensional space of the slow manifolds, in which a NEP follows straightforwardly. Although this procedure poses severe limitations on the dynamics, the fact that all the fixed points that organize it lie in this subspace make it suitable to calculate barrier heights.

By analyzing the dependence of the potential barriers on the amplitude of the external signal, we have shown that the dynamics can be explained in terms of noise-sustained transitions between attractors. We remark that all the activations are noise-induced and correspond to global decrease of the NEP.

As it occurs for related phenomena (e.g. coherence resonance in coupled FHN systems [8]), our results are expected to depend on both temporal and spatial noise correlations. The NEP approach would be useful even in those cases, since dynamics driven by space-correlated or colored (Ornstein–Uhlenbeck) noises can in principle be described in terms of a suitable NEP [51].

Acknowledgements

Financial support is acknowledged from the following institutions of Argentina: CONICET (PIP 12 220 100 100 315) and UNMdP (Project EXA633/13).

References

[1] J. García-Ojalvo, J.M. Sancho, Noise in Spatially Extended Systems, Springer, New York, 1999.
[2] F. Sagués, J.M. Sancho, J. García-Ojalvo, Rev. Mod. Phys. 79 (2007) 829.
[3] R. Toral, C.J. Tessone, J.V. López, Eur. Phys. J. Spec. Top. 143 (2007) 59.

[4] L. Gammaitoni, P. Hänggi, P. Jung, F. Marchesoni, Rev. Mod. Phys. 70 (1998) 223.
[5] K. Wiesenfeld, F.J. Jaramillo, Chaos 8 (1998) 539.
[6] T. Wellens, V. Shatokhin, A. Buchleitner, Rep. Prog. Phys. 67 (2004) 45.
[7] H.S. Wio, Phys. Rev. E 54 (1996) R3075.
[8] A.S. Pikovsky, J. Kurths, Phys. Rev. Lett. 78 (1997) 775.
[9] C. Zhou, J. Kurths, Phys. Rev. Lett. 88 (2002) 230602.
[10] C.J. Tessone, A. Sciré, R. Toral, P. Colet, Phys. Rev. E 75 (2007) 016203.
[11] A. Pikovsky, M. Rosenblum, J. Kurths, Synchronization: A Universal Concept in Nonlinear Sciences, CUP, Cambridge, UK, 2003.
[12] R. FitzHugh, Biophys. J. 1 (1961) 445.
[13] J. Nagumo, S. Arimoto, S. Yoshizawa, Proc. IRE 50 (1962) 2061.
[14] J.A. Acebrón, A. Bulsara, W.-J. Rappel, Phys. Rev. E 69 (2004) 026202.
[15] C. Koch, Biophysics of Computation: Information Processing in Single Neurons, UP, New York, 1999.
[16] L. Glass, P. Hunter, A. McCulloch (Eds.), Theory of Heart, Springer, Berlin, 1991.
[17] B. Lindner, J. García-Ojalvo, A. Neiman, L. Schimansky-Geier, Phys. Rep. 392 (2004) 321.
[18] D. Kaplan, J. Clay, T. Manning, L. Glass, M.R. Guevara, A. Shrier, Phys. Rev. Lett. 76 (1996) 4074.
[19] J.R. Clay, J. Comput. Neurosci. 15 (2003) 43.
[20] P. Hänggi, ChemPhysChem 3 (2002) 285.
[21] G. Schmid, I. Goychuk, P. Hänggi, Europhys. Lett. 56 (2001) 22.
[22] P. Jung, J.W. Shuai, Europhys. Lett. 56 (2001) 29.
[23] T. Yanagita, T. Ichinomiya, Y. Oyama, Phys. Rev. E 72 (2005) 056218.
[24] A.M. Lacasta, F. Sagués, J. Sancho, Phys. Rev. E 66 (2002) R045105.
[25] L. Callenbach, P. Hänggi, S.J. Linz, J.A. Freund, L. Schimansky-Geier, Phys. Rev. E 65 (2002) 051110.
[26] J.A. Freund, L. Schimansky-Geier, P. Hänggi, Chaos 13 (2003) 225.
[27] G.G. Izús, R.R. Deza, A.D. Sánchez, AIP Conf. Proc. 887 (2007) 89.
[28] G.G. Izús, A.D. Sánchez, R.R. Deza, Physica A 388 (2009) 967.
[29] A.D. Sánchez, G. Izús, Physica A 389 (2010) 1931.
[30] M. dell'Erba, G. Cascallares, A.D. Sánchez, G. Izús, Eur. Phys. J. B 87 (4) (2014) 1–7, http://dx.doi.org/10.1140/epjb/e2014-41029-2.
[31] L. Yan, N.C. Foley, J.M. Bobula, L.J. Kriegsfeld, R. Silver, J. Neurosci. 25 (2005) 9017.
[32] E. Ullner, A. Zaikin, E.I. Volkov, J. García-Ojalvo, Phys. Rev. Lett. 99 (2007) 148103.
[33] G. Balázsy, A. Cornell-Bell, A.B. Neimal, F. Moss, Phys. Rev. E 64 (2001) 041912.
[34] E.I. Volkov, E. Ullner, J. Kurths, Chaos 15 (2005) 023105.
[35] C.J. Tessone, E. Ullner, A.A. Zaikin, J. Kurths, R. Toral, Phys. Rev. E 74 (2006) 046220.
[36] A. Sherman, J. Rinzel, Proc. Natl. Acad. Sci. USA 89 (1992) 2471.
[37] Q. Zhao, C.G. Yao, M. Yi, Eur. Phys. J. B 84 (2011) 299.
[38] Y. Li, G. Schmid, P. Hänggi, L. Schimansky-Geier, Phys. Rev. E 82 (2010) 061907.
[39] X. Ao, G. Schmid, P. Hänggi, Math. Biosci. 245 (2013) 49.
[40] G.G. Izús, R.R. Deza, H.S. Wio, Phys. Rev. E 58 (1998) 93.
[41] G.G. Izús, R.R. Deza, H.S. Wio, Comput. Phys. Commun. 121–122 (1999) 406.
[42] R. Graham, in: E. Tirapegui, D. Villarroel (Eds.), Instabilities and Nonequilibrium Structures, D. Reidel, Dordrecht, 1987, pp. 271–290.
[43] H.S. Wio, R.R. Deza, Eur. Phys. J. Spec. Top. 146 (2007) 111.
[44] A. Destexhe, M. Rudolph, J.-M. Fellous, T.J. Sejnowski, Neuroscience 107 (2001) 13.
[45] N. Brunel, X.J. Wang, J. Comput. Neurosci. 11 (2003) 63.
[46] C. van Vreeswijk, L.F. Abbott, G.B. Ermentrout, J. Comput. Neurosci. 1 (1994) 313.
[47] M.V. Bennett, R.S. Zukin, Neuron 41 (2004) 495.
[48] M.L. Steyn-Ross, D.A. Steyn-Ross, J.W. Sleigh, Phys. Rev. X 3 (2013) 021005.
[49] N.B. Janson, A. Galanov, E. Schöll, Phys. Rev. Lett. 93 (2004) 010601.
[50] A. Zaikin, J. García-Ojalvo, R. Báscones, E. Ullner, J. Kurths, Phys. Rev. Lett. 90 (2003) 030601.
[51] A. Schenzle, T. Tél, Phys. Rev. A 32 (1985) 596.