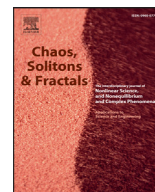




Contents lists available at ScienceDirect

Chaos, Solitons and Fractals

Nonlinear Science, and Nonequilibrium and Complex Phenomena

journal homepage: www.elsevier.com/locate/chaos

Emergence of biological complexity: Criticality, renewal and memory



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ARTICLE INFO

Article history:

Available online 11 August 2015

Keywords:

Biological criticality

Coherence and temporal complexity

Renewal

Memory

ABSTRACT

The key purpose of this article is to establish a connection between two emerging fields of research in theoretical biology. The former focuses on the concept of criticality borrowed from physics that is expected to be extensible to biology through a robust theoretical approach that although not yet available shall eventually shed light into the origin of cognition. The latter, largely based on the tracking of single molecules diffusing in biological cells, is bringing to the general attention the need to go beyond the ergodic assumption currently done in the traditional statistical physics. We show that replacing *critical slowing down* with *temporal complexity* explains why biological systems at criticality are resilient and why long-range correlations are compatible with the free-will condition necessary for the emergence of cognition. Temporal complexity generates ergodicity breakdown and requires new forms of response of complex systems to external stimuli. We concisely illustrate these new forms of information transport and we also address the challenging issue of combining *temporal complexity* with *coherence* and *renewal* with *infinite memory*.

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1. Introduction

The main purpose of this paper is to establish a connection between two emerging theoretical perspectives that physicists are adopting in their attempt at addressing fundamental biological issues beyond the limits of reductionism [1]. The former theoretical perspective is illustrated by the debate about the important work of Ref. [2]. In fact, the recent experiment of Ref. [2] is attracting [3] the attention of researchers on the key role of criticality in biology, thereby leading them to look, for instance, at the very interesting proposals of [4,5]. In the conclusion of their article the authors of Ref. [5] emphasize the phase-transition related property of *critical slowing down*, namely the infinitely slow regression to equilibrium of processes at criticality, pointing out however the existence of a possible conflict with the resilience

of complex biological systems that are expected to promptly adapt themselves to the changes of their environment, the swarm of birds of Ref. [2] being an outstanding example of biological resilience. An interesting experiment concerning the cognition of living beings is given by the work done at the Duke University by the group of Nicoliles [6]. In this experiment a rat *A* moving in a box transmits information to a rat *B* moving in a different box through a cable connecting the neural network of the brain of rat *A* to the neural network of the brain of rat *B*. This experiment is the *in vivo* counterpart of the *in vitro* experiment done in 1999 at the University of North Texas by the group of Guenter Gross [7], interpreted by the authors of this interesting paper as a form of chaos synchronization. Thus, we see a fast progress from the 1999 *in vitro* experiment of Ref. [7] to the 2013 *in vivo* experiment of Ref. [6] and from there to the 2014 experiment of Ref. [8] concerning the same kind of information transfer from the brain of one human subject to that of another. Actually, this form of synchronization seems to be a natural property of the dialogue between two individuals [9], leaving however

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unknown the statistical roots of these synchronization processes.

The latter remarkable example of biological complexity is illustrated in the recent review paper [10], reflecting the growing interest for anomalous diffusion in biological cells, as a paradigm of the special nature of biological processes. The authors of this review paper refer to two forms of non-conventional statistical mechanical processes to account for molecular diffusion. The former is the continuous time random walk (CTRW) model introduced by Scher and Montroll [11] and by Montroll and Weiss [12]. The random walker makes jumps that are not necessarily anomalous, but the time distance τ between two consecutive jumps rests on a waiting time distribution density $\psi(\tau) \propto 1/(\tau^\mu)$, with $\mu < 2$, thereby making these events crucial generators of ergodicity breakdown. The latter tool is given by a long-range correlation theoretical perspective, with no events, based on the generalized Langevin equation (GLE) by Mori [13] and the fractional Brownian motion (FBM) developed by Mandelbrot and van Ness [14] to assign infinite memory to the ordinary Brownian motion.

We plan to show that although apparently unrelated, the perspective of biological processes emerging from the work of Refs. [3–5] and that afforded by the authors of [10] may be unified by theoretical progress on open problems generated by ergodicity breaking: the response to stimuli of non-ergodic systems, and, even more challenging, the response to perturbation of systems under the joint action of both ergodicity breaking renewal events and of long-memory fluctuations compatible with coherence. The main aim of this article is to relate these forms of synchronization to criticality [15] through the key property of *temporal complexity*. In Section 2 we illustrate the difference between critical slowing down and temporal complexity. We devote Section 3 to illustrating another significant aspect of biological processes, anomalous diffusion in biological cells. In Section 4 we discuss the apparently conflicting presence of both temporal complexity and coherence in the brain, mirroring the surprising joint action of infinite memory and renewal events in the processes of anomalous diffusion in biology. Finally we devote Section 5 to express our wishes for future research work that may lead to a satisfactory solution of these problems.

2. Criticality

In this section we illustrate the difference between critical slowing down and temporal complexity. Let us consider a generic network of N units that have to make a choice between the state A and the state B . To make the model more attractive let us assume that the state A refers to altruism and the state B to selfishness. The concentration of nodes in A is denoted by the symbol p and the concentration of nodes in the state B by the symbol q . We set the normalization condition

$$p + q = 1. \quad (1)$$

The A to B transition rate is γ and the B to A transition rate is ω , thereby yielding

$$\dot{p} = -\gamma p + \omega q \quad (2)$$

and

$$\dot{q} = -\omega p + \gamma q, \quad (3)$$

so as to fit the normalization condition of Eq. (1) at all times. From Eq. (1) we get $q = 1 - p$, which plugged in Eq. (2) yields

$$\dot{p} = -(\gamma + \omega)p + \omega, \quad (4)$$

leading us to conclude that if we set the condition $\omega = 0$, the system naturally evolves toward the equilibrium condition $p = 0$, implying the extinction of altruism.

A much less pessimistic condition is possible if we make the assumption that the behavior of the units of the network is influenced by imitation [16]. In this case

$$\omega = Kp. \quad (5)$$

A small concentration of individuals in the state A may produce significant effects on the network if the imitation strength, here denoted by the symbol K , is very large. Actually, by plugging Eq. (5) into Eq. (4) we get

$$\dot{p} = -(\gamma - K)p - Kp^2, \quad (6)$$

which, for $K > \gamma$ admits the new equilibrium condition

$$p_{\text{eq}} = 1 - \frac{\gamma}{K}, \quad (7)$$

while the condition $p_{\text{eq}} = 0$ becomes unstable. We are in the presence of a phase transition with the critical value of K , K_c , being

$$K_c = \gamma. \quad (8)$$

2.1. Critical slowing down

At criticality the regression to equilibrium from a small out of equilibrium condition $p_0 \ll 1$ is given by

$$p(t) = \frac{p_0}{1 + \gamma p_0 t}. \quad (9)$$

The lifetime of this non-equilibrium condition is infinitely large. This is the *critical slowing down* pointed out in the work of [5].

2.2. Temporal complexity

To understand its connection with *temporal complexity* we have to keep in mind that Eq. (6) is an ideal condition implying that $N = \infty$. In the more realistic case of a network with a finite number of interacting units, we have to take into account that the number of neighbors of a given unit is not exactly determined by p . The dynamics of the network is established as follows. At each time step we have to assess if a given unit is either in the state A or in the state B . If it is in the state A , the transition from A to B is determined by the rate γ , independent of the concentrations p and q . If it is in the state B the probability of making a transition to A , $\Pi_{B \rightarrow A}$, namely the counterpart of ω of Eq. (5), is given by

$$\Pi_{B \rightarrow A} = K \frac{M_A}{M}, \quad (10)$$

where M is the total number of nearest neighbors of the unit under consideration and M_A is the number of them in the altruist state. It is evident that with a finite number of units

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