10-17-2013

Degeneracy and long-range correlations

D. Delignières
University Montpellier, France

Vivien Marmelat
University of Montpellier, France, vmarmelat@unomaha.edu

Degeneracy and long-range correlations

D. Delignières, and V. Marmelat

Citation: Chaos **23**, 043109 (2013); doi: 10.1063/1.4825250
View online: https://doi.org/10.1063/1.4825250
View Table of Contents: http://aip.scitation.org/toc/cha/23/4
Published by the American Institute of Physics

Degeneracy and long-range correlations

D. Delignières[a] and V. Marmelat
EA 2991 Movement To Health, Euromov, University Montpellier 1, 34090 Montpellier, France

(Received 28 July 2013; accepted 2 October 2013; published online 17 October 2013)

Degeneracy is a ubiquitous property of complex adaptive systems, which refers to the ability of structurally different components to perform the same function in some conditions and different functions in other conditions. Here, we suppose a causal link between the level of degeneracy in the system and the strength of long-range correlations in its behavior. In a numerical experiment, we manipulated degeneracy through the number of networks available in a model composed of a chain of correlated networks over which a series of random jumps are performed. Results showed that correlations in the outcome series increased with the number of available networks, and that a minimal threshold of degeneracy was required to generate long-range correlations. We conclude that degeneracy could underlie the presence of long-range correlations in the outcome series produced by complex systems. In turn, we suggest that quantifying long-range correlations could allow to assess the level of degeneracy of the system. Degeneracy affords a maybe more intuitive way than former hypotheses for understanding the effects of complexity on essential properties such as robustness and adaptability. © *2013 AIP Publishing LLC*. [http://dx.doi.org/10.1063/1.4825250]

In the present paper we try to establish a causal link between degeneracy and long-range correlations. The concept of long-range correlations refers to an intriguing statistical property of time series, characterized by the presence of long-term dependences between the current observation and a large set of previous observations. Long-range correlations have been evidenced in the behavior of a number of physical and biological systems of time series. Degeneracy, or partial redundancy, is a design principle that is supposed to underlie the organization of biological systems, providing them with adaptability and robustness. We show in a simulation study that the strength of correlations in the series produced by a complex network is related to its level of degeneracy, and that a minimum threshold of degeneracy is necessary for producing long-range correlated series. This hypothesis opens new perspectives for understanding the origins of the long-range correlations, their relationships with systems complexity, and their evolution with changing environmental constraints, learning, aging, and disease.

I. INTRODUCTION

A. Long-range correlations and complexity

The concept of long-range correlations refers to an intriguing statistical property, which has been evidenced in the behavior of a number of physical and biological systems. In a time series, the presence of serial correlation means that there exists some dependence between successive values. Such correlations could appear on the short term; for example, in a simple one-order auto-regressive model the current value is partially determined by a fraction of the just preceding one.[1]

In contrast long-range correlated series are characterized by the presence of dependencies that tend to persist over dozens or even hundreds of data. In this kind of process the current observation seems to keep the memory of a large set of previous observations. Long-range correlations can be understood through the fact that over multiple, interpenetrated time scales, an increasing trend in the past is likely to be followed by an increasing trend in the future, and conversely a decrease in the past is likely to be followed by a decrease in the future. Long-range correlated series are characterized by self-similarity, which means that similar statistical features are observed across different temporal or spatial scales. This kind of process has also been referred to as long-range dependence, long-term memory, fractal process, or $1/f$ noise.[2–6]

Long-range correlations have been evidenced in time series collected in a number of situations and covering a diversity of natural and physical systems, including for example the series of discharges of the Nile River,[7] the series of magnitudes of earthquakes,[8] or the evolution of traffic in Ethernet networks.[9] In the domain of living systems, long-range correlations have been evidenced in heartbeat fluctuations,[10] in serial reaction time,[11,12] in finger tapping,[13,14] in stride duration during walking or running,[15,16] or in relative phase in a bimanual coordination task.[17]

There is now a general agreement for considering long-range correlations as reflecting the complexity of the underlying system, defined as a flexible and adaptable coordination between its multiple components and sub-systems.[18] From this point of view, complexity is thought as an optimal comprise between complete disorder (no interaction between components) and total order (close and rigid coupling between components). Long-range correlations are not considered as arising from some specific component within the system but, rather, from the complex, multiplicative interactions between its multiple components, acting at different time scales. Several characteristic features of complex systems have been advocated to

[a]Author to whom correspondence should be addressed. Electronic mail: didier.delignieres@univ-montp1.fr

play a central role in the emergence of long-range correlations, such as self-organized criticality.[12] multiscale dynamics,[19] metastability,[3] or cascade dynamics.[20,21]

Long-range correlations are also considered to represent the hallmark of efficient and perennial systems. In the domain of living systems, they have been essentially discovered in experiments analyzing the behavior of young and healthy organisms. In contrast, the analysis of series produced by deficient systems (i.e., patients suffering from diverse pathologies, elderly) revealed a clear alteration of fractal properties, often towards disorder, and sometimes towards order. Hausdorff and colleagues, for example, showed that the series of stride intervals during walking typically presented long-range correlations in young and healthy adults. In contrast, stride series appeared less correlated in elderly, and in Huntington's or Parkinson's patients.[22,23] Goldberger *et al.*[24] reported some examples of cardiac interbeat interval series, showing that the series produced by healthy patients typically exhibited a correlated variability, close to $1/f$ noise. In contrast, in patients suffering from arrhythmia, the series were less correlated, close to white noise, and in patients with severe heart failure, series appeared less variable, and highly predictable. These results supported the idea of a close link between complexity and health and led to the theory of the loss of complexity with aging and disease.[25,26] Healthy systems are supposed to present a rich set of interactions between their many components and levels. In contrast, aging or disease are characterized either by a loss of interactions or by the dominance of few residual components. In the first case the absence of interactions cannot support coordinated patterns of activity across components, and in the second the system cannot flexibly reorganize in response to changing conditions. The main idea is that complexity provides systems with essential capacities of adaptation and flexibility. Complexity allows to protect against the possible deficiency of a given component or sub-system, in order to maintain stability despite external perturbations, and favors the emergence of innovative solutions when facing with a new problem.[24]

B. Degeneracy

Another theoretical framework allows to draw conceptual links between complexity, stability, and adaptability. This framework has been essentially developed in the domain of theoretical biology, especially evolution theory.[27,28] The authors examined the relationships between complexity, robustness, and evolvability in complex adaptive systems. In the context of biological evolution, robustness refers to the insensitivity of phenotypes to variations in both internal and external conditions and evolvability to the capacity to adapt to environmental constraints by generating heritable phenotypic variations. This conceptual framework has obviously a broader scope, and more generally robustness can be considered equivalent to the concept of stability, and evolvability is close to those of adaptability and flexibility, widely used in the analysis of complex systems dynamics.[29]

The authors show that these three properties are closely related. Complex systems are more likely to resist to environmental changes than simple ones, and an increase in complexity improves system's robustness. Evolvability appears a necessary prerequisite for complexity: the evolution of life forms is characterized by a general increase of systems complexity, and this trend cannot be understood without the capacity to generate heritable phenotypic changes.[27,28]

Robustness and evolvability could appear as opposite properties. Robustness suggests resistance to environmental changes, and evolvability a capacity of flexibility in order to adapt to changes. Paradoxically, these two properties appear both essential of the persistence of life. However, Whitacre and Bender[27] showed that under some conditions, robustness could naturally lead to evolvability. The key factor lies in the design principles that are used to achieve robustness in the system. For example, robustness can be obtained through redundancy: In a redundant system, similar components have similar functionality, and thus redundant components can be used to replace components that fail, or can be alternatively used for achieving a given function. One can easily conceive that redundancy provides the system with stability and robustness facing external perturbations. However, the authors show by means of simulation experiments that when robustness is obtained by pure redundancy, systems present a low evolvability.

In contrast robustness yields to evolvability when achieved through degeneracy (or partial redundancy). Degeneracy refers to a *partial overlap* in the functions of the multiple components within the system. In degenerate systems, structurally different components can perform similar functions under certain conditions but can also assume distinct roles in others conditions.[27]

Degeneracy and redundancy can be contrasted by comparing engineering and biological systems. Engineering systems are generally designed to be as simple and parsimonious as possible, with an explicit assignment of functions to each component. There is no place in such systems for *a priori* unnecessary or unplanned processes or interactions. Robustness can be afforded by duplicating essential components (i.e., by pure redundancy), in order to compensate for eventual failures, and errors are *a posteriori* corrected by feedback mechanisms, following deterministic rules.[30]

In contrast, biological systems do not work on the basis of such planned designs and deterministic functioning. There is no fixed assignment for a given function: a number of structurally different sets of components can produce a given output in a similar way, and components could assemble for producing new and different outputs under different constraints. Degeneracy allows biological systems to be adaptable to unpredictable changes in their environment or in terms of output requirements. Intuitively, one may indeed conceive that degeneracy provide systems with both robustness and evolvability. Because degeneracy appears as a key factor for the perennity and the evolution of species, its ubiquity in biological systems, at all levels of organization, is not surprising.[30]

Whitacre[28] showed that degeneracy plays a central role in the relationships between complexity, robustness, and

evolvability. As previously evoked, degeneracy appears as a precondition of evolvability and as an efficient principle for providing systems with robustness. Finally, degeneracy is conceptually close to complexity: Complex systems are usually described as systems in which components are functionally differentiated across a diversity of functions but also in which interacting components are integrated in more global functional units. Complex systems are then characterized by an interplay between functional segregation and functional integration,[31,32] yielding a multiscaled and hierarchical structure.[33] This definition of complexity implies degeneracy, as one can easily conceive that at low levels of complexity, there will be very few ways for structurally different parts to yield similar outputs.

C. A possible link between long-range correlations and degeneracy

The two previous lines of reasoning suggest a possible link between degeneracy and long-range correlations, which both seem to characterize complex, stable, and adaptive systems.

An interesting paradox has to be noticed at this level: If long-range correlations are supposed to sign the capabilities of adaptation of a system to environmental changes, they essentially arise when the system performs repeatedly a given function in a stable environment. Indeed, long-range correlations are experimentally observable when measurement focuses on intrinsic fluctuations but tend to transiently disappear under the influence of extrinsic perturbations.[3,34,35] Thus, degeneracy has been evoked as a property allowing resisting or adapting facing environmental changes, but long-range correlations are essentially observed when systems perform in stable conditions. What could be the effects of degeneracy for a system performing in such stable conditions?

We present in Figure 1 a very simple illustration of two systems performing a function by transmitting information from the upper level to the lower one. Each system presents alternative pathways, which allow to efficiently achieving the transduction of the signal. The left panel illustrates a typically redundant system, presenting independent alternative pathways, providing more or less equivalent outcomes. The system is supposed to randomly exploits the available pathways over successive trials. As pathways are not similar, but just equivalent, the series of outcomes will present fluctuations. However, because pathways are independent, successive performances are likely to be uncorrelated.

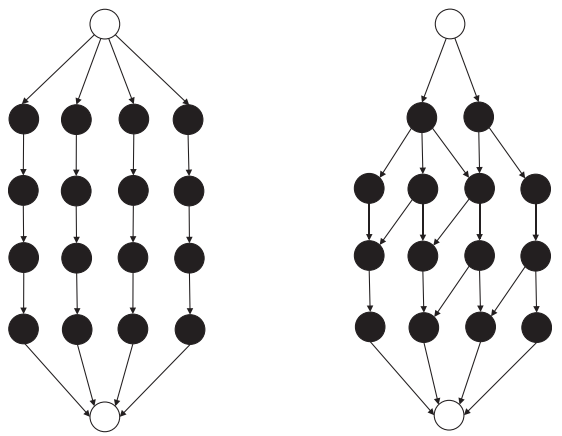

FIG. 1. Redundancy and degeneracy in networks models. These two panels represent hypothetical networks performing a function by transmitting information from the upper level to the lower one. The left panel illustrates a redundant system, in which several distinct pathways perform the same function. The right panel corresponds to a degenerate network: components could belong to several pathways, and then neighbor pathways share common components.

The right panel illustrates, in contrast, a degenerate network. In this network a number of pathways are available for achieving the outcome, but these pathways are not independent as in the previous system. Neighbor pathways share common elements, and in this case successive performances can be expected to be correlated.

Obviously, these oversimplified illustrations do not reflect the actual complexity of the physical of biological systems that produce long-range correlations. A better representation should consider networks composed of hundreds of alternative but correlated pathways, each composed of hundreds of components hierarchically organized and acting over diverse time scales.

II. A DEGENERATE MODEL

A model obeying these principles has been developed some years ago for accounting for the presence of long-range correlations in the series of stride durations during walking, and in the series of periods in forearm oscillations.[36–42] This model is composed of a hybrid self-sustained oscillator,[43] which stiffness is discretely determined, cycle-by-cycle, by a neural hopping model.

Consider the following second-order differential equation:

$$\ddot{x} = \alpha\dot{x} - \beta\dot{x}x^2 - \gamma\dot{x}^3 - \omega_i^2 + \sqrt{Q}\xi_t, \tag{1}$$

where x represents position. The dot notation indicates differentiation with respect to time. In this second-order differential equation $\alpha\dot{x}$ represents linear damping, $\beta\dot{x}x^2$ is a nonlinear van der Pol damping term, and $\gamma\dot{x}^3$ a nonlinear Rayleigh damping term, and ω_i^2 a cycle-dependent stiffness parameter. A noise term of strength Q is added to the model in order to simulate the perturbations that affect all dynamical systems. In the present notation, all coefficients are supposed to be positive. Under these conditions, this model yields a limit cycle attractor of frequency ω_i^2.

West and Scafetta[41] proposed a neural hopping model for providing the ω_i^2 series with long-range correlation properties. The key element of this model is a linear Markov process δ_j, generated by a first-order auto-regressive equation

$$\delta_j = \phi\delta_{j-1} + \eta\varepsilon_j, \tag{2}$$

where $0 < \phi < 1$ is a constant and ε_j a white noise process with zero mean and unit variance. This chain could be conceived as a set of alternative networks in the system, neighboring networks sharing common components and being then

mutually correlated. This chain then contains "correlated zones" of typical size r

$$r = -1/\log \phi. \tag{3}$$

The alternative networks in the system are supposed to be successively activated by a random walk along the chain, whose jump sizes follow a Gaussian distribution of width ρ (Figure 2). This random walk generates a series δ_i, representing the networks activated at each successive iteration. In this process, correlations within the δ_i series increase as the size of correlation within the chain (r) increases, and decrease as the width ρ of the distribution of jumps increases.

The frequency of the limit cycle (Eq. (1)) is determined, for each successive cycle i, by

$$\omega_i = \omega_0 + \mu\delta_i, \tag{4}$$

where ω_0 represents the baseline frequency.

Delignières *et al.*[37] analyzed the series produced by this model for values of ρ ranging from 15 to 45 and for values of r ranging from 5 to 45. These simulations showed, as expected, that correlations increased as r increased and as ρ decreased. More importantly, the authors found that most combinations produced long-range correlated series, except for the combinations of low r and high ρ, and conversely for high r and low ρ. They concluded that the production of long-range dependence was related to a kind of equilibrium between the two parameters.

In the initial formulation of the model, the Markov chain was conceived as possessing an infinite length. One can consider that the length of this chain represents the number of alternative pathways susceptible to satisfy the task at hand and then provides an index of degeneracy in the network. In order to test the hypothesis linking the level of degeneracy to the strength and the long-range nature of correlations in the produced series, we introduced a new variable in the model, γ, corresponding to the length of the chain over which jumps are performed. The chain is then bounded by two limits δ_1 and δ_γ. In order to allow the model to work despite this limited range, we just reversed the direction of the jump when one or the other limit of the chain was reached.

III. METHODS

We simulated a set of time series with this model, setting the parameters to the following values for the hybrid oscillator: $\alpha = 0.5$, $\beta = 1.0$, $\gamma = 0.02$, $\omega_0 = 4\pi$, and $Q = 0.1$. We tested three different values for r (40, 60, and 80). We used a fixed value for the width of jumps distribution ($\rho = 20$). η was set to 0.1 and μ to 1.0. The length of the chain (γ) was systematically varied from 10 to 200, by steps of 10. Two hundred series of 1024 data points were generated for each range value. Some example series generated by these simulations are shown in Figure 3, for γ values of 10, 50, 100, and 200 (from top to bottom). One can observe the progressive appearance of interpenetrated waves in the series, typical of $1/f$ fluctuations, with the increase of γ.

In order to test for the effective presence of long-range correlations in the series, we applied the ARMA/ARFIMA modeling procedure proposed by Torre *et al.*[44] ARMA refers to auto-regressive moving average models, and ARFIMA to auto-regressive fractionally integrated moving average models. This method consists in fitting 18 models to the studied series. Nine of these models are ARMA (p,q) models, p and q varying systematically from 0 to 2. These ARMA models do not contain any long-range serial correlation. The other nine models are the corresponding ARFIMA (p,d,q) models, differing from the previous ARMA models by the inclusion of the fractional integration parameter d representing persistent serial correlations. One supposes that if the series contains long-range dependence, ARFIMA models should present a better fit than the transient ARMA models. The best model is selected using a goodness-of-fit statistic that is

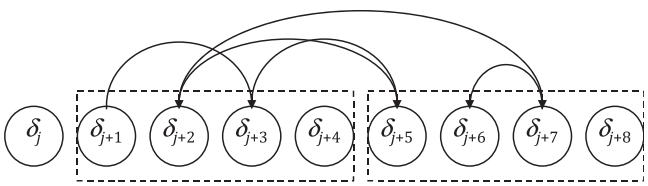

FIG. 2. Illustration of the hopping model. The random walk selects successively the variables δ_{j+1}, δ_{j+3}, δ_{j+5}, δ_{j+2}, δ_{j+7}, and δ_{j+6}. The dashed boxes indicate the size (here $r = 4$) of the correlated zones.

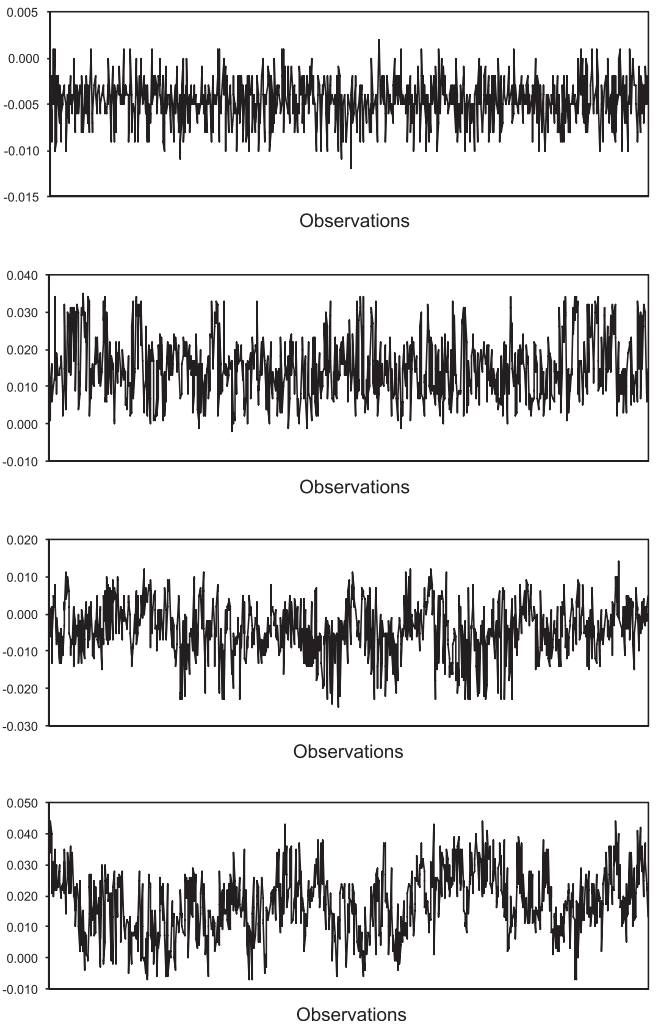

FIG. 3. Example series produced by the hopping model ($N = 1024$). From top to bottom, $\gamma = 10$, $\gamma = 50$, $\gamma = 100$, and $\gamma = 200$.

based on a trade-off between accuracy and parsimony. We used the Bayes Information Criterion (BIC) that was proven to give the best results in the detection of long-range dependence.[44] The ARFIMA/ARMA procedure provides two complementary criteria. The first one is the percentage of series that are better fitted by an ARFIMA model. The second is based on a transformation of the raw BIC values into weights (i.e., the probability that this model is the best over the set of candidate models). We then computed the sum of the weights captured by the nine ARFIMA models, considering that the weights of all tested model sum to one.

In a second step we measured correlations in the series with the Detrended Fluctuation Analysis (DFA).[10] This method is based on the analysis of the relationship between the mean magnitude of fluctuations in the series and the length of the intervals over which these fluctuations are observed. The algorithm of DFA consists first in integrating the series $x(t)$ and calculating for every t the cumulated sum of the deviations of the mean. This integrated series is then divided in non-overlapping intervals of length n. In each interval, a least squares line is fit to the data (representing the trend in the interval). The series is then locally detrended by subtracting to all values the theoretical value given by the regression. For each interval length n, the mean standard deviation $[F(n)]$ of these integrated and detrended series is computed. For fractal series, a power law is expected, as $F(n) \propto n^{\alpha}$, α being the scaling exponent. α is estimated by the slope of the graph representing $F(n)$ as a function of n, in log-log coordinates. $1/f$ fluctuations are characterized by α exponents close to 1, and uncorrelated series by exponents close to 0.5.

IV. RESULTS

The results of ARFIMA modeling are reported in Figure 4. The left panel reports the percentage of series recognized as long-range correlated, as a function of the length of the Markov chain (γ). The results appeared roughly similar for the three values of r. For γ values above 60, the percentage of long-range correlated series fluctuates between 80 and 90%. For lower range values, this percentage decreased dramatically. The median panel illustrates the evolution of the mean sum of ARFIMA weights with the increase of γ. We obtained similar results than with the previous variable, with a quite low plausibility of ARFIMA models for the lowest degeneracy levels. For γ values larger than 60, the mean sum of ARFIMA weights reached a plateau with a mean value of about 87%.

The results of DFA are illustrated in the right panel of Figure 4 and were consistent with ARFIMA results. DFA produced exponents close to 0.5 for the lowest degeneracy values ($\gamma = 10$ and $\gamma = 20$), suggesting the absence of any dependence in the series. The increase of γ yielded a negatively accelerated increase of mean α exponents that reached 0.8 for $\gamma = 80$ and 0.9 for γ values above 140. As expected, mean α exponents increased with the width of the correlation window (r), whatever the γ values.

We also generated a set of 200 series, using the same values for r and ρ, but with a Markov chain of infinite length. This set of series presented similar percentages of series best fitted by ARFIMA models and mean sums of ARFIMA weights than those observed for the highest γ values of the previous simulations. In contrast, mean α exponents were slightly higher, close to that expected from pure $1/f$ fluctuations ($r = 40$, $\alpha = 0.99 \pm 0.15$; $r = 60$, $\alpha = 1.04 \pm 0.16$, $r = 80$, $\alpha = 1.04 \pm 0.15$).

Note that the evolution of ARFIMA and DFA results presented different shapes: DFA suggested a gradual increase of the strength of correlations in the series, while ARFIMA modeling indicated a more abrupt and precocious emergence of long-range correlations. These results are not contradictory, as long-range correlations can appear in rather moderately correlated series.[45]

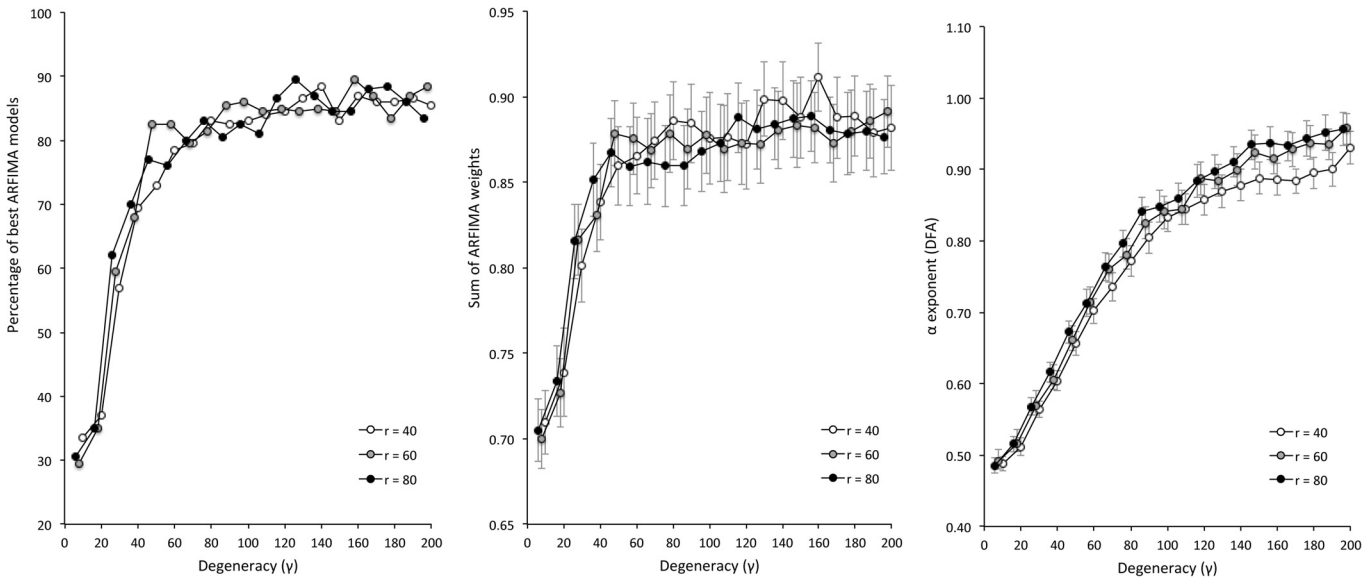

FIG. 4. Fractal properties of the series produced by the model, as a function of degeneracy (γ) and of the width of the correlation window (r). Left: Percentage of series best modeled by ARMIFA models, as a function of γ. Median panel: Mean sum of ARFIMA weights, as a function of γ. Right panel: Evolution of the mean α exponent with γ. Error bars represent 95% confidence intervals.

V. DISCUSSION

A. Degeneracy and long-range correlations

This simulation study confirms that degeneracy produces correlated series, and that a minimum threshold of degeneracy is necessary for obtaining genuine long-range correlated series. These results suggest a direct, causal link between degeneracy and long-range correlations. Increasing degeneracy in the model induces an increase in serial correlations in the produced series. The results obtained for the highest γ values, in the present study, tend asymptotically toward those obtained with a chain of infinite length.

Obviously, the exact values that have been tested in this simulation study should not be considered in absolute, and the architecture of the model should not be conceived as mirroring anatomical structures. This model is too simple for really accounting for the complexity of natural networks. However, we consider that it could reflect some global properties of biological systems and could be helpful for understanding the origins of long-range correlations in physiological time series.

The results show that long-range correlations appear when sufficiently distinct but correlated networks are involved in the production of performance over time. Conversely, long-range correlations tend to disappear when the length of the chain equals the theoretical length of correlated zones within the chain. In that case, one could suppose that all networks share a large amount of common components, and then variability in the resulting series mainly arises from the stochastic terms in the model.[37]

These results open new considerations about variability. Variability has been often conceived as the expression of random and non-significant noise in the system and discarded by means of averaging or filtering before analysis.[46] From this point of view, variability has often been decomposed into a determinist component, representing effective processes, and a stochastic component considered as unexpected fluctuations.[47]

The present approach suggests a completely different conception. The behavior of a complex system, in a given situation, cannot be considered as resulting from the performance of a fixed and immutable network, and its fluctuations as the hallmark of random perturbations. Degeneracy suggests that throughout repetitions, diverse sets of components can be recruited for achieving a given function. As such, variability does not result from "perturbations" within the system, but expresses its inherent complexity. In long-range correlation studies, experimenters try to strictly control for perturbations arising from external sources and then analyse focus of the intrinsic fluctuations of the system.[3] These intrinsic fluctuations provide essential information about the system and its design principles.

In turn, we argue that the strength of long-range correlations in the series produced by a system could be considered a relevant measure of its level of degeneracy. This appears of central interest for diagnosis and prognosis purposes, especially in the domain of rehabilitation. Indeed, if degeneracy represents a resource for adaptability and plasticity, long-range correlation measures could allow predicting the possible evolution of the system and its reactivity to rehabilitation programs.

B. Global or local degeneracy

This hypothetic link between long-range correlation and degeneracy poses interesting questions about the concept of "system." Some recent experimental results allow developing interesting considerations. In an experiment analyzing performance series obtained in finger tapping and circle drawing, Torre *et al.*[48] showed that long-range correlation properties were both individual and task specific. This experiment showed that although the goal of the two tasks was basically the same (producing a regular series of time intervals), there was no correlation between the samples of individual exponents, suggesting that long-range correlations are a task-specific property. However the results also revealed a significant individual effect: each participant tended to produce over tasks a specific level of serial correlation. The authors concluded that long-range correlations emerge from a unique assembly of cognitive–motor processes for each individual performing on a particular task, instead of reflecting some general characteristic of individuals.[48] In the same experiment, the authors tried to assess the reproducibility over trials of long-range correlation properties. They computed over seven successive completions of the task a Cronbach'α of about 0.59, suggesting that long-range correlation properties were also partly trial-specific.

Kello *et al.*[3] analyzed in serial reaction time tasks two simultaneous measures of key-press responses: reaction time and key-contact duration. Results showed that both series exhibited long-range correlations, but the two measures were uncorrelated with each other. The authors stated that even if reaction times and key-contact durations are measures of the same underlying system, they exhibit distinct long-range correlation patterns provided that they reflect the activities of distinct sets of components. Therefore, the intrinsic fluctuations in reaction times were free to vary independently of key-contact durations.

These results converge toward the idea that the presence and the strength of long-range correlations in an experimental series provide information about the specific network that was involved for producing a particular output, in this specific task and in the considered trial. In consequence, long-range correlations cannot be considered as an experimental index of overall degeneracy in the organism that produced the series, but rather of the specific and transient sub-system that was involved in the production of a particular outcome. Obviously, because sub-systems belong to the same organism, they could share sufficient components for explaining a partial consistency in long-range correlations measurements performed among different tasks, or among successive trials in the same task.

C. The origins of 1/f fluctuations

The main hypothesis of this paper suggested a causal link between degeneracy, a design principle that seems ubiquitous in most complex living systems, and the presence of long-range correlations in the times series produced in stable

conditions. This point of view, which suggests that long-range correlations could originate from the structural organization of systems, contrasts with other current hypotheses, which generally refer to more formal properties of complex systems. Currently two theoretical explanations are debated concerning the origins of long-range correlations. Their possible relationships with the degeneracy hypothesis merit some developments.

The first hypothesis that clearly explained long-range correlations by a generic property of complex systems was proposed by Van Orden, Kello, and colleagues.[3,12,34] The central concept of their hypothesis is criticality, a state where systems are in delicate balance between multiple behavioral solutions. Near critical states, systems are metastable, and a small and local perturbation can result in a global change in system's behavior. Critical states made new options for behavior available and then provide the system with adaptability and flexibility to cope with environmental constraints.

Living systems tends spontaneously to stay near critical states.[49] This so-called *self-organized critically* is possible when multiple interactions occur between multiple levels and individual components, a condition that directly refers to system's complexity. Self-organized criticality supposes that the dynamics of the system is dominated by interactions and not by some dominant components within the system.[50] Finally this *interaction-dominant* dynamics is known to produce statically self-similar, fractal fluctuations.[49]

This point of view does not apparently contradict the degeneracy hypothesis. Often degeneracy is described as a principle underlying the anatomical organization of the human nervous system and characterized by a balance between segregation and integration of neural pathways.[31] This balance is thought to allow cortical and subcortical areas to maintain some locality of processing while at the same time participating in globally coordinated patterns of activation. In other words, the balance of segregation and integration may allow metastable patterns to form.[51,52]

An alternative hypothesis has been recently proposed, relating long-range correlations to cascade dynamics.[20,21] As self-organized criticality, cascade dynamics models incorporate interactions across multiple time scales. Multiplicative cascade has been especially developed for modeling energy transfer across scales in complex systems.[53] In these models, energy transfer works from the coarsest to the finest scales, and variation on the finer scales is given by multiplying the variation on the coarser scales with random multipliers.

As previously stated, self-organized criticality produces mono-fractal fluctuations in behavior. In contrast, multiplicative cascade dynamics produce more complex patterns, called *multifractal* fluctuations. In the monofractal model, scale invariance is numerically defined by a single exponent. In multifractal series, the behavior around any point is described by a *local* scaling exponent, which varies over time, and then scaling invariance is defined by a spectrum of scaling exponents, rather than by a single average value.

Multifractals are able to explicitly define the width and shape of the spectrum of scaling exponents by the width and shape of the distribution of interaction multipliers, and there is a formal relationship between the local variation of scaling exponents and the multiplicative interactions between temporal scales. As such, seeking for the presence of multifractal fluctuations in the behavior of systems could allow to test the cascade dynamics hypothesis against that of self-organized criticality.[20] Multifractals have been evidenced, for example, in human gait, simple, and choice reaction time, word naming, or interval estimation.[20,21,42,54]

This cascade dynamics hypothesis could also be consistent with degeneracy. In the present experiment we limited our investigations to monofractal analyses. However, the very first attempts for analyzing the neural hopping model focused on multifractal properties.[36,41] These studies showed that the hopping model generated series possessing multifractal properties, and Ashkenazy *et al.*[36] found that increasing the width of jumps distribution (i.e., ρ in our notation) yielded a decrease in the width of the multifractal spectrum.

Degeneracy does not appear as a third hypothesis, competing with the two former. It shares with the two other theories the idea that long-range correlations arise from generic principles, common to most complex systems. However, in contrast with the two former hypotheses, which work at a very abstract level, degeneracy offers an explanation in terms of structural and functional organization that could allow novel and maybe more heuristic perspectives. Degeneracy seems particularly interesting from a neurobiological point of view, because research in this domain focuses on structures and organizations, especially through cortical imagery. This approach could allow a better dialogue with experimental psychologists, which are more inclined toward formal models.

Finally, the degeneracy hypothesis could afford new points of view about very classical concepts and theories. For example, Wijnants *et al.*[55] analyzed the effect of practice in a reciprocal aiming task. In order to obtain significant practice effects, participants were instructed to perform the task with their non-dominant hand. The results evidenced a gradual increase of long-range correlations in movement time series, over the five successive blocks of practice. This effect could be interpreted as the progressive emergence of degeneracy in the organization of the underlying network. In other words, initial attempts for performing a novel or unusual task could be essentially characterized by the involvement of rather simple, non-degenerate networks. Learning, or performance optimization, could be understood as a progressive increase of the complexity of the underlying network. This idea nicely challenges more traditional views considering learning as a process aiming at overcoming the initial complexity of the system toward a more simple and controllable organization.[56]

VI. CONCLUSION

In the present paper we show on the basis of theoretical arguments and a simulation experiment that degeneracy could underlie the presence of long-range correlations in the outcome series produced by complex systems. In turn, we suggest that quantifying long-range correlations could allow to assess the level of degeneracy of the system. We argue that degeneracy should be considered a function-specific

property, characterizing the networks involved in the production of a given outcome. We consider that this hypothesis does not compete with former theoretical explanations about the origins of long-range correlations but enriches the theoretical debate with a novel and complementary point of view. Especially, degeneracy affords a maybe more intuitive way than former hypotheses for understanding the effects of complexity on essential properties such as robustness and adaptability.

[1]G. E. P. Box and G. Jenkins, *Time Series Analysis: Forecasting and Control* (Holden-Day, San Francisco, 1976).
[2]A. Eke, P. Herman, J. B. Bassingthwaighte, G. M. Raymond, D. B. Percival, M. Cannon, I. Balla, and C. Ikrenyi, Pflugers Arch. Eur. J. Physiol. **439**, 403 (2000).
[3]C. T. Kello, B. C. Beltz, J. G. Holden, and G. C. Van Orden, J. Exp. Psychol. Gen. **136**, 551 (2007).
[4]B. Mandelbrot and J. W. Van Ness, SIAM Rev. **10**, 422 (1968).
[5]E. J. Wagenmakers, S. Farrell, and R. Ratcliff, Psychon. Bull. Rev. **11**, 579 (2004).
[6]B. J. West, *Where Medicine Went Wrong: Rediscovering the Path to Complexity* (World Scientific, London, 2006).
[7]B. E. Hurst, Trans. Am. Soc. Civ. Eng. **116**, 770 (1951).
[8]M. Matsuzaki, Philos. Trans.: Phys. Sci. Eng. **348**, 449 (1994).
[9]W. E. Leland, M. S. Taqqu, W. Williger, and D. V. Wilson, IEEE/ACM Trans. Netw. **2**, 1 (1994).
[10]C. K. Peng, S. Havlin, H. E. Stanley, and A. L. Goldberger, Chaos **5**, 82 (1995).
[11]D. L. Gilden, Psychol. Sci. **8**, 296 (1997).
[12]G. C. Van Orden, J. G. Holden, and M. T. Turvey, J. Exp. Psychol. Gen. **132**, 331 (2003).
[13]D. L. Gilden, T. Thornton, and M. W. Mallon, Science **267**, 1837 (1995).
[14]L. Lemoine, K. Torre, and D. Delignières, Can. J. Exp. Psychol. **60**, 247 (2006).
[15]J. M. Hausdorff, C. Peng, Z. Ladin, J. Wei, and A. Goldberger, J. Appl. Physiol. **78**, 349 (1995).
[16]K. Jordan, J. H. Challis, and K. M. Newell, Gait & Posture **24**, 120 (2006).
[17]K. Torre, D. Delignières, and L. Lemoine, Exp. Brain Res. **183**, 225 (2007).
[18]A. Diniz, M. L. Wijnants, K. Torre, J. Barreiros, N. Crato, A. M. T. Bosman, F. Hasselman, R. F. A. Cox, G. C. Van Orden, and D. Delignières, Human Mov. Sci. **30**, 889 (2011).
[19]J. M. Hausdorff, J. NeuroEng. Rehabil. **2**, 19 (2005).
[20]E. A. F. Ihlen and B. Vereijken, J. Exp. Psychol. Gen. **139**, 436 (2010).
[21]D. G. Stephen, J. R. Anastas, and J. A. Dixon, Fron. Physiol. **3**, 102 (2012).
[22]J. M. Hausdorff, S. L. Mitchell, R. Firtion, C. K. Peng, M. E. Cudkowicz, J. Y. Wei, and A. L. Goldberger, J. Appl. Physiol. **82**, 262 (1997).
[23]J. M. Hausdorff, Chaos **19**, 026113 (2009).
[24]A. L. Goldberger, L. a. N. Amaral, J. M. Hausdorff, P. C. Ivanov, C. K. Peng, and H. E. Stanley, Proc. Natl. Acad. Sci. U.S.A. **99**, 2466 (2002).
[25]L. Lipsitz and A. Goldberger, JAMA, J. Am. Med. Assoc. **267**, 1806 (1992).
[26]D. E. Vaillancourt and K. M. Newell, Neurobiol. Aging **23**, 1 (2002).
[27]J. M. Whitacre and A. Bender, J. Theor. Biol. **263**, 143 (2010).
[28]J. M. Whitacre, Theor. Biol. Med. Modell. **7**, 6 (2010).
[29]J. A. S. Kelso, *Dynamics Patterns: The Self-organization of Brain and Behavior* (MIT Press, Cambridge, MA, 1995).
[30]G. M. Edelman and J. A. Gally, Proc. Natl. Acad. Sci. U.S.A. **98**, 13763 (2001).
[31]G. Tononi, O. Sporns, and G. Edelman, Proc. Natl. Acad. Sci. U.S.A. **91**, 5033 (1994).
[32]G. Tononi, O. Sporns, and G. M. Edelman, Proc. Natl. Acad. Sci. U.S.A. **96**, 3257 (1999).
[33]J. M. Carlson and J. Doyle, Proc. Natl. Acad. Sci. U.S.A. **99**, 2538 (2002).
[34]B. B. Beltz and C. T. Kello, *in Focus on Cognitive Psychology Research*, edited by M. A. Vanchevsky (Nova Science Publishers, Hauppauge, NY, 2006), pp. 25–41.
[35]D. L. Gilden, Psychol. Rev. **108**, 33 (2001).
[36]Y. Ashkenazy, J. A. Hausdorff, P. C. Ivanov, and H. E. Stanley, Phys. A: Stat. Mech. Appl. **316**, 662 (2002).
[37]D. Delignières, K. Torre, and L. Lemoine, Acta Psychol. **127**, 382 (2008).
[38]D. Delignières and K. Torre, J. Appl. Physiol. **106**, 1272 (2009).
[39]J. M. Hausdorff, P. L. Purdon, C. K. Peng, Z. Ladin, J. Y. Wei, and A. L. Goldberger, J. Appl. Physiol. **80**, 1448 (1996).
[40]J. M. Hausdorff, Y. Ashkenazy, C. K. Peng, P. C. Ivanov, H. E. Stanley, and A. L. Goldberger, Phys. A: Stat. Mech. Appl. **302**, 138 (2001).
[41]B. J. West and N. Scafetta, Phys. Rev. E **67**, 051917 (2003).
[42]N. Scafetta, D. Marchi, and B. J. West, Chaos **19**, 026108 (2009).
[43]B. A. Kay, J. A. Kelso, E. L. Saltzman, and G. Schöner, J. Exp. Psychol. Hum. Percept. Perform. **13**, 178 (1987).
[44]K. Torre, D. Delignières, and L. Lemoine, Br. J. Math. Stat. Psychol. **60**, 85 (2007).
[45]V. Marmelat and D. Delignières, Med.-Lith. **47**, 393 (2011).
[46]A. B. Slifkin and K. M. Newell, Curr. Dir. Psychol. Sci. **7**, 170 (1998).
[47]M. A. Riley and M. T. Turvey, J. Motor Behav. **34**, 99 (2002).
[48]K. Torre, R. Balasubramaniam, N. Rheaume, L. Lemoine, and H. N. Zelaznik, Psychon. Bull. Rev. **18**, 339 (2011).
[49]P. Bak, *How Nature Works: The Science of Self-organized Criticality* (Springer-Verlag, New York, 1996).
[50]H. J. Jensen, *Self-Organized Criticality* (Cambridge University Press, Cambridge, England, 1998).
[51]K. J. Friston, Neuroimage **5**, 164 (1997).
[52]O. Sporns, *in Coordination Dynamics: Issues and Trends*, edited by V. Jirsa and J. A. S. Kelso (Springer-Verlag, Berlin, 2004), pp. 197–215.
[53]B. Mandelbrot, J. Fluid Mech. **62**, 331 (1974).
[54]N. Scafetta, L. Griffin, and B. J. West, Phys. A: Stat. Mech. Appl. **328**, 561 (2003).
[55]M. L. Wijnants, A. M. T. Bosman, F. Hasselman, R. F. A. Cox, and G. C. Van Orden, Nonlinear Dyn. Psychol. Life Sci. **13**, 79 (2009).
[56]R. Schmidt and T. Lee, *Motor Control and Learning: A Behavioral Emphasis*, 4th ed. (Human Kinetics, 2005).