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# Structural stability can shape biological evolution

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## Abstract

We analyze a model of biological evolution recently introduced in the literature and show that punctuated equilibrium and power-law behavior arise because of the differential structural stability of the mapping between genotype and phenotype that characterizes the model. Structural stability is a feature of biological organisms that had been introduced by biologists long ago, but never modelled until now, as far as we know. This model can be regarded as an alternative to those models that explain both the power-law behavior and the punctuated equilibrium of extinction curves by recourse to self-organized criticality (SOC). © 2000 Elsevier Science B.V. All rights reserved.

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## 1. Introduction

The last few years have witnessed great activity in the study of physical models that mimic facets of the biological evolution processes, shedding much light on diverse features of the Darwinian landscape. The goal in this respect has been that of devising ingenious and at the same time *simple* theoretical models that may provide one with some insight concerning punctuated equilibrium, self-organized criticality, and other features of biological evolution [1–8]. Recently, a somewhat more realistic model than those previously in existence was introduced [9–15].

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One sees in Ref. [11] that, appearances notwithstanding, the model advanced in Ref. [9] does not exhibit self-organized criticality (SOC) [16]. A different type of dynamical evolutive process is at work, mathematically grounded on phenotype–genotype correlations. Thus, this model constitutes a new approach on the study of the dynamics of biological evolution, representing an alternative to those models that need SOC to explain the power-law behavior and punctuated equilibrium of extinction curves.

SOC models are very interesting indeed, but, thus far, they appear too simple (in mathematical terms) to explain other biological facts than the ones mentioned in the preceding paragraph (the same can be said of some recent non-SOC models, see for instance, Refs. [5–8]). Our model, instead, is complex enough to explain some additional and interesting biological facts. Some of them are [9–15]:

(1) Punctuated equilibrium and power-law distributions of (i) species' lifetimes, (ii) extinction curves, and (iii) frequency spectra, of the extinction curves (all of them in total agreement with the data obtained from the fossil records).

(2) It exhibits oscillatory modes that introduce some periodicity into the system, as observed by some biologists. The model also integrates this fact with the apparently contradictory power-law decay of the power spectrum of the extinction curves (observed by other biologists).

(3) Explanation (qualitative and quantitative) of the mathematical reasons underlying the selectivity of extinction events.

(4) It is also the first mathematical model that gives numerical support to Kimura's neutral theory of molecular evolution.

(5) The model distinguishes between evolutive rules acting at the phenotypic and at the genotypic levels.

(6) The effect of the external environment, that “deforms” the power-law curves in the appropriate fashion, as observed in the fossil records (giving them a rather concave form). The model also generates a smooth integration (a non-trivial fact) at all scales of both inter-species interactions and environmental (external) influences, giving rise to a coherent dynamical picture a la Maturana and Varela.

(7) The model is also able to account for the the effect of symbiosis, that also deforms the power-laws curves. Also, the introduction of symbiosis gives numerical support to earlier speculations concerning an “adaptive grid lock” mechanism.

It should be pointed out that the aforementioned predictions are of a mathematically robust nature. Now, if one takes inventory of the results reported in Refs. [9–15], some intriguing questions remain that deserve further clarification. In this vein, we ask ourselves the following questions: (1) How should we interpret, in biological terms, the phenotype–genotype map, which constitutes the essential feature of the model?

(2) Assuming that we are able to interpret this map, is that interpretation itself able to generate *new predictions*?

It is the aim of the present communication to answer these questions, in the understanding that they will help not only to clarify the inner workings of a rather successful model, but also to try and elucidate which roads one should traverse in an investigation of mathematical evolutive models. Additionally, we hope to be able to

shed some modest amount of light on the actual biological process itself, as customarily expected from any *physical model*. Consequently, we will in this Communication concentrate our efforts in trying to relate the biological concept of structural stability to the above-mentioned phenotype–genotype mapping. It will be seen that interesting conclusions are arrived at. We begin the concomitant task, for the readers’ benefit, with a brief recapitulation of the model under consideration.

## 2. The model: a brief recapitulation

The model [9,11] deals with  $M$  distinct, interacting biological species, each of which is represented by a vector in  $\mathcal{R}^N$ . The components  $V_\alpha^i$  of  $V^i$  represent different phenotypic features ( $\alpha, \beta, \dots, N$ ) that are to be affected and modified by the evolutionary process. The degree to which the  $i$ th species is “adapted” to the environment is represented by a quantity  $F_i$ , to be called its “fitness”, which is given by the expression

$$F_i = \sum_j^M \sum_{\alpha\beta}^N g_{\alpha\beta}^{ij} V_\alpha^i V_\beta^j + \sum_\gamma^N V_\gamma^i A_\gamma^i, \quad i = 1, \dots, M, \quad (1)$$

where the hyper-matrix  $g_{\alpha\beta}^{ij}$  provides the details of the inter-species interaction and the second term on the r.h.s. is an “environmental” one. In Ref. [9] one assumed that  $g_{\alpha\beta}^{ij} = -g_{\beta\alpha}^{ji}$ .

There is a correlation between the components of  $V^i$  which is represented by recourse to mappings between a set of  $k+1$  real parameters and each of these components, i.e.,

$$V_\beta^i = f_\beta(a_0^i, \dots, a_k^i) \quad (2)$$

with  $f_\beta$  being an appropriate function. The set  $a_0^i, \dots, a_k^i$  defines in fact the  $i$ -species. As a result of biological mutations, these parameters are allowed to vary with time.

The conceptual difference between the  $a$ ’s (that span an “ $A$ -space”) and the  $V$ ’s (a “ $V$ -space”) should be here emphasized. The components of  $V$  denote different phenotypic features of the species that this vector represents. These features are correlated, via the  $a$ ’s, in the manner just described. Genetic changes, here mimicked by modifications in the  $a$  -values, drive the evolutionary process. The correlations just mentioned constitute an essential aspect of the model. Without them, a given species might (eventually) attain, after a series of appropriate mutations, *any* phenotypic feature whatsoever. This does not happen in nature. Summing-up, changes in our  $a$ ’s are to be understood as reflecting uncorrelated genetic modifications (changes in one or more bases in one or more genes) while changes in the  $V$ ’s represent the concomitant phenotypic modifications that, to a greater or lesser extent, will be mutually correlated. The  $a$ ’s do not represent the genetic code in itself, but only the correlations introduced by its existence.

Our model-system evolves in the following fashion: we start with an arbitrary initial configuration (the  $a_i$  are randomly chosen within  $[-1, 1]$ ) and, in each of a series of time steps, mutation effects are mimicked by slightly modifying the  $a_i$  for the  $l$ th

species. Both the selection of  $l$  and the nature of the changes are random. The condition  $|V^i| = 1$  is enforced so as to avoid unrestricted growth (with time) of  $|V^i|$ .

A particular mutation (change in a given  $a$ ) is “accepted” if it increases the corresponding fitness (as a consequence of such a mutation  $F_i$  grows). The  $a$ -change is, in this case, retained. Otherwise, it is discarded and the  $a_i$  end up with their previous values.

As the features of each species are in a state of continuous change, there is a considerable amount of “evolutive activity”, that we shall quantify with reference to the motion of a “center of mass”

$$X_{CM} = \sum_{i=1}^M V^i, \tag{3}$$

whose value is to be ascertained at a series of regularly spaced times. Then, we define the evolutive activity ( $EA$ ) as

$$EA = |\delta_t X_{CM}|^2,$$

where  $\delta_t$  represents variation in an arbitrary number  $t$  of time steps (in this and previous works we use  $t = 1000$ ).

For the sake of simplicity, we took in Refs. [9,11] (i) our hyper-matrix in the form

$$g_{\alpha\beta}^{ij} = k_{ij} \delta_{\alpha\beta} \tag{4}$$

and (ii)  $A_\gamma^i = 0$ . Thus, (1) reduces to

$$F_i = \sum_{j=1}^M k_{ij} V^i \cdot V^j, \quad i = 1, \dots, M, \tag{5}$$

where  $k_{ij} = -k_{ji}$ .

The  $k_{ij}$  are randomly chosen within  $[-1, 1]$  (for  $i < j$ ) and are kept constant throughout. The functional form for the  $\beta$ -component of  $V^i$  (cf. Eq. (2)) was chosen to be of a rather general form. We write

$$f_\beta = \sum_{n=0}^k a_n x^n, \quad x = \beta/N, \tag{6}$$

so that

$$V_\beta^i = \frac{\sum_{n=0}^k a_n^i (\beta/N)^n}{\sqrt{\sum_\beta \sum_{n,m=0}^k a_m^i a_n^i (\beta/N)^{n+m}}}. \tag{7}$$

### 3. Developmental mechanisms

It was shown in Ref. [11] that the source of the power-law behavior of the model advanced in Ref. [9] is to be located in the details of the  $A \rightarrow V$ -mapping. More precisely, it is linked to the fact that the vector  $\mathbf{a}$  traverses large regions of the  $A$ -space

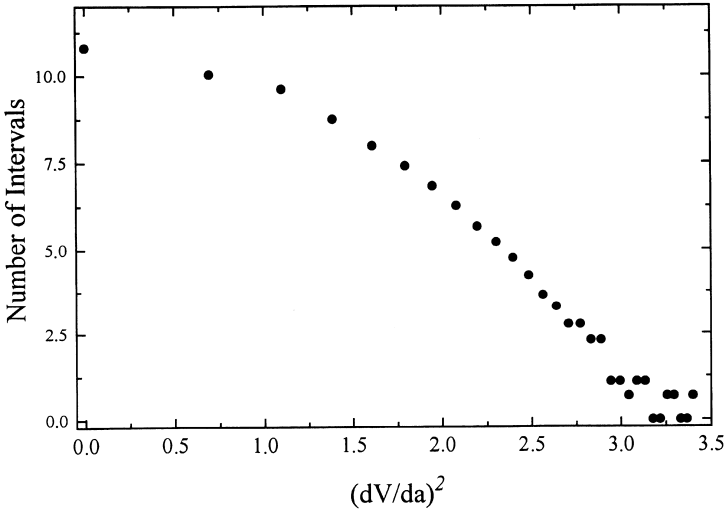


Fig. 1. Histogram depicting the distribution of  $(|\delta V|/|\delta \mathbf{a}|)^2$  (arbitrary units) over the unit hypersphere (see text).

in recurrent fashion. As the mapping of genotypes to phenotypes exhibits a strong change in its properties from region to region of the space of genotypes, the *same* evolutionary mechanism can give rise to either neutral evolution or to non-neutral selection. Which one is observed for a given species depends on the properties of this mapping at the species' current position in the space of genotypes.

This is easily appreciated by taking a look at the  $V$ -space mapping, focusing our attention specifically on the  $|\delta V|$  vs.  $|\delta \mathbf{a}|$  behavior, as we traverse the surface of an hypersphere  $\mathcal{S}$  of radius  $|\mathbf{a}| = \text{Constant}$ . Fig. 1 is a log.-log. plot of the distribution of  $(|\delta V|/|\delta \mathbf{a}|)^2$ . Randomly generated  $\mathbf{a}$ -vectors belonging to an  $\mathcal{S}$  of unit radius are subjected to small (and also arbitrary) variations  $\delta \mathbf{a}$  tangent to  $\mathcal{S}$  (see Ref. [11] for more details). Here, we find a remarkable resemblance with a graph depicting the distribution of evolutive activity. We display it in Fig. 2, with reference to examples examined in Ref. [11], which should be consulted for more details.

As demonstrated in Ref. [11], without correlations the power-law behavior (PLB) disappears, as a consequence of the fact that the mapping's features do not change as  $\mathbf{a}$  moves around. A natural question thus arises: what does this mathematical map mean? What is it really modelling?

Biologists have recognized at all times that evolution is a strongly constrained process [17,18]. The hypothesis that gene-variation might occur in any direction has been much criticized. Darwin considered internal correlations within the organism, such that selection for one feature would pull along other, correlated features, but notwithstanding this, he stressed the capacity for variation in almost any direction. It is fair to say that much of late-19th-century and early-20th-century biology was concerned with refuting this claim [18].

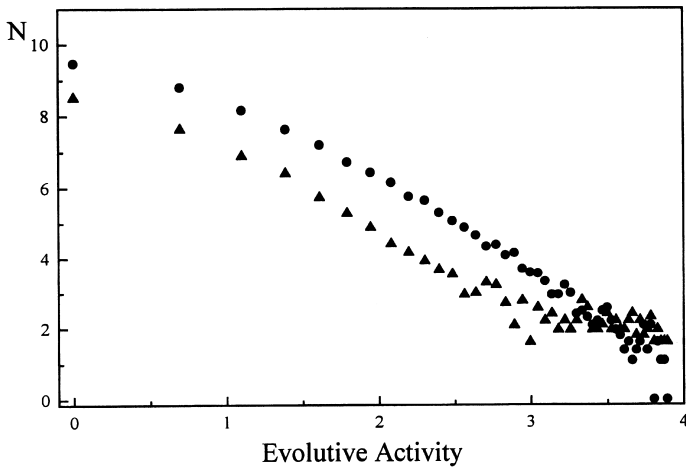


Fig. 2. Histogram depicting the distribution of evolute activities for two different examples described in Ref. [11] (arbitrary units).

Heterochronic studies posit that changes in the time of onset of particular developmental pathways in an organism, changes in the rate of progression of such developmental processes, and variations in their time of cessation can change the resulting organism. If, for instance, limb development were initiated earlier in one species and lasted longer in that species than in its neighbors, limbs in the first species would be expected to last longer. Such alterations in the timing of growth and differentiation are clearly a way to deform one organism into a closely neighboring one.

Allometric transformations, studied by Thompson [19] in his analysis of coordinate transformations mapping one form into a family of neighboring forms, and named and studied in detail by Huxley [20], belong to the same tradition as the analysis of heterochrony. The analysis of patterns in bio-neighborhoods can be based on the attitude of conceiving an organism as generated by a developmental algorithm. In such a situation, we could be in a position to determine the generative algorithm (more properly, the developmental mechanism), and consequently which the truly neighboring organisms are. Consider the phyllotaxis, seen in pine cones and sun flowers. The scales, as is well known, form in double spirals which radiate from the center, one clockwise, the other counterclockwise. The surprising feature is that the number of spirals in one direction is related to the number in the other direction as two adjacent numbers in the Fibonacci series  $1, 1, 2, 3, 5, 8, 13, 21, 34, \dots$ . A number of models to account for this double-spiral pattern have been suggested: see for instance the attractive example due to Mitchison [21], who suggests that tight packing of scale primordia on the conical meristem suffices to generate the observed phyllotactic series. The issue here is not the adequacy of his model, but the mode of analysis it exemplifies. First, given the presumptive developmental mechanism, we have an algorithm to generate a given form and, by modifying parameters of the model, a way of computing and predicting

neighboring forms. Thus the developmental mechanism predicts the family of forms it will generate. Moreover, unlike the cases of heterochrony and allomorphic transformation just noted, where a continuous gradation of neighboring forms occurs, here true neighbors are adjacent pairs in the Fibonacci series: either 8–13 or 13–21. Thus, true neighboring morphologies in evolution reflect transformations to neighboring forms in the family of forms generated by the underlying developmental mechanisms. The organism cannot vary equally in any direction but is strongly biased by the underlying developmental algorithm to vary among a defined set of neighboring morphologies. This point of view has been expressed by a number of authors [22–24].

As a consequence, developmental mechanisms give rise to families of forms, and transitions among these forms are due to shifts in the parameters of the underlying mechanisms. Morphological fitness landscapes might be smooth or rugged. If basin volumes in state space and bifurcation volumes in parameter space are like balloons, then small changes in states or parameters lead to small changes in morphology. If basins of attraction and parameter volumes are tortuous and intertwine, then small genetic changes yielding small smooth changes in initial states, or in parameters, would cross boundaries separating sharply different morphologies.

#### **4. Structural stability**

We introduce now the important concept of structural stability [25,26], that stems from the idea that, typically, volumes in parameter space defined by bifurcation surfaces are like soap bubbles. The volumes are reasonably large relative to the bifurcation surfaces which divide them. Thus, for most changes in the parameters, the system remains within one volume in parameter space and the dynamical behavior does not change in dramatic fashion. Dynamical systems having this property are said to be structurally stable. Their dynamics typically change only slightly as parameters vary but does occasionally “jump”, crossing bifurcation surfaces. For some time it was thought that almost all dynamical systems exhibit this property. However, recent work on strange attractors indicates that a large class of systems does not exhibit structural stability. In many systems with strange attractors, variation of the parameters leads to a succession of bifurcations at successively smaller intervals in parameter space [27]. Such an effect is seen in the famous period-doubling bifurcations studied by Feigenbaum [28], which may underlie the onset of turbulence in fluid flows. In these cases, the volumes separating qualitatively different behaviors become sinuous, intertwined labyrinths in parameter space. Here, tiny changes in parameters in almost any direction can lead to successive dramatic changes in the dynamical behavior of the correlated dynamical system.

One implication of the occurrence or non-occurrence of structural stability is that, in structurally stable systems, smooth walks in parameter space (our genotypic space) lead mostly to smooth changes in dynamical behavior (our phenotypic, or morphological space).

Having said that, it is evident now that the genotype to phenotype mapping of our model represents the differences in structural stability that are inherent to all organisms that evolve using genetic codes. Not all organisms (i.e., genomas or developmental algorithms) react in the same fashion, with similar intensity, to small perturbations (i.e., mutations, or small parameter changes).

Our model does not explicitly model the developmental algorithm, but *does it implicitly*, because it models differences in structural stability. As a result we can assert that all features of our model, from punctuated equilibrium to power-law behavior, have their origin in the differences of structural stability of the different genotypes.

Adopting this viewpoint represents an entirely new way of looking at evolution. In fact, we gather from our model that differences in structural stability alone are capable of explaining most features of the fossil record, with no need of invoking other mechanisms (which would not then play any important role), a somewhat unexpected result.

## 5. A prediction of our model

The observed independence between the molecular evolutionary clock and morphological evolution presents a strong challenge to any evolutive model. For a number of different proteins or DNA sequences, the rate of amino acid or nucleotide substitution appears approximately constant [29–31]. Thinking of a molecular clock ticking at a fairly uniform rate in protein evolution seems surprising when the protein sequences of chimpanzees and humans are compared. They are nearly identical. Nevertheless, the concomitant morphological variation is considered (at least by human classifiers) to be great. In contrast, divergent species of frogs are morphologically similar but exhibit strong differences at the DNA and protein levels. This important set of facts led Wilson and co-workers [32,33], and subsequently many others, to suppose that the major changes in morphological evolution were due to mutations not in structural genes but rather in regulatory ones, whose actions determine the action of other regulatory and structural genes. The general idea is that a single regulatory mutation can cause very large alterations in patterns of gene expression by disrupting the coordinating behavior of the genomic regulatory system [32,34–38].

Among mathematical models of evolution, ours is, as far as we know, the only one able to reproduce the above-mentioned uniform rate of genomic evolution (see Refs. [11,13]). We are thus in a position to make a prediction of a biological nature: *the relative proportions of structural and regulatory genes should change during the evolutive process*. For instance, in frogs the percentage of structural genes should be large, and in primates, small. The concomitant experiments are not yet feasible, but will eventually be performed. Thus, we find here a falsifiable prediction of our model. Consequently, as far as we know, our mathematical model is the only evolutive one that possesses the Popperian falsifiability characteristic, essential to the workings of the scientific endeavour.

## 6. Conclusions

We have shown, with reference to the model of biological evolution of Ref. [9], that punctuated equilibrium and power-law behavior arise because of the differential structural stability of the mapping between genotype and phenotype that characterizes the model.

We stress that the model advanced in Ref. [9] is, thus far, the only one based on the distinction between the genotypic and the phenotypic levels. We insist on the fact that punctuated equilibrium and power-law behavior arise, not because of self-organized criticality, but as a consequence of the above-mentioned differential structural stability of the mapping between genotype and phenotype.

Structural stability is a feature of biological organisms that has long been stressed by biologists, but *never modelled until now*. The essential content of the present Communication is the following: we exhibit, for the first time, the fact that our model is based on the structural stability concept. Ideas on the importance of this fact from a biological perspective have also been advanced. Finally, a concrete prediction has been made, on the basis of this model, that could be subjected to experimental testing.

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