

Dynamical mechanisms for biological evolution

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Both the power law and the punctuated equilibrium phenomena, which have lately been regarded as an essential requirement in order to model biological evolution, are analyzed here with reference to a simple model recently introduced in the literature. We show that these features are inherent in the associated dynamics, without recourse to external perturbations. It is seen, also, that correlations among phenotypic features constitute an essential ingredient. Comparison with previous evolutive models that exhibit self-organized criticality is also made. [S1063-651X(97)10306-3]

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I. INTRODUCTION

Gould [1] has conjectured that biological evolution takes place in terms of intermittent bursts of activity separating relatively long periods of quiescence, rather than in a gradual manner.

This intermittent pattern has been observed by Raup, Sepkosky, and Boyajian [2–5] by studying fossil records. Gould has coined the term “punctuated equilibrium” to describe the intermittent behavior of the evolution of single species.

It has been suggested that extinction events are caused by external forces (at least the larger ones), such as changing sea levels [6], worldwide climate pulses [7], or meteorites [8]. Plausible as this is, punctuated equilibrium may well be the natural consequence of the dynamics of biology itself, with no need for external triggering mechanisms.

Following this idea, it has been suggested that the ecology of interacting species has evolved to a self-organized critical state [9,10]. Self-organized criticality [11,12] refers to the tendency of some dynamical systems to organize themselves into a “poised” state far out of equilibrium (at the edge of chaos) with propagating avalanches of activity of all sizes.

However, theoretical investigations have been hampered by the difficulty of constructing even remotely realistic, yet tractable mathematical models. First, punctuated equilibria were observed by Bak, Chen, and Creutz [9] in the “Game of Life,” a simple computer model of a society of living and dying individuals living on a two dimensional lattice. However, it is not robust against small changes in the rules, as it should be in order to represent real evolution. Later, Kauffman and Johnsen [10] studied elaborate “NKC models” of coevolving species evolving at the edge of criticality, with periods of stasis interrupted by coevolutionary avalanches. However, as these models were driven they do not self-organize: some external tuning of the system was needed to obtain critical behavior [13,14].

Finally, Bak, Sneppen, and Flivbjerg [15,16] studied a very ingenious model of an evolving biology which self-organizes into a critical steady state. Notwithstanding its relevance and originality, the model is too simple to allow for

reasonable conjectures concerning terrestrial biology.

A more involved model, inspired in Refs. [15,16], has been recently proposed [17] that *mixes contraption and design with randomness*. The model exhibits some criticality features, in particular, in what pertains to power laws in the distribution of the intensities of the evolutive avalanches, as obtained by Raup [3] on the basis of fossil records.

More specifically, it was found in [17] that its dynamics is complex enough so as to be able to mimic some facets of actual biological evolution. On the basis of the model one may understand that several (simple) dynamical mechanisms exist that account for these facets without recourse to catastrophic events.

The main *new* feature of the model of [17] resides in that it allows species, say, A , to respond in *gradual* fashion to changes affecting another species (B) in such a manner that second-order effects arising out of the A - B interaction may influence a *third* species, and so on. A whole chain of changes thus ensues that takes place within the same time scale in which B is changing. Actions and reactions occur almost simultaneously, which is not the case of the model of Bak *et al.* [15,16].

In view of the relative success of the model advanced in Ref. [17] we think it appropriate to try to qualitatively understand the reasons that underlie that success, under the assumption that some qualitative features of the concomitant dynamics should be identified and considered responsible for it. If these features can be pinpointed, further progress can surely be made in constructing more realistic models.

In this vein we ask ourselves the following questions. (1) What exactly is the role that, in this model, correlations between phenotype and genotype play? Are they an essential dynamical ingredient responsible for the punctuated equilibrium (and power law) behavior or just a superfluous feature? (2) Are the mechanisms that originate the punctuated equilibrium and power law behavior in the present model similar to those of the pioneer models referred to above (in the sense that the dynamics of the model necessarily involves evolution towards a self-organized critical state) or do we find here *alternative* dynamical mechanisms?

It is the aim of the present paper to answer these questions. We begin the concomitant task with a brief review of the model under consideration.

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II. MODEL

We deal with M distinct, interacting biological species, each of which is represented by a vector in \mathbb{R}^N . The components V_α^i of \vec{V}^i represent different phenotypic features (α, β, \dots) 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,’’ a vocable that (in biology) would encompass fecundity, fertility, and other factors relevant to reproductive success. In our model F_i 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 hypermatrix $g_{\alpha\beta}^{ij}$ provides the details of the inter-species interaction and the second summand on the right-hand side is an ‘‘environmental’’ one (see below). As in Ref. [17] we assume $g_{\alpha\beta}^{ij} = -g_{\beta\alpha}^{ji}$. This is a reasonable assumption, because if the α feature of the species i gives it a competitive edge against the β feature of the j species, the latter gives it, of course, a competitive disadvantage against the former (symbiosis is excluded). The A_γ^i matrix mimics the environmental influence (such as climate, geography, etc.) over the γ feature of the i th species. Notice the strong similarity of the right-hand side above with that of the right-hand side of Volterra’s dynamics for the populations N_i of M interacting species [18]

$$\frac{dN_i}{dt} = \sum_{j=1}^M A^i N_i + g^{ij} N_i N_j, \quad (2)$$

where, of course, the symbols have different meanings which we do not need to explain here.

Of course, the components of \vec{V}^i must necessarily exhibit some degree of correlation, as genes are simultaneously involved in several phenotypic features. This correlation is represented by recourse to mappings between a set of $k+1$ real parameters and each of these components, i.e.,

$$f_\beta : a_0^i, \dots, a_k^i \rightarrow V_\beta^i, \quad (3)$$

that is,

$$V_\beta^i = f_\beta(a_0^i, \dots, a_k^i), \quad (4)$$

with f_β 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 family of all these (M) sets can be called the *configuration* of the system.

The conceptual difference between the a ’s and the V ’s should be emphasized here. 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 (see [17] for more details).

The 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 $|\vec{V}^i| = 1$ is enforced so as to avoid unrestricted growth (with time) of $|\vec{V}^i|$.

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 (‘‘selection’’ acting on ‘‘single genes’’). Otherwise it is discarded and the a_i end up with their previous, old values.

Extensive numerical studies suggest that such a system never reaches an equilibrium situation, a fact that could be guessed from the skew symmetry of g^{ij} [19]. The number of species, M , is kept constant for the sake of simplicity. However, as the features of each species are in a state of continuous change, there is a considerable amount of ‘‘evolutionary activity,’’ that we shall quantify with reference to the motion of a ‘‘center of mass’’

$$\vec{X}_{\text{c.m.}} = \sum_{i=1}^M \vec{V}^i \quad (5)$$

whose value is to be ascertained at a series of regularly spaced times. We define evolutionary activity (AE) in the fashion

$$AE = |\delta_t \vec{X}_{\text{c.m.}}|^2, \quad (6)$$

where δ_t stands for ‘‘variation in t temporal steps’’ (in this work we choose the number of steps to be 1000).

A species becomes extinct when it accumulates a succession of changes of a certain magnitude. More specifically, whenever the vector \vec{V}^i traverses in \mathbb{R}^N a distance larger than (a threshold) D_t .

For simplicity’s sake we choose (i) our hypermatrix in the form

$$g_{\alpha\beta}^{ij} = k_{ij} \delta_{\alpha\beta}, \quad (7)$$

and (ii) $A_\gamma^i = 0$. The latter is more than a mere way of simplifying things, however, as we intend to demonstrate that the main facets of biological evolution our model tries to mimic are of an *intrinsic dynamical origin*, so that ambient influences need not to be invoked.

Now, Eq. (1) reduces to

$$F_i = \sum_{j=1}^M k_{ij} \vec{V}^i \cdot \vec{V}^j, \quad i = 1, \dots, M \quad (8)$$

where $k_{ij} = -k_{ji}$. The k_{ij} are randomly chosen within $[-1, 1]$ (for $i < j$) and are kept constant throughout. These simplifications notwithstanding, a complex enough dynamics ensues that it can account for important details of fossil records.

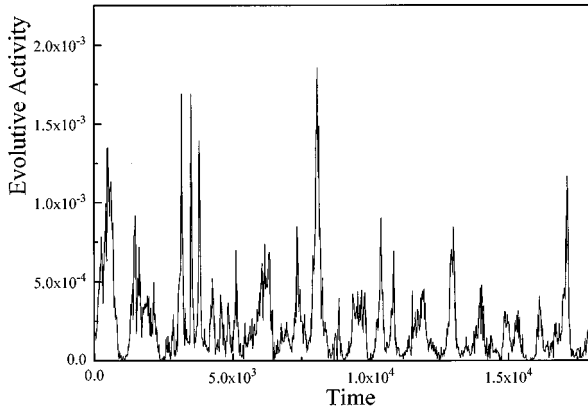


FIG. 1. Global evolution rate (arbitrary units) for $M=25$, $N=7$, and $k=5$. Each point represents the distance traversed by the center of mass of the system in an adimensional temporal unit equal to 1000 time steps.

As polynomials are a basis in any reasonable function space, the “correlation functions” discussed above can be chosen in a simple, and at the same time general, fashion as k -degree polynomials [cf. Eq. (4)]. In [17] the functional form was chosen in the (quite general) fashion

$$f_{\beta} = \sum_{n=0}^k a_n x^n, \quad x = \beta/N \quad (9)$$

so that

$$V_{\beta}^i = \sum_{n=0}^k a_n^i (\beta/N)^n. \quad (10)$$

Notice that in the limit $k \rightarrow \infty$ we have a *very* general representation for our functions. Indeed, in that limit Eq. (9) can represent *any* function [20].

III. DYNAMICS OF THE MODEL

Figure 1 depicts biological activity versus time for $M=25$, $N=7$, and [cf. Eq. (4)] $k=5$. One thousand time steps have been considered as the unit of time. The curve is qualitatively similar to Sepkosky’s extinction curve, obtained on the basis of the fossil record [5], and to that obtained in [17].

A more detailed analysis of the evolving system of Fig. 1 shows that the distributions of evolutive activity and lifetime follow a power law (see also the curves of Ref. [17]).

Power laws are typical of the self-organized criticality encountered in dealing with the celebrated (although simpler) models of Refs. [15,16]. The fact that both lifetime and number of extinct species follow there a power law is usually regarded as evidence for critical behavior. *This is not necessarily so, as will be shown in the present effort.*

An important difference between our model and the ones of [15,16] becomes apparent if we look at a graph of the number of “accepted” (good, beneficial) mutations versus time, for the series of Fig. 1. Contrary to what happens in [15,16], in *this* case the number of beneficial mutations *does not augment during the periods of frenetic biological activity*

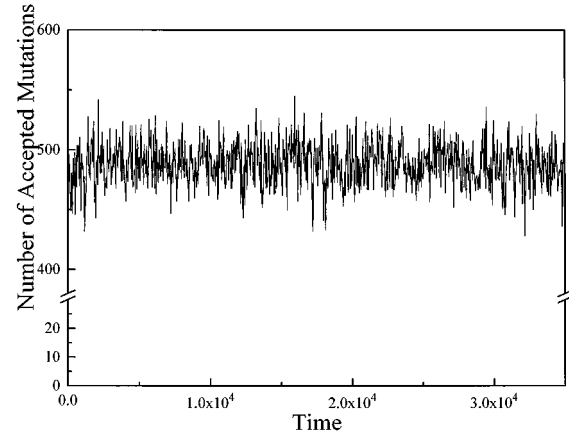


FIG. 2. Time evolution of the number of beneficial mutations for the data of Fig. 1. Each point corresponds to an adimensional temporal unit equal to 1000 time steps.

(i.e., of great morphological changes). It remains approximately the same at all times, with random fluctuations, as illustrated in Fig. 2. It is to be pointed out that, as far as we know, no other model is able to *quantitatively* exhibit such a behavior.

IV. ROLE OF THE CORRELATIONS

The parameters a_n define the species. The manner in which the different phenotypic characteristics of a given organism are correlated depends upon these parameters, which are being continuously modified at approximately the same mean rate. However, these changes do not affect the distinct species (or the same species at different times) in similar fashion. For a given set of a_n values a small variation δa_n may be translated into a correspondingly small change in the phenotypic features V_i . But it is also possible that for a different a_n set a tiny δa_n may result in an appreciable V_i change. Thus, during its evolutive history, a species enters (and leaves) diverse regions R_i of the space A of coordinates a_n , the nature of whose mapping on the V space (coordinates V_i) considerably varies with (strongly depends upon) i , from region to region.

In this model the correlation function f is the responsible for the critical phenomena it exhibits. This is easily verified. In a simulation run without f one has

$$V_j^i = a_j^i, \quad (11)$$

so that the different phenotypic features evolve in independent fashion. The corresponding results are depicted in Figs. 3–5.

Figure 3 depicts global evolutive activity vs time. Any resemblance to Sepkosky’s extinction one [5] is now lost. No sudden activity explosions ensue. For the single-species instance (Fig. 5) similar features are observed. Punctuated equilibrium has disappeared. Figure 4 displays the distribution of evolutive activities corresponding to the results shown in Fig. 3. No power law is obeyed and correlations among the diverse phenotypic features are thus seen to be an essential ingredient in order to attain criticality. The same happens with lifetime distributions. However, no important

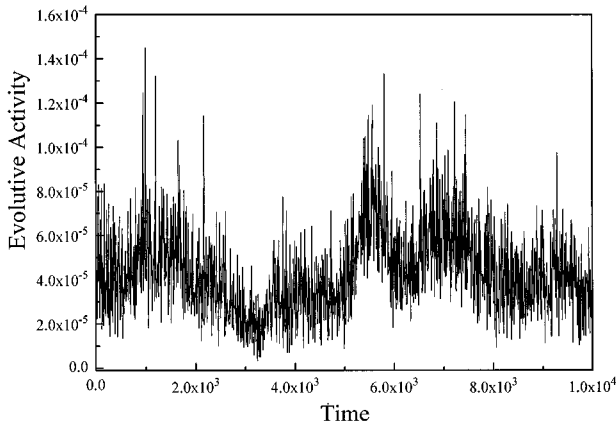


FIG. 3. Global evolutive activity (arbitrary units) for an uncorrelated system with $M=25$, $N=7$. Each point represents the distance traversed by the system's center of mass in the units of Fig. 1.

differences are observed in relation to the results depicted in Fig. 2 (number of mutations vs time).

Our model does not exhibit scaling behavior when N, M change, in contrast with the situation encountered when dealing with most dynamical systems possessing self-organized criticality. Some features of the mapping between A and V spaces, in turn, deserve special comment. The value of \vec{V} is constant along straight lines through the origin in A . Mutations (variations) $\delta\vec{a}$ that do not change vector orientation in \vec{a} leave \vec{V} invariant (see the Appendix), a fact that implies that a given $\delta\vec{a}$ will affect \vec{V} to a greater extent for small \vec{a} values than for large \vec{a} ones.

We now call attention to the results depicted in Fig. 6, which shows for an arbitrary species, $|\vec{a}|$ vs time. The concomitant behavior is the same whether (A) there are correlations (function f) or not (B) as illustrated by Eq. (11). We conclude that the entirely different dynamics associated to these two situations (to be hereafter referred to as instances A and B, respectively) cannot be attributed to the $|\vec{a}|$ variation. Of course, even in the case of Eq. (11) a residual degree of correlation remains on account of the condition $|\vec{V}|=1$, which constrains the variation of the components of \vec{V} .

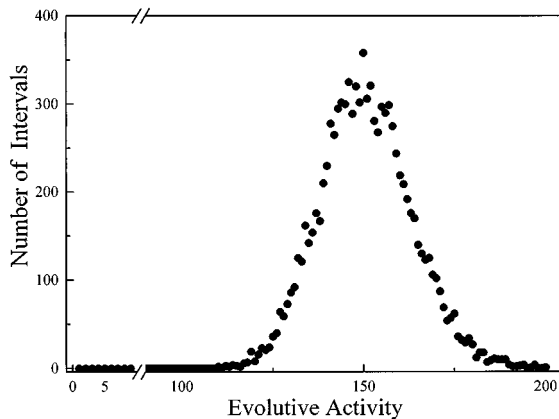


FIG. 4. Histogram of the data of Fig. 3 (arbitrary units). No power law is to be detected.

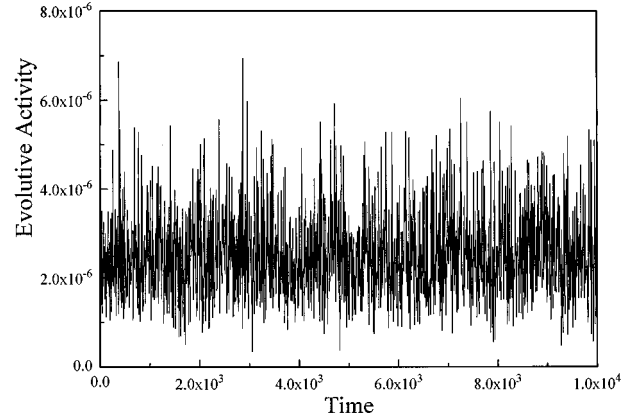


FIG. 5. Single-species evolutive activity (arbitrary units). The data are those of Fig. 3. Punctuated equilibrium has disappeared. The temporal units are those of Fig. 1.

The correlation coefficient between two quantities $x_1(n)$ and $x_2(n)$ is

$$C = \frac{\sum_{n=1}^N [x_1(n) - \bar{x}_1][x_2(n) - \bar{x}_2]}{\sqrt{\sum_{n=1}^N [x_1(n) - \bar{x}_1]^2 \sum_{n=1}^N [x_2(n) - \bar{x}_2]^2}}, \quad (12)$$

and varies in the interval $[-1, 1]$. The value 1 entails maximum degree of correlation, -1 ‘‘anticorrelation,’’ and zero, no correlation [$x_1(n)$, $x_2(n)$ vary in independent fashion].

In our case we take $N=10^5$ and $x_1(n)$, $x_2(n)$ are to be associated to variations δV_i and δV_j arising out of an arbitrary change $\delta\vec{a}$ in \vec{a} (randomly generated for each n).

Correlation coefficients in instances A and B do differ. For example, in case A the correlation coefficient for variations δV_1 and δV_2 is $C_{12}=0.9901$, and for δV_1 and δV_7 it is $C_{17}=-0.9205$. Other values are, for instance, $C_{67}=0.9266$ and $C_{14}=0.8942$. A high degree of correlation (anticorrelation) is observed. On the other hand, in instance B the corresponding figures are much lower. They do not vanish

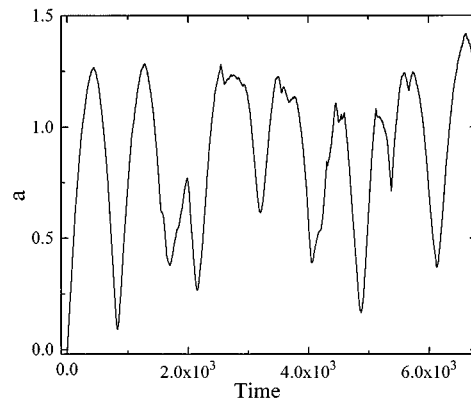


FIG. 6. Time evolution of $|\vec{a}|$ (arbitrary units) (see text). The temporal units are those of Fig. 1.

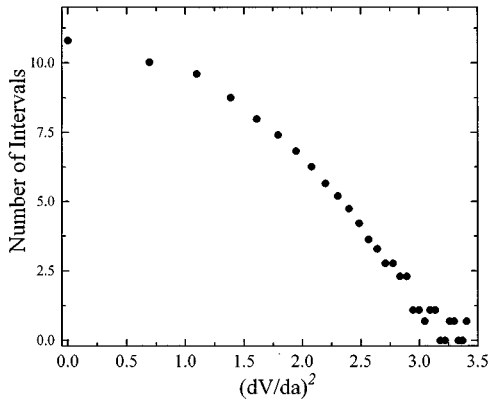


FIG. 7. Histogram showing the distribution of $(|\delta\vec{V}|/|\delta\vec{a}|)^2$ (arbitrary units) over the unit hypersphere (see text).

though, on account of the constraint $|\vec{V}|=1$. Indeed, symmetry demands that all C_{ij} be equal, the pertinent value being $C_{ij}=-0.23$. In order to understand the difference between these two dynamics (A and B) we look at V -space mappings, more specifically at the $|\delta\vec{V}|$ vs $|\delta\vec{a}|$ behavior as we move on the surface of an hypersphere \mathcal{S} of radius $|\vec{a}|=\text{const}$. In view of our preceding remarks we consider only $\delta\vec{a}$ variations tangent to \mathcal{S} .

In case (B) we have (see the Appendix)

$$\delta V_\beta = \frac{\delta A_\beta}{|\vec{A}|} - V_\beta \frac{\delta |\vec{A}|}{|\vec{A}|}, \quad (13)$$

with $A_\beta = a_\beta$. As $\delta|\vec{a}|=0$ ($\delta\vec{a}$ tangent to \mathcal{S}),

$$|\delta\vec{V}| \equiv \sqrt{\sum_{\alpha=1}^N (\delta V_\alpha)^2} = \frac{|\delta\vec{a}|}{|\vec{a}|}. \quad (14)$$

At any point on the surface of \mathcal{S} , a variation $|\delta\vec{a}|$ produces changes proportional to $|\delta\vec{V}|$. If we now introduce the correlation function [instance (A)], things really change. We cannot set $A_\beta = a_\beta$ so that the final expression for $|\delta\vec{V}|$ becomes more involved now, depending not only upon $|\vec{a}|$, but also on \vec{a} . Figure 7 is a log-log plot of $(|\delta\vec{V}|/|\delta\vec{a}|)^2$. Randomly generated \vec{a} vectors belonging to an \mathcal{S} of unity radius are subjected to small (and also arbitrary) variations $\delta\vec{a}$ tangent to \mathcal{S} . A remarkable resemblance to the graph depicting evolutive activity distribution is observed.

We conclude that the power law associated to this distribution is intimately related to the features of the $A \rightarrow V$ mapping. The ‘‘Gaussian’’ shape of the evolutive activity distribution in instance (B) simply reflects upon the nature of the $|\delta\vec{a}|$ distribution (remember that in such a case $|\delta\vec{V}|/|\delta\vec{a}|=\text{const}$).

Finally, we point out that the evolutive dynamics is ‘‘robust’’ against small changes in the correlation function f . For example, replacing Eq. (8) by

$$f_\beta = \sum_{n=0}^k \left(a_n \frac{\beta}{N} \right)^n, \quad (15)$$

no significant changes are detected.

V. DISCUSSION AND CONCLUSIONS

A. Generalities

An extremely simple model of biological, competitive co-evolution has been discussed here, that, its simplicity notwithstanding, is able to exhibit a rich, complicated dynamics.

Indeed, the dynamics is complex enough so as to mimic (even if in superficial fashion) *some* features of actual biological evolution. On the basis of this model one can conclude that *these* facets can be explained without recourse to catastrophic events. Further, no ambient influences need to be invoked.

In the model a nitid differentiation between genetic and phenotypic roles is appreciated. Evolution takes place because the ‘‘genes’’ (the a ’s) mutate, but fitness is measured with reference to phenotypic features (the V ’s). The mapping of the a ’s upon the V ’s is seen to play a leading role.

This model can be regarded as more ‘‘realistic’’ than that of [15]. Its main advantage *vis-a-vis* the model of Bak and Sneppen [15] resides in the fact that, as explained above, the interaction among species receives a much more detailed treatment: species A is here able to respond in gradual fashion to changes affecting species B and second-order effects arising out of this interaction and affecting *other* species (C, D, \dots) are also taken into account. A chain of changes thus ensues that takes place within the time scale in which B is changing. In other words, in our model ‘‘actions’’ (modifications in the features of a given species due to biological evolution) and ‘‘reactions’’ (concomitant changes in the remaining species) take place almost simultaneously, which is not the case of [15]. This difference between the models is to be attributed to the different temporal scales they encompass. In [15] the temporal scale is that for which a given species suffers drastic modifications, while ours allows for the description of very small ones.

B. Distinctive features of the present dynamical mechanism for biological evolution

(1) We must stress here that the dynamics being discussed here differs in a fundamental manner from previous ones that predict extinction curves (EC), e.g., game of Life [9], NKC Kauffman and Johnsen models [10], asymmetric spin glass models [13], or the Bak, Sneppen, and Flivbjerg model [15,16]. The EC’s in these models respond basically to an identical procedure: (i) Let the system evolve until it reaches a frozen state. (ii) After it comes to rest, perturb it by a single random mutation which does not increase the fitness of the corresponding species. This induces a coevolutionary avalanche, rather small in the beginning. When the system comes again to a stop repeat (ii). After a while, the system is pumped up to a ‘‘poised’’ state, where yet another mutation may induce an avalanche of any size.

However, in the present instance, sudden activity explosions *are not the product of perturbations of an equilibrium state*. They arise out of the intrinsic dynamics of the system. Thus, in our model, on the one hand, and in the previous ones, on the other, the origin of the ‘‘critical’’ behavior is different. Since we obtain critical behavior without recourse

to external perturbations, as the other models demand, our “criticality” can be regarded as being inherent to the system dynamics.

Moreover, *in the present model, power laws are NOT a consequence of self-organized criticality.* Other mechanisms are involved.

(2) A rather important difference between this and previous models resides in the fact that here we are able to mimic the interplay between different phenotypic features as evolution proceeds along its course. Without this interplay *power law behavior does not exist.*

This model exhibits a genetic “substructure” that one can mathematically express in closed and precise fashion. This is lacking in other models. We are able to mimic changes in just ONE gene (out of an entire genetic structure) of a given organism and afterwards ascertain what phenotypic variation this single modification induces.

(3) The physical nature of the mapping from A space to V space deserves some comment. Biological organisms are complex structures arising as the result of the dynamics of an involved genetic network. Of course, these structures are sensitive to subtle modifications in such a network (mutations). The concomitant structural changes are not, however, of an arbitrary character, due to the fact that we are speaking of highly correlated structures. Of course, some structures are more stable than others, and respond in diverse fashion to a network alteration. As their internal structure (the a ’s) is modified, some systems change behavior relatively smoothly, some relatively radically. Our model, although simple enough, does respect these biological “realities.” This is reflected in the fact that the number of beneficial mutations does not augment during periods of explosive biological activity but remains constant. Selective evolution is thus able to “tune up” in just this fashion the structure of the evolving system.

In previous models that predict self-organized criticality this is not the case. When avalanches occur, the number of “good” mutations grows.

C. Concluding remarks

Summing up, we can conclude that the model introduced in [17] generates a dynamical mechanism for punctuated equilibrium and power law behavior in biological evolution. The simplicity of the mechanism allows for its being expressed in concise, closed mathematical fashion, placing us thus in a position to predict outcomes. This mechanism is thus to be added to previously reported ones (self-organized criticality, periodic climate changes, recurrent catastrophes, etc.) and may be of some help in trying to unravel a small portion of the complexity that characterizes any biological process, and, in particular, to help to understand the mechanisms by which adaptive processes mold systems with their own inherent order.

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APPENDIX

We start with

$$V_\beta = \frac{\sum_{n=0}^k a_n(\beta/N)^n}{|\vec{A}|} = \frac{A_\beta}{|\vec{A}|}, \tag{A1}$$

where $A_\beta = \sum_{n=0}^k a_n(\beta/N)^n$ and $|\vec{A}| = \sqrt{\sum_{\alpha=1}^N A_\alpha^2}$.

Differentiation of V_β leads to a relation between variations $\delta\vec{a}$ and $\delta\vec{V}$ (we assume $\delta\vec{a}$ to be small), i.e.,

$$\delta V_\beta = \frac{\delta A_\beta |\vec{A}| - \delta |\vec{A}| A_\beta}{|\vec{A}|^2} = \frac{\delta A_\beta}{|\vec{A}|} - V_\beta \frac{\delta |\vec{A}|}{|\vec{A}|}, \tag{A2}$$

where $\delta A_\beta = \sum_{n=0}^k \delta a_n(\beta/N)^n$ (remember that $\delta |\vec{A}| = \sum_{\alpha=1}^N A_\alpha \delta A_\alpha / |\vec{A}|$).

Suppose now that variations $\delta\vec{a}$ are in the radial direction (parallel to \vec{a}). We can set

$$a_i = m_i t \tag{A3}$$

and

$$\delta a_i = m_i \delta t, \tag{A4}$$

with constant m_i . t is a parameter that adopts the same value for all a_i . Thus

$$\delta A_\beta = A_\beta \frac{\delta t}{t}, \tag{A5}$$

and

$$\frac{\delta |\vec{A}|}{|\vec{A}|} = \frac{\sum_{\alpha=1}^N A_\alpha^2 \delta t / t}{|\vec{A}|^2} = \frac{\delta t}{t} \frac{\sum_{\alpha=1}^N A_\alpha^2}{|\vec{A}|^2} = \frac{\delta t}{t}. \tag{A6}$$

Insertion into Eq. (A2) yields now

$$\delta V_\beta = \frac{\delta A_\beta}{|\vec{A}|} - V_\beta \frac{\delta t}{t} = \frac{A_\beta}{|\vec{A}|} \frac{\delta t}{t} - V_\beta \frac{\delta t}{t} = 0, \tag{A7}$$

showing that all lines through the origin (in A space) are mapped onto the surface of a hypersphere of radius unity in V space (notice that the no-correlation case is the particular instance $A_\beta = a_\beta$).

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