Macrodynamics in a model of biological evolution

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Abstract

Important macrodynamical features of the terrestrial ecosystem's evolution, like the selectivity of extinction events, are reproduced by a mathematical model recently introduced in the literature. We also show that the model is consistent with Kimura's neutral theory of molecular evolution. © 1998 Elsevier Science B.V.

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1. Introduction

In past years, a great deal of effort has been invested in devising ingenious and at the same time simple theoretical models that may provide one with meaningful insights concerning punctuated equilibrium and other features of biological evolution [1-6]. The relevant problem is, of course, that of explaining the observed patterns (fossil records) of biological evolutive activity, related to the extinction events. Gould and Eldredge [7] have conjectured that biological evolution takes place in terms of intermittent bursts of activity separating relatively long periods of quiescence, rather than in a gradual fashion. Such an intermittent pattern has been indeed observed by Raup, Sepkosky and Bayoijan [8-11] in their study of fossil records. When referring to this intermittent behavior of the single-species evolution, Gould and Eldredge speak of "punctuated equilibrium". It has been suggested that extinction events (at least the larger ones) are caused by external forces, such as changing sea levels [12] or big meteorites [13].

Recently, a somewhat more realistic model than those previously in existence was introduced [14,15]. It deals with several interacting species and its intrinsic dynamics clearly exhibit the punctuated equilibrium phenomenon. More importantly, the model predicts that the laws governing the extinction distribution curves be power laws [14] and is also able to reproduce not only Sepkosky's evolutive activity curves [11] but also the lifetime species distribution curves [8,11], both kinds of curves constructed on the basis of fossil records. As reported in Refs. [14,15], the model's workings are complex enough so as to be able to mimic some facets of actual biological evolution. On the basis of this model one may understand that several (simple) dynamical mechanisms exist that account for these (observed) facets without recourse to catastrophic events. It should be pointed out that the aforementioned predictions are of a mathematically robust nature [14,15].

For the convenience of the reader, we briefly summarize the findings of Refs. [14,15]:

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- in Ref. [14], in addition to introducing the model, it was shown that it exhibits the features of punctuated equilibrium and power-law behavior. Moreover, it is seen that critical behavior is a direct result of Darwinian competition (as simulated by the model workings). Elimination of such competition results in the disappearance of criticality.

- in Ref. [15] it was shown that, appearances notwithstanding, the model does not exhibit self-organized criticality (SOC) but a different type of critical dynamical evolution, that was mathematically grounded on phenotype-genotype correlations.

If one takes inventory of the results reported in Refs. [14,15], some intriguing questions remain that deserve further clarification. In this vein we ask ourselves the following question: to what extent do additional features of (actual) biological evolution, not yet investigated in Refs. [14,15], have a counterpart in our model?

It is the aim of the present Letter to answer some of 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 are indeed some of the 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 Letter:

- explain the mathematical reasons underlying the selectivity of extinction events, that had not previously (as far as we know) received numerical support;
- numerically test Kimura’s neutral theory of molecular evolution.

We begin the concomitant task with a brief recapitulation of the model under consideration.

### 2. The model

We deal [14,15] with $M$ distinct, interacting biological species, each of which is represented by a vector in $\mathbb{R}^N$. The components $V_i^j$ of $V$ represent different phenotypic features ($\alpha, \beta, \ldots$) 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

$$ F_i = \sum_{j} \sum_{\alpha \beta} g_{\alpha \beta} V_i^\alpha V_j^\beta + \sum_{\gamma} V_i^\gamma A_i^\gamma, \quad i = 1, \ldots, M, $$

where the hyper-matrix $g_{\alpha \beta}$ provides the details of the inter-species interaction and the second term on the r.h.s. is an “environmental” one (see below). In Ref. [14] one assumed $g_{\alpha \beta} = -g_{\beta \alpha}$.

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

$$ V_i^\rho = f_\rho (d_0^i, \ldots, d_k^i), $$

with $f_\rho$ an appropriate function. The set $d_0^i, \ldots, d_k^i$ 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 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. Summing up, changes in our $a$’s are to be

2 This can be illustrated with reference to the following example [17]. Assume that a shellfish could reduce the weight of its shell, so that it might have a better chance of escaping from some fast-moving predator. However, a lighter, thinner shell would also decrease its resistance to other predators. A compromise must be reached (conflict of design), which would give rise to one of our correlations. Called stabilizing selections, these phenotypic correlations may keep species from evolving at all: they can not go anywhere because moving in one direction has implications for its other competing functions. The two different shellfish features, speed and shell-hardness, must change together because they depend upon common genes. One may imagine changes that would be simultaneously beneficial for both features, but they will be very rare, in general.

3 Another example deserves to be cited here that shows how difficult it is to modify one feature without affecting others at the same time. Consider the period gene in the fruit-fly Drosophila.
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 does 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 \(|V'| = 1\) is enforced so as to avoid unrestricted growth (with time) of \(|V'|\).

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, old 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":

\[
X_{CM} = \sum_{i=1}^{M} V^i,
\]

(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 simplicity's sake we took in Refs. [14,15] (i) our hyper matrix in the form

\[
\delta_{ij}^{ab} = k_{ij} \delta_{aB},
\]

(4)

and (ii) \( A^f = 0 \). Thus, (1) is reduced to

\[
M.~F.~
\]

Melanogaster [18], whose mutation affects not only the fly's circadian rhythms (the timed cycles, such as for waking and sleeping, that are characteristic of all living things) but also the rhythm of the courtship song (and so the overall courtship routine and its efficacy).

![Fig. 1. Typical fitness versus time for a single species (arbitrary units). Each point corresponds to 1000 time intervals.](image)

**Fig. 1.** Typical fitness versus time for a single species (arbitrary units). Each point corresponds to 1000 time intervals.

\[
F_i = \sum_{j=1}^{M} k_{ij} V^i \cdot V^j, \quad i = 1, \ldots, M,
\]

(5)

where \( k_{ij} = -k_{ji} \). These simplifications will be removed below (see Section 4).

The \( k_{ij} \) are randomly chosen from \([-1, 1]\) (for \( i < j \)) and are kept constant throughout. The functional form of (2) was chosen in the (quite general) fashion

\[
f_{\beta} = \sum_{n=0}^{k} a_n x^n, \quad x = \beta/N
\]

(6)

so that

\[
V^i = \frac{\sum_{n=0}^{k} a_n^i (\beta/N)^n}{\sqrt{\sum_{\beta} \sum_{n,m=0}^{k} a_n^i a_m^j (\beta/N)^{n+m}}}
\]

(7)

3. Results

Let us study the time evolution of a single-species fitness. Fig. 1 is a (typical) fitness versus time graph, for a single species. Each point corresponds to 1000 time intervals. We learn from it that, even if an organism "accepts" beneficial mutations at a constant rate \( \delta \), periods of abrupt variations are nonetheless observed.

\[\text{\footnote{It was shown in Ref. [15] that in this model the number of beneficial mutations does not augment during the periods of very fast evolutive activity. It remains approximately the same at all times, with random fluctuations around the mean value.}}\]
We also notice the existence of periods of very slight fitness variation. The species accepts beneficial mutations without the benefit of a significant increase in fitness, which tantalizingly suggests that a large proportion of these mutations are of a neutral character.

Fig. 2 depicts, for an arbitrary species, the number of accumulated "accepted" mutations as a function of time. The straight line clearly is a stronger indication of the constant rate referred to above. Such a uniform behavior has been observed in proteins' molecular evolutionary rate for various lineages [16]. This may be construed as supporting Kimura's theory [16].

3.1. Kimura's theory

We are able to extract from the genetic substructure referred to above some support from a modified Kimura theory [16], as explained before. The neo-Darwinian theory of evolution through natural selection holds that most mutant genes are not selectively neutral. The mutated form of a gene is either more or less adaptive than the original form. On the other hand, Kimura's neutral theory [16] holds that most of the mutant genes (that are detected only by the chemical techniques of molecular genetics) are selectively neutral, that is, they are, from the adaptive point of view, neither more nor less advantageous than the genes they replace. At the molecular level, most evolutionary changes are caused by "random drift" of selectively equivalent mutant genes.

Two major findings with regards to molecular evolution demonstrate, in particularly clear manner, that (i) its patterns are quite different from those of phenotypic evolution, and (ii) the laws governing the two forms of evolution are different. Only one of these two items will be treated here. It is the finding, alluded to earlier, that for each protein the rate of evolution in terms of amino acid substitutions per year is approximately constant, and about the same, in various lineages, just as our model predicts. For any given species, plotting the accumulation of mutations versus time yields a straight line, as observed by Kimura [16]. However, we notice the existence of long periods during which no fitness changes are observed, which entails that mutations accepted in those periods are of an almost neutral type. As a compensation, abrupt fitness changes are also detected. At these times mutations cease to be neutral and their effects are manifested at the phenotypic level. Classical neo-Darwinian selective processes start then to play their customary role (Darwinian selection acts mainly on phenotypes).

Darwinian, or positive, selection cares little about just how these phenotypes are determined by genotypes. The laws governing molecular evolution are clearly different from those governing phenotypic evolution. Even if Darwin's principle of natural selection prevails in determining evolution at the phenotypic level, down at the level of the internal structure of the genetic material a great deal of evolutionary change is propelled by random drift.

3.2. Evolution acts at two different levels

Our model clearly exemplifies the difference between evolutive rules acting at the two distinct levels referred to above. At the phenotypic level, the main features are punctuated equilibrium and power-law behavior. As a consequence, many biologists assert that the neutral theory is not important biologically, because neutral genes are not involved in adaptation processes.

Such a position is not supported by the present model: random drift allows a species to escape, say, that subregion of A-space in which most mutations are neutral (i.e. do not significantly influence the pheno-
typic, V-space). This species reaches thus a different subregion of A-space in which mutations do produce phenotypic changes (i.e. appreciable "motion" in V-space). Only in this second type of A-space region Darwinian selection performs its important job. The nature of the mapping between A- and V-spaces becomes all important, as discussed in Ref. [15].

Our model allows one to understand just why evolutive rules at the genotypic and phenotypic levels act in diverse fashion, the mapping between A- and V-spaces becoming of fundamental importance in this respect.

3.3. Major extinction events are of a selective character

A notable feature of major extinction events is that they are selective (i.e. the victims and survivors are not random samples of the pre-extinction biota). In particular, biological groups that consistently show high background rates of speciation and extinction are most likely to be eliminated at mass extinction events [9]. In general, however, biological selectivity in extinction phenomena is little understood. This feature is hard to explain on the basis of previous models. In ours, it is a predictable outcome, as we explain below.

The key is provided by the nature of the mapping between spaces A and V investigated in Ref. [15]. As \( a_i \) traverses A it passes through regions the size of whose images in V varies enormously. Thus, some species remain in a given A-region during long periods without suffering noticeable changes. For other species, instead, small changes in \( a_i \) are translated by the mapping into large variations in \( V_j \) (a relatively small region of A-space is mapped into a large one in V-space). Large speciation ensues and, as changes grow, the species is replaced by a different one (in the model a species becomes extinct if it accumulates a sum of changes in \( V_j \) larger than a given threshold \( D \)). A quite definite sequence of the evolving species is then to be observed. Organisms that undergo a phase of rapid change, and thus face extinction (with a concomitant outburst of new species that are variants of just these) are precisely those that had already suffered previous significant changes (and from here higher speciation) than the "background" formed by the remaining species, i.e. they constitute a well-defined set.

Consider as an example the great extinction that took place near the end of the Cretaceous: amphibians and many aquatic reptiles were relatively unaffected. These not only are not highly speciated, but have remained largely unaffected until today. Our model predicts such an effect in natural fashion. Species unaffected in major extinctions are generally those that have not been suffering large changes (poor speciation). Moreover, they continue without evidence changes during long periods. On the contrary, species susceptible of detailed speciation are the ones most affected in great extinctions. The few among them that survive are the ones capable of originating the new species that will take advantage of the vacant ecological niches. The present interpretation constitutes a definite prediction of our model regarding selectivity of extinction events.

4. Conclusions

We wish to emphasize the analogy between certain dynamical mechanisms that the model mimics and other mechanisms observed by several investigators of biological evolution. Our model is very simple so that our effects can bear only a slight resemblance to the ones of Nature. To be stressed, however, is the fact that they do appear here, which should be construed as still another proof that the Galilean–Cartesian scientific approach (of studying simplified mathematical models of a given phenomenon, rather than trying to directly confront "reality" in all its complexity) also works for biological undertakings.

Summing up, a simple mathematical model is here seen to account for major features of biological evolution, which should reinforce the idea that natural phenomena, no matter how complex, are amenable to be treated à la Galileo. Of course, the analogies drawn in the present work between evolutive dynamics and simple dynamical trends are to be regarded as being of a mere illustrative character. Anyway, they may be of some help in trying to unravel a small portion of the complexity that characterizes very important kinds of dynamical processes: the biological ones.

References