Group selection models in prebiotic evolution

D. Alves, P. R. A. Campos, A. T. C. Silva, and J. F. Fontanari
Instituto de Física de São Carlos, Universidade de São Paulo, Caixa Postal 369, 13560-970 São Carlos, SP, Brazil
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The evolution of enzyme production is studied analytically using ideas of the group selection theory for the evolution of altruistic behavior. In particular, we argue that the mathematical formulation of Wilson's structured deme model [*The Evolution of Populations and Communities* (Benjamin-Cumings, Menlo Park, 1980)] is a mean-field approach in which the actual environment that a particular individual experiences is replaced by an *average* environment. That formalism is further developed so as to avoid the mean-field approximation and then applied to the problem of enzyme production in the prebiotic context, where the enzyme producer molecules play the altruists role while the molecules that benefit from the catalyst without paying its production cost play the nonaltruists role. The effects of synergism (i.e., division of labor) as well as of mutations are also considered and the results of the equilibrium analysis are summarized in phase diagrams showing the regions of the space of parameters where the altruistic, nonaltruistic, and the coexistence regimes are stable. In general, those regions are delimitated by discontinuous transition lines which end at critical points.

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I. INTRODUCTION

The controversial issue of the evolution and maintenance of altruism has probably entered the field of prebiotic evolution when Maynard Smith [1] remarked that giving catalytic support in a molecular catalytic feedback network, such as the hypercycle [2], is in fact an altruistic behavior. As a result, such systems are extremely vulnerable to the presence of parasites, i.e., molecules that receive catalytic support but do not give support to any other molecule in the network. However, the stability of this type of cooperative networks is crucial for the theories on the origin of life, as Eigen has shown that the lengths of competing self-replicating molecules are limited by their replication accuracies and so they cannot integrate sufficient information to code for a complex metabolism [3,4]. For the sake of concreteness, we define an altruistic behavior as one that is detrimental to the fitness of the individual who expresses it, but that confers an advantage on the group of which that individual is a member [5].

In the traditional group selection modeling, based on the Island models of Wright [6], it is assumed that the population is divided into reproductively isolated subpopulations or demes [5]. The stability of the altruists is achieved by postulating the existence of an external extinction mechanism acting on the demes that takes place at a rate depending on the deme composition. Of course, such extinctions will favor the occurrence of individuals that lower the probability of extinction of the deme they belong to which, in the case, are the altruistic individuals [7–9]. A more modern formulation of group selection put forward by Wilson [10] considers the demes as *trait groups*, in which the actual ecological, biochemical, or social interactions occur, but the individuals are allowed to access and compete for the total resources available in the environment. Clearly, in this formulation the notion of group or deme is somewhat blurred since, as will become clear in the examples given below, there is a stage of the life cycle of the individuals when they leave their demes to (effectively) interact with each other.

Actually, it is not so hard to envision physical systems described by Wilson's trait group or structured deme model. For instance, some basic features of viral selection dynamics can be modeled by viewing the cells as demes [11,12]. In this case, it is assumed that only N free viruses enter and hence infect a cell; however inside the cell the viruses undergo exponential growth leading to the burst of the cell and the consequent release of free viruses which will again infect (colonize) other cells, and so on. As only N viruses can infect each cell, there is an effective competition between all individuals in the population. This restriction, though very farfetched, does not seem to change qualitatively the behavior of the system [11] and, in addition, it suits very well to describing *in vitro* serial passage of viruses [12]. Another interesting application of Wilson's formalism, which will be the main concern of this paper, is the evolution of enzyme production in the prebiotic context [13,14]. Here the demes are rock crevices or suspended water droplets of some fixed size. As before, although the macromolecules inside the demes undergo exponential growth, they are regularly washed away by tides or distributed by winds, and only a small fraction of them is then re-adsorbed to the cracks or droplets. Both examples show that the spatial localization of viruses or macromolecules facilitates the selection against parasites. Henceforth we will refer to the individuals that do not display altruistic behavior as non-altruists instead of parasites, since they can subsist even in the complete absence of altruists.

The mathematical formulation of Wilson's structured deme model is centered on the concept of the average subjective frequencies of altruists, which are defined as the frequencies of altruists experienced by the *average* altruist and non-altruist in the population [10]. These quantities differ from the global frequency of altruists because the variance of the distribution of the deme compositions is nonzero, i.e., the population is not homogeneous. In particular, the stability of the altruists is achieved by assuming that the fitness of both altruists and non-altruists are proportional to their subjective frequencies. This formulation may be viewed as a sort of mean-field approach in the sense that the fitness of a given individual, say a non-altruist, in a particular deme is not

proportional to the frequency of altruists it actually experiences (i.e., the fraction of altruists in its deme) but to the frequency of altruists experienced by the average non-altruist in the population. In this paper we show that going beyond this mean-field approach does not make the theory any more complicated and, in addition, it allows the identification of a recently proposed model for the evolution of altruism [15,16], as well as of a population genetics formulation of Eigen's model of molecular evolution [17], as variants of Wilson's group selection model.

The remainder of the paper is organized as follows. In Sec. II we present the general formalism that takes into account that the fitness of an individual depends on the fraction of altruists it actually experiences in its deme. Otherwise the model conforms to Wilson's trait group model with the unlimited growth inside the demes followed by the destruction of the demes, and the random sampling of N individuals to form each new deme. The formalism is then applied to the detailed study of a model for the evolution of enzyme production proposed by Michod [13] in Sec. III. Building on the work of Donato [15,16], in Sec. IV we apply our formalism to investigate the effects of synergism or division of labor in the prebiotic problem of enzyme evolution. A variant of the quasispecies model of molecular evolution in which the replicating entities are the individual monomers that build up the molecules is considered in Sec. V. Finally, some concluding remarks are presented in Sec. VI.

II. MODEL

The population is composed of an infinite number of demes, each of which is composed of N haploid, asexually reproducing individuals. The individuals can be of two types, A or B, depending on whether they present altruistic or non-altruistic behavior, respectively. By definition, altruistic individuals increase the fitness or reproductive rate of all individuals in the deme they belong to, but pay a price for that by reducing their own fitness. Thus the key ingredient of any group selection model is that the fitness of the individuals depends on the composition of the demes, which are classified according to the number of altruists they have: there are $N+1$ different types, labeled by the integers $i=0,1,\ldots,N$. Hence, an altruistic individual living in a deme of type i has fitness $F_A(i)$ while a non-altruistic individual living in the same deme has fitness $F_B(i)$, with $F_B(i)\geqslant F_A(i)$. Clearly, either in the viral dynamics or in the enzyme production problem mentioned before, the occurrence of errors in the replication of the individuals (viruses or macromolecules) may have important implications to the equilibrium composition of the population. In order to take this possibility into account we introduce the mutation rate $u\in[0,1/2]$, which gives the probability that type A mutates to type B and *vice versa*.

To derive a recursion equation for the frequency of altruists p_t in the population at generation t it is more convenient to introduce the frequency of demes with $i=0,\ldots,N$ altruists in generation t, denoted by $Y_t(i)$. According to the discussed above, and assuming, as usual, nonoverlapping generations (i.e., all individuals in generation t are replaced by their offspring in generation $t+1$) the *average* number of altruists $\mathcal{N}_A$ and non-altruists $\mathcal{N}_B$ generated during the stage of unlimited growth inside the demes are

$$\mathcal{N}_A=\sum_{i=0}^{N}[(1-u)iF_A(i)+u(N-i)F_B(i)]Y_t(i) \tag{1}$$

and

$$\mathcal{N}_B=\sum_{i=0}^{N}[(1-u)(N-i)F_B(i)+uiF_A(i)]Y_t(i), \tag{2}$$

respectively. Hence the global frequency of altruists in the (free) population at generation $t+1$ is given by

$$p_{t+1}=\frac{\mathcal{N}_A}{\mathcal{N}_A+\mathcal{N}_B}=u+\frac{1-2u}{w_t}\sum_i iF_A(i)Y_t(i), \tag{3}$$

where

$$w_t=\sum_{i=0}^{N}[iF_A(i)+(N-i)F_B(i)]Y_t(i) \tag{4}$$

is the average fitness of the population. The next step in modeling is to distribute these individuals in infinite demes, each of which contains exactly N individuals. In the absence of additional information, the most conservative assumption that can be made about the regrouping mechanism is that the individuals are picked randomly from the (free) population. This leads to the binomial distribution

$$Y_{t+1}(i)=\binom{N}{i}(p_{t+1})^i(1-p_{t+1})^{N-i}, \tag{5}$$

which together with Eqs. (3) and (4) allow the complete description of the life cycle of the individuals.

For the sake of completeness and to facilitate comparisons between the two formalisms, at this point it is convenient that we introduce the basic ingredients of the original structured deme formalism as proposed by Wilson [10]. The conditional probability distributions of type A given type $l=A,B$ at generation t are defined by

$$\mathcal{P}_t(i|A)=\frac{iY_t(i)}{\sum_{i=0}^{N}iY_t(i)}, \tag{6}$$

$$\mathcal{P}_t(i|B)=\frac{(N-i)Y_t(i)}{\sum_{i=0}^{N}(N-i)Y_t(i)}, \tag{7}$$

which must be interpreted as follows: considering a particular replicator of type l then $\mathcal{P}_t(i|l)$ is the probability that such a replicator belongs to a deme containing i individuals of type A. Hence the average subjective frequency of altruists as seen by altruists is given by

$$f_A(t)=\frac{1}{N}\sum_{i=0}^{N} i\mathcal{P}_t(i|A)=p_t+\frac{\sigma_t^2}{N^2 p_t}, \tag{8}$$

where $\sigma_t^2=\Sigma_i i^2 Y_t(i)-[\Sigma_i i Y_t(i)]^2$ is the variance of the deme distribution and $p_t=\Sigma_i i Y_t(i)/N$ is the global frequency of altruists in the population. Similarly, the average subjective frequency of altruists as seen by non-altruists is

$$f_B(t)=\frac{1}{N}\sum_{i=0}^{N} i\mathcal{P}_t(i|B)=p_t-\frac{\sigma_t^2}{N^2(1-p_t)}. \tag{9}$$

In the case where the demes are assembled randomly, obeying a binomial distribution, one has $\sigma_t^2=Np_t(1-p_t)$ so that for large N the subjective frequencies tend to the global one. Since $f_A(t)\geqslant p_t\geqslant f_B(t)$, the main point of introducing the subjective frequencies is to show that a population structured in groups of distinct compositions can simultaneously enhance the effects of the presence of altruists on themselves and diminish those beneficial effects on the non-altruists. Of course, the assumption that the distribution of deme compositions $Y_t(i)$ affects the dynamics only through the average subjective frequencies $f_l(t)$, $l=A,B$ is too restrictive, limiting, for instance, the choices for the dependence of the fitness of the individuals on the deme composition.

III. EVOLUTION OF ENZYME PRODUCTION

According to the scenario proposed by Michod [13], we consider two types of replicators, *A* and *B*, and assume that only replicator *A* can produce a catalyst (enzyme) which, however, can catalyze the replication of both types of replicators, but with different efficiencies. Since replicator *A*, which produces the catalyst, must suffer some cost in its noncatalyzed self-replication rate, while replicator *B* attains all the benefits of the catalyst without paying the cost for its production, we have here a typical situation of altruistic behavior. The cost associated with being altruistic is modeled by assigning the noncatalyzed self-replication rate $1-r$, with $r\in[0,1]$, to *A* and the rate 1 to *B*. Moreover, the rate of catalyzed replication is proportional to the concentration of enzymes in the deme, which in turn is proportional to the concentration of replicators *A* in that deme. Hence, assuming that self-replication and the replication catalyzed by the enzyme are separate processes, the fitness of a replicator $l=A,B$ belonging to a deme of type i can be written as

$$F_l(i)=1-\alpha_l r+k_l\frac{i}{N}, \quad i=\alpha_l,\alpha_l+1,\ldots,N-1+\alpha_l, \tag{10}$$

where $\alpha_l=1$ if $l=A$ and 0 if $l=B$. Here the parameters k_l represent the beneficial effect of enzyme mediated replication. In particular, $k_B=0$ implies that the enzyme is specific for the replicator which produced it, as in the one-membered hypercycle [2]. However, it seems more plausible to assume that the primordial enzymes were some kind of general catalysts which would facilitate the replication of a wide spectrum of replicators, so in the following we will assume that $k_A\geqslant k_B$. We note that Michod considers the case $k_A=k_B$ and $u=0$ only [13]. The recursion equation (3) thus becomes

$$p_{t+1}=u+(1-2u)\times\frac{p_t(1-r)+k_A p_t^2+\frac{1}{N}k_A p_t(1-p_t)}{1+p_t(k_B-r)+(k_A-k_B)\left[\frac{1}{N}p_t(1-p_t)+p_t^2\right]}, \tag{11}$$

which is identical to that obtained using Wilson's original (mean-field) formulation [13]. In fact, we note that the coefficient of k_A in the numerator of Eq. (11) can be written as $p_t f_A(t)$, so that the rate of increase of altruists in the population due to the replication catalyzed by the enzymes is proportional to the average subjective frequencies of altruists.

It is instructive to consider first the case where mutations are not allowed ($u=0$) since the steady-state equation obtained by setting $p_{t+1}=p_t=p^*$ can be solved analytically in this case. Explicitly, we find three fixed points: $p^*=0$, $p^*=1$, and

$$p^*=\frac{r-k_A/N}{(k_A-k_B)(1-1/N)}. \tag{12}$$

A physically meaningful fixed point must be in the simplex $[0,1]$ and satisfy the standard stability condition

$$\left.\frac{dp_{t+1}}{dp_t}\right|_{p_t=p^*}<1. \tag{13}$$

We find that $p^*=0$ is stable for $k_A/r<N$, while $p^*=1$ is stable for $k_A/r>1+(1-1/N)k_B/r$. Interestingly, for $k_B/r>N$ there is a region where both fixed points are unstable and so the stable one is the intermediate fixed point (12) which corresponds to a regime of coexistence between altruists and non-altruists. These distinct regimes are illustrated in Fig. 1 where we show the steady-state frequencies p^* for two different values of the initial frequency of altruists. We note that in the case $u=0$ the analysis is considerably simplified as only the ratios k_l/r, $l=A,B$ matter for the stability of the fixed points. We identify four different phases in the steady-state regime: the pure altruistic phase (*A*) associated to the fixed point $p^*=1$; the pure non-altruistic phase (*B*) associated to the fixed point $p^*=0$; the coexistence phase (*C*) associated to the fixed point (12); and the phase labeled $(A)-(B)$ where both $p^*=1$ and $p^*=0$ are stable. In this phase the two kinds of replicators compete such that there is an all-or-none selection, though the winner is not determined by the fitness only, but also by its initial abundance in the population. In fact, the basins of attraction of the two stable fixed points are delimited by the intermediate fixed point (12). These results are conveniently summarized in a phase diagram in the plane $(k_A/r,k_B/r)$ as shown in Fig. 2(a). We note that the transitions between phases (*B*) and (*C*) as well as between phases (*C*) and (*A*) are continu-

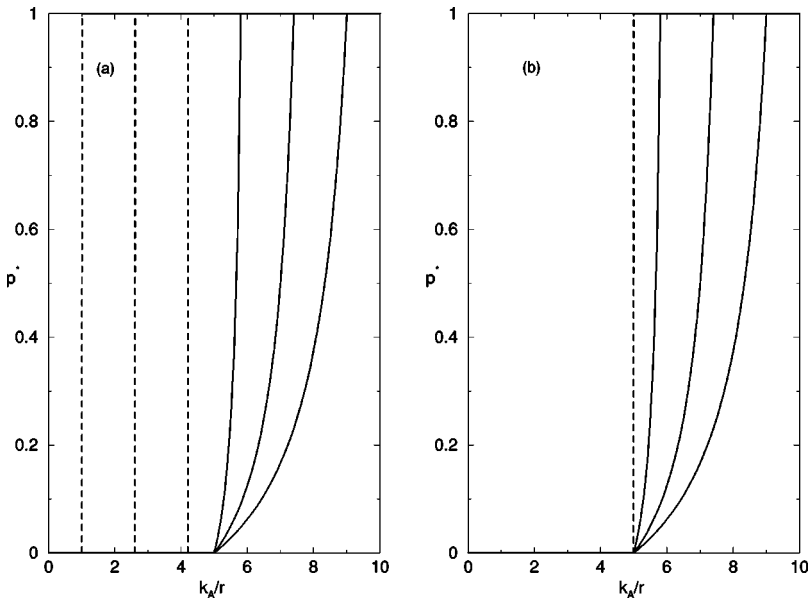

FIG. 1. Steady-state frequency of type A replicators for $u=0$, $N=5$, and (from left to right) $k_B/r=0$, 2, 4, 6, 8, and 10. The initial frequencies are (a) $p_0=0.999$ and (b) $p_0=0.001$. The first three lines in part (a) collapse into a single line in part (b).

ous, in the sense that p^* increases continuously as those transition lines are crossed. It is important to note that even in the case of completely nonspecific catalysis $k_A=k_B$ the altruistic replicators can dominate the entire population provided that the condition $k_A>rN$ is satisfied.

We turn now to the more general case where the mutation rate u is nonzero. The obvious complication in this case is that $p=0$ and $p=1$ are no longer fixed points and so, in principle, the phases identified before cannot be unambiguously defined. However, the threshold phenomena observed in the dependence of the steady-state frequency of type A replicators on the scaled catalyst specificity k_A/r (see Fig. 3) indicates that a unique extension of the definitions of phases (A),(B) and $(A)-(B)$ is possible indeed, provided that k_B/r is not larger than some critical value. As expected, phase (C) disappears since its defining characteristic, namely, $0<p^*<1$, occurs for all parameter settings in the case of nonzero mutation rates. The rich interplay between the stable fixed points is illustrated in the phase diagrams of Fig. 2. The prominent feature of those phase diagrams is the existence of *critical points* at which the two discontinuous transition lines intersect and, as a result, above which it is no longer possible to distinguish between phases (A) and (B). For fixed u, r, and N the critical point coordinates (k_A^c,k_B^c) are determined by requiring that the three fixed points of the recursion equation (11) collapse into a single one. Accordingly, in Fig. 4 we show the critical point coordinates as function of the mutation rate u. As expected, for $u=0$ we find $k_B^c/r=N$ regardless of the value of r. Of particular interest is the mutation rate at which k_B^c vanishes, henceforth denoted by u_e, as it signalizes the disappearance of all traces of the two distinct regimes associated to altruistic and nonaltruistic behaviors, leading to the phase diagram of Fig. 2(d). Interestingly, at this value of the mutation rate we find

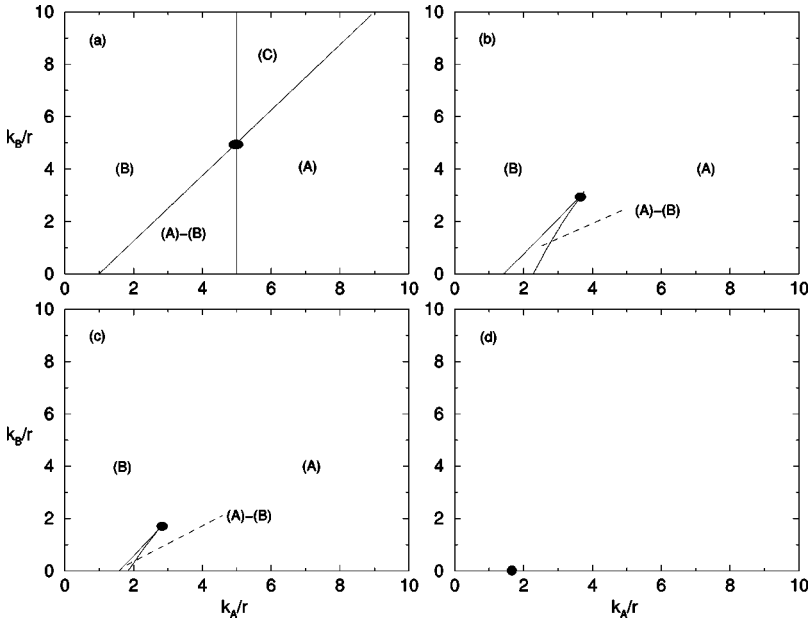

FIG. 2. Phase diagrams for $N=5$ and $r=0.1$ showing the regions of stability of the different fixed points for (a) $u=0$, (b) $u=0.005$, (c) $u=0.01$, and (d) $u=0.0158$. The intersection point touches the coordinate axis at $k_A/r=5/3$.

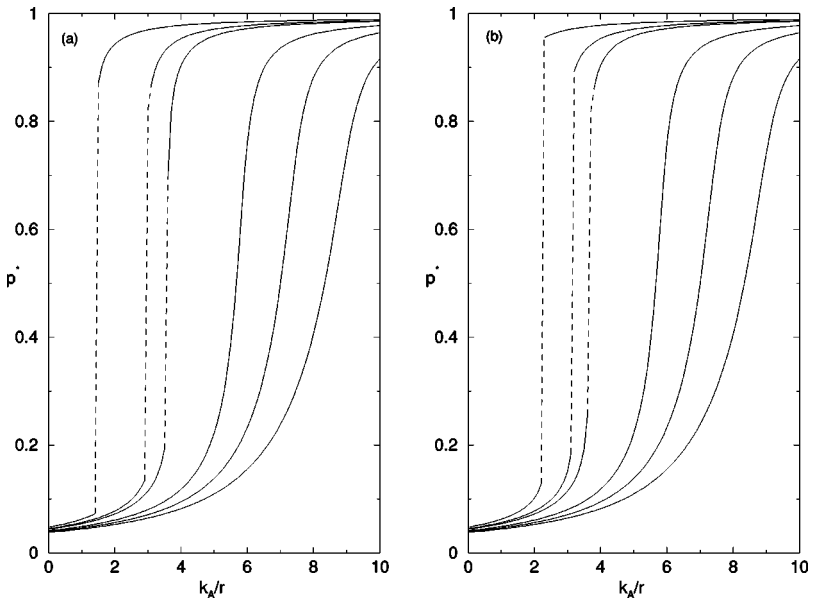

FIG. 3. Steady-state frequency of type A replicators in the population for $u=0.005$, $N=5$, $r=0.1$, and (from left to right) $k_B/r=0$, 2, 2.9, 6, 8, and 10. The initial frequencies are (a) $p_0=1$ and (b) $p_0=0$.

$k_A^c/r=2N/(N+1)$ independently of r. The dependence of u_e on the altruistic cost r for several values of the deme sizes is illustrated in Fig. 5.

The importance of the finitude of the deme sizes N to the stabilization of the altruists can be appreciated by considering the limit $N\to\infty$, which corresponds to a homogeneous population, in the case of absence of mutations $u=0$. In fact, in this case the fixed point $p^*=0$ is always stable, while $p^*=1$ becomes stable only for $k_A>r+k_B$, which is a very uninteresting situation from the point view of the evolution of altruism since in this case the effective fitness of an altruist $(1-r+k_A)$ is larger than the fitness of a non-altruist $(1+k_B)$ belonging to the same deme.

IV. SYNERGISM

A puzzling problem in evolution is the existence of complex structures that are of value to the organism only when fully formed [18]. It might be possible that enzyme production has become a reality due to the combined work of several molecules, each being responsible for the synthesis of different pieces of the catalyst. This situation of division of labor between the altruists, termed synergism, can result in highly nonadditive fitness interactions. To model this case, we assume that the advantage to the deme is accrued only if the number of altruists reaches some minimal value. Explicitly, we will assume that only individuals belonging to demes composed of $i\geqslant i_m$, with $i_m=0,1,\ldots,N$, altruists have their fitness enhanced: for such demes all individuals have their fitness increased by the factor $1/(1-c)$ with $c\in[0,1]$. The dependence of the fitness of types A and B on the composition of the deme is summarized by the following equation:

$$F_l(i)=\begin{cases}1-\alpha_l r, & \text{if } i<i_m,\\ (1-\alpha_l r)/(1-c) & \text{otherwise,}\end{cases} \tag{14}$$

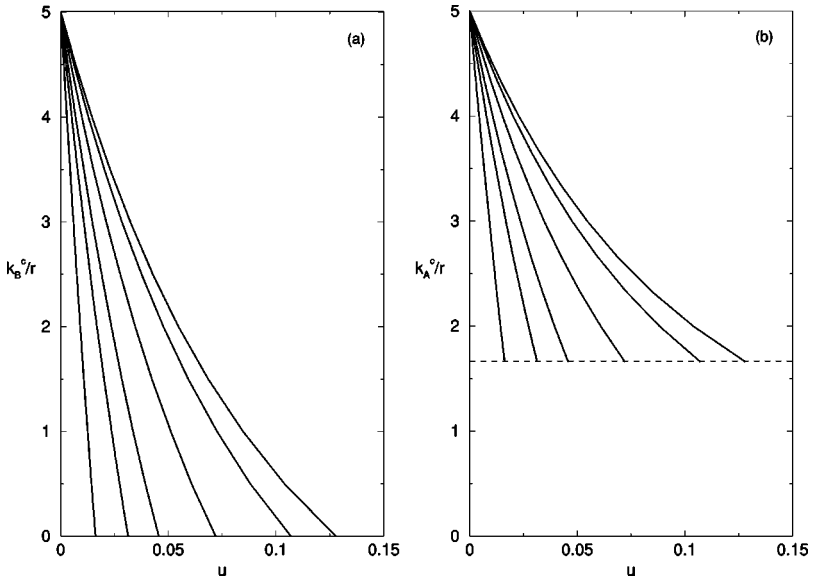

FIG. 4. Coordinates of the critical point (a) k_B^c/r and (b) k_A^c/r as functions of the mutation rate u for $N=5$ and (from left to right) $r=0.1$, 0.2, 0.3, 0.5, 0.8, and 1.0. At the points where $k_B^c/r=0$ we find $k_A^c/r=5/3$.

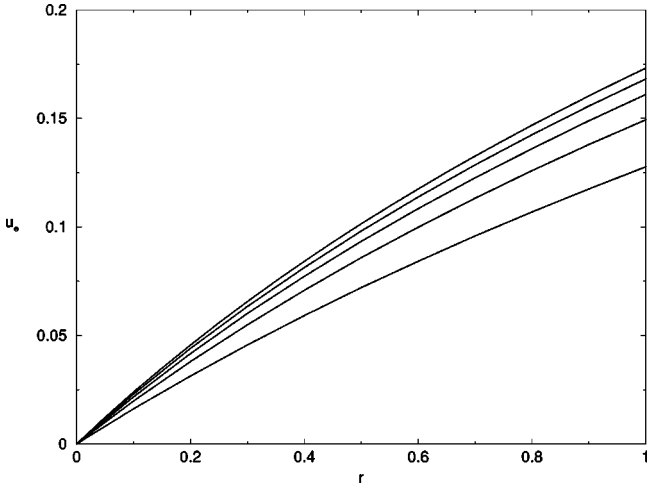

FIG. 5. Mutation rate u_e beyond which the discontinuous transitions disappear as function of the altruistic cost r for (from bottom to top) $N=5$, 10, 20, 50, and ∞.

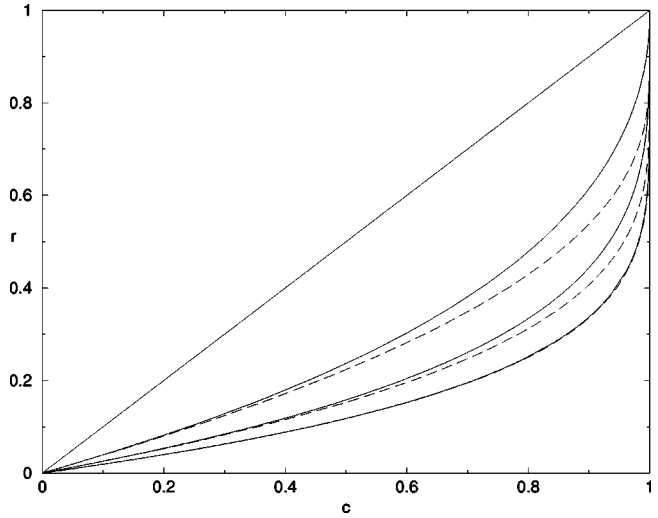

FIG. 6. Transition lines for $N=20$, $u=0$ and, from top to bottom, $i_m=1$, 2, 4, 10 (solid lines), and $i_m=19$, 17 (dashed lines). The curves for $i_m=20$ and $i_m=11$ coincide with those for $i_m=1$ and $i_m=10$, respectively.

where, as before, $r\in(0,1)$ is the cost for being altruistic and $\alpha_l=1$ if $l=A$ and 0 if $l=B$. The recursion equation (3) is then written as

$$p_{t+1}=u+(1-2u)\times\frac{(1-r)\left[p_t(1-c)+\frac{1}{N}c\sum_{i=i_m}^{N}iY_t(i)\right]}{(1-c)(1-rp_t)+c\sum_{i=i_m}^{N}Y_t(i)\left(1-\frac{1}{N}ri\right)}. \tag{15}$$

The formalism based on the average subjective frequencies cannot be applied to describe this dynamics because of the highly nonlinear dependence of the fitness F_l on the number of altruists in the deme.

Before we proceed with the analysis of the steady-state solutions of recursion equation (15), we must note that the fitness assignment summarized in Eq. (14) was used by Donato [15] in an alternative model for the selection of altruistic behavior, which, similarly to Wilson's structured deme model, though not explicitly acknowledged by that author, has a stage of the life cycle of the individuals when they interact with all other individuals in the population. In fact, this must be so because in Donato's model the relative fitness of an individual, which is related to the number of offspring it generates, is defined as the ratio between the fitness of that individual and the fitness of the *whole* population [15,16]. However, that model has two other ingredients that differ from Wilson's: (i) the sizes of the demes are not fixed *a priori*, but there is a maximal deme size that once reached leads the deme to split in two smaller ones; and (ii) the offspring of the individuals of one deme in one generation form one deme in the next generation. These rules were motivated by the analogy with social animals which live in groups not too large and whose offspring remain in the group of their parents. An interesting outcome of the model is the possibility of stable coexistence between altruists and nonaltruists within a same group, which is in fact the situation observed in nature since the altruistic behavior is usually exhibited only by some individuals in the group. This result contrasts with that predicted by the Island group selection models, namely, that in the absence of mutations there are either fully altruistic ($i=N$) or fully non-altruistic ($i=0$) groups only [7–9]. As we will show in the sequel, using the fitness assignment of Eq. (14) this coexistence regime is associated to one of the stable steady-state solutions of the recursion equation (15).

As before, we will consider first the simpler case where $u=0$. As expected, $p=1$ and $p=0$ are always fixed points and, depending on the values of the control parameters i_m, c, and r, there can be either one or two additional fixed points. On the one hand, $p^*=0$ is always stable for $i_m>1$, while for $i_m=1$ it becomes unstable in the region $c>r$. In fact, for fixed r a stable fixed point appears at $c=r$, increasing continuously from 0 as c increases. This behavior signalizes the occurrence of a continuous transition from a regime characterized by fully non-altruistic demes only ($p^*=0$) to a regime where inhomogeneous demes formed of both altruistic and non-altruistic individuals are allowed also ($0<p^*<1$). On the other hand, $p^*=1$ is always unstable for $i_m<N$, while for $i_m=N$ it becomes stable in the region $c>r$. In this case both fixed points $p^*=1$ and $p^*=0$ are stable but, as pointed out before, only one of the two types of individuals will take over the population. In the other cases $1<i_m<N$ the intermediate, stable fixed point $0<p^*<1$ appears in a discontinuous manner, i.e., p^* is nonzero already at the outset.

In Fig. 6 we present the transition lines separating the region in the plane (c,r) where the altruistic individuals persist in the population (region below the curves) from the region where the only stable fixed point is the non-altruistic one $p^*=0$. Since those curves satisfy $c\geqslant r$, it seems that the surviving altruists are those belonging to demes with $i\geqslant i_m$

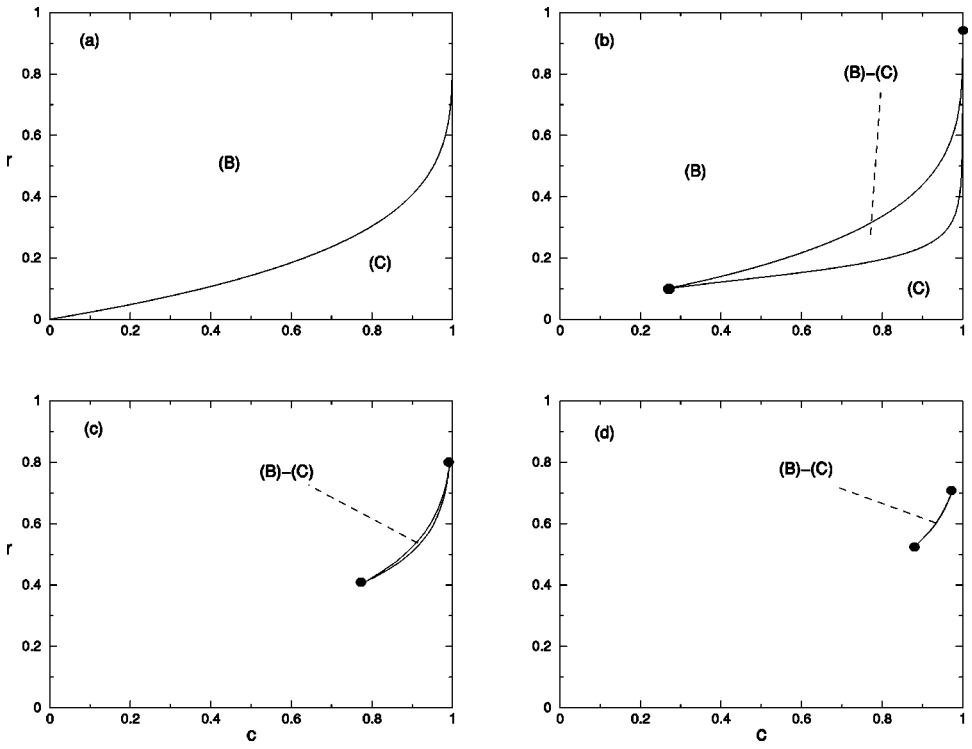

FIG. 7. Phase diagrams for $N=20$ and $i_m=5$ showing the regions of stability of the different fixed points for (a) $u=0$, (b) $u=0.0085$, (c) $u=0.0282$, and (d) $u=0.0330$.

since they have a larger fitness than non-altruists living in demes with $i<i_m$. Interestingly, the size of the region of existence of altruists decreases with increasing i_m, reaches a minimal value for $i_m=N/2$, and then increases towards its initial size as i_m approaches N (the transition lines for $i_m=1$ and $i_m=N$ coincide). However, it must be noted that the most favorable situation to the altruists is the case of no synergism $i_m=1$, since only then the fixed point $p^*=0$, associated to the non-altruistic regime, becomes unstable. Moreover, the basin of attraction of the intermediate fixed point decreases with increasing i_m and so, unless there is already a large number of altruists at the outset, the non-altruists will take over the population. For instance, for $i_m=N$ the basin of attraction of $p^*=1$ is vanishingly small close to the transition line $c=r$. This rather frustrating result simply reflects the difficulty, already pointed out in the beginning of the section, of evolving a synergistic system in nature. A possible solution to this problem is provided by the so-called Baldwin effect [18] which, in the framework proposed by Hinton and Nowlan [19,20], assumes the existence of a third type of individual, say X, which either by learning, guessing, or imitation can act as an individual of type A or B but whose offspring are, of course, of type X. These plastic individuals may provide the appropriate conditions (i.e., a large number of altruistically behaving individuals) to start the synergistic effects and, once this is done, they will become extinct due to the competition with the born altruists, leaving thus no trace of their early presence in the population. We will leave the investigation of this avenue of research for a future contribution.

Taking into account the effect of mutation ($u>0$) leads to a very rich interplay between the different steady-state regimes of the recursion equation (15) as illustrated by the phase diagrams shown in Fig. 7. In the absence of mutation the phase labeled (B) is associated to the non-altruistic regime characterized by the fixed point $p^*=0$, while phase (C) is associated to the coexistence regime characterized by the intermediate fixed point $0<p^*<1$. As before, although for nonzero mutation rates $p=0$ is no longer a fixed point it is still possible to distinguish between the fixed points corresponding to the non-altruistic and the coexistence regimes, due to the occurrence of threshold phenomena similar to those shown in Fig. 3. The main effect of mutation is to produce, at the expense of phase (C), a bounded region, labeled $(B)-(C)$, where both phases are stable. This region is delimited by two discontinuous transition lines that intersect and end at two critical points. As the mutation rate u increases the size of the bounded region decreases and disappears altogether at the critical end point u_e at which the two critical points coalesce. Hence for $u\geqslant u_e$ it is no longer possible to distinguish between phases (B) and (C). The dependence of u_e on i_m is shown in Fig. 8. As expected $u_e=0$ for $i_m=1$, regardless of the value of the deme size N, since the transition between those two phases is continuous already for $u=0$.

V. QUASISPECIES MODEL

Another interesting application of the formalism presented in Sec. II is the study of the error threshold transition in Eigen's molecular quasispecies model [3]. Such an approach has recently been proposed as an (uncontrolled) approximation to the original kinetics formulation of the quasispecies model [17], without the realization of its close connection with Wilson's trait group framework. In this case, the monomers play the role of the individuals, and the molecules the role of the demes. However, there is no distinction between altruistic and non-altruistic monomers (i.e.,

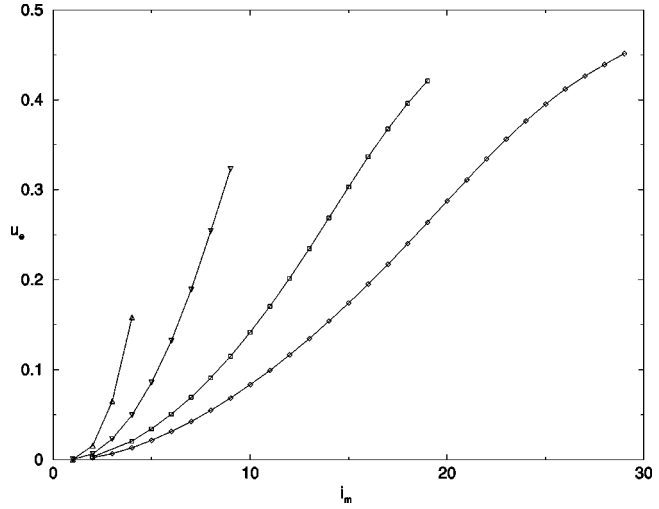

FIG. 8. Mutation rate u_e beyond which the discontinuous transitions disappear as function of i_m for $N=5$ ($\triangle$), $N=10$ ($\triangledown$), $N=20$ ($\square$), and $N=30$ ($\diamond$). The lines are guides to the eye.

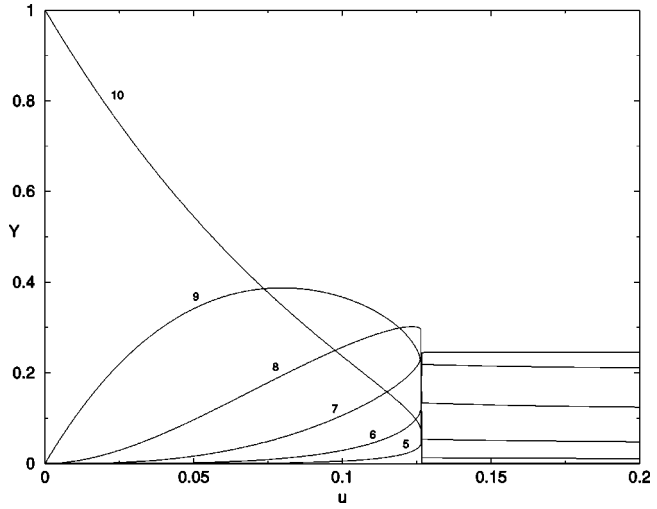

FIG. 9. Steady-state frequencies of molecules composed of 10 (master), 9, 8, 7, 6, and 5 monomers of type A as functions of the error rate for $N=10$ and $s=0.9$. The initial type A monomer frequency is $p_0=1$.

there is no altruistic cost) but the self-replication rates of the monomers depend on the molecule they belong to. Thus, contrasting to Eigen's original proposal, in this formulation the molecules are not self-replicating entities, being only passive carriers of monomers. In this context, it is more appropriate to think of the mutation rate u as the replication error rate per monomer. Explicitly, for the single-sharp-peak replication scenario we have $F_B(i)=1-s$, $i=0,\ldots,N-1$ and

$$F_A(i)=\begin{cases}1-s, & \text{if } i=1,\ldots,N-1,\\ 1, & \text{if } i=N,\end{cases} \tag{16}$$

where $0\leqslant s\leqslant 1$ is the selective advantage of the so-called master molecule, namely, the molecule composed of N monomers of type A [4]. The general recursion equation (3) then becomes

$$p_{t+1}=u+(1-2u)\frac{(1-s)p_t+sp_t^N}{1-s+sp_t^N}. \tag{17}$$

The only stable fixed point for $u=0$ is $p^*=1$ which corresponds to the domination of the population by the master molecules. As the mutation rate increases, two distinct regimes are observed in the composition of the population: the *quasispecies* phase (Q) characterized by the master molecule and its close (in the sense of the Hamming distance) neighbors, and the *uniform* phase (U) where the 2^N molecules appear in the same proportion. More pointedly, phase (Q) is associated to the fixed point $p^*\approx 1$ and phase (U) to $p^*\approx 1/2$. In Fig. 9 we illustrate the dependence of the steady-state molecule frequencies $Y(i)$, given in Eq. (5), on the error rate u. Although these results show a remarkable similarity to those obtained with the original kinetics formulation of the quasispecies model [4], the agreement is qualitative only: a full analysis of the location of the error-threshold transition for the original model indicates that the predictions based on the recursion equation (17) are very inaccurate [21].

In the present study the error-threshold transition corresponds to the discontinuous transition between the phases $(Q)-(U)$ and (U) (see Fig. 10). As in the previous models, the discontinuous transition lines intersect and end at a critical point (u^c,s^c) given by [17]

$$u^c=1-\frac{1}{2}\left(\frac{N+1}{N-1}\right)^2 \tag{18}$$

and

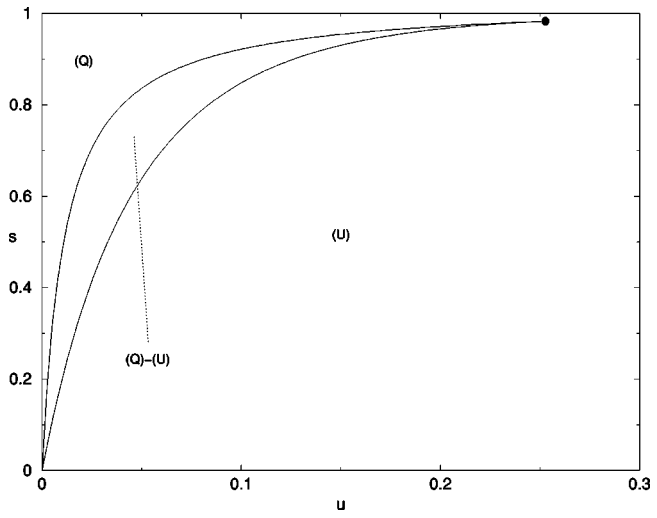

FIG. 10. Phase diagram for $N=10$ showing the regions of stability of the quasispecies (Q) and uniform (U) regimes. The discontinuous transitions end at the critical point $u^c=0.251$ and $s^c=0.983$.

$$\frac{1}{1-s^c}=1+2^N\left(\frac{N-1}{N+1}\right)^N\frac{(N-1)^2-4N}{N^2-1}. \qquad (19)$$

It must be noted, however, that the rich interplay between phases (Q) and (U) depicted in the phase diagram of Fig. 10 is a consequence of Wilson's trait group framework, in which the molecules are disassembled and then randomly assembled during the life cycle of the population. Clearly, the use of that framework is inadequate in the context of the quasispecies model, in which the molecules and not the monomers are the self-replicating entities.

VI. CONCLUSION

In this paper we have basically attempted to re-interpret and unify several models dealing with the evolution of altruistic behavior [13,15,16] in a single framework, namely, the ''extended'' Wilson's structured deme model of group selection [10]. In doing so, we have carried out a thorough analysis of the steady-state regime of a model for the evolution of enzyme production proposed originally by Michod [13], without resorting to the mean-field approximation implicit in Wilson's concept of average subjective frequencies [10]. Furthermore, the effect of synergism (i.e., division of labor) was considered by assuming that the presence of altruists accrues benefits only to groups containing some minimal number of that type of individual, following thus Donato's alternative group selection model [15,16]. In particular, we have obtained the phase diagrams showing the regions of stability of the altruistic and non-altruistic regimes. A particularly relevant result is the finding of a regime of stable coexistence within a same group of altruists and non-altruists which, though expected from observation, is not predicted by the Island group selection models [7–9]. We have also identified a recently proposed variant of the quasispecies model, in which the macromolecules are viewed as vehicles for the self-replicating monomers [17], as a particular realization of the ''extended'' Wilson's structured deme formalism presented in this paper. In addition, we have found that taking into account the possibility of mutations leads to interesting qualitative changes on the steady-state regime of the model dynamics as, for instance, the appearance of critical points in the phase diagrams of the models. In particular, we have shown that there is a value of the mutation rate u_e (see Figs. 5 and 8) above which the selective pressures are no longer operative, in the sense that it is no longer possible to distinguish between the altruistic and the non-altruistic regimes.

To conclude, we note that in the prebiotic context error-prone replication (mutation) has played a crucial role in revealing the limitations of noncooperative molecular systems, such as Eigen's quasispecies model, to function as efficient information integrators [3,4]. Furthermore, it was shown recently that mutation can have disastrous effects over the stability of altruistic demes in the more traditional Island formulation of group selection theory [9]. In view of this, mutation should not be viewed as merely another complication to be added to a model, but as a basic test for probing the robustness of any model of integration of information in prebiology, this being thus the main motivation for the present contribution.

ACKNOWLEDGMENTS

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[1] J. Maynard Smith, Nature (London) **280**, 445 (1979).
[2] M. Eigen and P. Schuster, Naturwissenschaften **65**, 7 (1978).
[3] M. Eigen, Naturwissenschaften **58**, 465 (1971).
[4] M. Eigen, J. McCaskill, and P. Schuster, Adv. Chem. Phys. **75**, 149 (1989).
[5] S. A. Boorman and P. R. Levitt, *The Genetics of Altruism* (Academic, New York, 1980).
[6] S. Wright, Genetics **16**, 97 (1931); **28**, 114 (1943).
[7] I. Eshel, Theor Popul. Biol. **3**, 258 (1972).
[8] K. Aoki, Evolution (Lawrence, Kans.) **36**, 832 (1982).
[9] A. T. C. Silva and J. F. Fontanari, Eur. Phys. J. B **7**, 385 (1999).
[10] D. S. Wilson, *The Evolution of Populations and Communities* (Benjamin-Cumings, Menlo Park, 1980).
[11] E. Szathmáry, J. Theor. Biol. **157**, 383 (1992).
[12] E. Szathmáry, J. Theor. Biol. **165**, 341 (1993).
[13] M. R. Michod, Am. Zool. **23**, 15 (1983).
[14] E. Szathmáry and J. Maynard Smith, J. Theor. Biol. **187**, 555 (1997).
[15] R. Donato, J. Phys. I **6**, 445 (1996).
[16] R. Donato, L. Peliti, and M. Serva, Theory Biosci. **116**, 309 (1997).
[17] D. Alves and J. F. Fontanari, Phys. Rev. E **54**, 4048 (1996).
[18] J. Maynard Smith, Nature (London) **329**, 761 (1987).
[19] G. E. Hinton and S. J. Nowlan, Complex Syst. **1**, 495 (1987).
[20] J. F. Fontanari and R. Meir, Complex Syst. **4**, 401 (1990).
[21] D. Alves and J. F. Fontanari, Phys. Rev. E **57**, 7008 (1998).