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Deterministic group selection model for the evolution of altruism

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Abstract. We study the evolution of an infinite population of asexually reproducing individuals, each of which can be either altruist or non-altruist, subdivided into reproductively isolated groups (demes) of finite size under the action of two opposed selective pressures, namely, differential individual reproduction and differential deme extinction. We derive a recursion equation for the deterministic, discrete time evolution of the frequencies of the different types of demes, classified according to the number of altruistic individuals they have. We give emphasis to the detrimental effects of mutation and migration on the stability of the altruistic demes, which are the only stable demes in the absence of these processes. Furthermore, we draw an analogy between the proposed deterministic group selection model and the quasispecies model for molecular evolution.

PACS. 87.10.+e General, theoretical, and mathematical biophysics (including logic of biosystems, quantum biology, and relevant aspects of thermodynamics, information theory, cybernetics, and bionics) – 87.90.+y Other topics in biophysics and medical physics – 89.90.+n Other areas of general interest to physicists

1 Introduction

Among the three major alternative theories for the evolution and maintenance of altruistic behavior in nature, namely, *kin* selection [1], *reciprocity* selection [2,3] and *group* selection [4,5], the latter is doubtless the most controversial and also the most elusive to mathematical analysis (see [6] for a thorough discussion of the genetics of altruism). Group selection is based on an analogy between individuals (or genes) and reproductively isolated subpopulations, termed demes. If the extinction of demes, similarly to the death of individuals, takes place at a rate depending on their genetical composition, then such extinctions may favor the occurrence of a gene that lowers the probability of extinction of the deme it belongs to. Since, on the other hand, this gene may be detrimental to the individual carrying it and thus its occurrence disfavored by selection at the individual level, group selection has been invoked as a possible explanation for the existence of altruistic traits in nature. Such a trait is defined 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. Though this sort of argument had been advanced in the early sixties [7], a mathematical framework to investigate the mechanisms needed by group selection was proposed much later by Levins [5,6]. The extreme complexity of the resulting mathematical theory, based on a nonlinear integral partial differential equation as well as the need for too restrictive assumptions, have motivated the proposal and the numerical study of a variety of discrete time versions of Levins' model [8,9]. However, the results from these studies have largely been equivocal, due mainly to the limited computational resources available at that time.

Although group selection is often studied as a mechanism for the evolution of altruism, it has a great interest on its own. In particular, the subdivision of a population into demes which can exchange individuals through migration yields an important class of problems in classical population genetics, the so-called Island models [4]. More recently, this arrangement has been successfully used to improve the performance of genetic algorithms in the search for quasi-optimal solutions of optimization problems [10,11]. Moreover, since modern theories of integration of information in prebiotic systems involve the compartmentation of a small number of self-replicating molecules in a large number of almost isolated compartments (primeval cells), the mathematical framework developed in the study of group selection can be readily applied to the prebiotic evolution problem [12,13].

Building on the work of Aoki [9], we consider in this paper the evolution of a population of haploid, asexually reproducing individuals divided into a countable infinity of demes of equal size N. Henceforth we will refer to such a population as a metapopulation. An individual can be either altruist or non-altruist. The cost associated with being altruistic is modelled by assigning the reproductive rate $1-\tau$, with $\tau \in [0,1]$, to the altruists and the reproductive rate 1 to the non-altruists. Furthermore, it is

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assumed that the probability of a given deme surviving extinction is a nondecreasing function of the number of altruists belonging to that deme [5]. Also, according to Levins' original model, as soon as a deme is extinct, immediate recolonization takes place with the substitution of the extinct deme by a replica of one of the surviving demes chosen at random [5,6]. Within this framework, a truly remarkable result was obtained by Eshel [14], namely, that for sufficiently small migration rate the gene for altruism is fixed in the metapopulation.

The aim of this paper is to investigate the effect of mutation on the stability of the altruistic equilibrium state predicted by Eshel [14], as well as to complement the numerical analysis of Aoki [9]. The remainder of the paper is organized as follows. In Section 2 we present the model and describe in detail the diverse events that comprise the life cycle of the individuals and demes. The results obtained by solving numerically the recursion equations for the frequencies of the different types of demes that compose the metapopulation are analyzed and discussed in Section 3. Also in that section, we comment on the similarities between the deterministic group selection formulation and Eigen's quasispecies model for molecular evolution [15]. Finally, some concluding remarks are presented in Section 4. In particular, we compare our standard group selection model with a recently proposed alternative model for the evolution of altruistic traits [16,17].

2 Model

The metapopulation is composed of an infinite number of demes, each of which is composed of N haploid, asexually reproducing individuals. The alleles A or B at a single locus determine whether a given individual is altruist or non-altruist, respectively. The fitness or reproductive rate of the individuals is determined solely by this trait: the non-altruistic individuals are assigned the reproductive rate 1, and the altruistic ones the reproductive rate $1-\tau$. In this sense, we will use interchangeably the term gene and individual to refer to the unit of selection. The demes are classified according to the number of altruistic individuals they have, so that there are $N+1$ different types of demes, labeled by the integers $i = 0, 1, \ldots, N$. We will focus on the time evolution of the frequencies of demes of type i, denoted by Y_i^t, where t stands for the discrete generation index. Clearly, $\sum_{i=0}^{N} Y_i^t = 1$ for all t. As usual, it is assumed non-overlapping generations, *i.e.*, all the individuals in generation t are replaced by their offspring in generation $t+1$. Of particular interest are the average fraction of altruistic individuals in the metapopulation, defined by

$$p_t = \frac{1}{N} \sum_i i\, Y_i^t, \tag{1}$$

and the variance

$$\sigma_t^2 = \frac{1}{N^2} \sum_i i^2 Y_i^t - p_t^2. \tag{2}$$

The life cycle (*i.e.*, one generation) consists of the following events, which are discussed in detail in the sequel: extinction, recolonization, reproduction, mutation, and migration.

2.1 Extinction and recolonization

Differential survival probability favoring demes with a large number of altruists, and the subsequent recolonization of the extinguished demes by the surviving ones, is practically the only generally accepted mechanism to produce group selection in nature. In such setting, a deme of type i survives extinction with probability α_i given by

$$\alpha_i = \begin{cases} \frac{1}{2}\left(1 + \frac{i}{i_c}\right) & \text{if } i < i_c \\ 1 & \text{otherwise,} \end{cases} \tag{3}$$

where $i_c = 0, 1, \ldots, N$ is a parameter measuring the intensity of the group selection pressure. The larger the number of altruists in a deme, the larger its chances of surviving extinction. As the result of extinction, a fraction $1 - \sum_i \alpha_i Y_i$ of demes disappear and must then be recolonized (*i.e.* replaced by the surviving demes). This is achieved by replicating the existing demes in proportion to their frequencies in the surviving metapopulation, yielding thus the following new deme frequencies

$$\frac{\alpha_i Y_i^t}{\sum_j \alpha_j Y_j^t} \tag{4}$$

for $i = 0, \ldots, N$. This procedure is termed interdemic selection since the normalization condition enforced by recolonization yields an effective, indirect interaction between the demes.

2.2 Reproduction

The reproduction process described here takes place inside the demes and hence is termed intrademic selection. Since the size of the demes is fixed and finite (N), random drift occurs. As usual, we assume that the number of offspring that an individual contributes to the new generation is proportional to its relative reproductive rate. Thus, using the standard genetic algorithm prescription [18], the probability that a deme of type j changes to a deme of type i is written as

$$R_{ij} = \binom{N}{i} w_j^i \left(1 - w_j\right)^{N-i}, \tag{5}$$

where

$$w_j = \frac{j\,(1-\tau)}{N - j\tau} \tag{6}$$

is the relative reproductive rate of the subpopulation of altruists in a deme of type j. We note that $\sum_i R_{ij} = 1\ \forall j$ and $\sum_i i R_{ij} = N w_j$.

2.3 Mutation

To take into account the fact that the replication of a gene may not be perfect, we introduce the mutation rate $u \in [0, 1/2]$, which gives the probability that the allele A mutates to B and *vice versa*. Hence the probability that a deme of type j changes to a deme of type i due to mutations of its members is given by

$$U_{ij} = \sum_{l=l_l}^{l_u} \binom{j}{l} \binom{N-j}{i-l} u^{i+j-2l} (1-u)^{N-i-j+2l}, \quad (7)$$

where $l_l = \max(0, i+j-N)$ and $l_u = \min(i, j)$. Clearly, $\sum_i U_{ij} = 1\ \forall j$ and $\sum_i i U_{ij} = Nu + j(1-2u)$.

2.4 Migration

In the procedures described before, there was no *explicit* interaction between demes, although, as mentioned already, the recolonization of extinguished demes by the surviving ones gives rise to an effective interaction between the demes. Migration, however, allows for a direct interchange of individuals between demes. It is implemented as follows. In each deme, firstly J randomly chosen individuals are eliminated and then replaced by individuals picked randomly from the so-called migration pool. This pool is composed of an infinite number of individuals of which the fraction p_t is altruist (*i.e.*, the migration pool is the metapopulation before the migration procedure). The derivation of the transition matrix between demes j and i due to migration is more involved, since in this case we must calculate the number of altruists among the J individuals that are eliminated. This, however, is readily recognized as the classic combinatorial problem that leads to the hypergeometric distribution [19] and so the desired transition matrix is simply given by

$$M_{ij} = \sum_{k=k_l}^{k_u} \frac{\binom{j}{k}\binom{N-j}{J-k}}{\binom{N}{J}} \binom{J}{i-j+k} \times p_t^{i-j+k} (1-p_t)^{J-i+j-k}, \quad (8)$$

where $k_l = \max(j-i, 0, J-N+j)$ and $k_u = \min(j, J-i+j, J)$. As before, $\sum_i M_{ij} = 1\ \forall j$.

2.5 Recursion equations

Given the above four discrete events that comprise the life cycle of the individuals, we can easily write a recursion equation for the frequencies of demes of type $i = 0, 1, \ldots, N$, namely,

$$Y_i^{t+1} = \frac{\sum_{j,k} M_{ij}(p_t) T_{jk} \alpha_k Y_k^t}{\sum_k \alpha_k Y_k^t} \quad (9)$$

where $T_{jk} = \sum_l U_{jl} R_{lk}$ and we have made explicit the dependence of the migration transition matrix on the average frequency of the altruistic gene in the entire metapopulation given by equation (1). Of course, choosing a different order for the occurrence of these basic events will give distinct recursion equations, but we have verified that the main results are qualitatively the same whatever the sequence of the life cycle events.

3 Analysis of the results

In this section we analyze the results obtained by solving numerically the recursion equations (9). For the sake of clarity, in the following we will focus mainly on the average frequency of the altruistic gene as well as on the squared deviations around this average (variance) given by equations (1, 2), respectively. Of course, the set of deme frequencies $Y_i,\ i = 0, \ldots, N$ provides much more information about the structure of the metapopulation. We note, in particular, that the variance reaches its maximal value, namely $\sigma^2 = 1/4$, in the case that $Y_0 = Y_N = 1/2$, while for the uniform distribution $Y_i = 1/(N+1)\ \forall i$ the variance is $\sigma^2 = 1/12 + 1/6N$. In order to better appreciate the effects of the interdemic selection and migration, we will study first the case of independent demes, *i.e.*, $i_c = J = 0$.

3.1 Independent demes

Clearly, in the absence of mutation ($u = 0$), either the gene A or the gene B will reach fixation within a given deme. Thus one has $Y_i^\infty = 0$ for $i \neq 0, N$ and so, in this case, the average frequency of the altruistic gene in the metapopulation yields exactly the fraction of altruistic demes, *i.e.*, $p_\infty = Y_N^\infty$. This quantity is shown in Figure 1 as a function of the altruistic gene disadvantage τ for several deme sizes. The initial deme distribution was uniform, *i.e.* $Y_i^0 = 1/(N+1)$ for $i = 0, \ldots, N$. As expected, for $\tau = 0$ half of the demes are altruist and the other half non-altruist. For $\tau = 1$ only the demes that started altruist will continue so, yielding $p_\infty = Y_N^0 = 1/(N+1)$. Moreover, for $N \to \infty$ one finds $p_\infty = 1/2$ for $\tau = 0$ and $p_\infty = 0$ otherwise. In order to appreciate the magnitude of the deviations from the mean values, we present in Figure 2 the variance σ^2 as a function of τ. Using the same arguments as before, we can easily show that $\sigma^2 = N/(N+1)^2$ and $\sigma^2 = 1/4$ for $\tau = 1$ and $\tau = 0$, respectively. These results indicate that, in the case of small isolated demes, the random drift is quite effective to maintain unfavorable genes ($\tau > 0$). However, we have verified that the altruistic demes are very unstable against migration: setting $J = 1$ leads to the rapid fixation of the non-altruistic gene in the metapopulation, independently of the deme size. Interestingly, there is also a certain instability against mutation as depicted in Figure 3 which presents p_∞ as a function of the mutation rate u. The frequency of the altruistic gene decreases very abruptly for a very small increment of the mutation rate, as shown in the inset, and for $\tau > 0.3$

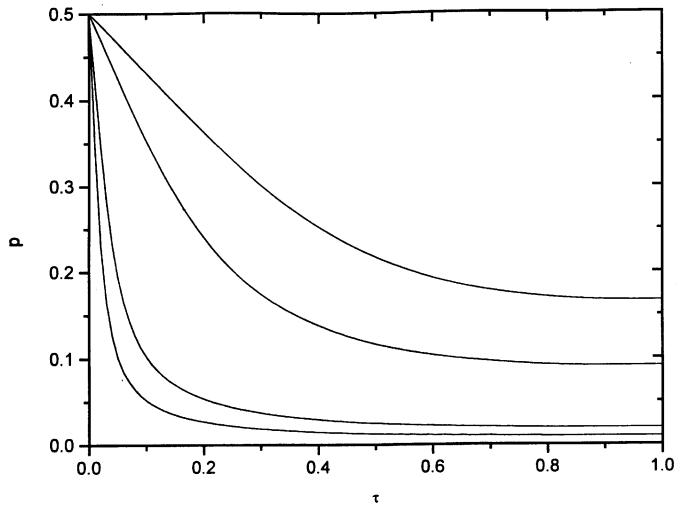

Fig. 1. Steady-state average frequency of the altruistic gene in the metapopulation p as a function of its selective disadvantage τ for (from top to bottom) $N = 5, 10, 50$, and 100. The parameters are $u = 0$, $i_c = 0$, and $J = 0$.

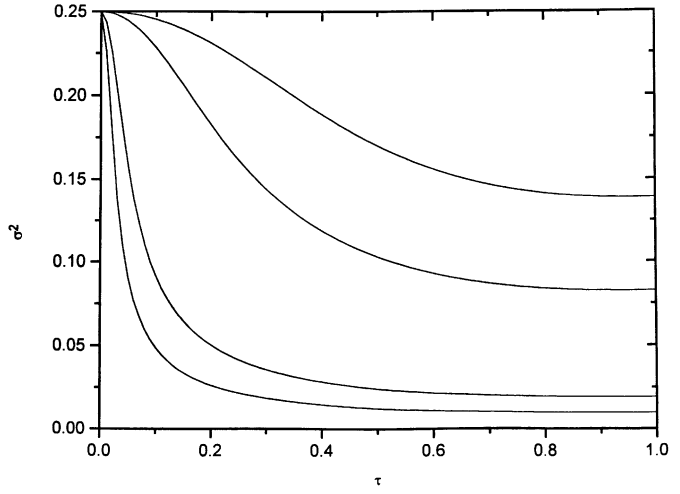

Fig. 2. Steady-state variance of the frequency of the altruistic gene in the metapopulation σ^2 as a function of its selective disadvantage τ. The parameters and convention are the same as for Figure 1.

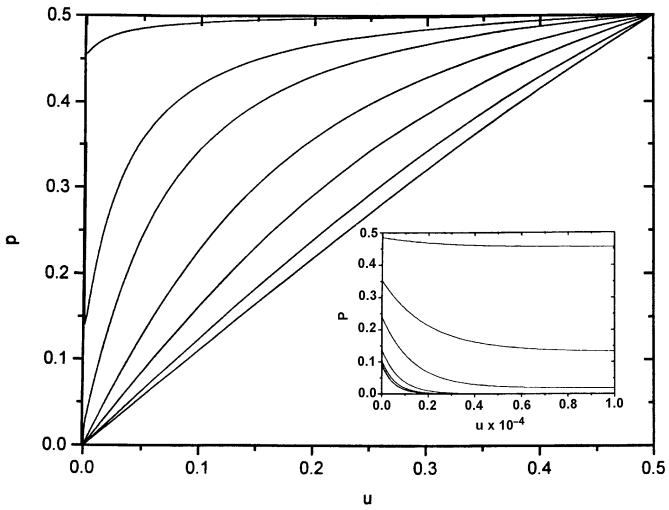

Fig. 3. Steady-state average frequency of the altruistic gene in the metapopulation p as a function of the mutation rate u for (from top to bottom) $\tau = 0.01, 0.1, 0.2, 0.4, 0.6,\ 0.8$, and 0.9. The inset shows the abrupt decrease of p for small mutation rates. The parameters are $N = 10$, $i_c = 0$, and $J = 0$.

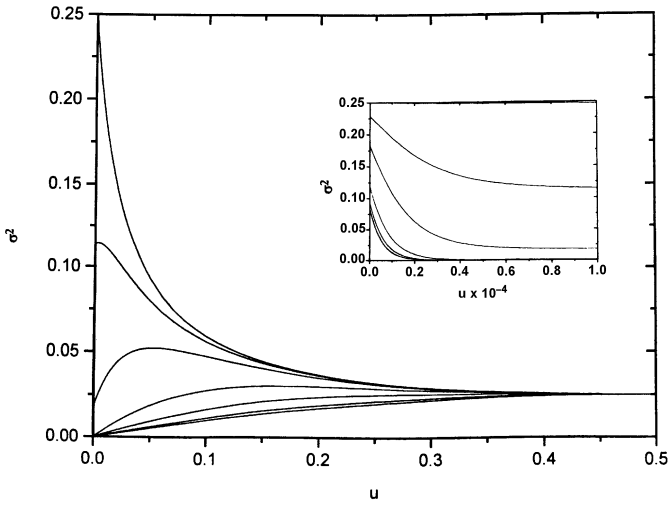

Fig. 4. Steady-state variance of the frequency of the altruistic gene in the metapopulation σ^2 as a function of the mutation rate u. The parameters and convention are the same as for Figure 3.

this effect almost leads to the loss of that gene from the metapopulation. Of course, this sudden decrease is a result of the inevitable take over of the initially altruistic demes by a mutant, non-altruistic gene. The dependence of the variance on the mutation rate is depicted in Figure 4. The steady-state for $u = 1/2$ is completely random: within each deme the genes A and B are equally probable, so that $p_\infty = 1/2$ and $\sigma^2_\infty = 1/4N$.

3.2 Interacting demes

Now we turn to the more general case, where both extinction and migration are allowed. Perhaps, as remarkable and unexpected as Eshel's result mentioned in Section 1 is the time evolution of the metapopulation that leads ultimately to the fixation of the altruistic gene. This evolution is illustrated in Figure 5 that shows p_t as a function of the discrete time index t for several initial populations parameterized by the initial frequency of the altruistic gene p_0. More pointedly, given p_0 the initial deme frequencies are set by

$$Y_i^0 = \binom{N}{i} (p_0)^i (1 - p_0)^{N-i}, \tag{10}$$

so that the next generations can be readily obtained through the recursion equations (9). For $p_0 < 0.5$, the average frequency of the altruistic gene first decreases

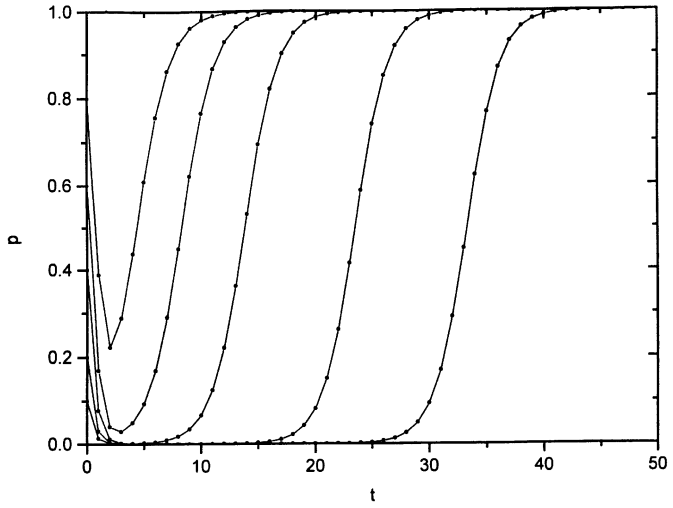

Fig. 5. Time evolution of the average frequency of the altruistic gene in the metapopulation p for (from right to left) $p_0 = 0.1, 0.2, 0.4$, 0.6, and 0.8. The parameters are $N = 10$, $u = 0$, $\tau = 0.9$, $i_c = 10$, and $J = 0$. The lines are guide to the eye.

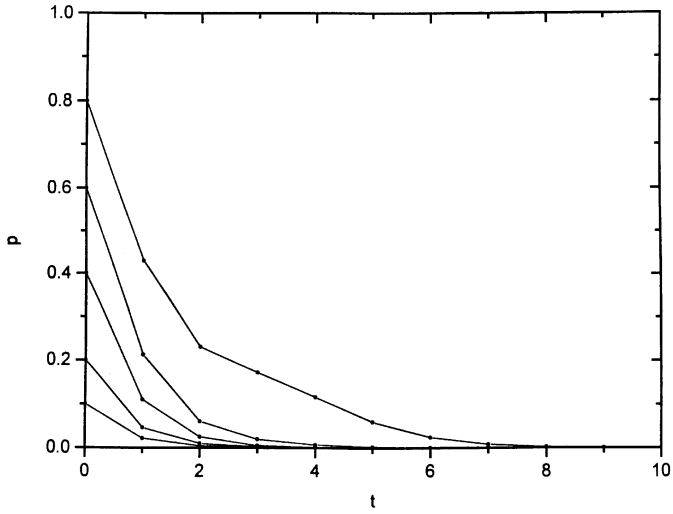

Fig. 6. Same as for Figure 5 but for $J = 1$.

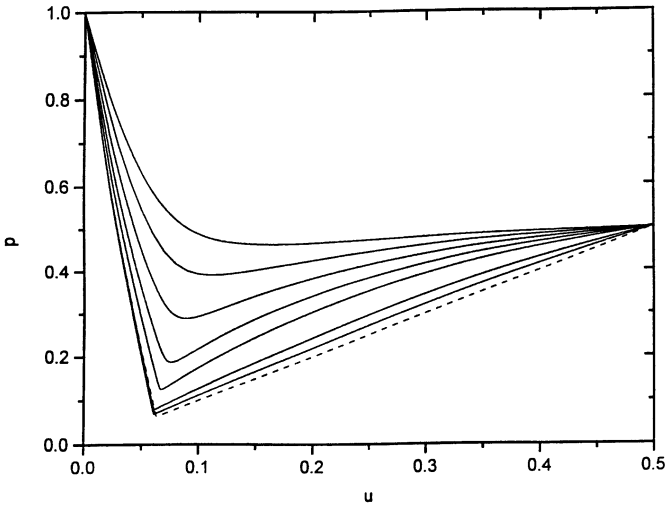

Fig. 7. Steady-state average frequency of the altruistic gene in the metapopulation p as a function of the mutation rate u for (from top to bottom) $\tau = 0.2, 0.3$, 0.4, 0.5, 0.6, 0.8, and 0.9. The parameters are $N = 10$, $i_c = 10$, and $J = 0$. The dashed curves are the analytical predictions for $\tau = 1$.

rapidly, almost leading to the irreversible lost of that gene from the population ($u = 0$ in this case), then enters into a period of stasis, and finally grows rapidly towards fixation. We note that this rather surprising behavior pattern has not been observed in a previous analysis of the model [9]. Obviously, since this model is deterministic the same value of p at distinct generations must correspond to different deme frequencies Y_i. It is clear then that the dynamics takes the metapopulation very close to the unstable fixed point, $p_\infty = 0$, before driving it towards the stable one, $p_\infty = 1$. Although this result suggests that this equilibrium situation may be easily disrupted by changing the control parameters so as to stabilize the fixed point $p_\infty = 0$, this is not so since different choices of the parameters N, τ, and $i_c > 0$ yield qualitatively similar results. In fact, the sole relevant parameter capable of stabilizing $p_\infty = 0$ is the migration parameter J as shown in Figure 6 which presents the same time evolution as before, except that for $J = 1$.

The dependence of the steady-state average frequency of the altruistic gene p_∞ on the mutation rate u is presented in Figure 7 in the case that migration is not allowed ($J = 0$). The main effect of differential extinction ($i_c > 0$) is to increase dramatically the robustness of the altruistic demes to mutation. In particular, while for $i_c = 0$ the inset in Figure 3 shows that the decrease of p_∞ is observed for mutation rates of order 10^{-4}, for $i_c = N$ it occurs for u of order 10^{-1}. Moreover, for small u and large τ, p_∞ decreases linearly with increasing u. The effect of migration is illustrated in Figure 8. As mentioned before, for τ not too small only one migrant per deme is sufficient to disrupt the equilibrium characterized by the fixed point $p_\infty \approx 1$. For $u > 0.2$ the results for $J = 0$ and $J = 1$, shown in Figures 7 and 8, become indistinguishable, indicating then that all selection pressures are eliminated by the random perturbations due to mutation.

In Figures 9 and 10 we show the dependence of the steady-state deme frequencies Y_i on the mutation rate u for $\tau = 0.9$ and $\tau = 0.3$, respectively, in the case of isolated demes ($J = 0$).

The similarity between the present deterministic group selection model and Eigen's molecular quasispecies model [15] becomes apparent from these figures. More specifically, the demes play the role of the molecules, and the altruistic deme, which has the largest survival probability, is analogous with the master sequence, *i.e.*, the molecule with the largest replication rate. (See [20,21] for a similar discrete time formulation of the quasispecies model.) Furthermore, as shown in Figure 9, there is a critical mutation rate, termed error threshold in the molecular evolution context, at which the concentration of altruistic demes practically vanishes (it does vanish for $\tau = 1$

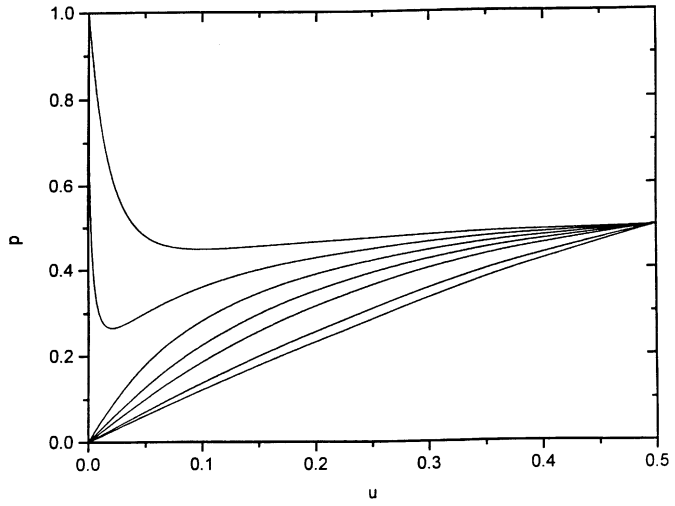

Fig. 8. Same as for Figure 7 but for $J = 1$.

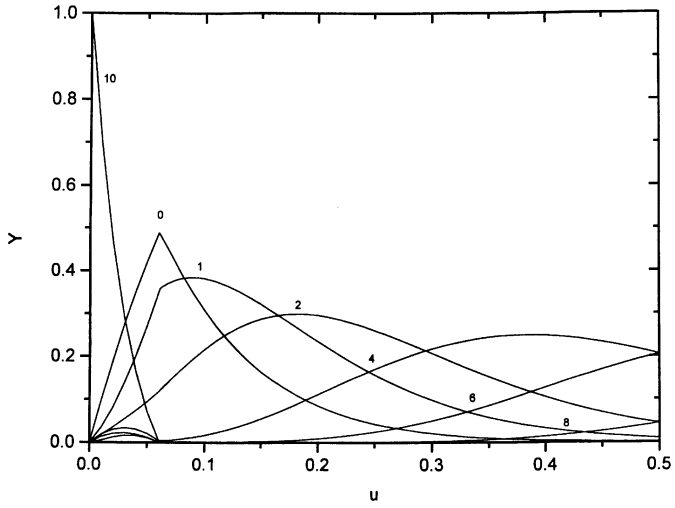

Fig. 9. Steady-state frequencies of demes with $i = 0, 1, 2, 4, 6, 8$ and 10 altruistic individuals as a function of the mutation rate u for $N = 10$, $\tau = 0.9$, $i_c = 10$ and $J = 0$.

as we will show in the sequel). Of course, the main difference between these models is the intrademic selection (reproduction) which has no counterpart in the quasispecies model. This is reflected in the fact that, for small u, the second most frequent deme in Figure 9 is the non-altruistic one, which is not related to the altruistic deme by mutations. In this case the metapopulation is composed mostly of these two opposed demes. A similar coexistence of two quasispecies or, more precisely, of a species and a quasispecies, though highly desirable, is not possible in the standard formulation of the quasispecies model [15]. As shown in Figure 10, for smaller values of τ this critical behavior disappears and the distribution of deme frequencies becomes more similar to that of the quasispecies model, with the deme frequencies decreasing as their distance from the altruistic deme increases. Interestingly, the migration process as modelled in this paper may have an unexpected counterpart in molecular evolution, since

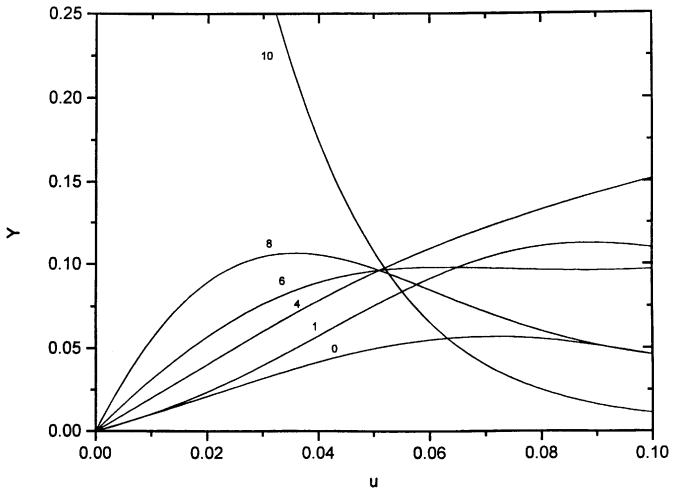

Fig. 10. Steady-state frequencies of demes with $i = 0, 1, 4, 6, 8$ and 10 altruistic individuals as a function of the mutation rate u for $N = 10$, $\tau = 0.3$, $i_c = 10$ and $J = 0$. We note that $Y_{10} \to 1$ for $u \to 0$.

it has been demonstrated recently that the swapping of genes between bacteria is not at all rare in nature [22].

The apparent discontinuities in the derivatives of the steady-state deme frequencies Y_i, and consequently of the steady-state frequency of the altruistic gene p, with respect to the mutation rate u observed in Figures 7 and 9, as well as the almost vanishing of Y_N at a certain critical mutation rate u^* deserve a more careful analysis. Fortunately, the steady state can be calculated analytically in the case $\tau = 1$, $i_c = N$ and $J = 0$, for which those features are more pronounced. In this case we have $w_j = 0$ if $j < N$ and $w_N = 1$, which leads to $R_{0j} = R_{NN} = 1$ for $j < N$ and $R_{ij} = 0$ otherwise. Inserting this matrix in the recursion equations (9) and noting that $M_{ij} = \delta_{ij}$ yield

$$Y_i^{t+1} = U_{i0} + (U_{iN} - U_{i0}) \frac{\alpha_N Y_N^t}{\sum_k \alpha_k Y_k^t} = U_{i0} + 2 (U_{iN} - U_{i0}) \frac{Y_N^t}{1 + p_t}, \tag{11}$$

where we have used $\alpha_N = 1$ and $\sum_k \alpha_k Y_k^t = (1 + p_t)/2$ for $i_c = N$. It is now straightforward to derive the following equation for p_{t+1}:

$$p_{t+1} = u + 2 (1 - 2u) \frac{Y_N^t}{1 + p_t} \cdot \tag{12}$$

Moreover, setting $i = N$ in equation (11) yields

$$Y_N^{t+1} = u^N + 2 \left[(1 - u)^N - u^N \right] \frac{Y_N^t}{1 + p_t} \cdot \tag{13}$$

At the steady state $p_{t+1} = p_t = p_\infty$ and $Y_N^{t+1} = Y_N^t = Y_N^\infty$ we can readily solve equations (12, 13) for p and Y_N simultaneously. In particular, in the regime that $u^N \approx 0$

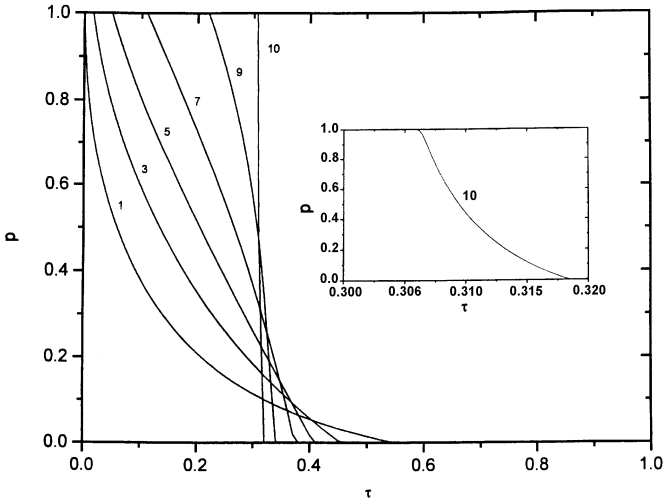

Fig. 11. Steady-state average frequency of the altruistic gene in the metapopulation p as a function of its selective disadvantage τ for $i_c = 1, 3, 5, 7, 9$, and 10. The very abrupt but continuous decrease of p for $i_c = 10$ is shown in the inset. The parameters are $N = 10$, $u = 0$, and $J = 1$.

and $(1-u)^N \approx e^{-Nu}$ we find

$$p_\infty = 2e^{-Nu} - 1$$
$$Y_N^\infty = e^{-Nu}\ \frac{2e^{-Nu} - 1 - u}{1 - 2u} \tag{14}$$

for $u < u^*$, and

$$p_\infty = u$$
$$Y_N^\infty = 0, \tag{15}$$

otherwise. Here u^* satisfies of the equation

$$2e^{-Nu^*} - 1 = u^*, \tag{16}$$

which yields $u^* \approx 0.063$ for $N = 10$. Actually, these two solutions exist for all $u \in [0, 1/2]$, but they are stable only inside the ranges of u given above. We note that though this discontinuous behavior occurs for $\tau = 1$ only, the knees in the curves of Figure 7 are already very pronounced for $\tau = 0.6$.

It is also worth to consider the effect of extending to other demes, besides the altruistic one, the certainty of surviving extinction. Of course, in our formulation this amounts to set $i_c < N$. In the case that neither migration nor mutation are allowed ($J = u = 0$), the altruistic gene reaches fixation (*i.e.*, $p_\infty = 1$) whatever the values of $i_c > 0$ and $\tau < 1$. The case $J = 1$ is illustrated in Figure 11, which shows p_∞ as a function of τ for several values of i_c. There is a rich, nontrivial interplay between the parameters i_c and τ. For instance, given $i_c > 1$, one may have the fixation of the altruistic gene ($p_\infty = 1$) or of the non-altruistic one ($p_\infty = 0$) depending on the value of τ. Rather surprisingly, the range of τ for which the fixation of the non-altruistic gene occurs actually increases with increasing i_c. However, we note that for $i_c = 0$ one has $p_\infty = 0$ for all $\tau > 0$.

Although our analysis has concentrated mainly on a fixed deme size ($N = 10$) and on two values of the migration parameters ($J = 0$ and 1), we have verified that the effect of increasing the values of these parameters is to reduce the frequency of the altruistic gene smoothly, leaving its qualitative dependence on the other parameters unaffected.

4 Conclusion

As the group selection model studied in this paper builds heavily on a discrete time formulation of Levins' classical model [5] proposed by Aoki [9], it is appropriate that we highlight our original contributions to the subject in this concluding section.

The main focus of our analysis was on the effects of mutation on the evolution and stability of altruistic demes under group selection. We note that mutation has not been considered in the previous analyses of variants of this model [5,8,9]. In this line, several interesting results were obtained: (i) the disastrous effect of mutation on the altruistic demes produced by genetic drift only (Fig. 3); (ii) the dramatic reduction of that deleterious effect resulting from the group selection pressure acting *via* differential extinction and recolonization (Fig. 7); and (iii) the appearance of a critical mutation rate u^* for $\tau \approx 1$ at which the frequency of the altruistic deme almost vanishes (Fig. 9).

Moreover, the formulation of the deterministic dynamics using the transition matrices R_{ij}, U_{ij} and M_{ij} that yield the probabilities that a deme with j altruists changes into a deme with i altruists due to reproduction, mutation and migration, respectively, can be easily generalized so as to take into account the effect of finite deme number. The resulting stochastic dynamics can be investigated using approaches proposed originally to study the finite population quasispecies model [20,21]. Furthermore, that formulation facilitates the comparison between group selection and molecular evolution models and, in particular, give some clues on how the coexistence of different quasispecies may be achieved: two opposite selective pressures favoring different classes of molecules must be introduced in the model. In fact, the balance between two opposite chemical reactions, namely, autocatalysis and heterocatalysis, is the basis of the hypercycles – a model that allows the coexistence of several self-replicative molecules [23].

To conclude, some comments regarding the comparison of the standard group selection model presented in this paper with an alternative model for the evolution of altruistic genes [16,17] are in order. In that model, the fitness (reproductive rate) of an individual depends on the genetic composition of the deme it belongs to. In particular, an altruistic individual may be fitter than a non-altruistic one located in a different deme. Furthermore, reproduction is not an intrademic affair: 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* metapopulation. Clearly, this last assumption blurs completely

the notion of group or deme and so, despite being of interest on its own, the model proposed in references [16, 17] cannot be taken as an alternative to the standard mathematical formulation of group selection proposed by Levins [5].

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