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A Stochastic Model for Evolution of Altruistic Genes

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Abstract. — We study numerically a stochastic model for evolution and maintenance of a population which reproduces asexually under selective pressure and is divided into smaller groups of variable size. An altruistic trait is defined as lowering the fitness of the carrier, but the survival probability of all the members of a group with a large enough number of altruists is enhanced. Numerical results show that there is a transition in the average proportion of altruists versus the relative advantage conferred by the presence of altruists to all the members of the group. At the transition the distribution of altruistic frequency in the groups is not trivial, and average deme size reaches a minimum. We found also an error–threshold in mutation rate analogous to the quasi–species model of M. Eigen.

1. Introduction

Natural selection is an egostic process: individual organisms compete for representation of their genes in the next generation, and only those genomes better able to survive and reproduce are likely to be maintained in the evolutionary process. In this framework it is difficult to explain how is possible the evolution and the maintenance of "altruistic" genes, that is genes which determine a behaviour disadvantageous to the carrier, but beneficial for other individuals (benefits and disadvantages are measured in term of individual fitness).

Nevertheless, in nature we can find some examples of altruistic behaviour: the social organization in himenoptera and in hisoptera [1], parental care [2] (including "self sacrificing" behaviour such as injury–feigning), homeostatic regulation of population density [3] (i.e. territorial behaviour capable of adjusting population density to the available food supply), warning calls in birds [3], and so on. A recent report concerns territorial defense by prides of lions. Female lions living in Serengeti National Park and Ngorongoro Crater (Tanzania) live in groups that hunt together and defend a common territory against other groups. Heinsohn and Packer report in [6] that some honess act more aggressively and swiftly in defense, while others tend to lag behind. Territorial contests between lions are violent, frequently leading to serious injury or death of the combatants. On the other hand, a pride of lions without territory has small probability to feed itself and reproduce. No consistent relations were observed between physical size and defensereadiness. Furthermore, when engaging in aid of a pride–mate mounting a defense, no consistent relation was found between the readiness of the helper, and the kin relation between the helper and the first defender. Territorial defense in groups of female lions therefore seems to be a case of altruistic behaviour where kin relation does not play a role.

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So far, there is no evidence of a genetical basis for altruistic behaviour. However, it is an interesting problem in itself to understand how interactions between individuals may be reflected on the action of individual natural selection.

The biological world may be divided in a hierarchy of increasing complexity: gene, genome, cell, organism, group, species, type, up to the whole biosphere. Selective pressure acts on each of these levels; in the following I will call a level on which natural selection operates a selection unit. A phenomenon or a behaviour which increases the chances of survival for a selection unit may be disadvantageous at another level. This is what happens in altruism: the presence of altruists increases the chances of the group which it belongs to. The general problem is not only to understand what is the level of selection action, but also how the different levels may interact under the selective pressure.

Classically, two main mechanisms have been proposed to explain the origin and evolution of altruism: kin selection [1, 2, 7], and group selection [4, 5].

In both mechanisms it is necessary to suppose that individual fitness depends not only on individual genotype, but also on the rest of population. In kin selection the individual probability of survival is proportional to the relatedness that the individual shares with the altruist. The selection mechanism thus is limited only to related individuals, and the selection unit is the individual. In group selection, the interaction is between individuals belonging to the same group, and groups are defined by external constraints (for example spatial boundaries), disregarding any relatedness between individuals. Here there are two selection levels and two selection units, the individual and the group.

In the model discussed below we disregard relatedness between individuals. The selection unit is the individual, but the individual fitness depends on the composition of the group. Although this model is extremely simple, we are able to observe numerically the maintenance of altruistic genes.

2. The Model

We consider a population $\Omega$, made up of a fixed numbers of individuals, $M$. This population is divided into groups (demes) of variable size. We assume that generations are non-overlapping; that heredity acts according to the usual Mendelian mechanism and that there is a behavioural locus with two alleles: A (recessive), E (dominant). An individual of genotype AA is an altruist, while AE and EE are not.

To each individual $\alpha$ in a generation we associate its reproduction probability $f(\alpha)$, which in the model is the same as the survival probability of this genome in the population over one step. The fitness $f(\alpha)$ depends on two factors:

1. on its genotype: if the individual is altruist (genotype AA), its fitness is reduced by a factor $(1 - r)$ with respect to the other members of its group;

2. if the individual (whatever its genotype) belongs to a group with a large enough fraction $x$ of AA individuals (i.e. $x \geq x^*$), its fitness is enhanced by a factor $(1 + c)$ with respect to groups which do not satisfy this condition.

The factor $r$ is called intrademic selection rate and the factor $c$ is called interdemic selection rate.

The transition between generations is a discrete process, involving the following three steps:

1. Selection and reproduction. Reproduction may be asexual or sexual. In the following, we will consider only the asexual case. If $\alpha$ is an individual of the population $\Omega_t$, the
probability that an individual $\alpha' \in \Omega_{t+1}$ is one of its offspring is given by:

$$P(\alpha' \in \Omega_{t+1}|\alpha \in \Omega_t) = \frac{f(\alpha)}{\sum_{\beta=1}^{M} f(\beta)},$$

where $f$ is the fitness defined in Table I and the sum is over the whole population. The offspring of the individuals of one deme in one generation form one deme in the next generation.

2. Mutation. Each genotype may mutate into another one; if $u$ is the mutation rate per allele, then the mutation probabilities $p_{g \rightarrow g'}$ are given in Table II.

3. Deme splitting. There is a maximal finite size of a deme, call it $M^*$. If a deme grows to be larger than $M^*$ it is split in two, and the members of the original deme are randomly distributed into the two new demes. This rule is motivated by the analogy with social animals, which typically live in groups that are not too large. It is also a necessary ingredient for purely modelling reasons: if there is no maximal size of demes, sooner or later one of the demes will take over the whole population, and the interdemic selection ceases to operate.

Then, we compute the fraction of AA individuals for each deme and for the whole population, and iterate the process.

Table I. — Table of the fitness. Rows: individual genotypes; columns: genetical group composition.

<table>
<thead>
<tr>
<th></th>
<th>$x &lt; x^*$</th>
<th>$x \geq x^*$</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA</td>
<td>$1 - r$</td>
<td>$(1 - r)(1 + c)$</td>
</tr>
<tr>
<td>EA, EE</td>
<td>$1$</td>
<td>$1 + c$</td>
</tr>
</tbody>
</table>

Table II. — Mutation probability $p_{g \rightarrow g'}$. Rows: genotypes $g'$; columns: genotypes $g$.

<table>
<thead>
<tr>
<th></th>
<th>AA</th>
<th>AE</th>
<th>EE</th>
</tr>
</thead>
<tbody>
<tr>
<td>AA</td>
<td>$(1 - u)^2$</td>
<td>$u(1 - u)$</td>
<td>$u^2$</td>
</tr>
<tr>
<td>AE</td>
<td>$2u(1 - u)$</td>
<td>$(1 - u)^2 + u^2$</td>
<td>$2u(1 - u)$</td>
</tr>
<tr>
<td>EE</td>
<td>$u^2$</td>
<td>$u(1 - u)$</td>
<td>$(1 - u)^2$</td>
</tr>
</tbody>
</table>

3. Numerical Results

As a consequence of the ergodic theorem of stochastic processes [9] (which in our model holds for mutation rates different from zero), in a few generations the system reaches a stationary probability distribution, independent on the initial conditions. This steady state is characterized by a dynamical equilibrium between the intrademic and the interdemic dynamics; fluctuations in the concentration of altruists in different demes are very large. We therefore present results both about distributions and average values.

The altruistic gene is kept in the population by the relative advantage it confers to its group in the interdemic competition. Its distribution is not trivial, at least in the phases in which its concentration is different from zero. This is shown in Figures 1, 2, 3, 4.
We found a transition in the equilibrium average value of the fraction $x_{\text{tot}}$ of AA individuals in the whole population, as function of the interdemic selection rate $c$, keeping fixed the other parameters (the intrademic selection rate $r$, the mutation rate $u$, the threshold concentration of AA individuals $x^*$). This is shown in Figure 5.
If the mutation rate increases, the transition becomes more and more broad: both intrademic and interdemic selection have no more effect. This result reminds of the error threshold in the quasi-species model of M. Eigen [8]. See Figure 6 below.

Another interesting quantity to study is the deme size. In the model this quantity is a result of the dynamics of the system, it is not a parameter. We found that at the transition (i.e. in
Fig. 5. — Average value of altruist concentration $x_{tot}$ in the population versus interdemic selection rate $c$. $M = 500, M^* = 50, r = 0.2, u = 0.03$.

Fig. 6. — Average value of altruist concentration $x_{tot}$ in the population versus interdemic selection rate $c$. $M = 500, M^* = 50, r = 0.2, u = 0.05, 0.08, 0.1, 0.3$.

correspondence of the interdemic selection value $c^*$) the average deme size reaches a minimum. Moreover, we can see a change in the shape of the distribution above and below the transition. See Figures 7, 8, 9.
4. Discussion

We have studied a model where a population of $M$ individuals is divided in demes of smaller size. The fitness of an individual depends on its genetic make-up and on the presence of altruistic individuals in the deme.
Fig. 9. — Average value of deme size versus interdemic selection rate $c$. $M = 500$, $M^* = 50$, $u = 0.03$, $r = 0.2$.

Numerical results demonstrate the maintenance of the altruistic gene; in particular, there is a transition in the value of concentration of altruists as a function of interdemic selection rate. The interesting feature of this result is that in the simulated model the selection unit is the individual. The individual probability of reproduction depends also on the genetic composition of the deme, and for this reason the effects of individual selection “propagate” to the deme level. The individual probability of reproduction is enhanced if the concentration of altruists, in its deme, is above the threshold: this may be considered as a kind of “interaction” between individuals, without involving any kin relationship between them.

Since the genome of our model consists of just one locus with only three possible states (AA, AE, EE) and we consider asexual reproduction, the only meaning one could give to kin relationship would be the genetic overlap at this locus between individual in a deme. A kin selection mechanism would imply that altruism is more frequent in genetically homogeneous demes. In our model altruism is more prominent in genetically inhomogeneous demes, and the most genetically homogeneous are the ones with the smallest fraction of altruists.

In our model deme selection is a consequence of individual selection: a deme is the collection of individuals who survived (reproduced) in the previous generation. This is the reason which allows us to consider the deme size as a variable and not as a parameter. We saw that in the range of $c$ corresponding to the transition, the deme size reaches a minimum; it means that altruism is most successful in small demes: this is a result, not a starting point!

The other important result is that the transition becomes broader with increasing mutation rate: this is analogous to the error threshold in the quasi–species model [8], where there is a transition between a localized phase and a disperse one. Below the threshold the equilibrium distribution of sequences is localized near the “master” sequence, and the transition happens when the individual selective pressure, which tends to localize sequences, cannot anymore balance the dispersive effect of mutation. In our model, the changes in the genetical composition of population are due not only to mutation and to individual selection, but also to the effects on selective process of the “interaction” in the reproduction probability. Hence our numerical
results suggest that the error–threshold remains also in presence of interaction between the selective units.

I have also studied in collaboration with Luca Peliti and Maurizio Serva a simplified model where selection acts directly on the deme level, with intensity proportional to the interactions between the individuals in the deme [10]. Analytical results on this model fully support the numerical results on the more detailed model studied here.

Acknowledgments

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References