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Vol. 11, No. 3, June 1977
Printed in Belgium

On the Founder Effect and the Evolution of Altruistic Traits: An Ecogenetical Approach

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Received May 24, 1976

The evolution of some altruistic traits in a population repeatedly recolonizing temporal habitats is studied. Special consideration is given to the joint effect of the population density and gene frequencies.

1. A GENETICAL AND AN ECOGENETICAL MODEL FOR THE EVOLUTION OF ALTRUISTIC TRAITS DUE TO A FOUNDER EFFECT

Conditions for the evolution of altruistic traits in a population repeatedly recolonizing temporal habitats have been studied by Maynard Smith (1964), Matessi and Jayakar (1976), Wilson (1974a), and Cohen and Eshel (1976). Such habitats are fruits, exhaustable sources of food, or short-living hosts. Among the biological examples analyzed were those of active and passive cleaning mechanisms, self-restraint from over-use of natural resources, and host preservation by bacteria and viruses (see also Cohen (1976)). In this connotation, the term *altruist* should be understood only in its technical meaning (e.g., see Eshel, 1972; Haldan, 1932; Hamilton, 1964) as an inherited trait which is advantageous for the population as a whole, but which is disadvantageous for its individual carrier when competing with nonaltruistic neighbors.

The population studied has been assumed to consist of infinitely many, temporarily isolated colonies, each being established by a relatively small group of founders taken at random from colonies of the previous generation. The growth of each colony (and, therefore, its average contribution to colonies of the next generation) has been assumed to depend statistically on the genetical makeup of the random founding group. The larger the frequency of the altruist gene within the founding group of the colony, the larger the growth rate of the entire colony. However, it is assumed that in each colony the selfish type (this is the term we use for the nonaltruistic type), if it exists, propagates faster than the altruist. Assuming independent colonization of available habitats by either altruistic or selfish individuals, it was assumed by Cohen and Eshel that

ISSN 0040-5809

the probability of a habitat being colonized by exactly n altruistic and m selfish colonizers is

$$a(m, n) = e^{-\nu} \frac{\nu^{m+n}}{m!\, n!} p^n q^m,$$

where ν is the density of the colonizing population (i.e., the expected number of individuals colonizing a new habitat. For other plausible statistical assumptions about colonization of new habitats see Matessi and Jayakar, 1976).

The average rate of increase $h_\nu(p)$ and $g_\nu(p)$ of the altruist and the selfish in a population of a density $\nu > 0$ and an altruistic frequency $0 \leqslant p \leqslant 1$ have been obtained by averaging altruistic and selfish rates of reproductions over colonizers of all possible habitats. In probability 1 the frequency of the altruist within the next generation of this population is readily given by:

$$p' = f_\nu(p) = \frac{ph_\nu(p)}{ph_\nu(p) + qg_\nu(p)}. \tag{1.1}$$

As a detailed analysis of various typical situations has shown, the shape of $f_\nu(p)$ and, therefore, some of the rules governing the selection forces in the population, appear to be different for different sorts of altruistic traits. But in all cases the density of the colonizing population has proved crucial for the evolution of the altruistic trait in this population. More specifically, for any frequency $0 < p < 1$ of the altruistic gene, it is shown that:

(i) The frequency $p' = f_\nu(p)$ of the altruist in the next generation is a monotone decreasing function of the density ν.

(ii) For low enough density, selection always favors the altruist, i.e., $f_\nu(p) > p$.

Under quite general assumption it has also been shown that:

(iii) For high enough density, selection is always unfavorable for the altruist, i.e., $f_\nu(p) < p$.

It, thus, follows that for any altruistic frequency $0 < p < 1$, a critical density $\nu^* = \nu^*(p) > 0$ exists such that $p' = f_\nu(p) > p$ if and only if $\nu > \nu^*(p)$, i.e., selection operates in favor of the altruist if the population density is below the frequency-dependent critical value and vice versa if the population density is above this value. Taking into consideration various values assumed for the population density, more specific questions about the stability of fixed points and polymorphism maintenance have been asked from different viewpoints both by Matessi and Jayakar (1976) and by Cohen and Eshel (1976).

A factor not incorporated into the present studies is the simultaneous effect of altruism on the density and genetical structure of the population. Realistically, the effect of altruism should be an increase in the carrying capacities of habitats where it is frequent. That is, the density of a population might be expected to be a function of the frequency of the altruist within the population as likely as the reverse.

Furthermore, analysis of the ecogenetical forces governing simultaneous changes in both the density ν of the population and the frequency p of the altruist within it very often yields results that are essentially different from those being drawn from models studying each of these parameters separately.

Thus, in such situations as nonpolluting altruism, self-restraint from overexhaustion of environmental resources, and host preservation, the critical population density $\nu^*(p)$ has been shown to be an increasing function of p. For such situations it has been readily shown (Cohen and Eshel, 1976) that at any fixed density of the population neither a stable polymorphism nor protection of both types can possibly be maintained in the population (see Fig. 1).

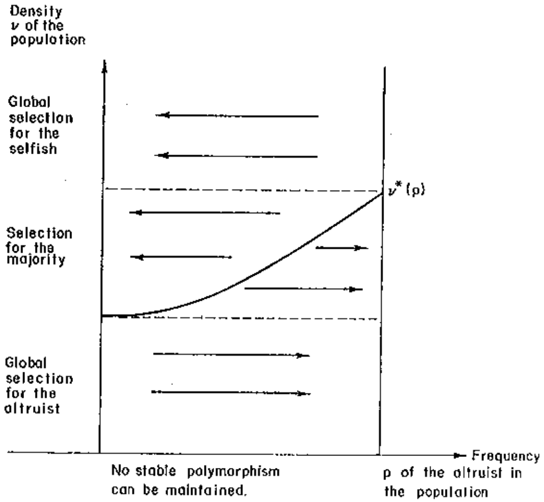

FIG. 1. Selection for and against the altruist in various fixed population densities.

When simultaneous changes in both p and ν are taken into consideration, it will be shown that there are always appropriate ecological conditions of population growth for which a stable polymorphism of altruist and selfish can be maintained. (See Fig. 2 and next section for details.)

For ecological conditions determining different control of the population size, the same selection forces within the population can cause either permanent cycling, global convergence to one of the monomorphic equilibria or local convergence toward both. On the other hand, it is shown that, under certain conditions, an inner ecogenetical fixed point may be locally unstable even when both ecological and selection forces each operate separately to stabilize the appropriate parameters of this fixed point. In this case a permanent cycling is always achieved.

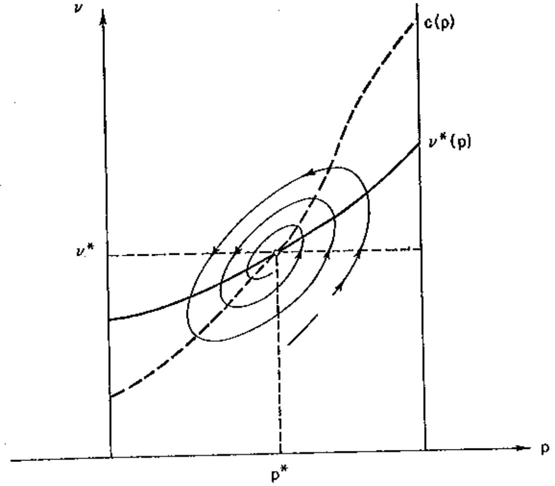

FIG. 2. An ecogenetic version of the situation shown in Fig. 1. Simultaneous changes of gene frequencies and population densities. Global convergence to an ecogenetical equilibrium (p^*, ν^*) is possible (see Section 2 for the appropriate conditions) even though for $\nu = \nu^*$ changes in p are always to a frequency further from p^*.

In the next section mutual effects of the genetical and the ecological systems on each other are analyzed. Special consideration is given to possible effects of either improvement or deterioration of environmental conditions (to be rigorously defined in due course) on the ecogenetical system and, in particular, on its stable population density. For example, we show that stable population density may not be a monotone function of improvement in environmental conditions, a result which cannot be attributed to ecological effects alone. In general, it is shown that such an improvement, when being expressed in terms of an increase in the carrying capacity of the population, can cause a genetical switch of this population from a stable altruistic monomorphism to a stable selfish monomorphism. In some cases we show that this switch results in a drop in the density of the population to a level that is beyond the one which has been stably maintained under less favorable conditions.

The paper concludes with some speculations about the implications of the ecogenetical model for the theory of epidemiology.

2. AN ECOGENETICAL MODEL FOR THE EVOLUTION OF ALTRUISTIC TRAITS DUE TO FOUNDER EFFECT

For a frequency p of altruists and a population density ν let

$$T\colon (p, \nu) \to (p', \nu') = (f(p, \nu), \varphi(p, \nu)) \tag{2.1}$$

be the transformation determining the simultaneous change in altruistic frequency and population density from one generation to next. The rule $p \to p' = f(p, \nu)$, determining the *genetical change* in the population is given by (1.1). For many important cases the function $f(p, \nu)$ is specifically calculated in Cohen and Eshel, (1976). From basic results obtained by Cohen and Eshel it is generally shown that

(i) $f(p, \nu)$ is an increasing function of p and a decreasing function of ν (i.e., the higher the frequency of the altruist and the lower the density of the population in a given generation, the higher the frequency of the altruist in the next generation).

As usual in population biology, we know less about the specific structure of the *ecological rule* $\nu \to \nu' = \varphi(p, \nu) = ph(p, \nu) + qg(p, \nu)$ determining the change in the population density from one generation to next. It is quite reasonable, however, to assume most generally that:

(ii) $\nu' = \varphi(p, \nu)$ is a monotone increasing function of both p and ν (i.e., the more dense is the population in a given generation and the higher the frequency of the altruist within this population, the more dense the population of the next generation).

(iii) For any value $0 \leqslant p \leqslant 1$, the growth rate $(1/\nu)\, \varphi(p, \nu)$ of the population is a decreasing function of its density ν, assuming values smaller than 1 as the density ν tends to infinity.

As an immediate result of (ii) and (iii) we infer the existence of a carrying capacity function $c(p) \geqslant 0$ satisfying $c(p) = \varphi(c(p), p)$ such that for all $0 \leqslant p \leqslant 1$:

$$\begin{aligned} \nu < c(p) &\Rightarrow \nu < \nu' = \varphi(p, \nu) < c(p), \\ \nu > c(p) &\Rightarrow \nu > \nu' = \varphi(p, \nu) > c(p). \end{aligned} \tag{2.2}$$

From (ii) it is also implied that $c(p)$ is a monotone nondecreasing function of p, and we generally assume that it is also continuous and differentiable. We also assume that the solution $\nu^*(p)$ of $p = f(p, \nu^*)$ is differentiable.

The fixed points of the ecogenetical system (2.1), simultaneously satisfying the equations $p = f(p, \nu)$ and $\nu = \varphi(p, \nu)$, are $(0, c(0))$, $(1, c(1))$ and all the intersections $(\hat{p}, \hat{\nu})$ of the carrying capacity function $c(p)$ and the critical density function $\nu^*(p)$, if exist. We start by studying the monomorphic fixed points.

PROPOSITION 1. *The purely selfish fixed point* $(0, c(0))$ *is stable if* $c(0) > \nu^*(0)$ *and unstable if* $c(0) < \nu^*(0)$.

The purely altruistic fixed point $(1, c(1))$ *is stable if* $c(1) < \nu^*(1)$ *and unstable if* $c(1) > \nu^*(1)$.

Proof. If $c(0) < \nu^*(0)$, then there is a vicinity of $(0, c(0))$ on which $\nu < \nu^*(p)$ and, therefore, $p' = f(p, \nu) > p$. No convergence to $(0, c(0))$ is possible in this vicinity except, maybe, for points $(0, \nu)$ on the boundary of this vicinity, and $(0, c(0))$ is then unstable.

Suppose, on the other hand, $c(0) > \nu^*(0)$. From the continuity of c and ν^* it follows that for some $\delta > 0$ and all $0 < p < \delta$, $c(p) > \nu^*(p)$. Consider the vicinity $U = \{(p, \nu) \mid \nu > \nu^*(p), 0 \leqslant p < \delta\}$ of $(0, c(0))$. Let $(p, \nu) \in \mathrm{Cl}(U)$. We show that $T(p, \nu) = (p', \nu') \in U$. Since $\nu > \nu^*(p)$ we know that $p' \leqslant p \leqslant \delta$ with a sharp inequality $p' < p$ unless $p = 0$. As for ν', let us distinguish between two possibilities. If $\nu \geqslant c(p)$, then from (2.2) and the monotonicity of $c(p)$ we infer that

$$\nu' \geqslant c(p) > c(p') > \nu^*(p').$$

If, on the other hand, $\nu^*(p) \leqslant \nu \leqslant c(p)$, then $\nu' \geqslant \nu \geqslant \nu^*(p) > \nu^*(p')$. Thus, in all cases $(p', \nu') \in U$ and $T\colon \mathrm{Cl}(U) \to U$ is a continuous transformation decreasing the value of p. The iteration $T^n(p, \nu)$ thus tends to the line $p = 0$ over which the transformation decreases the value $|\nu - c(0)|$, and convergence to $(0, c(0))$ follows immediately.

The proof is quite similar for $(1, c(1))$.

As a result of Proposition 1 we get:

PROPOSITION 2. *If for all* $0 \leqslant p \leqslant 1$ $\nu^*(p) > c(p)$, *then the pure altruistic fixed point* $(1, c(1))$ *is globally stable in the ecogenetic system.*

If for all $0 \leqslant p \leqslant 1$, $\nu^*(p) < c(p)$, *then* $(0, c(0))$ *is globally stable.*

Proof. Assume $c(p) > \nu^*(p)$ for all $0 \leqslant p \leqslant 1$. If, for a given point (p, ν), $\nu > \nu^*(p)$, then the convergence $T^n(p, \nu) \to (0, c(0))$ is proved in quite the same way as in Proposition 1. In the region $\{(p, \nu) \mid \nu \leqslant \nu^*(p)\}$, on the other hand, $p' > p$ and, therefore, for any (p, ν) in this region there is an iteration $T^n(p, \nu) = (p^{(n)}, \nu^{(n)})$ with $\nu^{(n)} \geqslant \nu^*(p^{(n)})$ or else the sequence $\{T^n(p, \nu)\}$ converges to a fixed point in the region. But since there is no such a fixed point $\lim_{m\to\infty} T^m(p, \nu) = \lim_{m\to\infty} T^m(p^{(n)}, \nu^{(n)}) = (0, c(0))$.

The proof of the global convergence of the system to $(1, c(1))$ is similar.

Since an increase in the carrying capacity function of a population may indicate and, by definition, measure an improvement of environmental conditions (see next section for a more detailed consideration of some specific situations), Proposition 2 may be restated as follows:

A sufficient improvement of environmental conditions (i.e., a global increase of the carbying capacity function to above the level of the function $\nu^*(p)$*) always results in the extinction of the altruist. On the other hand, a sufficiently drastic environmental deterioration, if not dropping the carrying capacity to zero, is due to result in the fixation of the altruist, provided this type exists in the population.*

Since for any function $c(p)$ of frequency-dependent carrying capacity, $c(1)$ is always larger than the selfish carrying capacity $c(0)$, Proposition 2, in a certain sense, suggests a natural mechanism of selection for a regulation of the population size. For suppose that an improvement of environmental conditions increases the carrying capacity function from $c_1(p)$, which is below the critical density function $\nu^*(p)$, to $c_2(p)$, which is above $\nu^*(p)$. The density of the population will then, first, increase from its original stable level $c_1(1)$ to values close to $c_2(1)$. Yet, selection against the altruist will eventually drop the density of the population to its lower, only stable level, $c_2(0)$. Moreover, as shown in Fig. 3, when $\nu^*(p)$ is an increasing function of p (as has been found to be the case in most relevant situations studied in Cohen and Eshel (1975)), a moderate increase of $c(p)$ from a little below $\nu^*(p)$ to values not far above $\nu^*(p)$ may end with an eventual decrease in the globally stable population density. The assumption being made here is that the selection forces operating within habitats and, therefore, the critical density function $\nu^*(p)$ are not affected by changes of the carrying capacity function $c(p)$. (See the next section for the natural conditions justifying this assumption.)

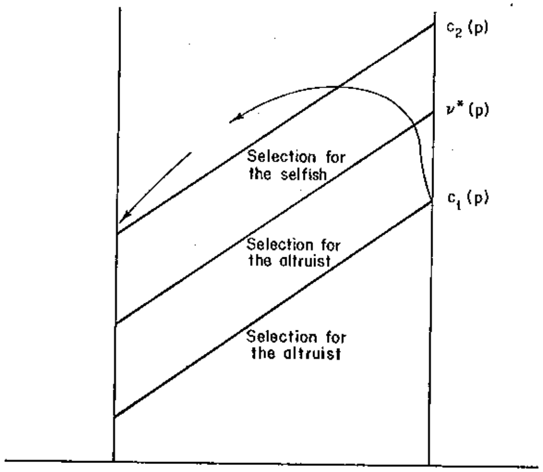

FIG. 3. An increase in the carrying capacity function resulting in an eventual drop in the stable population density. When the carrying capacity function changes from $c_1(p)$ to $c_2(p)$ the altruist becomes extinct and the globally stable ecogenetical equilibria shift from $(1, c_1(1))$ to $(0, c_2(0))$ with the density being decreasing from $c_1(1)$ to $c_2(0)$.

As an immediate result of Proposition 1 we also get:

PROPOSITION 3. *If* $c(p)$ *intersects* $\nu^*(p)$ *from above (a single or an odd number of simple intersections), then both the monomorphic fixed points are stable. If there is exactly one intersection, then these are the only stable fixed points of the system and no polymorphism can be stably maintained.*

PROPOSITION 4. *If $c(p)$ intersects $\nu^*(p)$ from below (with a single or an odd number of simple intersections), then both the monomorphic fixed points are unstable and a protected polymorphism is maintained by the ecogenetical system.*

Note that the result of Proposition 4 is independent of the question of whether $\nu^*(p)$ is an increasing or a decreasing function of p. Thus, if the critical density $\nu^*(p)$ is increasing with the altruist frequency, but at a slower rate than the carrying capacity $c(p)$, *then the result of the ecogenetical model appears to stand in disagreement with the prediction of the pure genetical model.* (Indeed, the results of the two models always agree under the assumption $c(p) \equiv$ constant, tacitly made in Cohen and Eshel (1975).) As is indicated by the following two propositions, a protected polymorphism of altruist and selfish can either be maintained as a stable polymorphism or else be a result of a permanent cycling. Moreover, for any rule of natural selection given by $p' = f(p, \nu)$, each of these two possibilities can occur, depending on the appropriate ecological rule $\nu' = \varphi(p, \nu)$ of the population growth. Actually, we attempt to characterize the behavior of the system by two ecological parameters:

(i) The slope $c'(\hat{p})$ of the carrying capacity at the point $(\hat{p}, \hat{\nu})$ of its intersection with $\nu^*(p)$. This parameter naturally measures *the susceptibility of the ecological system to genetical variations near* $(\hat{p}, \hat{\nu})$.

(ii) The value $0 \leqslant 1 - \partial\varphi/\partial\nu \leqslant 1$, namely, *the ecological stability of the system at* $(\hat{p}, \hat{\nu})$. This parameter measures the rate in which the population density approaches its carrying capacity near $(\hat{p}, \hat{\nu})$.

In accordance with the definition of ecological stability, it is convenient to define also the *genetical stability* $1 - \partial f/\partial p(\hat{p}, \hat{\nu}) \leqslant 1$ at the point $(\hat{p}, \hat{\nu})$. Since unlike $\partial\varphi/\partial\nu$, $\partial f/\partial p(\hat{p}, \hat{\nu})$ may be larger than 1 (this is the case when $\nu^{*\prime}(\hat{p}) > 0$), we also speak of the *genetical instability* $\partial f/\partial p(\hat{p}, \hat{\nu}) - 1$.

Proposition 4 indicates that an appropriate carrying capacity function $c(p)$ can always guarantee a protected polymorphism of altruist and selfish. In order to study the behavior of the system in this case, let $(\hat{p}, \hat{\nu})$ be any interior fixed point of the transformation T (i.e., $(\hat{p}, \hat{\nu})$, is an intersection of $\nu^*(p)$ and $c(p)$), and let the linear approximation matrix of T at this point be

$$\frac{\partial(f, \varphi)}{\partial(p, \nu)} = \left\| \begin{matrix} \dfrac{\partial f}{\partial p} & \dfrac{\partial f}{\partial \nu} \\ \dfrac{\partial \varphi}{\partial p} & \dfrac{\partial \varphi}{\partial \nu} \end{matrix} \right\|$$

Note that all entries of the matrix except for $\partial f/\partial\nu$ are nonnegative (immediate from the conditions (i)–(iii) given above). Thus, having $\partial\varphi/\partial\nu + \partial f/\partial p \geqslant 0$, the largest eigenvalue of the matrix (in absolute value) is

$$\lambda_1 = \frac{1}{2}\left\{\frac{\partial f}{\partial p} + \frac{\partial \varphi}{\partial \nu} + \left(\left(\frac{\partial f}{\partial p} - \frac{\partial \varphi}{\partial \nu}\right)^2 + 4\,\frac{\partial f}{\partial \nu}\,\frac{\partial \varphi}{\partial p}\right)^{1/2}\right\}. \qquad (2.3)$$

It is readily shown that this eigenvalue is less than 1 in absolute value if and only if

$$1 - \frac{\partial f}{\partial p}\frac{\partial \varphi}{\partial \nu} > -\frac{\partial f}{\partial \nu}\frac{\partial \varphi}{\partial p} > -\left(1 - \frac{\partial f}{\partial p}\right)\left(1 - \frac{\partial \varphi}{\partial \nu}\right). \tag{2.4}$$

If one of the inequalities in (2.4) is replaced by an equality, then $|\lambda_1| = 1$. Hence, (2.4) is a sufficient condition for stability of $(\hat{p}, \hat{\nu})$, whereas with weak inequalities it serves as a necessary condition for stability.

But by implicit differentiation of $p = f(p, \nu^*(p))$ it is implied that for any point on $\nu^*(p)$

$$\frac{\partial f}{\partial p} + \nu^{*\prime}\frac{\partial f}{\partial \nu} = 1 \tag{2.5}$$

and for any point on $c(p)$

$$\frac{\partial \varphi}{\partial p} + c'\frac{\partial \varphi}{\partial \nu} = c'. \tag{2.6}$$

Hence, in the intersection point $(\hat{p}, \hat{\nu})$ we have

$$\nu^{*\prime}(\hat{p})\frac{\partial f}{\partial \nu}\frac{\partial \varphi}{\partial p} = c'(\hat{p})\left(1 - \frac{\partial \varphi}{\partial \nu}\right)\left(1 - \frac{\partial f}{\partial p}\right). \tag{2.7}$$

From (2.4) and (2.7) we readily get:

PROPOSITION 5. (i) *If*

$$1 - \frac{\partial \varphi}{\partial \nu} \leqslant \frac{\partial f}{\partial p} - 1 \tag{2.8a}$$

(*i.e., if the* genetical instability *of* $(\hat{p}, \hat{\nu})$ *is larger than its* ecological stability), *then* $(\hat{p}, \hat{\nu})$ *is always unstable.*

(ii) *If*

$$1 - \frac{\partial \varphi}{\partial \nu} > \frac{\partial f}{\partial p} - 1 > 0 \tag{2.8b}$$

(*i.e., if* $(\hat{p}, \hat{\nu})$ *is* genetically unstable *but if* genetical instability *is less than the* ecological stability), *then* $(\hat{p}, \hat{\nu})$ *is stable for appropriate medium values of* $c'(\hat{p})$, *namely, for*

$$\nu^{*\prime}(\hat{p}) < c'(\hat{p}) < K\left(\frac{\partial f}{p}, \frac{\partial \varphi}{\partial \nu}\right)\nu^{*\prime}(\hat{p}), \tag{2.9}$$

where we define

$$K(x, y) = -\frac{1 - xy}{(1 - x)(1 - y)} \tag{2.10}$$

With weak inequalities, (2.9) *stands for a necessary condition for stability of* $(\hat{p}, \hat{\nu})$. (*Note that with* (2.8a), *the right side of* (2.9) *is always larger than its left side.*)

(iii) *If* $\partial f/\partial p < 1$ (*i.e., if the system is* genetically stable), *then* $(\hat{p}, \hat{\nu})$ *is stable if and only if* $c'(\hat{p})$ *is sufficiently low. More specifically, it is stable if the right inequality of* (2.10) *is satisfied, and only if it is satisfied at least as a weak inequality.*

Proof. As is immediately implied from (2.5), the condition $\partial f/\partial p > 1$ of (i) and (ii) corresponds to a situation in which $\nu^{*\prime}(\hat{p}) > 0$. The condition $\partial f/\partial p < 1$ of (iii) corresponds to the opposite situation. In both cases,

$$\frac{\nu^{*\prime}(\hat{p})}{((\partial f/\partial p) - 1)(1 - (\partial\varphi/\partial\nu))} > 0.$$

Thus, (2.9) is obtained from (2.4) by multiplying all its terms by this positive value and inserting (2.7) into it. It is easy to verify now that (2.9) is void under condition (2.8a) and meaningful under condition (2.8b). Finally, if $\partial f/\partial p < 1$, the right inequality of (2.4) and, therefore, the left inequality of (2.9), automatically hold and this completes the proof of (iii).

Remarks. The right inequality of (2.9) simply guarantees that at the point $(\hat{p}, \hat{\nu})$, $c(p)$ will intersect $\nu^*(p)$ from below. Indeed, this is automatically guaranteed in case (iii), in which $\nu^*(p)$ decreases at $\hat{p}$. The right inequality puts some restriction on the *genetical susceptibility* of the ecological system near $(\hat{p}, \hat{\nu})$. The role of the ecological stability $1 - \partial\varphi/\partial\nu$ in the stabilization of $(\hat{p}, \hat{\nu})$ is quite remarkable because even though in the case $\nu^{*\prime}(\hat{p}) > 0$ of genetical instability, the right side of (2.9) is an increasing function of the ecological stability $1 - \partial\varphi/\partial\nu$; the opposite is true in the case $\nu^{*\prime}(\hat{p}) < 0$ of genetical stability. In this case the right side of (2.9) monotonically decreases from infinity to the value

$$\frac{\nu^{*\prime}(\hat{p})}{(\partial f/\partial p) - 1} = \left(\frac{\partial f}{\partial\varphi}\right)^{-1}$$

as $1 - \partial\varphi/\partial p$ increases from 0 to 1. We thus, quite surprisingly, obtain:

COROLLARY. (i) *If* $\nu^{*\prime} > 0$ (*i.e., if* $(\hat{p}, \hat{\nu})$ *is genetically unstable*) *then for any value* $c'(\hat{p})$ *of genetical susceptibility of the ecological system which is above* $\nu^{*\prime}(\hat{p})$ *there is a critical value of ecological stability above which* (*and only above which*) $(\hat{p}, \hat{\nu})$ *is stable.*

(ii) *If* $\nu^{*\prime}(\hat{p}) < 0$ (*i.e., if* $(\hat{p}, \hat{\nu})$ *is* genetically stable) *and if* $c'(\hat{p}) < (\partial f/\partial\nu)^{-1}$, *then* $(\hat{p}, \hat{\nu})$ *is always stable.*

(iii) *If* $\nu^{*\prime}(\hat{p}) < 0$ (*i.e., if* $(\hat{p}, \hat{\nu})$ *is* genetically stable) *and if the* genetical susceptibility *of the ecological system is high enough, i.e.,* $c'(\hat{p}) > (\partial f/\partial\varphi)$, *then* $(\hat{p}, \hat{\nu})$ is stable if and only if its ecological stability is sufficiently low.

At first sight it seems quite surprising that with genetical stability and a high genetical susceptibility of the ecological system near $(\hat{p}, \hat{\nu})$, ecogenetical stability of this point depends on *its ecological stability being sufficiently low* rather than vice versa, as is true in the case of genetical instability. Heuristically it may be understood, however, that if both the slope of $c(p)$ and the ecological stability are too high, relatively small deviations of (p, ν) to the right or to the left of $(\hat{p}, \hat{\nu})$ will result in too drastic deviations of (p', ν') to above or to below $(\hat{p}, \hat{\nu})$, respectively.

In order to study the behavior of the system when none of the fixed points is stable, let us separate all possible ecogenetical states (p, ν) into four regions:

$$\begin{aligned} A&: \quad \nu > c(p);\ \nu > \nu^*(p), \\ B&: \quad \nu^*(p) \geqslant \nu > c(p), \\ C&: \quad \nu \leqslant c(p);\ \nu < \nu^*(p), \\ D&: \quad c(p) < \nu \leqslant \nu^*(p). \end{aligned}$$

We now prove

PROPOSITION 7. *If* $c(p)$ *intersects* $\nu^*(p)$ *exactly once from below at a point* $(\hat{p}, \hat{\nu})$, *then for all* $0 < p < 1$ *and* $\nu > 0$, *the iteration* $T^n(p, \nu)$ *either tends to* $(\hat{p}, \hat{\nu})$ *as* $n \to \infty$ *or else it cycles around* $(\hat{p}, \hat{\nu})$, *passing infinitely many times through the regions* A, B, C, D *in this order* (*see Fig.* 4).

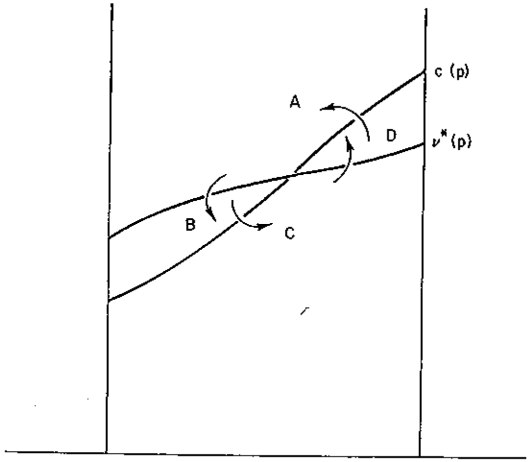

FIG. 4. The case of a permanent cycling. $c(p)$ intersects $\nu^*(p)$ in a too slope (see explanation in the text).

Proof. It is easy to verify that the regions A, B, C, and D have been defined in such a way that in each of them the transformation T is either monotone increasing or monotone decreasing in each of the two parameters p and ν. As has been assumed, T is also continuous. Thus, from monotonicity arguments it follows that for any point (p, ν) in one of these regions either there is a finite iteration $T^N(p, \nu)$ which lies outside the original region of (p, ν) or else, the sequence of iterations $T^n(p, \nu)$ approaches a fixed point which is in the closure of this region. But we know from the proof of Proposition 3 that for any interior point (p, ν), $T^n(p, \nu)$ cannot approach either $(0, c(0))$ or $(1, c(1))$. Hence, if $T^n(p, \nu)$ converges it should converge to $(\hat{p}, \hat{\nu})$.

Suppose $T^n(p, \nu)$ does not converge and let $(p, \nu) \in A$. In this case $\nu > c(p)$ and from the monotonicity of T we know that also $\nu' > c(p)$. But since $\nu > f(p)$, we have $p' < p$ and, therefore, $c(p') < c(p)$ and $\nu' > c(p')$. This means that $(p', \nu') \in A \cup B$ or $T: A \rightarrow A \cup B$. Thus, the first iteration $T^n(p, \nu)$ which is not in A should be in B. In the same way $T: B \rightarrow B \cup C$. $T: C \rightarrow C \cup B$ and $T: D \rightarrow D \cup A$, and this completes the proof.

Remark. Since $c(p)$ may intersect $\nu^*(p)$ more than once, the behavior of a general ecogenetical system with fitness selection in favor of one type (namely, the selfish) and group selection based on founder effect in favor of another type (namely, the altruist) may involve more complicated phenomena. Yet, since the empirical data now available about such systems is meager, it seems quite premature to go into further analysis of very specific situations. Instead it was the intention of the analysis being carried on throughout this section to study possible impacts of the simplest ecogenetical factors on the evolution of altruistic traits in a population repeatedly colonizing temporal habitats.

3. POSSIBLE IMPLICATIONS CONCERNING BACTERIA AND VIRUS POPULATIONS

This section concentrates on possible theoretical implications of the ecogenetical model studied above, concerning phenomena of altruistic efficiency, altruistic restraint, and host preservation among bacteria, viruses, and possibly other parasites (for biological references and discussion the reader is referred to Cohen and Eshel (1975) and Cohen (1975)). More specifically, by altruistic efficiency we mean the ability of an individual to increase its ratio of reproduction to consumption at the expense of its absolute rate of reproduction *when natural resources are shared by a large group of individuals of its own species.* Common resources are indeed typical to cultures of bacteria, wherein selection always seems to favor highly reproductive, nonefficient types.

By altruistic restraint we mean a restraint from overpolluting the common group environment rather than from overexhausting its resources. Host preservation is a special case of altruistic efficiency, altruistic restraint, or both.

A well-recorded example of host preservation has been given by the evolution of nonlethal mutation of the *Myxoma* virus in Australia, where it was introduced in order to reduce the level of the rabbit population (Fenner, 1965). In this case, rapid death of the host decreased the probability of further infection. In other, less drastic, cases it is the antibody production of the host, elicited by its nonrestrained parasite, which endangers the entire parasite colony. Note, however, that the necessity for self-restraint of the parasite population does not automatically guarantee a mechanism for its selection within this population. On the contrary, local disadvantage of the self-restrained type, when directly competing with nonrestrained neighbors, makes its natural selection possible only under quite restrictive conditions.

Quite generally, bacteria and virus populations are far too large and local random drift within each of them is too small to provide interdeme variance which is sufficient for group selection among them (e.g., see Williams, 1966; Wilson, 1974a; Eshel, 1972). Thus, the random component of the founder effect was suggested as the main cause for the evolution of altruistic traits within such populations (Cohen and Eshel, 1975). In this case, low density of the population proved crucial for the evolution of any altruistic traits. On the other hand, it appears that in many cases the very density of the population is, in turn, affected by the establishment of the altruistic trait within it. It has been the aim of this work to analyze mutual effects of the density of a population and the frequency of an altruistic trait within it.

One result of the analysis of the previous section concerns the effect of changes in the carrying capacity function of the population on the fate of the altruist within this population. An increase in the carrying capacity function of a population is naturally interpreted as an improvement in the environmental conditions. In the case of a parasite population, such an increase may occur in two different ways:

(i) by an increase in the number or density of available habitats (hosts);

(ii) by an improvement of the environment for the parasite within each habitat.

In the first case, $f(p, \nu)$ and, therefore, $\nu^*(p)$ are not likely to change since they are determined by the selection forces within each habitat and by the two-dimensional Poisson distribution of founding groups among habitats which is, in turn, uniquely determined by p and ν (see Cohen and Eshel, 1975). On the other hand, the new carrying capacity of the parasite population is apt to increase proportionally to the density of the host population.

The second case, involving more than one parameter, is more complicated to analyze generally, but for most applications its effect is likely to be very much the same as that of the first one.

An obvious result of the two cases is, indeed, an immediate increase in the number of hosts being infected. But, as predicted by Propositions 2 and 3, a possibly more serious long-run effect of crowdedness and lack of sanitation may be due to a selection force being imposed on the parasite population in favor of more harmful types within it. Actually, fixation of the harmful parasitic type may, in turn, cause a relative decrease in the total frequency of infections, possibly (though not necessarily) to a level which is even below the one being kept by the less harmful parasite within the less crowded situation.

Other results of the analysis determine conditions for either a stable polymorphism of the altruist and the selfish type, selection for the majority (depending also on the initial density), or a permanent ecogenetical cycling.

Periodic erruptions of many parasitic bacteria and virus populations (generally not fully predictable because of mutation effects, e.g., see Kessler (1970)) may well be explained on the basis of the model suggested here: In a dense parasite population, infection is often multiclonal with an intrahost selection toward fast growing. As a result, more harmful mutations are selected and infected hosts are either killed (in the case of a lethal disease) or stimulated to develop antibodies to the disadvantage of the parasite population. At this stage the parasite, still mainly in its harmful version, becomes more and more scarce. Infections, in the rare cases of their occurrence, are quite generally monoclonal with an advantage given to the more restrained, host-preserving clones. It is in this stage that selection operates in favor of non harmful (or less harmful) mutation and the disease in its harmful form disappears. Yet relative tolerance of the host toward the nonharmful parasite enables the parasite population to increase in density up to a level in which natural selection again favors the less-restrained mutant, with the result of a new ecogenetical cycle starting.

Note that the mechanism of founder effect selection which is suggested here favors fast-growing types only when the population density is already dense. In this sense, founder effect operates in a direction opposite to a mechanism of density regulation and may, thus, not be advantageous to the population as a whole. But since, under conditions of low density, this mechanism always operates in favor of the altruist, it cannot possibly lead to a deterministic extinction of the population.

Finally, a situation in which one type of behavior (i.e., that of a selfish fast reproduction) is selected in one situation, whereas the alternative behavior (i.e., that of an altruist self-restraint) is selected in the alternative situation, is likely to enable evolution of a globally advantageous discriminative altruism (for comparison, see Eshel and Cohen, 1975).

Thus, at least with ecogenetical conditions, under which a permanent cycling of an altruist–selfish population is predicted, there will be a global advantage to a third, discriminative type, which is characterized by the following traits:

(i) It is able to detect the existence of a dense, competing, parasite population in its vicinity.

(ii) With the absence of strong competition it stays in a self-restrained, host-preserving form.

(iii) Above a critical density of competing neighbors it switches to a fast-growing, harmful form and vice versa if the density of competing neighbors falls below that critical density.

The third requirement for the reproductive behavior of the discriminative type is likely to determine an almost simultaneous chain reaction switch of all individuals in the parasite population from one form to another. The evolutionary explanation being suggested here for this empirically well-known phenomena (e.g., see Kessler, 1970) is, indeed, speculative. Yet, it is suggested that the extensive study of biochemical signals stimulating virulent reaction in latent forms of viruses and bacteria can, possibly, be helped by a theoretical analysis of this hypothetical situation.

ACKNOWLEDGMENT

I am grateful to Dr. Uri Lieberman, to Dr. Nira Richter-Dyn, and especially to Professor Marcus W. Feldman for many helpful remarks on the manuscript.

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Printed by the St Catherine Press Ltd., Tempelhof 37, Bruges, Belgium.