

# A species near its equilibrium size in a fluctuating environment can evolve a lower intrinsic rate of increase

(density-dependent selection/evolutionary ecology/stochastic environments)

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**ABSTRACT** In one specific model of a density-regulated population undergoing natural selection in a fluctuating environment there is a systematic evolutionary pressure favoring a lower intrinsic rate of increase, which can sometimes even overcome an evolutionary pressure favoring a higher carrying capacity.

Mathematical models of evolving populations that are set in an ecological context have been the subject of increasing attention (1-19). These models include both population sizes and gene frequencies as variables and thus forge a link between the classical disciplines of population ecology and population genetics. These models have largely assumed that environmental conditions remain constant in time. Results in purely population genetic models have been obtained when the selective coefficients are permitted to fluctuate (20-24). In this report we extend this work to explore the evolution of the pattern of population growth for a special choice of a population dynamic model.

The model we consider here describes the growth of a population and evolution within it, combining the ingredients of density-dependent population regulation, genetic variability for growth parameters, and fluctuating resource levels. Consider a population (of size  $N_t$  at time  $t$ ) of diploids segregating two alleles ( $A, a$ ) at a single locus. We denote the frequency of  $A$  at time  $t$  by  $p_t$  and let  $1 - p_t = q_t$  be the frequency of  $a$ . We assume the population has nonoverlapping generations, each of which arises from random union of gametes that were shed into a common gamete pool by the surviving members of the parent generation. Thus, the zygotic genotype frequencies are in the Hardy-Weinberg proportions  $AA:Aa:aa::p_t^2:2p_tq_t:q_t^2$ . The contribution of each individual to the next generation depends on its genotype and on the current population size and resource level. We assume that the genotype-specific per capita contributions to the next generation are given by

$$\begin{aligned} W_{AA,t}(N_t) &= 1 - r_{AA} + \frac{r_{AA}K_{AA,t}}{N_t} \\ W_{Aa,t}(N_t) &= 1 - r_{Aa} + \frac{r_{Aa}K_{Aa,t}}{N_t} \\ W_{aa,t}(N_t) &= 1 - r_{aa} + \frac{r_{aa}K_{aa,t}}{N_t} \end{aligned} \quad [1]$$

in which  $r_{ij}$  is in  $(0,1)$  and  $K_{ij,t} > 0$ . The fitnesses are strictly positive and are convex decreasing functions of the population size.  $K_{ij,t}$  and  $r_{ij}$  are analogous, respectively, to the carrying capacity at time  $t$  and to the intrinsic rate of increase of the logistic model in ecology, as noted in more detail below. To find the population size in generation  $t + 1$ , we add the total con-

tributions of the three genotypes to obtain

$$N_{t+1} = N_t \cdot \bar{W}_t(N_t, p_t), \quad [2a]$$

in which  $\bar{W}_t(N_t, p_t) = p_t^2 W_{AA,t}(N_t) + 2p_tq_t W_{Aa,t}(N_t) + q_t^2 W_{aa,t}(N_t)$ . Provided  $N_t$  is initially positive, it remains so. The frequency of allele  $A$  at time  $t + 1$  is given by the standard formula for natural selection at one locus (25),

$$p_{t+1} = p_t [p_t W_{AA,t}(N_t) + q_t W_{Aa,t}(N_t)] / \bar{W}_t(N_t, p_t). \quad [2b]$$

The functional form (Eq. 1) of the absolute fitnesses is related to a familiar discrete-time logistic growth law,

$$N_{t+1} = N_t [1 + r - (rN_t/K_t)] = f(K_t, N_t).$$

In the logistic model, the parameter  $K_t$  is called the carrying capacity at time  $t$  and denotes the amount of resources then present, measured in units of the number of individuals those resources could support. The parameter  $r$  is called the intrinsic rate of increase. In the logistic model,  $r$  is approximately the geometric growth rate at low population sizes; but also when  $K_t = K$  for all times  $t$ ,  $(1 - r)$  is the asymptotic geometric rate of approach of  $N_t$  to the equilibrium  $K$ . Suppose that in the vicinity of the point  $N_t = K_t = \bar{K}$  (the mean value of  $K_t$ ), the function  $f$  is approximated by the constant and linear terms of its Taylor expansion:

$$f(K_t, N_t) \cong N_t [1 - r + (rK_t/N_t)] = g(K_t, N_t).$$

The per capita contribution to the next generation with the function  $g$  is the same form as the fitnesses (Eq. 1). The "linear" growth law

$$N_{t+1} = g(K_t, N_t)$$

and the logistic growth law are quite similar in the trajectories they produce in the region near  $\bar{K}$  (26). In particular,  $(1 - r)$  in the linear growth law is the asymptotic geometric rate of approach of  $N_t$  to an equilibrium when  $K$  is constant in time, just as in the logistic model. However, the  $r$  in the linear growth law is not related to the geometric growth rate at low population sizes in the same manner as in the logistic model. With the linear growth model,  $N_t$  can be expressed as an average of all the past carrying capacities the population has experienced:

$$N_t = \sum_{i=1}^{\infty} r(1-r)^{t-i} K_{t-i}. \quad [3]$$

In this expression, it is assumed that the population was initiated at  $t = -\infty$  so that  $N_t$  will be viewed as exhibiting the stationary distribution determined by the  $K_t$  process. We stress that our model is defined by 1 itself; the relationship of this model to the various possible formulations of the logistic equation in discrete time is complicated and is sensitive to the details of both the formulation and its analysis.

If the carrying capacities are constant in time in Eq. 2, then the qualitative outcome of evolution at the  $A-a$  locus depends

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Table 1. Equilibria and local stability for the model (Eq. 2) when  $K_{AA,t} = K_{AA}$ ,  $K_{Aa,t} = K_{Aa}$ , and  $K_{aa,t} = K_{aa}$  for all  $t$

Condition on $K$ s	Equilibria		
	Fixations		Polymorphism
	$\hat{p} = 0, \hat{N} = K_{aa}$	$\hat{p} = 1, \hat{N} = K_{AA}$	$\hat{p} = p^*$ in $(0,1)$ , $\hat{N} = N^*$
Case 1: $K_{AA} \geq K_{Aa} \geq K_{aa}$ †	Unstable	Stable	‡
Case 2: $K_{AA} \leq K_{Aa} \leq K_{aa}$ †	Stable	Unstable	‡
Case 3: $K_{AA} < K_{Aa} > K_{aa}$	Unstable	Unstable	Stable
Case 4: $K_{AA} > K_{Aa} < K_{aa}$	Stable	Stable	Unstable
Case 5: $K_{AA} = K_{Aa} = K_{aa}$	Neutral curve of equilibria		

† Except for case 5.

‡ The polymorphic equilibrium does not exist for these parameter values.

solely on the ordering relationships among the  $K$ s (see Table 1) (2). The ordering relationships among the  $r$ s do not affect the qualitative conclusions of existence and stability of equilibria although they do affect the position of the polymorphic equilibrium and the shapes of the trajectories before the stable equilibria are attained. We call attention to case 5 in Table 1:  $K_{AA} = K_{Aa} = K_{aa} \equiv K$ . If this condition holds, then any value of the gene frequency  $p$  is an equilibrium so long as  $N = K$ . That is, instead of the two or three equilibria of Table 1 existing in  $(N,p)$ -space, there is a curve of neutrally stable equilibria. The existence of this curve is independent of the values of  $r$  although they affect the shapes of the trajectories leading to the curve.

To motivate the criterion for instability that we will introduce when the carrying capacities fluctuate according to a stochastic process, we exhibit the situation where the fluctuations in  $K$  (and hence the fluctuations in the density-dependent fitness functions) follow a deterministic, periodic sequence with period  $T$ . We are interested in the condition under which a rare allele,  $a$ , will increase. Suppose that for season  $i$  ( $i = 1, \dots, T$ ), we have

$$p' = f_i(p, N)$$

$$N' = g_i(p, N),$$

in which the primes denote the values after the transformation has operated. If, for example,  $T = 2$ , the transformation that projects  $p$  and  $N$  across a full cycle of seasons is

$$p' = f_2(f_1(p, N), g_1(p, N))$$

$$N' = g_2(f_1(p, N), g_1(p, N)).$$

An equilibrium  $(\hat{p}, \hat{N})$  censused at the end of the cycle will be locally stable if the Jacobian matrix of the composite transformation has eigenvalues in the unit circle and unstable if at least one eigenvalue lies outside the unit circle. If the transformation across each season type is of the form 2 with a density-dependent fitness function that is specific to each season type, then the Jacobian matrix is easily found to be triangular when evaluated at  $\hat{p} = 1$  and  $N = \hat{N}$ . For example, with  $T = 2$  we have

$$\begin{pmatrix} \frac{\partial p'}{\partial p} & \frac{\partial p'}{\partial N} \\ \frac{\partial N'}{\partial p} & \frac{\partial N'}{\partial N} \end{pmatrix} = \begin{pmatrix} \frac{\partial f_2}{\partial(f_1)} \frac{\partial f_1}{\partial p} & 0 \\ \cdot & \frac{\partial g_2}{\partial(g_1)} \frac{\partial g_1}{\partial N} \end{pmatrix},$$

in which the “ $\cdot$ ” indicates a term that is generally nonzero. In-

deed, the triangular matrix that pertains to  $T$  generations is the product of  $T$  triangular matrices. Because the matrix is triangular, the eigenvalues are given by the diagonal terms. Furthermore, the eigenvector corresponding to the eigenvalue that appears in the lower right of the main diagonal is parallel to the  $N$  axis (i.e., lies in the  $p = 1$  boundary itself). The magnitude of this eigenvalue is determined solely by the population dynamic equations for the  $AA$  homozygote. The other eigenvector, that corresponding to the element in the top left, is the one of principal evolutionary importance; it points to the interior of  $(p, N)$  space. If its eigenvalue exceeds 1, then the rare allele is certain to increase. It is easy to compute that the magnitude of this eigenvalue will exceed 1 if the geometric mean of the ratio of the homozygote to the heterozygote fitnesses taken over the cycle is less than 1; that is,

$$\sqrt[T]{\prod_{i=1}^T \frac{W_{AA,t}}{W_{Aa,t}}} < 1$$

or equivalently,

$$\frac{1}{T} \sum_{i=1}^T \log \frac{W_{AA,t}}{W_{Aa,t}} < 0. \tag{4}$$

The fitness ratios are evaluated, for each  $i$  within the full cycle of length  $T$ , at the corresponding  $\hat{N}_i$  assuming that  $p = 1$ . This criterion is seen to be an extension of the Haldane–Jayakar condition (20). The condition is sufficient for instability. To ascertain stability, the magnitude of the other eigenvalue, the one pertaining to the purely ecological process for the  $AA$  homozygote, would also have to satisfy certain conditions.

We now return to the stochastic form of the model (Eq. 2) when the  $K$ s are allowed to fluctuate in time. The result we wish to report is that now the ordering relationships among the  $r$ s do influence and may completely control the qualitative outcome of density-dependent selection. One striking result is the evolution of a lower  $r$  by a  $K$ -selected species in a fluctuating environment in this model.

We assume the fluctuating  $K$ s are of the form

$$K_{AA,t} = \bar{K}_{AA} + k_t; K_{Aa,t} = \bar{K}_{Aa} + k_t; K_{aa,t} = \bar{K}_{aa} + k_t,$$

in which  $k_t$  is an ergodic bounded random variable having zero expectation. The bounds on  $k_t$  are chosen to ensure that the  $K$ s are positive and that the  $W$ s are bounded away from zero and infinity when  $N_t$  is bounded away from zero. If we consider a population whose size has been varying randomly but that has been confined to the boundary at  $p = 1$ , from Eq. 3 we know that its size is given by the random variable  $N_t$  in

$$N_t = \sum_{i=1}^{\infty} r_{AA} (1 - r_{AA})^{i-1} (\bar{K}_{AA} + k_{t-i}). \tag{5}$$

In the bivariate process, where  $p$  is not confined exactly to equal 1, there exist realizations with  $p$  sufficiently near 1 so that  $N_t$

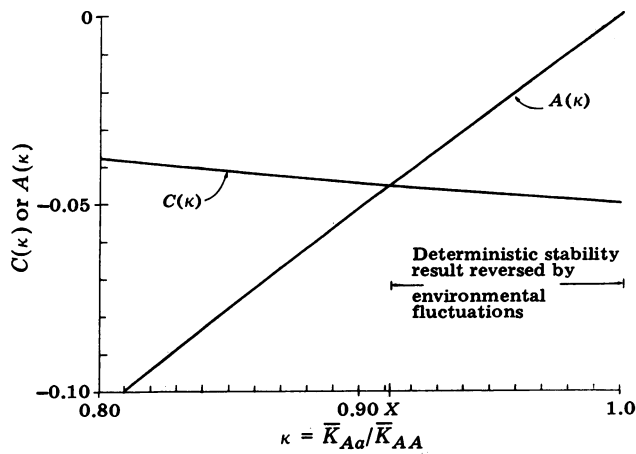


FIG. 1. Relative magnitudes of the functions  $A$  and  $C$  determine the sign of Eq. 7. The point of intersection  $X$  occurs to the left of 1 if  $r_{AA} > r_{Aa}$ . To the left of  $X$ ,  $C$  is greater than  $A$  and the expression in Eq. 7 is positive. To the right of  $X$ , Eq. 7 is negative and  $\hat{p} = 1$  is unstable in a stochastic environment. In the segment between  $X$  and 1,  $\hat{p} = 1$  would be stable in a constant environment because  $K_{AA} > K_{Aa}$ . Hence, in the interval between  $X$  and 1 the deterministic stability result is reversed by the environmental fluctuations.  $r_{AA} = 1$ ;  $r_{Aa} = 0.5$ ;  $\sigma_k^2 / \bar{K}_{AA}^2 = 0.1$ .

is approximately given by 5. But no such realization (with  $N_t$  sufficiently near 5) can actually converge to  $p = 1$  if

$$E \log \frac{W_{AA}}{W_{Aa}} < 0. \quad [6]$$

This follows from the fact that if  $p = 1$ , then by the strong law of large numbers.

$$\Pr \left\{ \lim_{T \rightarrow \infty} \frac{1}{T} \sum_{t=1}^T \log \frac{W_{AA,t}}{W_{Aa,t}} = E \log \frac{W_{AA}}{W_{Aa}} \right\} = 1.$$

Hence 6 implies that 4 holds in the limit as  $T \rightarrow \infty$ . Karlin and Liberman (24) showed that 6 is sufficient to guarantee that  $\Pr\{\lim_{t \rightarrow \infty} p_t = 1\} = 0$  for a univariate gene frequency process. In the bivariate process, 6 is a sufficient condition for instability of a marginal distribution for  $N$  and  $p$  that is concentrated on the boundary of  $p = 1$  in the sense that, if 6 is satisfied, then the probability of the process converging to the boundary  $p = 1$  is zero. The proof of this statement for fitnesses of the form 1 is similar to Karlin and Liberman's proof for the univariate process.

We now investigate how the size of the left-hand side of Eq. 6 depends on the parameters of the model when the carrying capacity fluctuations,  $k_t$ , are small by examining constant, linear, and quadratic terms in the Taylor expansion about zero. For ease of exposition, we now assume that the  $k_t$  are identically and independently distributed (but we relax this assumption later). Hence,

$$\begin{aligned} E \log \frac{W_{AA}}{W_{Aa}} &\approx -\log(1 - r_{Aa} + r_{Aa}\kappa) + \frac{\sigma_k^2}{\bar{K}_{AA}^2} \frac{r_{Aa}}{(2 - r_{AA})} \\ &\times \frac{-r_{AA}r_{Aa}\kappa^2 - 2r_{AA}(1 - r_{Aa})\kappa + r_{Aa}(2 - r_{AA})}{(1 - r_{Aa} + r_{Aa}\kappa)^2} \\ &= -A(\kappa) + C(\kappa), \end{aligned} \quad [7]$$

in which  $\kappa = \bar{K}_{Aa} / \bar{K}_{AA}$ ,  $\sigma_k^2 = \text{Var}(k_t)$ , and terms of  $o(\|k_t\|^2)$  are neglected.

By way of comparison with the deterministic model, consider first the case  $\bar{K}_{AA} = \bar{K}_{Aa} = \bar{K}_{aa}$ . If the  $k_t$  have nonzero variance, there is no longer any analogue to a neutral curve of gene-frequency equilibria. Indeed, the  $r$ s now determine whether the fixation states are unstable, unlike the constant-environment

case. The sign of Eq. 7 is always the same as the sign of the quantity  $(r_{Aa} - r_{AA})$ , and so the equilibrium  $\hat{p} = 1$  is unstable if  $r_{Aa} < r_{AA}$ . Thus a major qualitative difference between evolution in a constant environment and evolution in a fluctuating environment is that  $r$  is not generally selectively neutral. In this model, natural selection favors a lower  $r$ . Furthermore, we have established that the presence of autocorrelation in carrying capacities does not affect our results for this model.

Expanding our discussion to the general case in which the genotypes can have different  $K$ s, we find that the outcome of evolution is dependent on both the  $r$ s and the  $\bar{K}$ s. Fig. 1 illustrates how the two components  $A$  and  $C$  affect the sign of Eq. 7 and the instability of the fixation state. Considered as functions of  $\kappa$ , the curves  $C$  and  $A$  are monotonic in the region of interest (decreasing and increasing, respectively) and intersect at a single point  $\kappa = X$ . The segment between  $X$  and 1 consists of those  $\kappa$  values for which the presence of environmental fluctuations reverses the stability result found in the deterministic model. That is, for  $\kappa$  between  $X$  and 1,  $\hat{p} = 1$  is unstable despite the fact that  $\bar{K}_{AA} > \bar{K}_{Aa}$  (see cases 1 and 4 in Table 1). Furthermore, we have established that increasing the autocorrelation in carrying capacities tends to decrease the width of the interval between  $X$  and 1. Thus, in this model the advantage to a lower  $r$  in a stochastic environment may offset the disadvantage of a lower  $K$ .

We have not addressed questions of the global behavior of Eq. 2, having concentrated on questions of gene-frequency fixation instability. Keeping in mind, therefore, the limited information of the whole interior picture that knowledge of the boundary behavior can give, we conclude by mentioning a major implication of our result for the theory of  $r$  and  $K$  selection. Many authors (27-31) have invoked energy allocation arguments to explain why selection for "high- $K$ " traits precludes simultaneous evolution of "high- $r$ " traits. Our result—that in this model there is an inherent selection pressure in a stochastic environment in favor of a lower  $r$ —provides an evolutionary mechanism for the appearance of an  $r$ - $K$  tradeoff at one locus, which does not invoke an energetic constraint between  $r$  and  $K$ .

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