

## Ecogenetic Models, Competition, and Heteropatry

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We develop a system of equations to analyse the existence of genetic polymorphisms under disruptive selection in heterogeneous environments. These equations have both a genetic and a population density regulation component. In the absence of the genetic component, the equations reduce to a discrete time description of competition between interacting clonal lines or species. We use these equations to demonstrate that different populations, competing along a resource spectrum, are able to dynamically coexist, as asymptotically periodic or chaotic solutions to our system of equations, despite the fact that a coexistence equilibrium—stable or unstable—does not exist. We then extend these results to environments in which several niches are explicitly defined. Our analysis of the ecological component of our model establishes that the answer to questions of coexistence among groups of individuals cannot rely on analyses of the existence of equilibria and their stability properties.

In the most general model presented here, we allow for an assortative mating structure that is induced by the spatial heterogeneity of the environment. The level of assortative mating is controlled by a parameter so that at one extreme mating is panmictic, while at the other extreme individuals mate within their natal niches before dispersing to oviposit in other niches. We refer to this spatial mating structure as heteropatry.

We investigate, through numerical studies, the properties of a heteropatric model containing both ecological and genetic components. First we address the question of the existence of protected genetic polymorphisms (i.e., the different alleles at a particular locus all increase in frequency when rare) under a wide range of model parameter values in a diallelic one-locus version of our model, assuming panmixis and partial dominance selection of varying direction. We make the point that establishing the instability of monomorphic population equilibria is insufficient to guarantee the existence of a protected polymorphism, since the instability may be in the population size component rather than the genetic frequency component of the monomorphic equilibrium solution. The results indicate that density

dependence serves to decrease the likelihood that a protected polymorphism exists, while the degree of selection of natal habitat for oviposition purposes serves to increase this likelihood. The situation is complicated, though, since conditions which promote the existence of protected polymorphisms may actually reduce the possibility that a stable polymorphism exists. Further, the way we introduce density dependence (either by scaling the population interaction coefficients or by altering the shape of the response function) differentially affects the results. Finally, our results suggest that if individuals prefer to oviposit in their best niche (where they are most fit), rather than their natal niche (where they mature), then allele fixation very probably occurs. © 1989 Academic Press, Inc.

## 1. INTRODUCTION

Models combining ecological and genetic components (hereafter referred to as ecogenetic models) have been used to establish that stable and/or protected genetic polymorphism<sup>1</sup> can exist under disruptive selection in density regulated sympatric populations exploiting heterogeneous environments. Levene (1953) was the first to use an ecogenetic model to demonstrate that heterosis (viz., heterozygote  $Aa$  is fitter than either homozygote  $AA$  or  $aa$  in a diallelic one-locus system) is not required for the existence of a stable polymorphism. Levene demonstrated that it is possible for all genotypes ( $AA$ ,  $Aa$ , and  $aa$ ) to coexist when the environment is heterogeneous and each homozygote outcompetes the other two genotypes in one of the available niches. Several studies have used Levene's approach to obtain sharper results, and have extended it to include one or more of the following: migration between niches; temporal heterogeneity; some level of habitat preference by females; and assortative mating under the control of Mendelian genes (Deakin, 1966; Levins and MacArthur, 1966; Maynard Smith, 1966, 1970; Prout, 1968; Ewing, 1979; Felsenstein, 1981; Rausher, 1984; Hoekstra, Bijlsma, and Dolman, 1985; for a review see Hedrick, Ginevan, and Ewing, 1976). Models of the Levene genre are ecologically deficient because niche size is used as a surrogate for a true intra-niche density-dependent fitness or survival response.

The main purpose of our paper, however, is to place ecogenetic analyses of polymorphisms in spatially heterogeneous environments on a sound ecological footing, and to dispel the idea that an equilibrium analysis is sufficient for addressing questions relating to the existence of stable and/or protected genetic polymorphisms. Our results demonstrate that genotypes may coexist in the absence of both stable and unstable polymorphic equilibria, and that chaotic coexistence of genotypes is possible. We also introduce the notion that assortative mating may be induced in a sympatric

<sup>1</sup> A system or model has a protected polymorphism if, for example, alleles  $A$  and  $a$  at a diallelic locus both increase when rare.

population solely by the heterogeneous structure of the environment. We refer to this phenomenon as heteropatry. As expected, we are able to demonstrate that this spatially induced assortative mating extends the range of conditions under which polymorphisms can exist. More importantly, however, it emerges from our analysis (presented below) that conditions which ensure the local stability of a polymorphic equilibrium may be out of consonance with those that ensure the existence of a protected polymorphism.

In contrast to Levene (1953), Dempster (1955) developed a model in which he allowed differential selection to take place between niches as a consequence of selective forces within niches: an approach that has been extended to a density-dependent setting by Arnold and Anderson (1983). In Dempster's model the proportions of individuals that enter each niche before selection acts are *a priori* fixed. Christiansen (1975), however, demonstrated that Dempster's model is a linear transformation of Levene's model: the transformation matrix is constructed from the intra-niche selection coefficients.

Clarke (1972) was one of the first to rigorously present an intra-niche density-dependent competition analysis in which he assumed that a concave saturating function (Holling type II response function) of a weighted function of the combined densities of the different genotypes controlled fitness. This allowed him to obtain conditions under which a rare allele is able to increase its representation in the population. His model did not explicitly define the niche structure in the environment; rather, the niches were implicitly defined through the competitive interaction functions which he used. The existence of polymorphisms came about because one of the genotypes (phenotypes when one of the alleles is assumed dominant) was fittest at low densities and the other was fittest at high densities. In the case of three phenotypes (a diallelic one-locus model) the heterozygote was assumed intermediate between the two homozygotes at both low and high densities. Maynard Smith and Hoekstra (1980) embedded the density-dependent approach of Clarke (1972) in an explicit multi-niche environment. They conducted an insightful comparative analysis between their approach and the models of Levene, Clarke, and others. Also, Maynard Smith and Hoekstra (1980) demonstrated that polymorphisms can exist under much weaker conditions than suggested by analyses of the Levene genre of models (cf. Hoekstra *et al.*, 1985; and also see Gillespie, 1976).

Asmussen (1983a, b) analysed density-dependent selection in an intra-specific competition setting for both haploid and diploid genetic systems. She concludes that "...genetic polymorphism is greatly increased when intraspecific competition is incorporated within a density-regulated framework..." The analysis we present only confirms this to be true in the context of stable genetic polymorphisms. In fact the opposite is true in the

context of protected genetic polymorphisms, which once again stresses the need to keep the two concepts of polymorphism separate.

Polymorphism studies have also been undertaken that explicitly include competition through Lotka–Volterra type dynamics (Arnold and Anderson, 1983) and MacArthur’s (1972) resource–consumer type dynamics (Wilson and Turelli, 1986). In fact, Wilson and Turelli (1986) demonstrated that a lack of competition allows maladapted genotypes to invade ‘empty niches’ so that it is possible for stable polymorphisms to exist in which the most heterozygous individuals are in fact the least fit at equilibrium.

All the above studies assumed that mating in the population as a whole either is random or, if assortative, is explicitly under the control of Mendelian genes (except for Hoekstra *et al.* (1985) who also analysed the case where mating occurs before any of the adults move out of their natal niches). Here we assume that assortative mating can be induced in a sympatric population occupying a heterogeneous environment, if the scale of heterogeneity is small relative to typical mating dispersal distances. Dickenson and Antonovic’s (1973) analysis of pollen-borne gene flow between different plant genotypes in two niches satisfies these assumptions. Their model, like Levene’s (1953), however, lacks an adequate density–dependent component.

The theory we present here is developed around a ‘model’ organism whose immature stages develop on single host species, where each host species constitutes an ecological niche. Unlike Dickinson and Antonovic’s (1973) pollen transfer model, we allow the mature stages to disperse and deposit eggs or give birth to individuals on the same or other host species, under the control of a preference parameter. This model is applicable, for example, to polyphagous insects feeding on several host species (Futuyma, 1986). As a function of spatial structure, we assume that adults are more likely to mate with individuals from the same than from different host species. In addition, further assortative mating may be under the control of genes influencing mate selection.

We begin by outlining the general structure of our model. We then present, in some detail, the ecological component of the model. This is necessary because discrete time models of competition have received little attention in the literature. Finally we present simulation results obtained using a complete ecogenetic model.

## 2. ECOGENETIC MODEL

We develop a general model by letting  $N_{ij}(t)$  denote the number of individuals of genotype  $i$ ,  $i = 1, \dots, n$ , that are born or hatch at time  $t$  in

niche  $j$ ,  $j = 1, \dots, m$ . Later we use the more informative, but less general, notation exemplified by  $N_{Aa2}$ , which denotes the number of individuals of genotype  $Aa$  in niche 2. For convenience, we will refer to a set of individuals of type  $ij$  as a phenotype; that is, we have  $n \times m$  distinct phenotypes each consisting of  $N_{ij}(t)$  individuals in our population at time  $t$ .

The ecological phase of the process will be the maturation of each genotype, in a niche, from a newly hatched or newborn individual at time  $t$  to a sexually mature adult at time  $t + t'$ , where  $0 < t' < 1$ . The number of individuals maturing in niche  $j$  is assumed to depend on a proportional survival rate  $s_{ij}(\cdot) \in [0, 1]$ , where the functional argument of  $s_{ij}(\cdot)$  is assumed to be the weighted sum

$$N_j^w(t) = \sum_{i=1}^n \alpha_{ij}^l N_{ij}(t), \quad j = 1, \dots, m, \quad (1)$$

of individuals in niche  $j$ . Note that the weighting parameters  $\alpha_{ij}^l$  can be interpreted as interaction coefficients since they represent the relative impact individuals of each phenotype have on one another, where the index  $j$  relates to explicit niche competition, the index  $l$  relates to implicit niche competition, while the index  $i$  relates to the genotype's being influenced by the density of the weighted population  $N_j^w$  in niche  $j$ .<sup>2</sup> In selecting values  $\alpha_{ij}$ , it is important to consider the fact that density-dependent effects are influenced by the number of individuals of each phenotype which survive through to maturation. Thus, if individuals of a particular genotype are unable to feed on a specific host, their initial presence in the niche should have a minimal effect on the development of the other genotypes in that niche.

From the above it follows that the number of individuals of each phenotype that mature is

$$N_{ij}(t + t') = s_{ij}(N_j^w(t)) N_{ij}(t). \quad (2)$$

For purposes of exposition we will think of each niche as consisting of a number of patches, where the patches themselves are randomly distributed throughout some region of space (Fig. 1). After individuals mature in a niche, they begin around time  $t'$  a process of dispersal and mating. If we assume that a proportion  $(1 - \delta_{ij})$  of individuals  $N_{ij}(t + t')$  mate within their patch, while a proportion  $\delta_{ij}$  mate outside of their patch, then (assuming random mating within patches and between those that

<sup>2</sup> We can also view explicit and implicit niche competition respectively as inter- and intra-habitat competition. In this case, we cannot equate habitat with niche, since implicit niche competition that supports competitive coexistence is equivalent to a several niche structure in a single habitat.

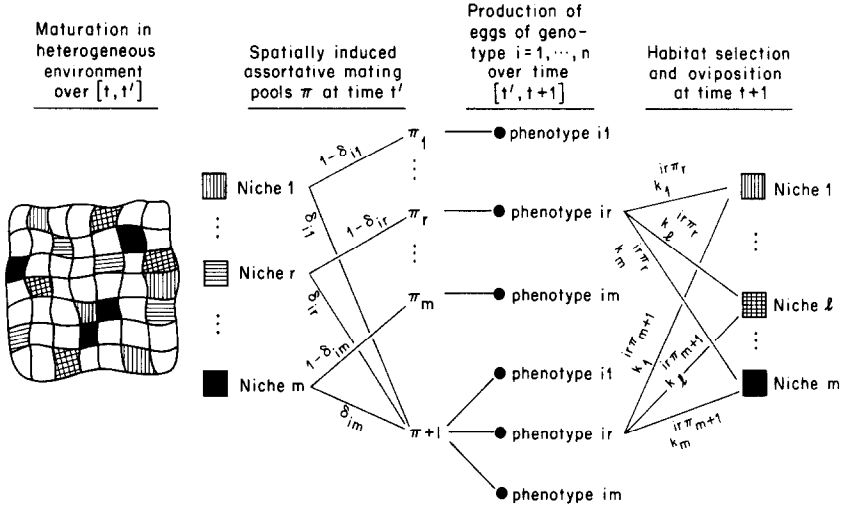


FIG. 1. Schematic outline for the construction of a model of genotypes competing in a heterogeneous multi-niche environment. See text for details.

disperse out of patches) we can represent this process by a set of random mating pools  $\pi_j, j = 1, 2, \dots, m + 1$ , as illustrated in Fig. 1. Thus the number of individuals of phenotype  $ij$  in each mating pool is for genotype  $i = 1, \dots, n$

$$\begin{aligned} \text{mating pool } \pi_j: & N_{ij}^{\pi_j}(t + t') = (1 - \delta_{ij}) N_{ij}(t + t') \quad j = 1, \dots, m, \\ \text{mating pool } \pi_{m+1}: & N_{ij}^{\pi_{m+1}}(t + t') = \delta_{ij} N_{ij}(t + t') \quad j = 1, \dots, m, \end{aligned} \tag{3}$$

where  $0 \leq \delta_{ij} \leq 1$  for  $i = 1, \dots, n$  and  $j = 1, \dots, m$ . Note that mating pools  $\pi_j, j = 1, \dots, m$  have individuals from niche  $j$ , while mating pool  $\pi_{m+1}$  has individuals from all niches. From the structure of these mating pools it is evident that  $\delta_{ij} = 0$ , for all  $i$  and  $j$ , corresponds to the case where mating is wholly within the particular niche in which each individual developed; while  $\delta_{ij} = 1$ , for all  $i, j$ , corresponds to panmixis in the population as a whole. Our notion of heteropatry corresponds to the assumption that  $\delta_{ij} < 1$ , even when mate selection behavior has no genetic component at all. Of course more complicated mating pool structures can emerge if spatial associations between niches are themselves non-random and/or there is a genetic component to mate selection.

The individuals in these mating pools produce zygotes that depend on both their own genotype and the genotypes of the other individuals in the mating pool. Since we have assumed that mating within each pool is panmictic, the genotype frequencies of the progeny are easily calculated. For purposes of this calculation, it is convenient to introduce an intermediate variable  $E_{\mu}^{ij\pi_r}(t + 1)$  to represent the number of eggs of genotype  $\mu$  that are

produced by individuals of phenotype  $ij$  in mating pool  $\pi_r$ ,  $r = 1, \dots, m + 1$ . To calculate  $E_\mu^{ij\pi_r}(t + 1)$  we assume that each adult of phenotype  $ij$  produces an average of  $b_{ij}$  eggs (average taken over both sexes where appropriate). The genotypes of the eggs can be calculated only if the genotypes of the parents are known. Thus, the details of this calculation can only be given for specific cases, as discussed in the diallelic one-locus two-niche model presented below.

After mating has occurred, we assume that individuals lay their eggs in the different niches, where the proportion laid in each niche is a function of both their phenotype  $ij$  and the mating pool from which they come. If preference for oviposition site depends on  $i$ , then oviposition is, by definition, under genetic control (see Rausher, 1984; Jaenike, 1986). On the other hand, oviposition preference may be linked to diet or imprinting cues and so be dependent on the niche  $j$  in which the individual matured. As depicted in Fig. 1, we use parameters  $k_l^{ij\pi_r}$  to represent the proportion of eggs that individuals of phenotype  $ij$  from mating pool  $\pi_r$  lay in niche  $l$ ,  $l = 1, \dots, m$ . With this definition it follows that

$$\sum_{l=1}^m k_l^{ij\pi_r} = 1 \quad (4)$$

for  $i = 1, \dots, n$  and for  $j = r$  when  $r = 1, \dots, m$ ; or  $j = 1, \dots, m$  when  $r = m + 1$ .

Using our intermediate egg variable  $E_\mu^{ij\pi_r}(t + 1)$ , it easily follows that

$$N_{\mu l}(t + 1) = \sum_i^n \sum_{j=1}^m [k_l^{ij\pi_j} E_\mu^{ij\pi_j}(t + 1) + k_l^{ij\pi_{m+1}} E_\mu^{ij\pi_{m+1}}(t + 1)]. \quad (5)$$

This completes the formulation of the equations that describe a one generation transformation from  $N_{ij}(t)$ , appearing on the right-hand side of Eq. (2), to (renaming the indices  $\mu$  and  $l$ )  $N_{\mu l}(t + 1)$ , appearing on the left-hand side of Eq. (5).

### 3. ECOLOGICAL MODEL

#### *Basic Growth Equation*

We begin our analysis by considering the dynamics of a homogeneous population in a homogeneous environment; that is, the single phenotype case. Here Eq. (2) simply reduces to

$$N(t + t') = s(N(t)) N(t). \quad (6)$$

During the reproductive phase, assume each individual produces  $b$  zygotes which become the new individuals at time  $t + 1$ ; that is,

$$N(t + 1) = bN(t). \quad (7)$$

Combining Eqs. (6) and (7) we obtain

$$N(t + 1) = bs(N(t)) N(t). \quad (8)$$

Equation (8) is obviously most suitable for populations with discrete non-overlapping generations, such as are found in many insect species; although, if generations overlap, as happens in many species of birds, fish, and mammals, then Eq. (8) can be expanded to include age structure (Bergh and Getz, in press). The equilibrium and stability properties of this class of equations have been extensively analysed (May and Oster, 1976; reviewed in May, 1981). In much of the population modeling literature, specific density-dependent survival functions  $s(N)$  have a negative slope at  $N=0$  (e.g., the linear function  $s(N) = \sigma - \beta N$ , the 'Ricker' function  $s(N) = \sigma e^{-\beta N}$ , and the 'Hassell' function  $s(N) = \sigma(1 + \beta N)^{-\gamma}$ —see May and Oster, 1976). Density-independent growth at low population densities is more realistically modeled, however, by survival functions which satisfy

$$\left. \frac{ds}{dN} \right|_{N=0} = 0. \quad (9)$$

To ensure that Condition (9) is satisfied, we select the proportion survival rate function (Maynard Smith, 1973)

$$s(N) = \frac{\sigma}{1 + (N/\beta)^\gamma}, \quad \gamma > 1, \quad (10)$$

where  $0 < \sigma \leq 1$  is the density-independent proportional survival rate,  $\beta$  is the population density at which this density-independent rate is reduced by a factor of 2, and  $\gamma$  is a shape parameter that determines, for increasing  $N$ , how rapid the transition is from a density-independent survival rate of  $\sigma$  to almost zero. Note that if  $\gamma = 1$  (the form adopted by Hoekstra *et al.* 1985), then Condition (9) is not satisfied. As  $\gamma$  tends to infinity, however, we obtain a step function which corresponds to intra-niche density-independent survival when the initial population density is below the carrying capacity  $\beta$  and a survival rate of zero when the initial population density is above  $\beta$  (i.e., the environment runs out of food before any of the individuals mature). Sometimes it is useful to retain  $\beta$  explicitly, since  $s(N)$  approaches the density-independent unlimited carrying-capacity case as  $\beta \rightarrow \infty$ ; and we may want to investigate how the properties of certain



solutions are affected by the transition from density-dependent to density-independent survival.

When  $s(N)$  has the form expressed by Eq. (10), then Eq. (8) has the explicit form

$$N(t+1) = \frac{\beta^\gamma b\sigma N(t)}{\beta^\gamma + N(t)^\gamma}. \quad (11)$$

It is straightforward to show that Eq. (11) has an equilibrium value (Maynard Smith, 1973)

$$\hat{N} = \beta(b\sigma - 1)^{1/\gamma}. \quad (12)$$

Thus, a nontrivial biologically meaningful equilibrium ( $\hat{N} > 0$ ) exists when and only when  $b\sigma > 1$ ; that is, at low densities each individual must at least be able to replace itself if an equilibrium is to exist.

The existence and local stability properties of the equilibrium  $\hat{N}$  are easily deduced using standard techniques (Maynard Smith, 1973; May, 1975; May and Oster, 1976). Specifically, it is possible to show that the equilibrium  $\hat{N}$  is always locally stable when  $1 < \gamma \leq 2$ , while, for  $\gamma > 2$ , local solutions

approach $\hat{N}$ monotonically	when	$1 < b\sigma < \frac{\gamma}{\gamma - 1}$	
oscillate and approach $\hat{N}$	when	$\frac{\gamma}{\gamma - 1} < b\sigma < \frac{\gamma}{\gamma - 2}$	(13)
oscillate and move away from $\hat{N}$	when	$b\sigma > \frac{\gamma}{\gamma - 2}$	

Finally, for  $\gamma > 2$ , the solutions are chaotic provided  $b\sigma$  is sufficiently large (May, 1975). In keeping with life-table terminology, we refer to  $b\sigma$  as the reproductive value of the population.

### *Implicit Niche Competition*

One needs first to understand the dynamics of competition between reproductively separate groups of individuals (different species or clonal lines of the same species) before addressing the more complex question of coexistence of interbreeding genotypes. Competition between species has been studied in much depth, but never in the context of the proportional survival rate function defined in Expression (10). Here we do so and obtain a result that has not been found in any other analysis of competition between two species. Specifically we show that two species can theoretically coexist even if no coexistence equilibrium—stable or unstable—exists.

Before analysing the case of competition among species or clonal lines

competing in an explicitly defined multi-niche environments, we begin with the simpler case of analysing competition when the different niches are only implicitly defined. This approach (taken in most continuous time analyses of competition) assumes that the impact individuals in different population groups have on each other can be characterized by the interaction coefficients introduced in Eq. (1), but which now have only two indices (and are written as  $\alpha_{ij} \geq 0$ ,  $i, j = 1, \dots, n$ ,) since no explicit niche structure is defined (cf. May, 1973). In this case, Eq. (11) expands to

$$N_i(t+1) = \frac{\beta_i^{\gamma_i} b_i \sigma_i N_i(t)}{\beta_i^{\gamma_i} + (\sum_{j=1}^n \alpha_{ij} N_j(t))^{\gamma_i}}, \quad i = 1, \dots, n, \quad (14)$$

where  $i$  refers to individuals of type  $i$  (species or clonal lines, depending on applicability). This type of implicit niche competition model is applicable when an environment contains some resource gradient (e.g., seed size) and one group is more efficient at one end of the resource spectrum, while the other is more efficient at the other end of the resource spectrum.

The dynamic properties of this model can now be investigated to establish under what conditions several different groups of individuals (species, clonal lines) are able to coexist. The first requirement for a stable coexistence is that a positive equilibrium solution  $\bar{\mathbf{N}} = (\bar{N}_1, \dots, \bar{N}_n)' > \mathbf{0}$  exist (the inequality is taken element-wise and ' denotes vector transpose). Under equilibrium conditions,  $N_i(t+1) = N_i(t) = \bar{N}_i$ ,  $i = 1, \dots, n$ , whence System (14) reduces to

$$\sum_{j=1}^n \alpha_{ij} \bar{N}_j = \beta_i (b_i \sigma_i - 1)^{1/\gamma_i}, \quad i = 1, \dots, n; \quad (15)$$

or, using Expression (12), we obtain

$$\sum_{j=1}^n \alpha_{ij} \bar{N}_j = \hat{N}_i, \quad i = 1, \dots, n. \quad (16)$$

From Expression (12), however, the right-hand side of this equation is  $\hat{N}_i$ , the equilibrium level of the  $i$ th population in the absence of competition. Since we assume  $\hat{N}_i > 0$ , we require that

$$b_i \sigma_i > 1, \quad i = 1, \dots, n. \quad (17)$$

Whether the linear system of Eqs. (16) has a positive solution  $\bar{\mathbf{N}} > \mathbf{0}$  depends on both the values of the parameters  $\alpha_{ij}$  and the isolated population levels  $\hat{N}_i$ .

Since we have assumed that  $b_i \sigma_i > 1$ , it follows from Eqs. (15) that

$$b_i \sigma_i = \frac{\beta_i^{y_i} + (\sum_{j=1}^n \alpha_{ij} \bar{N}_j)^{y_i}}{B_i^{y_i}}, \quad i = 1, \dots, n, \quad (18)$$

or from Eqs. (16) that

$$b_i \sigma_i = \frac{\beta_i^{y_i} + \hat{N}_i^{y_i}}{\beta_i^{y_i}}, \quad i = 1, \dots, n. \quad (19)$$

In two dimensions the situation is easy to analyse (see Hassell and Comins, 1976, for an analysis of a similar two-dimensional model). Specifically, if the interaction coefficients are normalized so that  $\alpha_i = 1$ ,  $i = 1, 2$ , then the intersection of the two equations defined by System (16) will only permit  $\bar{N}_1 > 0$  and  $\bar{N}_2 > 0$  (Fig. 2) when

$$\alpha_{21} < \hat{N}_2 / \hat{N}_1 < 1 / \alpha_{12}, \quad (20)$$

or when

$$\alpha_{21} > \hat{N}_2 / \hat{N}_1 > 1 / \alpha_{12}. \quad (21)$$

In both cases the equilibrium solution  $(\bar{N}_1, \bar{N}_2)$  is given by

$$\begin{aligned} \bar{N}_1 &= (\hat{N}_1 - \alpha_{12} \hat{N}_2) / (1 - \alpha_{12} \alpha_{21}) \\ \bar{N}_2 &= (\hat{N}_2 - \alpha_{21} \hat{N}_1) / (1 - \alpha_{12} \alpha_{21}). \end{aligned} \quad (22)$$

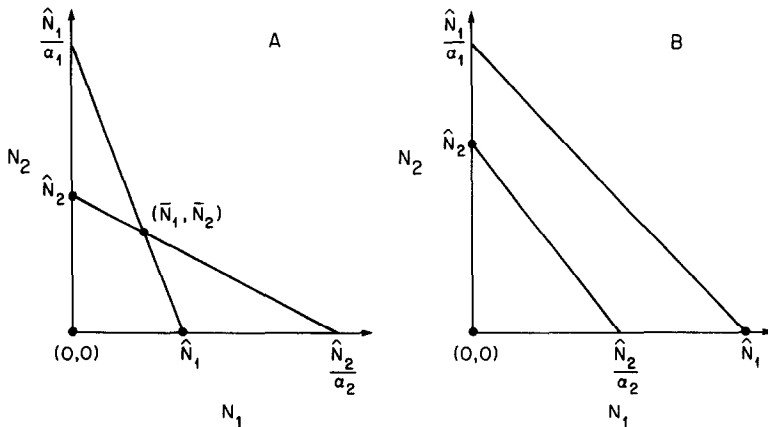


FIG. 2. The equilibrium isoclines equations (see (16) with  $i=2$ ) plotted in the  $N_1$ - $N_2$  phase plane in A and B. In A Inequality (20) is satisfied and this leads to the existence of an interior equilibrium  $(\bar{N}_1, \bar{N}_2)$ . In B neither Inequality (20) nor (21) is satisfied and no coexistence equilibrium exists.

Conditions (20) and (21) for the existence of an equilibrium, and the equilibrium values expressed in (21), are identical to those for the continuous time Gause–Lotka–Volterra model of competing species. The stability properties, however, are different. The local stability properties of the equilibrium  $(\bar{N}_1, \bar{N}_2)$  depend on whether the eigenvalues of the Jacobian matrix associated with System (14) have modulus greater or less than 1 and are real or complex. Using linearization techniques and defining

$$\kappa_i = \frac{\gamma_i \bar{N}_i (b_i \sigma_i - 1)}{\hat{N}_i b_i \sigma_i}, \quad (23)$$

for  $i = 1, 2$ , it is easily shown (Edelstein-Keshet, 1988) that the eigenvalues of the Jacobian stability matrix associated with the equilibrium  $(\bar{N}_1, \bar{N}_2)$  are given by

$$\lambda^\pm = 1 - \frac{\kappa_1 + \kappa_2}{2} \pm \frac{\sqrt{(\kappa_1 - \kappa_2)^2 + 4\alpha_{12}\alpha_{21}\kappa_1\kappa_2}}{2}. \quad (24)$$

It follows from Inequality (17) and Expression (23) that the discriminant in (24) (term under the square root) is positive. Further, if  $\alpha_{12}\alpha_{21} < 1$  (this condition can be expected to hold in situations where each competitor is superior in some region of the resource spectrum, since this implies that both  $\alpha_{12} < 1$  and  $\alpha_{21} < 1$ ), then the discriminant is less than  $(\kappa_1 + \kappa_2)^2$  and, consequently,

$$1 - (\kappa_1 + \kappa_2) < \lambda^- < \lambda^+ < 1. \quad (25)$$

Thus the equilibrium  $(\bar{N}_1, \bar{N}_2)$  is stable whenever  $\kappa_1 + \kappa_2 < 2$ , which is likely to occur when  $\gamma_1$  and  $\gamma_2$  are relatively small (not much larger than 2). If  $\gamma_1$  and/or  $\gamma_2$  is much larger than 2, then the left-hand side of Inequality (25) (see Identities (23)) is likely to be less than  $-1$ , and the equilibrium  $(\bar{N}_1, \bar{N}_2)$  may be unstable.

Even if the equilibrium  $(\bar{N}_1, \bar{N}_2)$  is unstable, dynamic coexistence is possible if the populations oscillate around  $(\bar{N}_1, \bar{N}_2)$  but are not attracted to the other three equilibria  $(0, 0)$ ,  $(\hat{N}_1, 0)$ , and  $(0, \hat{N}_2)$  (Fig. 2A). The eigenvalues of the Jacobian stability matrices associated with these three equilibria (also the three equilibria depicted in Fig. 2B) are easily determined. They are (since we don't know which of the eigenvalues is the larger, we use the notation  $\lambda_1$  and  $\lambda_2$  instead of  $\lambda^+$  and  $\lambda^-$ )

$$\begin{aligned} (0, 0): \quad & \lambda_1^{(1)} = b_1 \sigma_1 \quad \text{and} \quad \lambda_2^{(1)} = b_2 \sigma_2 \\ (\hat{N}_1, 0): \quad & \lambda_1^{(2)} = 1 - \gamma_1 \frac{b_1 \sigma_1 - 1}{b_1 \sigma_1} \quad \text{and} \quad \lambda_2^{(2)} = b_2 \sigma_2 \frac{\beta_2^{\gamma_2}}{\beta_2^{\gamma_2} + (\alpha_{21} \hat{N}_1)^{\gamma_2}} \\ (0, \hat{N}_2, ): \quad & \lambda_1^{(3)} = b_1 \sigma_1 \frac{\beta_1^{\gamma_1}}{\beta_1^{\gamma_1} + (\alpha_{12} \hat{N}_2)^{\gamma_1}} \quad \text{and} \quad \lambda_2^{(3)} = 1 - \gamma_2 \frac{b_2 \sigma_2 - 1}{b_2 \sigma_2}. \end{aligned} \quad (26)$$

From this it is clear that these three equilibria are unstable provided

$$b_i \sigma_i > \frac{\beta_i^{\gamma_i} + (\alpha_{ij} \hat{N}_j)^{\gamma_i}}{\beta_i^{\gamma_i}} > 1 \quad i = 1, j = 2 \quad \text{and} \quad i = 2, j = 1, \quad (27)$$

which, as a consequence of Identity (19), is always true for the case depicted in Fig. 2A ( $\hat{N}_1 > \alpha_{12} \hat{N}_2$  and  $\hat{N}_2 > \alpha_{21} \hat{N}_1$ ). Note that equilibrium point (0, 0) is unstable under the weaker condition

$$b_i \sigma_i > 1 \quad i = 1, 2. \quad (28)$$

For the case depicted in Figure 2B, it similarly follows that  $\lambda_2^{(2)} < 1$  and  $\lambda_1^{(3)} > 1$ . Thus one expects the superior competitor  $N_1$  (dominating isocline in Figure 2B) to drive the inferior competitor to extinction, as happens in the Lotka–Volterra competition model (Edelstein-Keshet, 1988). However, a very interesting situation occurs when  $\gamma_1$  is large enough to ensure that  $\lambda_1^{(2)} > 1$ . Now the isolated equilibrium associated with the competitively dominant population is also unstable; that is, no interior equilibrium exists and all three axis equilibria are unstable.

Two situations can arise, as illustrated in Figure 3A, where either the inferior competitor is driven to extinction while the dominant competitor oscillates around its isolated equilibrium ( $\gamma = 4.5, 5.0$ ), or both populations coexist despite the fact that no interior equilibrium, stable or unstable, exists ( $\gamma = 1.0, 2.0, 3.0, 4.0$ ). In all these cases, the solution asymptotically approaches a two-point cycle in the interior of the phase plane. If both  $\gamma_1$  and  $\gamma_2$  are sufficiently large, however, chaotic behavior ensues (Figure 3B); that is, the behavior of the solution is governed by a strange attractor in the interior of the  $N_1$ – $N_2$  plane (Guckenheimer and Holmes, 1983).

These results have important implications for the analysis of competing subpopulations. They imply that the absence of an interior (coexistence) equilibrium point, in a discrete time competition model, does not automatically imply that coexistence is impossible. We have demonstrated here that coexistence for this case is possible, and could be cyclic (Figure 3A) or chaotic (Figure 3B). Of course, in some cases one or both populations may pass through a level that is sufficiently low to threaten their existence under stochastic conditions not considered here.

### *Explicit Niche Competition*

As already discussed, some ecological systems can be explicitly separated into several distinct niches rather than implicitly defined by coexistence of competitors exploiting resources along a spectrum. For example, fruit flies lay their eggs on several different host plant species, and the larvae develop in those fruit on which they hatch. If several hosts occur sympatrically, then a single population may simultaneously exploit these hosts as different

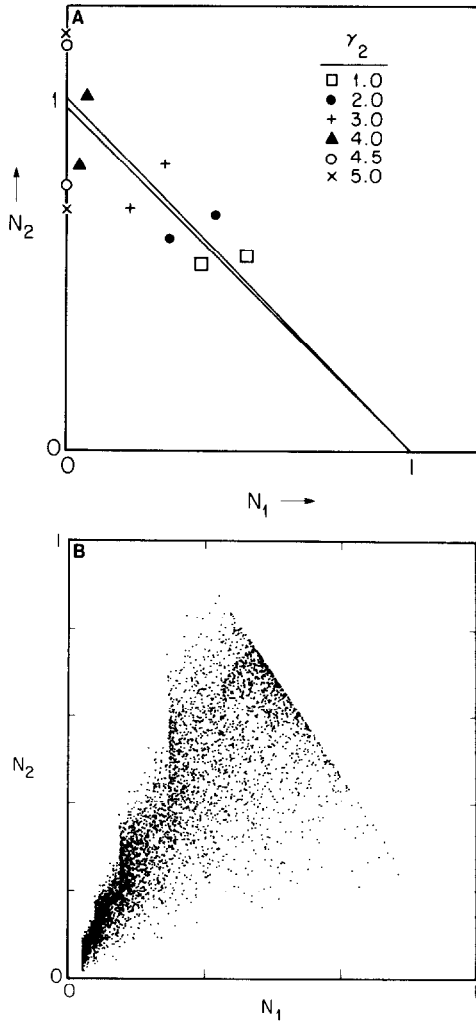


FIG. 3. When the parameters in the implicit niche two-population competition model (Eqs. (14) for  $i=2$ ) have the values  $\beta_1\sigma_1 = \beta_2\sigma_2 = 2$ ,  $\alpha_{12} = 0.98$ ,  $\alpha_{21} = 1.00$ ,  $\gamma_1 = 8.0$ , and  $\gamma_2$  takes on a range of values from 1.0 to 5.0, as indicated in A, then the solutions starting with initial condition  $N_1(0) = N_2(0) = 1$  converge on a cyclic solution with period 2. Coexistence is lost as  $\gamma_2$  increases. The two lines in the  $N_1$ - $N_2$  phase plane (intersecting at  $\bar{N}_1 = 1$  and  $\bar{N}_2 = 0$ ) are the isoclines satisfying Eqs. (16) for  $i=1, 2$ . If  $\alpha_{12}$  and  $\alpha_{21}$  are respectively changed to 0.90 and 1.02 and  $\gamma_2 = 11$  then chaotic coexistence is possible, as illustrated in B. The initial condition for the simulation in B is  $N_1(0) = N_2(0) = 0.5$ , although diagrams exhibiting the same density structure of points results from other initial conditions. In particular, the same line-like accumulations of points are apparent. Connecting consecutive points in this diagram reveals that the solution oscillates between points lying on these "lines" until an edge area of the line is reached, at which time the solution bounces around before running up and down a pair of lines again.

niches. In some of these niches, individuals of one type (genotype or species as the case may be) may compete with individuals of a different type, and the suite of competitors may vary from niche to niche. In this case we are back to the model presented in Eqs. (1) and (2); except, if the different types are non-interbreeding or non-clonal, we interpret the index  $i$  as a species rather than a genotype index.

If a survival function of the form given in Expression (10) holds with its own set of parameters for each phenotype  $ij$ , then Eqs. (1) and (2) can be combined to obtain

$$N_{ij}(t+t') = \frac{\beta_{ij}^{\gamma_{ij}} \sigma_{ij} N_{ij}(t)}{\beta_{ij}^{\gamma_{ij}} + (\sum_{l=1}^n \alpha_{il}^j N_{lj}(t))^{\gamma_{ij}}}, \quad i = 1, \dots, n, j = 1, \dots, m. \quad (29)$$

If the various population types are reproductively distinct (i.e., clonal lines or different species), then we do not need to consider dispersal with respect to mating: we only need to consider the proportions  $k_l^j$  (cf. Eq. (4)) of eggs from species  $i$  in habitat  $j$  that are oviposited in habitat  $l$ , i.e.,

$$\sum_{l=1}^m k_l^j = 1. \quad (30)$$

Thus at time  $t+1$  it follows that

$$N_{il}(t+1) = \sum_{j=1}^m k_l^j b_{ij} N_{ij}(t+t'), \quad (31)$$

where we recall that  $b_{ij}$  is the average number of zygotes produced by an individual of phenotype  $ij$ . We can now combine Eqs. (29) and (31) to finally obtain

$$N_{il}(t+1) = \sum_{j=1}^m \frac{k_l^j \beta_{ij}^{\gamma_{ij}} b_{ij} \sigma_{ij} N_{ij}(t)}{\beta_{ij}^{\gamma_{ij}} + (\sum_{r=1}^n \alpha_{ir}^j N_{rj}(t))^{\gamma_{ij}}}, \quad i = 1, \dots, n, l = 1, \dots, m. \quad (32)$$

This model is considerably more complicated than the implicit niche competition model, because we now have an explicit niche dimension and a dispersal component between distinct niches.

We will not analyse this model in detail. Analytical results are more complicated to derive than for the implicit niche model. It does, however, form the ecological backbone of the ecogenetic model discussed in the next section where numerical simulation studies are carried out in a full ecogenetic framework.

## 4. DIALLELIC ONE-LOCUS TWO-NICHE MODEL

*Model Equations*

The simplest genetic component of an ecogenetic model for a diploid species involves two alleles at one locus. Consider the case where the alleles  $A$  and  $a$  differentially influence an individual's ability to exploit two niches. For example, if individuals in a herbivorous species require an enzyme coded by allele  $A$  to detoxify a chemical compound produced by a plant, then only genotypes  $Aa$  and  $AA$  (assuming codominance or dominance of the allele  $A$ ) are capable of exploiting this plant as an alternative niche.

Let  $N_{AAj}^{\pi_r}(t+t')$ ,  $N_{Aaj}^{\pi_r}(t+t')$ , and  $N_{aaj}^{\pi_r}(t+t')$ ,  $j=1, 2$ , denote the number of individuals of each of the six designated phenotypes that mate at random within mating pools  $\pi_r$ ,  $r=1, 2, 3$  (cf. Identities (3)). If we define  $N^{\pi_r}$  as the total number of individuals in mating pool  $\pi_r$ , that is,

$$N^{\pi_r} = \sum_{j=1}^2 (N_{AAj}^{\pi_r} + N_{Aaj}^{\pi_r} + N_{aaj}^{\pi_r}),$$

then it follows that the proportion of gametes, produced by individuals in mating pool  $\pi_r$ , containing either  $A$  or  $a$  alleles are respectively

$$p_A^{\pi_r} = \frac{\sum_{j=1}^2 (N_{AAj}^{\pi_r} + \frac{1}{2}N_{Aaj}^{\pi_r})}{N^{\pi_r}}$$

$$p_a^{\pi_r} = \frac{\sum_{j=1}^2 (N_{aaj}^{\pi_r} + \frac{1}{2}N_{Aaj}^{\pi_r})}{N^{\pi_r}}. \quad (33)$$

Now, if mating is random in  $\pi_r$ , it follows that the number of eggs  $E_{\mu}^{ij\pi_r}(t+1)$  of genotype  $\mu$  ( $\mu=AA, Aa, aa$ ) produced by individual of phenotype  $ij$  ( $i=AA, Aa, aa, j=1, 2$ ) in mating pool  $\pi_r$ ,  $r=1, 2, 3$ , is

$$E_{AA}^{AAj\pi_r}(t+1) = p_A^{\pi_r}(t+t') b_{AAj} N_{AAj}^{\pi_r}(t+t')$$

$$E_{AA}^{Aaj\pi_r}(t+1) = \frac{1}{2} p_A^{\pi_r}(t+t') b_{Aaj} N_{Aaj}^{\pi_r}(t+t')$$

$$E_{Aa}^{AAj\pi_r}(t+1) = p_a^{\pi_r}(t+t') b_{AAj} N_{AAj}^{\pi_r}(t+t')$$

$$E_{Aa}^{Aaj\pi_r}(t+1) = \frac{1}{2} b_{Aaj} N_{Aaj}^{\pi_r}(t+t') \quad (34)$$

$$E_{Aa}^{aaj\pi_r}(t+1) = p_A^{\pi_r}(t+t') b_{aaj} N_{aaj}^{\pi_r}(t+t')$$

$$E_{aa}^{Aaj\pi_r}(t+1) = \frac{1}{2} p_a^{\pi_r}(t+t') b_{Aaj} N_{Aaj}^{\pi_r}(t+t')$$

$$E_{aa}^{aaj\pi_r}(t+1) = p_a^{\pi_r}(t+t') b_{aaj} N_{aaj}^{\pi_r}(t+t')$$

where we recall that  $b_{ij}$ ,  $i=AA, Aa, aa, j=1, 2$ , are the zygote production



rates for the designated phenotypes. Finally, we can now calculate the number of individuals of each genotype hatching in either habitat at time  $t + 1$  (assuming all eggs hatch), by substituting Expressions (34) in Eq. (5).

### *Likelihood of Protected Polymorphisms*

As we discussed in the Introduction, several studies analyse conditions for the existence of genetic polymorphisms using Levene type ecogenetic models. To provide a frame of reference for our results, we compare them with results obtained by Hoekstra *et al.* (1985).

Before we do this, we reconcile the notation and assumptions in Hoekstra *et al.* (1985) with our approach. First, the assumption by Hoekstra *et al.* that mating is random throughout the population is equivalent to our assumption that  $\delta_{ij} = 1$  for  $i, j = 1, 2$  (see Fig. 1); that is, all individuals end up in mating pool  $\pi_3$  (cf. Expressions (3)).

Inherent in the Levene model is the assumption that the relative proportion of adults produced in each niche is constant. Thus the proportion of genotypes maturing in each niche is regulated by their relative fitness in that niche, but the number of individuals of each genotype entering the mating pool is readjusted so that the total number from each niche is in proportion to the relative carrying capacity of that niche. Specifically, the proportion of individuals from niche 1 entering  $\pi_3$ , in each time step, is the fixed value  $0 < c < 1$ . Hence  $1 - c$  is the relative proportion from niche 2. Unfortunately, there is no analogue in our model for delivering a fixed proportion of individuals from each niche independent of differential survival rates that affect the density of individuals from one time period to the next. In our formulation, however, the parameters  $\beta_{ij}$  (cf. System (29)) are niche size parameters, so we set  $\beta_{i1} = c$  and  $\beta_{i2} = 1 - c$ , for  $i = AA, Aa, aa$ . Note, however, that we are unable to capture Levene's approach as a special case of our own, because the concept of intra-niche competition is entirely missing from Levene's model (only differential survival is possible).

After mating, following Maynard Smith (1970), we assume that the oviposition preference coefficients  $k_l^{ij\pi_3}$  ( $i = AA, Aa, aa, j, l = 1, 2$ ) depend on the niche size parameter  $c$ , but are independent of the genotype  $i$ . In addition we use the parameter  $h$ , varying in value between 0 and 1, to control the degree of habitat preference, such that  $h = 0$  corresponds to no habitat preference (selection is purely proportional to habitat size) while  $h = 1$  corresponds to the case where all individuals oviposit in the same niche in which they mature (natal niche). Specifically, the habitat preference components of Hoekstra *et al.*'s (1985) and our model are equivalent if we set  $k_1^{i1\pi_3} = c + h(1 - c)$  and  $k_2^{i2\pi_3} = 1 - c + hc$  for  $i = AA, Aa, aa$ . (Note from Relationship (4) that it also follows that  $k_2^{i1\pi_3} = (1 - h)(1 - c)$  and  $k_1^{i2\pi_3} = c - hc$  for  $i = AA, Aa, aa$ .)

Using the relative fitness expressions in Table I, Hoekstra *et al.* (1985)

TABLE I

Relative Fitness under Partial Dominance of Varying Direction,  $0 \leq v, s \leq 1$   
(Hoekstra *et al.*, 1985)

Genotype	<i>AA</i>	<i>Aa</i>	<i>aa</i>
Niche 1	$(1 - s)$	$(1 - vs)$	1
Niche 2	1	$(1 - vs)$	$(1 - s)$

derived conditions which guarantee the existence of a protected genetic polymorphism:

$$\frac{v(2 - h + hvs - 2s)}{(1 - vs)(2 - h)} < c < \frac{(1 - v)(2 - h + hvs)}{(1 - vs)(2 - h)}. \tag{35}$$

Note from Table I that if  $0 \leq v \leq 0.5$ , then in either niche the heterozygote is at least as fit as the arithmetic mean of the two homozygotes. This latter condition originally was thought to be necessary for the existence of genetic polymorphisms in a heterogeneous environment (Maynard Smith and Hoekstra, 1980), although Arnold and Anderson (1983) have weakened this condition by replacing the arithmetic mean with a weighted harmonic mean. In the simulations presented below, we set  $v = 0.5$ .

We now address the question of how intra-niche density-dependent competition influences the likelihood that genetic polymorphisms exist. To do this, we compare the range of parameter values defined by Inequality (35) with results obtained from our model, when the parameters  $b_{ij}\sigma_{ij}$  are selected according to the fitness values in Table I. Specifically, the survival coefficients  $\sigma_{ij}$  were given the values in Table I (e.g.,  $\sigma_{Aa1} = 1 - vs$ ) and the fecundity parameters were assumed to be independent of the phenotype; i.e.,  $b_{ij} = b$  for all  $i, j$ . Since any value of  $b$  maintains the same relative fitness values in Table I, we are free to choose any positive value for  $b$ . Our choice of  $b$ , however, affects the stability properties of solutions, although we can still evaluate how changes in the density-related parameters affect the existence of protected polymorphisms for a given value of  $b$ . We note from the results presented in Figure 2A for  $\gamma_2 = 4.5$  and 5.0, however, that it is not sufficient to check the stability properties of the monomorphic solution to establish whether the polymorphism persists through time. A monomorphism equilibrium may be unstable and yet the population may asymptotically approach the monomorphic axis on which the equilibrium lies; that is, the monomorphic gene frequency solution is stable but the equilibrium population size is unstable.

For simulation purposes, we set  $b = 10$ . Initially we also assumed that each individual has the same effect on density-dependent growth regardless

of genotype; that is, we assumed  $\alpha_{il}^j = 1$  for  $i, l = AA, Aa,$  or  $aa,$  and  $j = 1, 2$  (see Eq. (32)).

When the habitat selection parameter has a value  $h = 0.8,$  the results illustrated in Figure 4A indicate that an increase in the intra-niche density dependence associated with a reduction in the value of the parameter  $\gamma$  leads to a reduction in the likelihood that a protected polymorphism exists. These results suggest that by increasing the value of  $\gamma$  we obtain a system in which protected polymorphisms become increasingly likely. From our earlier purely ecological analyses, however, we might suspect that increasing  $\gamma$  also leads to less stable behavior including, as we demonstrate below, periodic and chaotic solutions. Thus, large  $\gamma$  may permit polymorphisms to exist under a wider range of conditions, but it also means that these polymorphisms are less likely to be stable equilibria and more likely to exhibit chaotic behavior.

A second way of decreasing density dependence is to reduce the weighted population size in each niche by decreasing the interaction parameter values (cf. Expression (1)). In fact, as a consequence of differential survival of the different genotypes in each niche, we expect that some of the inter-

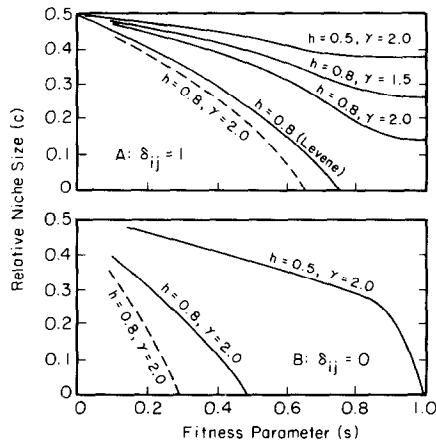


FIG. 4. Comparative analysis between Levene's and our model in terms of the set of parameter values under which a protected genetic polymorphism exists (i.e., the points in  $c$ - $s$  parameter space, above the lines in question, support a protected genetic polymorphism). Recall that when  $c = 0.5$  both niches are equal in size while when  $c = 0.0,$  niche 1 is of size 0. The parameter  $s$  relates to relative fitness such that  $s = 0$  implies that both homozygotes are equally fit in both niches and  $s = 1$  implies that each homozygote can only survive in one of the niches (see Table I). Note that symmetry considerations imply the reflection of this diagram around the line  $c = 0.5$  holds for  $c \in [0.5, 1]$ . In A we have panmixis ( $\delta_{ij} = 0$  for all  $i, j$ ) and in B we have extreme heteropatry ( $\delta_{ij} = 1$  for all  $i, j$ ). The solid lines correspond to interaction parameters  $\alpha_{il}^j = 1,$  while the broken lines correspond to interaction parameters  $\alpha_{il}^j = \sigma_{ij},$  for all  $i, j, l.$  Note that the lines were not plotted for  $s < 0.1$  because the simulations took too long to converge when stable equilibria did exist.

action parameters are less than 1. To explore the effects of this, we set  $\alpha_{ii}^j = \sigma_{ij}$ , for  $i, l = AA, Aa, aa$  and  $j = 1, 2$  (i.e., the weighted population size depends on the survivability of each genotype in each niche, but no additional structure is assumed for the way different genotypes interact in the same niche). The broken line in Fig. 4A, again, indicates that a reduction in competition increases the likelihood of a protected polymorphism; in this case beyond the limits of the Levene model. This does not contradict Asmussen's (1983b) finding that '...intergenotypic interactions may allow genetic variation to be more easily maintained than in the corresponding model of purely density-dependent selection' (the latter corresponds to setting  $\alpha_{ii}^j = 0$  for all  $i, l, j$ ). We need to bear in mind, as discussed above, that conditions which increase the likelihood of a protected polymorphism may decrease the likelihood of a stable polymorphism; the notions of protected and stable genetic polymorphisms must be kept distinct.

We also explored the effects of habitat selection on the existence of protected polymorphisms. In particular, we see in Fig. 4A that the likelihood of a protected polymorphism is decreased with a decrease in the habitat selection parameter  $h$  from 0.8 to 0.5. Note from Fig. 4A, for the selectively extreme condition  $s = 1$ , that stable polymorphisms can be maintained in Levene's model for all  $c \in [0, 1]$ , but in our model this does not hold under panmixis when the interaction coefficients are all one. It is also apparent from these results that, in a heterogeneous environment where density-dependent selection acts and is important, assumptions related to the way that density dependence acts strongly influence the existence of stable and/or protected genetic polymorphisms.

The question of how density dependence affects the existence of protected polymorphisms was also posed by Hoekstra *et al.* (1985), who employed a density-dependent model introduced by Arnold and Anderson (1983). In this model, the fitness of the different genotypes in each environment was assumed to be of the logistic form. Hoekstra *et al.* (1985) concluded that the added density dependence decreases the likelihood of a protected polymorphism when compared with Levene's model. Our results support this conclusion, with the caveat that although increasing density dependence appears to reduce the likelihood of a protected polymorphism we were still able to formulate a model that is more likely to support a protected polymorphism than is Levene's.

As mentioned above, we can expect the onset of oscillatory solutions when the value of the parameter  $\gamma$  is increased, even though the likelihood of a protected polymorphism is increased as well. For example, when we select parameter values  $c = 0.4$ ,  $h = 0.8$ ,  $b = 10$ , set  $\sigma_{ij}$  according to values in Table I with  $s = 0.8$  and  $v = 0.5$ , and let  $\gamma$  take on values in the range 2.6–3.4, the simulations indicate that the solutions are asymptotically periodic in both the gene frequencies in each niche (Figure 5A) and the

total number of individuals in each niche (Figure 5B). However, when we increase  $\gamma$  to 3.6, both the gene frequency in each niche (Figure 5C) and the total number of individuals in each niche (Figure 5D) exhibit chaotic behavior. Interestingly, this chaotic behavior has an intricate structure with points preferentially clustering along certain curves in the plane.

In all cases, the population size fluctuates much more than the gene frequencies. This is probably due to the fact that the gene frequency component is inherently stable (Asmussen, 1979), while the population regulation mechanism destabilizes as the value of  $\gamma$  is increased.

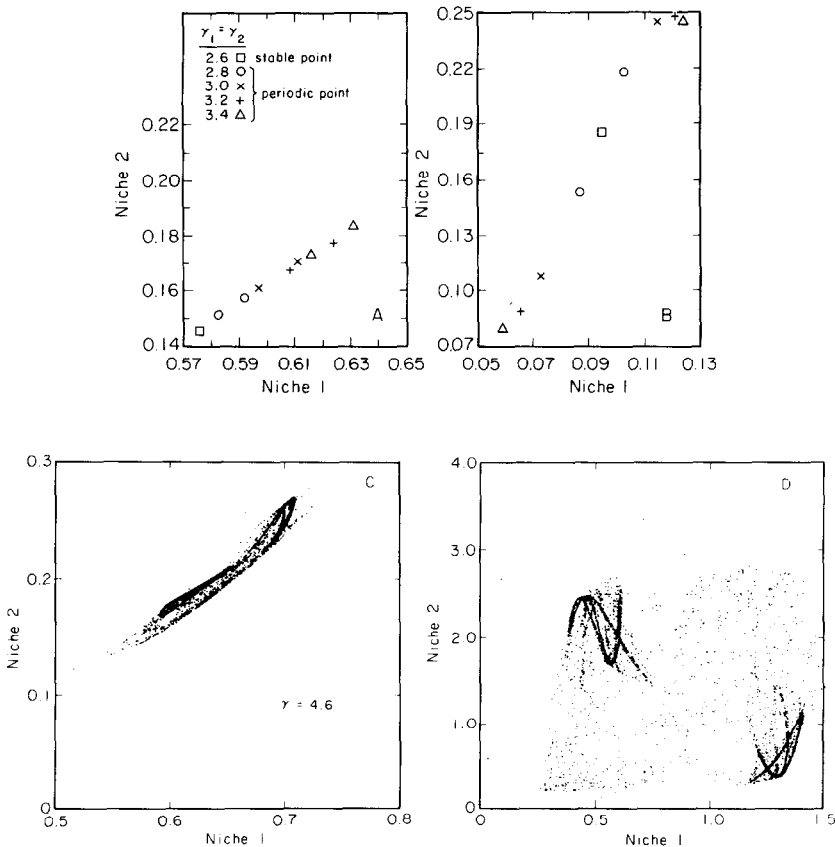


FIG. 5. Simulation results using the diallelic one-locus model described by Eqs. (29), (33), and (34), and for the parameter values discussed in the text. Figures A and B, respectively, illustrate the asymptotic frequency and population size solutions in each niche for various values of the shape parameters  $\gamma_1$  and  $\gamma_2$ . When  $\gamma_1 = \gamma_2 = 2.6$  the asymptotic solution is a stable point. For increasing values of  $\gamma_1 = \gamma_2$  it is a stable two-cycle until  $\gamma_1 = \gamma_2 = 3.6$  when the solution, as illustrated in C (frequency) and D (population size), becomes chaotic.

*Genotype Habitat Preference*

So far, we have considered the situation where adults have a tendency to choose their natal habitat for oviposition purposes, regardless of genotype. This was incorporated in the model using the habitat preference parameter  $h$ , as discussed in the previous section. Now, we study the effects of genotype controlled habitat preference process, where each genotype prefers the habitat in which it is most fit according to the relative fitness values listed in Table I. To do this we redefine the preference parameters  $k_i^{j\pi_3}$  in terms of the previously introduced parameters  $h$  and  $c$  in the following manner. (Note that our definitions are just one of many possible definitions of  $k_i^{j\pi_3}$  that will satisfy the assumption that each genotype preferentially migrates to the habitat in which it is most fit). Specifically, in niche 1 we set  $k_1^{AA1\pi_3} = c - hc$ ,  $k_1^{Aa1\pi_3} = c$ , and  $k_1^{aa1\pi_3} = c + h(1 - c)$ , while in niche 2 we set  $k_2^{AA2\pi_3} = 1 - c + hc$ ,  $k_2^{Aa2\pi_3} = 1 - c$ , and  $k_2^{aa2\pi_3} = 1 - c - h(1 - c)$ . Note from Identity (4) that it follows that  $k_2^{AA1\pi_3} = 1 - c + hc$ ,  $k_2^{Aa1\pi_3} = 1 - c$ ,  $k_2^{aa1\pi_3} = 1 - c - h(1 - c)$ ,  $k_1^{AA2\pi_3} = c - hc$ ,  $k_1^{Aa2\pi_3} = c$ , and  $k_1^{aa2\pi_3} = c + h(1 - c)$ .

From the above expressions it is apparent that  $h = 0$  corresponds to no preference (selection of niche is purely proportional to niche size). Heterozygotes continue to display no niche preference for all values of  $h$  (same relative fitness in both niches), but as  $h$  increases each homozygote shows an increasing preference for the niche in which it is most fit. In fact, for  $h = 1$ , the homozygotes  $aa$  all choose niche 1 and the homozygotes  $AA$  all choose niche 2. After extensive simulations, using the same set of productivity rates, interaction coefficients, and shape parameter  $\gamma$  as discussed in the natal niche preference studies, we were unable to establish the existence of protected polymorphism for  $h = 0.2, 0.5, \text{ or } 0.8$ .

This is a surprising result, but must be interpreted circumspectly. First, it is dependent on the form of the fitness table (Table I): for example, if allele  $A$  is completely dominant so that there are only two phenotypes ( $A-$  and  $aa$ ), then we have found in additional simulation studies that stable polymorphisms are possible. Second, there is no way that individuals can teleologically know in which habitat they are most fit. It is easy to see how natal habitat preference can evolve, say, through some type of imprinting mechanism. It is hard to conceive of a mechanism that allows individuals to assess which niche provides the greatest relative fitness to their genotype, given that they only experience one niche in their maturation phase, and that some individuals are more fit in other niches (they may all be able to assess which niches are inhospitable to the species as a whole). If a mechanism does exist for all individuals to assess niche quality for their genotype prior to oviposition (perhaps individuals can sample and evaluate each niche) and preferentially oviposit in the most favorable niche,

then it appears that fixation always occurs. The allele that is fixed will depend on many factors including population and fitness parameters, initial gene frequencies, and relative niche size (e.g., it is more likely the homozygote  $aa$  will win if niche 1 is much larger than niche 2, etc.).

### *Heteropatry*

So far we have assumed that mating is panmictic for the population as a whole (i.e.,  $\delta_{ij}=1$  for all  $i, j$  in Eqs. (3)). We now analyse the other extreme where individuals mate within their niche before dispersing to oviposit (i.e.,  $\delta_{ij}=0$  for all  $i, j$ ). Depending on the values of  $k_i^{ij\pi_j}$  in Eq. (5), oviposition preference may or may not be random. As in the previous section we use the habitat preference parameter  $h$  to model oviposition behavior. Recall that ( $h=0$ ) corresponds to no habitat preference, while  $h=1$  now implies that there is no genetic interaction between the niches; that is, the niches are genetically isolated entities.

Figure 4A illustrates the simulation results for  $h=0.5$  and  $0.8$  with the two different sets of interaction parameters,  $\alpha_{ij}^j=1$  and  $\alpha_{ij}^j=\sigma_{ij}$  for all  $i, j, l$ . All three curves illustrated in Fig. 4B can be directly compared with corresponding curves in Fig. 4A (the three curves for which  $\gamma=2$ ), since the only parameters that differ between corresponding curves are the assortative mating parameters  $\delta_{ij}$  (see Fig. 1 and Expressions (3)). Recall that the results in Fig. 4A correspond to panmixis in the population as a whole ( $\delta_{ij}=1, i, j=1, 2$ ) while the results in Fig. 4B correspond to mating within the habitat before dispersing to oviposit ( $\delta_{ij}=0, i, j=1, 2$ ).

The condition  $\delta_{ij}=0$  for  $i, j=1, 2$ , is the most extreme form of heteropatry, while the other extreme,  $\delta_{ij}=1$ , for  $i, j=1, 2$ , is sympatry. From the comparison, it is clear that strong heteropatry leads, as expected, to a noticeable increase in the likelihood that a protected polymorphism exists; that is, the area above comparable curves in the  $s-c$  plane is noticeably larger in Fig. 4B than in Fig. 4A.

It is clear from the simulation studies compared in Fig. 5A and B that, much as one would expect, spatially induced assortative mating together with some level of niche oviposition preference can dramatically enhance conditions for the existence of protected polymorphisms. If, in addition, genic assortative mating mechanisms become linked with habitat preference (i.e., the values of the parameters  $\delta_{ij}$  become increasingly determined by genetic factors) then heteropatry enhances the possibility of sympatric speciation (but see Futuyma and Mayer, 1980). In essence we have heteropatry in a polyphagous organism when dispersal to mate is on a scale comparable with environmental heterogeneity, but dispersal to oviposit or bear young is on a larger scale. Biologists should evaluate the scales in appropriate populations (i.e., possible polyphagous ancestor,

sessile young, and adults highly dispersive after mating) when they argue that sympatric speciation might have occurred.

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