THE EVOLUTION OF HABITAT PREFERENCE IN
SUBDIVIDED POPULATIONS

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Accounting for the maintenance of genetic variation in natural populations has been a major goal of population biologists. One character that has largely escaped theoretical examination in this regard is habitat preference. It has been established that variation in habitat preference can facilitate the maintenance of genetic diversity affecting viability (Maynard Smith, 1966; Dickinson and Antonovics, 1973; Taylor, 1975, 1976) and that natural selection may favor fixation of a habitat preference over random habitat selection in heterogeneous environments (Maynard Smith, 1966; Templeton and Rothman, 1978). However, in most of these studies it is assumed that variation in habitat preference is environmentally induced (by larval or adult conditioning) or that genetic differences in preference are indirect, pleiotropic manifestations of genetic variation affecting viability. Theoretical investigations of the properties of genetic variability affecting only habitat selection behavior are virtually nonexistent (but see Templeton and Rothman, 1978). It is thus not clear under what conditions genetic variation in preference itself will be maintained. Standard "multiple niche" models (Christiansen and Feldman, 1975; Felsenstein, 1976; Hedrick et al., 1976) are not obviously applicable to studies of habitat preference because they assume that genotypes differ in viability within a particular habitat rather than in habitat preference.

Several recent investigations on phytophagous insects have demonstrated that populations of many species vary genetically in host (habitat) preference (Tabashnik et al., 1981; Tavormina, 1982; Jaenike and Grimaldi, 1983; Via, 1983; Lofdahl, 1984). Here I describe and analyze a genetic model that can account for the maintenance of such variation. The model has been cast in terms general enough that it should be applicable to habitat selection by other organisms, such as the settling larvae of marine invertebrates, which in some cases exhibit individual variation in substrate preference (Meadows and Campbell, 1972).

I show here that when density-dependent population regulation occurs independently in different habitats, a form of frequency-dependent selection can maintain genetic variation at a single locus affecting habitat preference. The conditions under which variation is maintained are not unduly restrictive. In addition, I show that Fretwell's (1972; Fretwell and Lucas, 1970) suggestion that selection modifies habitat preference in such a way as to equalize viabilities in different habitats is true if habitat preference does not affect fecundity; when fecundity is affected, a generalization of Fretwell's suggestion to equalization of parental investment is valid under certain realistic assumptions about how variation in preference affects fecundity. Finally, I discuss the relevance of these results to the process of host race formation and sympatric speciation in phytophagous insects.

The Model

The model is completely deterministic and pertains to an animal population whose environment is comprised of two habitats or niches. Mated females place offspring in one or both of the two habitats. The offspring develop and mature in their natal habitat, then mate randomly with respect to habitat of origin. The

cycle then repeats itself. Several additional assumptions are made:

(1) The proportion of eggs or offspring placed in each habitat is governed by a single autosomal locus with two alleles. In particular, AA females place a proportion P_1, Aa females a proportion P_2, and aa females a proportion P_3 of their offspring in habitat I and the remainder in habitat II. The parameters P_i can also be interpreted as representing the proportion of individuals of genotype i that settle in habitat I.

(2) Within a habitat, genotypes have equal viability. Genotypes may, however, differ in fecundity, since preference for oviposition in the rarer habitat may decrease oviposition rate and hence total number of eggs laid. The fecundities of genotypes AA, Aa and aa are represented by F_1, F_2, and F_3 respectively.

(3) Population size is regulated independently in the two habitats. In particular, habitat I contributes a constant fraction c of all individuals in the mating pool, whereas habitat II contributes a fraction $(1 - c)$. This assumption of "soft selection" (Wallace, 1968) is similar to that made by Levene (1953) and others subsequently (see Christiansen, 1975; Felsenstein, 1976).

The basic recursion equations for gene and genotype frequencies of this system are as follows:

$$G'_1 = p\left[\frac{cA}{T_I} + \frac{(1-c)B}{T_{II}}\right] = p\tilde{p} \qquad (1a)$$

$$G'_2 = q\left[\frac{cA}{T_I} + \frac{(1-c)B}{T_{II}}\right]$$
$$+ p\left[\frac{cM}{T_I} + \frac{(1-c)N}{T_{II}}\right]$$
$$= q\tilde{p} + p\tilde{q} \qquad (1b)$$

$$G'_3 = q\left[\frac{cM}{T_I} + \frac{(1-c)N}{T_{II}}\right] = q\tilde{q} \qquad (1c)$$

$$p' = \frac{1}{2}p + \frac{1}{2}\left[\frac{cA}{T_I} + \frac{(1-c)B}{T_{II}}\right]$$
$$= \frac{1}{2}p + \frac{1}{2}\tilde{p} \qquad (1d)$$

where $A = G_1P_1F_1 + (\frac{1}{2})G_2P_2F_2$, $B = G_1(1 - P_1)F_1 + (\frac{1}{2})G_2(1 - P_2)F_2$, $M = (\frac{1}{2})G_2P_2F_2 + G_3P_3F_3$, $N = (\frac{1}{2})G_2(1 - P_2)F_2 + G_3(1 - P_3)F_3$, $T_I = A + M$, $T_{II} = B + N$, G_1, G_2, and G_3 are the genotype frequencies of AA, Aa, and aa in the mating pool, and p is the gene frequency of A.

To illustrate the origin of these equations, consider the recursion equation for G_1. This value is simply the weighted average of the genotype frequencies ($G'_{1,I}$ and $G'_{1,II}$) of AA in the two groups of adults derived from the two habitats:

$$G'_1 = cG'_{1,I} + (1 - c)G'_{1,II}.$$

Since, by assumption (2), selection does not operate within habitats, $G'_{1,I}$ is equal to the frequency of AA among offspring placed in habitat I by females of the previous generation. Mothers with genotypes AA, Aa, and aa produce respectively the fractions $G_1P_1F_1/T_I$, $G_2P_2F_2/T_I$, and $G_3P_3F_3/T_I$, of these offspring. Of the offspring produced by AA mothers, a fraction p will be AA because the mother always contributes an A allele to the offspring, whereas the father contributes an A allele with a probability equal to the gene frequency of A in the population. Similarly, of the offspring in habitat I produced by Aa mothers, a fraction $p/2$ will be AA. No AA offspring are produced by aa mothers. Consequently, the proportion of offspring placed in habitat I that are AA is given by

$$G'_{1,I} = p\left[\frac{A}{T_I}\right]. \qquad (2)$$

An analogous expression for $G'_{1,II}$ is $G'_{1,II} = pB/T_{II}$, which when combined with (2) to give the average frequency of AA in the mating pool yields (1a). Equations (1b–1d) are obtained by similar reasoning.

ANALYSIS OF THE MODEL

Equation (1d) implies that at equilibrium $p = \hat{p}$. In turn, equations (1) imply that at equilibrium genotype frequencies are unchanging and are in Hardy-Weinberg proportions.

Equilibria of the System

When gene frequencies are at equilibrium, $p' = p$ and (1d) can be rewritten, after factoring out the fixation equilibria corresponding to $p = 0$ and $p = 1$, as

$$0 = c\left(p - \frac{\alpha_1}{\beta_1}\right)\beta_1 T_{\text{II}}$$

$$+ (1 - c)\left(p - \frac{\alpha_2}{\beta_2}\right)\beta_2 T_{\text{I}} \quad (3)$$

where $\alpha_1 = P_3 F_3 - P_2 F_2$, $\alpha_2 = (1 - P_3)F_3 - (1 - P_2)F_2$, $\beta_1 = P_1 F_1 + P_3 F_3 - 2 P_2 F_2$, and $\beta_2 = (1 - P_1)F_1 + (1 - P_3)F_3 - 2(1 - P_2)F_2$.

Equation (3) represents the most general condition that genetic equilibria of the model must satisfy; all internal equilibria of the model correspond to roots of (3). In general, (3) is a cubic equation and hence explicit expressions for the internal equilibria and their stability can not be obtained. However, several deductions can be made about the internal equilibria satisfying (3). First, there are at most three such equilibria. Second, if both fixation equilibria are stable or both are unstable, there is an odd number (1 or 3) of valid internal equilibria, whereas if exactly one fixation equilibrium is stable, there is an even number (0 or 2). Third, computer simulations suggest that whenever both fixation equilibria are unstable, each internal equilibrium nearest each fixation equilibrium is stable. Finally, the stability conditions for the fixation equilibria are easily derived (see Appendix) and are given by

for $p = 1$

$$\begin{cases} \dfrac{-c\beta_1}{(1 - c)\beta_2} > \dfrac{P_1}{1 - P_1} \text{ if } (\beta_2 - \alpha_2) < 0 \\[3mm] \dfrac{-c\beta_1}{(1 - c)\beta_2} < \dfrac{P_1}{1 - P_1} \text{ if } (\beta_2 - \alpha_2) > 0 \end{cases}$$

and

for $p = 0$

$$\begin{cases} \dfrac{-c\beta_1}{(1 - c)\beta_2} > \dfrac{P_3}{1 - P_3} \text{ if } (\beta_2 - \alpha_2) < 0 \\[3mm] \dfrac{-c\beta_1}{(1 - c)\beta_2} < \dfrac{P_3}{1 - P_3} \text{ if } (\beta_2 - \alpha_2) > 0. \end{cases}$$

Since these inequalities can simultaneously fail to be satisfied, there are conditions under which both fixation equilibria are unstable, i.e., under which genetic variation for preference will be maintained in the population.

Analytical solutions for the equilibria are obtainable for one class of biologically interesting special cases of (3), those which satisfy the condition

$$\begin{aligned} F_1 F_3(P_1 - P_3) &- F_1 F_2(P_1 - P_2) \\ &- F_2 F_3(P_2 - P_3) = 0. \end{aligned} \quad (4)$$

This condition holds, for example, if fecundities of all three genotypes are equal or if fecundity is inversely proportional to search time (see below). When (4) is true, (3) can be reduced to a quadratic equation by factoring and two classes of equilibria may be recognized, by analogy with Uyenoyama and Bengtsson (1979). At equilibria of one class, designated symmetric, gene frequencies are equal in the two habitats, i.e.,

$$p_{\text{I}} \equiv \frac{A}{T_{\text{I}}} = \frac{B}{T_{\text{II}}} \equiv p_{\text{II}} \quad (5)$$

where p_i is the gene frequency of A in habitat i. In addition to the two fixation equilibria, one root of (3) may also be symmetric. The remaining equilibria are designated asymmetric because at those equilibria gene frequencies are unequal in the two habitats.

From (1d) and (5) it can be shown that an internal symmetric equilibrium, when it exists, is given by

$$p = \hat{p}_s = \frac{\alpha_1}{\beta_1} = \frac{\alpha_2}{\beta_2} = \frac{F_3 - F_2}{F_1 + F_3 - 2F_2}. \quad (6)$$

Moreover, (4) is a necessary and sufficient condition for the existence of a non-

TABLE 1. Existence and stability criteria for equilibria of the model.[1]

Equilibrium p	Existence criteria	Stability criteria
0	Always exists	$\dfrac{-c\beta_1}{(1-c)\beta_2} < \rho_1$ if $(\beta_2 - \alpha_2) > 0$ $\dfrac{-c\beta_1}{(1-c)\beta_2} > \rho_1$ if $(\beta_2 - \alpha_2) < 0$
1	Always exists	$\dfrac{-c\beta_1}{(1-c)\beta_2} < \rho_3$ if $\alpha_2 > 0$ $\dfrac{-c\beta_1}{(1-c)\beta_2} > \rho_3$ if $\alpha_2 < 0$
p_s	$0 = F_1F_3(P_1 - P_3) - F_1F_2(P_1 - P_2) - F_2F_3(P_2 - P_3)$ and $P_2 > P_1, P_3$ or $P_2 < P_1, P_3$	$\dfrac{-c\beta_1}{(1-c)\beta_2} > \rho_2$ if $(\beta_2 - \alpha_2) > 0$ $\dfrac{-c\beta_1}{(1-c)\beta_2} < \rho_2$ if $(\beta_2 - \alpha_2) < 0$
p_a	c lies within range of values spanned by ρ_1, ρ_3 and, if p_s lies within $(0, 1)$, ρ_2	Always

[1] Symbols as defined in Figure 1. Stability criterion for $\hat{p}_a$ holds only when (4) is valid.

fixation symmetric equilibrium, though that equilibrium may not lie within (0,1). When (4) is valid, $\hat{p}_s$ lies within (0,1) if $P_2 > P_1, P_3$ or $P_2 < P_1, P_3$, i.e., if there is overdominance for preference.

When (4) is true, $p - (\alpha_1/\beta_1) = p - (\alpha_2/\beta_2)$ may be factored out of (3) to yield

$$0 = c\beta_1 T_{II} + (1 - c)\beta_2 T_I. \qquad (7)$$

The valid roots of this quadratic equation represent the remaining (asymmetric) equilibria of the system and are given by

$$p = \hat{p}_a = \hat{p}_s \pm \frac{R}{\beta_1}$$

where $R = \left(P_2^2 F_2^2 - P_1 P_3 F_1 F_3 \right.$

$$\left. + \frac{c[F_1 F_3(P_3 - P_1) - 2F_2 F_3(P_3 - P_2)]\beta_1}{\beta_2} \right)^{1/2}.$$

Stability of the Equilibria

The local stability criteria for the equilibria are easily derived (see Appendix) and are presented in Table 1. By analogy with Uyenoyama and Bengtsson (1979),

both the existence and stability of all equilibria under condition (4) can be represented by a simple graphical analysis.

From (7) it is clear that any asymmetric equilibrium satisfies the following equation when (4) is true:

$$K \equiv -\frac{c\beta_1}{(1-c)\beta_2} = \frac{T_I}{T_{II}}. \qquad (8)$$

Moreover, under (4), an internal extremum of the curve

$$f(p) = \frac{T_I}{T_{II}}$$

$$= \frac{p^2 P_1 F_1 + 2pq P_2 F_2 + q^2 P_3 F_3}{p^2(1-P_1)F_1 + 2pq(1-P_2)F_2 + q^2(1-P_3)F_3}$$

corresponds to $p = \hat{p}_s$. This result is obtained by showing that $\hat{p}_s$ satisfies $\partial f(p)/\partial p = 0$.

The equilibria under (4) are thus obtained by graphing $f(p)$ on the interval (0,1). The extrema (including fixation equilibria) correspond to the symmetric equilibria. The value of p at which $f(p)$ intersects $g(p) = K$ then satisfy (8) and hence constitute the asymmetric equilib-
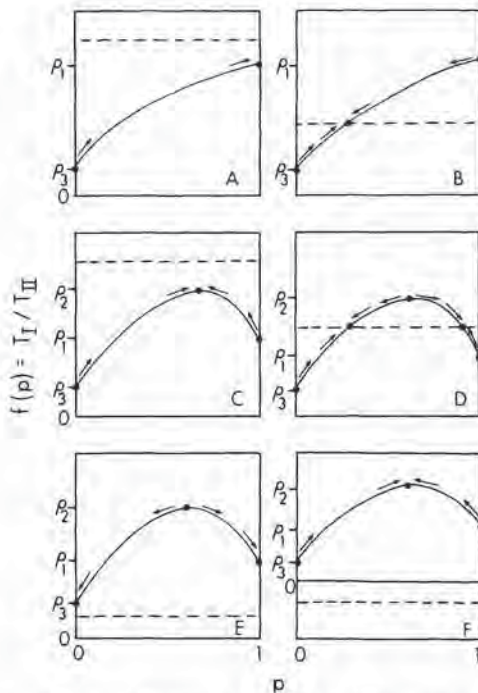
MARK D. RAUSHER

FIG. 1. Equilibria and stability for various relationships between the parameters of the model. Solid line is function $f(p)$ defined in text. Broken line is $f(p) = -c\beta_1/(1 - c)\beta_2 = K$. $\rho_1 = P_1/(1 - P_1)$. $\rho_3 = P_3/(1 - P_3)$. $\rho_2 = \tau_s$, the value of $f(p)$ at internal symmetric equilibrium (see Appendix). Arrows indicate direction in which gene frequencies change. A through E are cases in which $-c\beta_1/(1 - c)\beta_2 > 0$ and hence $g(p)$ acts as an attractor for gene frequencies. F is a case in which $-c\beta_1/(1 - c)\beta_2 < 0$ and in which $g(p)$ acts as a repeller of gene frequencies. A, $P_1 > P_2 > P_3$ and $K > \rho_1$. B, $P_1 > P_2 > P_3$ and $\rho_3 < K < \rho_1$. C, $P_2 > P_1, P_3$ and $K > \rho_2$. D, $P_2 > P_1, P_3$ and $\rho_1, \rho_3 < K$. E, $P_2 > P_1, P_3$ and $K < \rho_1, \rho_3$. F, $P_2 > P_1, P_3$ and $K < 0$.

ria. From the stability criteria it can be deduced that, as long as $K > 0$, the line $g(p) = K$ acts as an attractor for gene frequencies. Consequently, the stability of any equilibrium can be determined by the position of $g(p) = K$ relative to $f(p)$ (see Fig. 1). Under certain restricted circumstances, K may be less than zero. The line $g(p) = K$ may then act as a repeller of gene frequencies (see Fig. 1f) and the stability of the symmetric equilibria must be determined by the criteria listed in

Table 1. (No asymmetric equilibria exist because $g(p) = K$ does not intersect $f(p)$, since $f(p) > 0$ always.)

Special Cases Satisfying (4)

Equal Fecundities.—Although if habitats differ in abundance, fecundities of genotypes with different habitat preferences may also differ because the rate of discovery of acceptable habitats will differ, this generalization need not always be correct. In particular, animals, such as many herbivorous insects, that oviposit in bouts separated by periods of searching might compensate for reduced discovery rates associated with a particular preference scheme by simply increasing the number of eggs laid per bout and thereby maintain an oviposition rate equal to that of genotypes with different preferences (e.g., Rausher, 1983b). An important special case of the model is thus one in which fecundities of the genotypes are equal, i.e., $F_1 = F_2 = F_3$.

When genotypes have equal fecundities, (4) is always valid, and hence all asymmetric equilibria are stable. Moreover, at the asymmetric equilibria, $T_I + T_{II} = 1$ and (8) reduces to $c = T_I$, i.e., the proportion of offspring placed in habitat I is exactly equal to the proportion of individuals in the mating pool that are derived from that habitat. Consequently, at the asymmetric equilibria survivorship is the same in both habitats, as predicted by Fretwell (1972; Fretwell and Lucas, 1970).

With no overdominance, i.e., when P_2 lies between P_1 and P_3, the conditions for the existence of an asymmetric equilibrium, and hence for the maintenance of genetic variation, reduce to $P_1 < c < P_3$ or $P_1 > c > P_3$. In other words, as long as one homozygote genotype places a fraction larger than c, while the other homozygote genotype places a fraction smaller than c, of its offspring in habitat I, genetic variation for preference will be maintained in the population. The regions of the $P_1 \times P_3$ parameter space that allow maintenance of genetic variation in preference are thus extensive (Fig. 2).
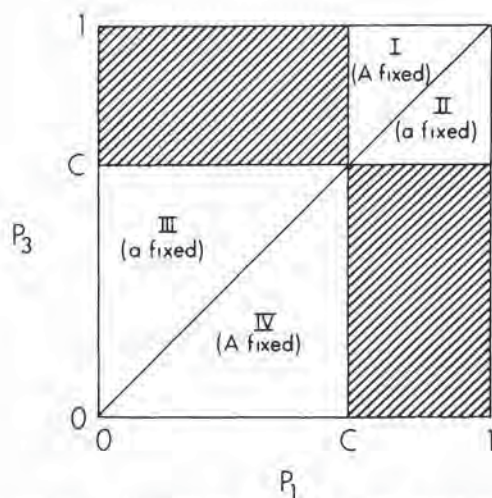
FIG. 2. Regions of parameter space (P_1, P_3) in which polymorphism for habitat preference is maintained when $F_1 = F_2 = F_3$. Shaded areas indicate regions in which a polymorphism is always maintained. When there is no overdominance, either A or a is fixed, as indicated, in regions I–IV. When there is overdominance, a polymorphism will also be maintained in regions III and IV if $P_2 > P_1$, P_3 and in regions I and II if $P_2 < P_1$, P_3.

Criteria for the existence of a preference polymorphism are thus not unduly restrictive.

With overdominance, $P_1 < c < P_3$ or $P_1 > c > P_3$ remains a sufficient condition for maintenance of variation. Variation will also be maintained if 1) $c > P_1$, $P_3 < P_2$ or, 2) $c < P_1$, $P_3 > P_2$ (see Fig. 2). In case 1), if $c < P_2$, there will be two stable asymmetric equilibria, at each of which survivorship is the same in both habitats. By contrast, if $c > P_2$, there is only one stable, symmetric equilibrium. Since at this equilibrium $c = T_1$, viabilities in the two habitats are not equal. Nevertheless, since $f(p) = T_I/T_{II}$ is a maximum at this equilibrium, selection acts to minimize the discrepancy in viability between the two habitats (i.e., to minimize the difference between $f(p) = T_I/T_{II}$ and $g(p) = c/(1 - c)$. Similarly, in case 2), if $c > P_2$, two stable asymmetric equilibria exist, whereas if $c < P_2$, one stable symmetric equilibrium exists, at

which the difference in viability between habitats is minimized.

Fecundity Inversely Proportional to Search Time.—As suggested previously, fecundities of the three preference genotypes may differ because habitats differ in abundance. The genotype with the strongest preference for the less abundant habitat is likely to spend more time searching than do other genotypes and hence is likely to lay fewer eggs before dying. One way of modelling the effect of search time on fecundity is to assume that, on average, an individual searches for a period S_I prior to ovipositing in patch of habitat I and for a period S_{II} before ovipositing in a patch of habitat II. The average time searching per egg laid is then, for genotype i,

$$\bar{S}_i = P_i S_I + (1 - P_i)S_{II}.$$

By letting $\phi = S_{II}/S_I$, it is evident that

$$\bar{S}_i = S_I[P_i + (1 - P_i)\phi].$$

If adults of all genotypes live on average a fixed amount of time, L, then the number of eggs laid is simply $F_i = L/\bar{S}_i$. Consequently, apart from the proportionality constant L/S_I, the fecundities of the three genotypes are given by

$$\frac{1}{F_1} = P_1 + (1 - P_1)\phi,$$

$$\frac{1}{F_2} = P_2 + (1 - P_2)\phi,$$

$$\frac{1}{F_3} = P_3 + (1 - P_3)\phi. \qquad (9)$$

The parameters S_I and S_{II} can be thought of as the amount of parental investment (in time units) associated with eggs laid in habitats I and II, respectively. The parameter ϕ then represents the parental investment in an egg laid in habitat II relative to the investment in an egg laid in habitat I.

When fecundity depends inversely on search time in this manner, natural selection acts to equalize the total parental investment per surviving adult in the two habitats, i.e.,

$$\frac{E_\mathrm{I}}{c} = \frac{E_\mathrm{II}}{(1 - c)}, \qquad (10)$$

where E_i is the total parental investment in eggs laid in habitat i.

This result is derived by noting that whenever (9) is true, (4) is true. Consequently, by substituting (9) into (8) and rearranging, one obtains, at the asymmetric equilibria,

$$\tau = \frac{T_\mathrm{I}}{T_\mathrm{I} + T_\mathrm{II}}$$
$$= \frac{c\phi}{(1 - c) + c\phi}$$

and

$$1 - \tau = \frac{T_\mathrm{II}}{T_\mathrm{I} + T_\mathrm{II}}$$
$$= \frac{(1 - c)}{(1 - c) + c\phi}. \qquad (11)$$

Next, by noting that, in relative units, parental investment in offspring placed in habitats I and II are 1 and ϕ, respectively, and that τ and $1 - \tau$ are the proportions of offspring placed by the population of mated females in habitats I and II respectively, one obtains

$$E_\mathrm{I} = \tau \cdot 1 = \frac{c\phi}{(1 - c) + c\phi}$$

and

$$E_\mathrm{II} = (1 - \tau) \cdot \phi = \frac{(1 - c)\phi}{(1 - c) + c\phi}.$$

Dividing E_I by c and E_II by $(1 - c)$ then yields equal expressions, proving (10). It is also easily shown that any stable symmetric equilibria represent the locally minimal deviation from (10) allowed by the genetic constraints of the system. Thus, in general, when (9) holds, selection acts to move the population toward an equilibrium at which parental investment in the two habitats is proportional to the contribution each habitat makes to the mating pool.

DISCUSSION

The model reported here describes a mechanism for maintaining genetic vari-
ation in habitat preference. The forces preserving variation in this model arise due to a type of frequency-dependent selection that favors the rare allele. These forces can be visualized most easily by considering the special case in which fecundities of the genotypes are equal. Consider first a situation in which allele A is near fixation. Most females will be AA and most offspring will be placed in habitat I. Consequently, density-dependent mortality in habitat I will be high and this mortality will affect AA individuals disproportionately, since this genotype is overrepresented in the offspring placed in habitat I. By contrast, because few offspring are placed in habitat II, density-dependent mortality will be low. Because genotypes Aa and aa are overrepresented in the offspring placed in habitat II, the overall survival probability of these genotypes will be high relative to that of AA. Allele a will therefore increase in frequency. By an analogous argument, allele A will tend to increase in frequency when a is near fixation.

Jaenike and Grimaldi (1983) have recently reported the existence of genetic variation for host preference in *Drosophila tripunctata*. Some genotypes prefer to oviposit in rotting fruits while others prefer to oviposit on fungi in the genus *Amanita* and related genera. Moreover, Grimaldi and Jaenike (1984) have also demonstrated the existence of intense larval competition within fungal hosts for several other related species of mycophagous *Drosophila*. Although because of logistic limitations they have not yet been able to examine competitive interactions among larvae of *D. tripunctata*, the results with the other species suggest they are probably intense. Such competition in turn is likely to provide strong density-dependent regulation within larval habitats. At least qualitatively, the *D. tripunctata* system would seem to satisfy the assumptions of the model presented here; the model may thus provide a valid explanation for the maintenance of genetic variation in habitat preference in that species. It may also explain similar

genetic variation in cactophilic *Drosophila* species (Lofdahl, 1984), in which interspecific, and hence presumably intraspecific, competition among larvae is intense (Fellows and Heed, 1972; Mangan, 1982).

Two conclusions of general relevance to field biologists emerge from the analysis presented here. First, at evolutionary equilibrium there may or may not be a correlation between the habitat an individual is collected from and the habitat preference of that individual. If a species exhibiting variation in habitat preference is at an asymmetric equilibrium, such a correlation will exist. However, if the species is at a stable symmetric equilibrium, which can occur only if there is overdominance in preference, then such a correlation will not exist because gene and genotype frequencies are the same in both habitats. Variation in habitat preference is thus not automatically accompanied by genetic divergence between habitats. In fact, the existence of such divergence might be used as a criterion to determine whether an equilibrium is symmetric or asymmetric.

Second, under a given set of environmental conditions, there may be two or more stable equilibria with very different gene frequencies (e.g., Fig. 1d). Consequently, geographic variation in gene frequencies at loci affecting preference is not necessarily due to geographic variation in environmental conditions (e.g., relative abundances, suitabilities, or carrying capacities of different habitats) as is often assumed (Singer 1971, 1982; Gilbert and Singer, 1975; Fox and Morrow, 1981; Rausher, 1983a).

Viability and Parental Investment at Equilibrium

Fretwell (1972; Fretwell and Lucas, 1970) has suggested that when survivorship within habitats is density-dependent, habitat preference will evolve so as to ensure equal viabilities of offspring in different habitats. This suggestion is based on the assumption that habitat selection behavior produces an "ideal free distribution" of individuals among habitats. Such a distribution is generated if individuals first distribute offspring to the habitat in which viability at low density is highest and offspring continue to be placed in that habitat until increasing density-dependent mortality reduces viability to a value just equal to that of the next-best empty habitat. At that point individuals begin placing offspring in each habitat in such a way as to maintain equal survivorship in the two habitats. While several authors (Taylor, 1975, 1976; Wiens, 1976; Whitham, 1980) have echoed Fretwell's suggestion, it rests on the tenuous assumption that behavior of individuals without perfect knowledge of the distribution of conspecifics among habitats can produce an ideal free distribution (Comins and Hassell, 1979).

The genetic model presented here, which makes no such assumption, substantiates Fretwell's suggestion as long as preference genotypes do not differ in fecundity. Under these conditions, at asymmetric equilibria, which are always stable, the proportion of offspring placed into a habitat equals the proportion of individuals in the mating pool that are derived from that habitat; hence, percent survival is the same in all habitats. When asymmetric equilibria do not exist, viabilities are not equal at the stable symmetric equilibria, but selection moves the system as far toward equalization of viabilities as the genetic constraints allow.

When genotypes differ in fecundity, equalization of viabilities does not occur at any equilibrium. Such equalization would not be expected, however, because then all genotypes would have equal viabilities but different fecundities, and hence different fitnesses. Nevertheless, under conditions represented by (9), a generalization of Fretwell's suggestion remains valid: at equilibrium the total parental investment per surviving offspring is equal in the two habitats, i.e., $E_1/c = E_2/(1 - c)$.

This result is reminiscent of Fisher's (1930) well-known theorem regarding the equalization of parental investment in

males and females at evolutionary equilibrium. In fact, the structure and many of the properties of this model of habitat selection are very similar to those of the model of sex ratio evolution described by Uyenoyama and Bengtsson (1979). The similarities in formal properties of these models describing the evolution of two very different traits presumably derive from a basic underlying similarity in the processes governing gene frequency change in those traits: in both situations, two groups of individuals contribute a fixed proportion of the individuals involved in all matings. In the habitat selection model, the two groups of individuals are those deriving from the two habitats, whereas in any model of the evolution of sex ratio in diploid organisms, the two groups correspond to the two sexes, with each sex contributing equally to the matings, as first pointed out by Fisher (1930). These similarities suggest that models describing the evolution of any other trait sharing this common property will exhibit properties and dynamics similar to those of the two models discussed here.

Sympatric Speciation in Phytophagous Insects

The importance of sympatric speciation in the evolution of phytophagous insects is currently disputed (e.g., see Bush, 1974, 1975; White, 1978; Futuyma and Mayer, 1980; Mayr, 1982). One problem that has impeded acceptance of sympatric speciation as a common phenomenon in natural populations has been the difficulty of envisioning how genetic variation at loci affecting preference and at loci affecting viability on different host plants could exist simultaneously. For example, consider an insect species that oviposits only on host I and that has maximum possible fitness on host I but lower than maximal (though not zero) fitness on host II. If a mutation affecting viability arises such that the mutant genotype has superior fitness on host II, there will be no selection favoring the

mutant allele because feeding and oviposition behavior will not have been altered and the mutant individual will use only host I. Moreover, since the mutant allele will likely have pleiotropic effects lowering fitness on host I (Rausher, 1983a), selection will probably tend to eliminate the very variation that would permit high survivorship on the novel host species. Elimination will usually occur even with independent population regulation on each host, since the conditions for maintenance of a polymorphism (harmonic mean overdominance; Felsenstein, 1976) are rather stringent. In turn, when there arises a mutant allele causing oviposition and feeding on host II, selection will tend to act against this allele because individuals carrying it will have relatively low survival and fecundity on that host. Selection will thus tend to eliminate variability at loci affecting both preference for and viability on a novel host and hence will tend to prevent colonization of such a host, the initial stage of sympatric speciation.

Analysis of the model presented here indicates that this difficulty in putting together variation at two loci diminishes greatly if populations are regulated independently on each host species. As long as fitness on the novel host is not zero, mortality on that host due to genetically based inability to process its foliage ("hard" selection) will be compensated for by the overall density dependence ("soft" selection) in population regulation. Variation in host preference will then often be preserved indefinitely, since the conditions for maintenance of variation in this model are not stringent. Such variation will thus often be present when variation increasing viability on the novel host arises. Once both types of variation are present, preference and viability may evolve together to produce host races differentially adapted to the two host species. Breakdown of coadapted preference-viability gene complexes by recombination may then provide selection pressures favoring development of re-

productive isolation between the host races by a mechanism similar to that described by Felsenstein (1981).

SUMMARY

When density-dependent population regulation occurs independently in different habitats within a mosaic environment, a form of frequency-dependent selection can maintain genetic variation at a single locus affecting habitat preference. The conditions under which variation is maintained are not unduly restrictive. In addition, Fretwell's suggestion that selection modifies habitat preference in such a way as to equalize viabilities in different habitats is true if habitat preference does not affect fecundity. When fecundity is affected, a generalization of Fretwell's suggestion to equalization of parental investment is valid under certain realistic assumptions about how variation in preference affects fecundity. The relevance of the analysis to the processes of host race formation and sympatric speciation in phytophagous insects is discussed briefly.

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APPENDIX 1

In this appendix I derive the stability conditions for the various equilibria in the model. From equations (1) it can be seen that the dynamics of the system can be described in terms of two variables, p and $\bar{p}$, where

$$\bar{p} = \frac{cA}{T_{\mathrm{I}}} + \frac{(1-c)B}{T_{\mathrm{II}}}$$

and $\bar{q} = 1 - \bar{p}$. The recursion equation for $\bar{p}$ is

$$\bar{p}' = c\left[\frac{P_1F_1p\bar{p} + \frac{1}{2}P_2F_2(p\bar{q} + q\bar{p})}{P_1F_1p\bar{p} + P_2F_2(p\bar{q} + q\bar{p}) + P_3F_3q\bar{q}}\right]$$
$$+ (1-c)\left[\frac{(1-P_1)F_1p\bar{p} + \frac{1}{2}(1-P_2)F_2(p\bar{q} + q\bar{p})}{(1-P_1)F_1p\bar{p} + (1-P_2)F_2(p\bar{q} + q\bar{p}) + (1-P_3)F_3q\bar{q}}\right].$$

The stability of any equilibrium is ascertained by examining the eigenvalues of the matrix

$$\begin{pmatrix} \dfrac{\partial p'}{\partial p} & \dfrac{\partial p'}{\partial \bar{p}} \\[2ex] \dfrac{\partial \bar{p}'}{\partial p} & \dfrac{\partial \bar{p}'}{\partial \bar{p}} \end{pmatrix} = \begin{pmatrix} \dfrac{1}{2} & \dfrac{1}{2} \\[2ex] \dfrac{\partial \bar{p}'}{\partial p} & \dfrac{\partial \bar{p}'}{\partial \bar{p}} \end{pmatrix}$$

evaluated at the equilibrium in question. At all equilibria of the system, this matrix is positive.

If the absolute value of both eigenvalues is less than 1, then the equilibrium is locally stable. If the absolute value of either eigenvalue exceeds 1, then the equilibrium is unstable. Since at all equilibria $p = \bar{p}$, $\partial \bar{p}'/\partial p = \partial \bar{p}'/\partial \bar{p}$. The eigenvalues are thus $\frac{1}{2}$ and $\frac{1}{2} + \partial \bar{p}'/\partial \bar{p}$. Since the matrix is positive, $\frac{1}{2} + \partial \bar{p}'/\partial \bar{p}$

is the larger eigenvalue; moreover, by Frobenius' theorem, this eigenvalue is positive. Hence, an equilibrium will be stable if $1 > \frac{1}{2} + \frac{\partial \bar{p}'}{\partial \bar{p}}$, i.e.,

$$\frac{1}{2} > c\left[\frac{P_1F_1p + \frac{1}{2}P_2F_2(1 - 2p)}{P_1F_1p^2 + 2P_2F_2pq + P_3F_3q^2} - \frac{(P_1F_1p^2 + P_2F_2pq)(P_1F_1p + P_2F_2(1 - 2p) - P_3F_3q)}{(P_1F_1p^2 + 2P_2F_2pq + P_3F_3q^2)^2}\right]$$
$$+ (1 - c)\left[\frac{(1 - P_1)F_1p + \frac{1}{2}(1 - P_2)F_2(1 - 2p)}{(1 - P_1)F_1p^2 + 2(1 - P_2)F_2pq + (1 - P_3)F_3q^2}\right.$$
$$\left. - \frac{[(1 - P_1)F_1p^2 + (1 - P_2)F_2pq][(1 - P_1)F_1p + (1 - P_2)F_2(1 - 2p) - (1 - P_3)F_3q]}{[(1 - P_1)F_1p^2 + 2(1 - P_2)F_2pq + (1 - P_3)F_3q^2]^2}\right].$$
$$\text{(A.1)}$$

Boundary Equilibria

For $p = 1$, (A.1) reduces to

$$\frac{-c\beta_1}{(1 - c)\beta_2} > \frac{P_1}{(1 - P_1)} \quad \text{if} \quad [(1 - P_1)F_1 - (1 - P_2)F_2] < 0$$

$$\frac{-c\beta_1}{(1 - c)\beta_2} < \frac{P_1}{(1 - P_1)} \quad \text{if} \quad [(1 - P_1)F_1 - (1 - P_2)F_2] > 0.$$

When $F_1 = F_2 = F_3$, this condition reduces further to

$$0 > (P_2 - P_1)(c - P_1),$$

i.e., the equilibrium with A fixed is stable if $P_1 < c$ and $P_1 > P_2$ or if $P_1 > c$ and $P_1 < P_2$. Similarly, for $p = 0$, (A.1) reduces to

$$\frac{-c\beta_1}{(1 - c)\beta_2} > \frac{P_3}{(1 - P_3)} \quad \text{if} \quad [(1 - P_3)F_3 - (1 - P_2)F_2] < 0$$

$$\frac{-c\beta_1}{(1 - c)\beta_2} < \frac{P_3}{(1 - P_3)} \quad \text{if} \quad [(1 - P_3)F_3 - (1 - P_2)F_2] > 0,$$

and when $F_1 = F_2 = F_3$, the equilibrium is stable if $0 > (P_2 - P_3)(c - P_3)$, i.e., if $P_3 < c$ and $P_3 > P_2$ or if $P_3 > c$ and $P_3 < P_2$.

Symmetric Equilibria

Substituting (6) into (A.1) and simplifying yields the following conditions for stability of the internal symmetric equilibrium, when it exists:

$$\frac{-c\beta_1}{(1 - c)\beta_2} > \tau_s \text{ if } [(1 - P_1)F_1 - (1 - (P_2)F_2] > 0$$

$$\frac{-c\beta_1}{(1 - c)\beta_2} < \tau_s \text{ if } [(1 - P_1)F_1 - (1 - P_2)F_2] < 0,$$

where

$$\tau_s = \frac{(P_1P_3F_1F_3 - P_2^2F_2^2)[(1 - P_1)F_1 + (1 - P_3)F_3 - 2(1 - P_2)F_2]}{(P_1F_1 + P_3F_3 - 2P_2F_2)[(1 - P_1)(1 - P_3)F_1F_3 - (1 - P_2)^2F_2^2]}.$$

Asymmetric Equilibria when (4) is Valid

From (1d) it can be seen that at equilibrium

$$1 = c\left[\frac{P_1F_1p + P_2F_2q}{P_1F_1p^2 + 2P_2F_2pq + P_3F_3q^2}\right] + (1 - c)\left[\frac{(1 - P_1)F_1p + (1 - P_2)F_2q}{(1 - P_1)F_1p^2 + 2(1 - P_2)F_2pq + (1 - P_3)F_3q^2}\right].$$

Moreover, it can be shown easily that

$$P_1F_1p + P_2F_2(1 - 2p) = P_1F_1p + P_2F_2q - \frac{1}{2}P_2F_2,$$

$$(1 - P_1)F_1p + (1 - P_2)F_2(1 - 2p) = [(1 - P_1)F_1p + (1 - P_2)F_2q] - \frac{1}{2}(1 - P_2)F_2,$$

and

$$c\left[\frac{P_2 F_2}{T_1}\right] + (1 - c)\left[\frac{(1 - P_2)F_2}{T_{11}}\right] = 1.$$

Using these relationships, and recognizing that

$$\hat{p}_s = \frac{P_3 F_3 - P_2 F_2}{P_1 F_1 + P_3 F_3 - 2 P_2 F_2},$$

(A.1) reduces to

$$0 > \frac{pq(p - \hat{p}_s)^2}{T_1 T_{11}} \frac{\alpha^2}{\beta^2}[P_1 F_1 - P_2 F_2][(1 - P_1)F_1 - (1 - P_2)F_2], \qquad (A.2)$$

where $\alpha = F_1(P_1 - P_3) - 2F_2(P_2 - P_3)$, $\beta = F_1(P_1 - P_3) - F_2(P_2 - P_3)$.

But q, p, T_1 and T_{11} are always positive, as are the squared terms. Consequently, asymmetric equilibria are stable if and only if

$$0 > [P_1 F_1 - P_2 F_2][(1 - P_1)F_1 - (1 - P_2)F_2]. \qquad (A.3)$$

But it can be shown that if $0 < [P_1 F_1 - P_2 F_2] \cdot [(1 - P_1)F_1 - (1 - P_2)F_2]$, then no internal asymmetric equilibria exist. Consequently, whenever assymetric equilibria exist, (A.3) is satisfied. Hence, whenever an asymmetric equilibrium exists and (6) is true, that equilibrium is stable.