

## The Evolution of Habitat Preference II. Evolutionary Genetic Stability under Soft Selection

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A previous analysis of a single-locus, two-allele model of the evolution of habitat selection revealed that preference evolves toward equalization of parental investment in offspring placed in two different habitats in which population size is regulated independently (soft selection). That analysis is extended here to the case of a single locus with multiple alleles. The major result of this analysis is that regardless of how many alleles are present at equilibrium, new alleles will invade if and only if they tend to make parental investment in the two habitats more equal. In other words, the population evolves toward an Evolutionary Stable State (ESS) characterized by equalization of parental investment. The model may explain the maintenance of genetic variation for host (habitat) preference exhibited by many insect species. © 1987 Academic Press, Inc.

### 1. INTRODUCTION

Several authors have used game-theoretical or optimality analyses to predict how natural selection will mold habitat preference (Levins and MacArthur, 1966; MacArthur and Pianka, 1966; Charnov, 1976; Pyke *et al.*, 1977; Jaenike, 1978; Pyke, 1984). These authors have assumed that behavior associated with habitat selection is plastic, that animals can estimate the relative abundances and suitabilities of various habitats, and that habitat preference is altered to maximize fitness. One principle emerging from these analyses is that when density is regulated independently in different habitats, the optimal behavior is that which causes individuals to distribute themselves or their offspring among habitats in a way that produces equal survivorship in all habitats (Fretwell and Lucas, 1970; Fretwell, 1972).

With good reason, these analyses omit any direct treatment of the evolutionary dynamics of genes that influence behavior. Historically, models of habitat selection have been used to understand the behavior of

mammals, birds and other organisms with highly plastic, complex behavior that does not have an obvious genetic component of variation. In these animals it is not a simple, single behavior that evolves, but a complex behavioral "norm of reaction" (Dobzhansky, 1970), the modelling of which is currently beyond the capabilities of population geneticists. Instead, the optimality approach assumes that natural selection produces flexible behavior that yields the "optimal" (fitness-maximizing) behavior under the most common environmental conditions an organism encounters and attempts to predict the characteristics of that behavior (Dawkins, 1982). This approach is primarily useful in predicting correlations between behavioral responses and environmental conditions.

By contrast with birds and mammals, habitat selection by insects and other invertebrates is often much less plastic. Acceptance or rejection of a particular habitat can be determined by the presence or absence of one or a few token stimuli (Beck, 1965; Rees, 1969; Mitchell, 1977; Feeny *et al.*, 1983) and individual variation in habitat preference often has a strong genetic component (Tabashnik *et al.*, 1981; Wasserman and Futuyma, 1981; Carson and Ohta, 1981; Tavormina, 1982; Sokolowski, 1982, 1985; Sokolowski *et al.*, 1984; Jaenike and Grimaldi, 1983; Lofdahl, 1984). Simple genetic models are thus likely to provide insight into the evolution of habitat preference in these organisms.

Previously Rausher (1984) described a one-locus, two-allele model of the evolution of habitat preference when density-dependent regulation occurs independently in different habitats. Under these conditions, the requirements for maintenance of genetic variation in preference are unrestrictive, suggesting that such variation may be common in nature. Analysis of the model confirmed the prediction from optimality theory that, at evolutionary equilibrium, survivorship in different habitats is equal, as long as there is no indirect effect of habitat preference on fecundity. More generally, it was shown that parental investment per surviving offspring is equalized in different habitats.

Although in many cases the equilibria in the two-allele model are globally stable to perturbation of gene frequencies, it is not clear whether they are stable to invasion by other alleles and thus whether they represent Evolutionary Stable Strategies (*sensu* Maynard Smith and Price, 1973). The purpose of the present study is to determine whether this is the case. We show here that regardless how many preference-determining alleles are present at equilibrium, new alleles will invade if they tend to make survivorship (or, more generally, parental investment) more equal in different habitats and will not invade if they increase the difference in survivorship between habitats. In other words, the ESS for habitat preference is characterized by equalization of survivorship (parental investment) between habitats. The analysis also suggests the number of alleles present at an ESS

is determined by a balance between mutation and random perturbation of gene frequencies due to sampling in finite populations.

## 2. THE MODEL

The model is deterministic and pertains to an animal population whose environment is composed of two habitats or niches. Mated females place offspring in one or both habitats. The offspring develop 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 by a female in each habitat is determined by a single autosomal locus with  $n$  alleles. Females of genotype  $A_i A_j$  place a proportion  $P_{ij}$  of their offspring in habitat I and the remainder in habitat II.

(2) Within a habitat, genotypes have equal viability. In the analytical treatment that follows, it is also assumed that genotypes do not differ in fecundity. Numerical iterations were performed to examine cases in which genotypes differ in fecundity.

(3) 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 (Christiansen, 1975; Felsenstein, 1976).

The recursion equations of the model are derived as follows: Let  $X_{ij}$  be the genotype frequency of  $A_i A_i$  and let  $X_{ij}$  be half the frequency of  $A_i A_j$  in habitat I immediately after oviposition. Similarly, let  $Y_{ij}$  and  $Y_{ij}$  be the corresponding frequencies in habitat II. Then  $X_i = \sum_j X_{ij}$  and  $Y_i = \sum_j Y_{ij}$  are the gene frequencies of allele  $A_i$  in habitats I and II, respectively. Finally, let  $G_{ii}$  be the frequency of  $A_i A_i$  and  $G_{ij}$  be one half the genotype frequency of  $A_i A_j$  in the mating pool.

For any offspring individual in the population, the probability that its mother was  $A_i A_j$  and that the mother transmitted allele  $A_i$  to it is  $G_{ij}$ . Consequently, the probability  $Z_{ij}$  that an individual has  $A_i$  transmitted to it by a mother of genotype  $A_i A_j$  and that the individual is placed in habitat I is

$$Z_{ij} = P_{ij} G_{ij}.$$

The probability,  $T_1$ , that an individual will be placed in habitat I is then

$$T_1 = \sum_i \sum_j P_{ij} G_{ij} = \sum_i Z_i,$$

where  $Z_i = \sum_j Z_{ij}$ . Furthermore, the probability that an offspring will receive allele  $A_k$  from its father is simply equal to the gene frequency of  $A_k$  in the mating pool (gene frequencies are equal in the two sexes),  $p_k$ , which is given by

$$p_k = cX_k + (1 - c) Y_k.$$

Therefore, the probability that an offspring receives  $A_k$  from its father,  $A_i$  from a mother that is genotype  $A_i A_j$ , and is placed in habitat I is simply  $p_k P_{ij} G_{ij}$ . The overall probability that an individual is  $A_i A_k$  and is placed in habitat I is thus  $p_k \sum_j P_{ij} G_{ij} + p_i \sum_j P_{kj} G_{kj}$  (note that the second term is absent if  $i = k$ ). Dividing this by  $T_1$  gives the new genotype frequency in that habitat,

$$2X'_{ik} = \frac{p_k \sum_j P_{ij} G_{ij}}{T_1} + \frac{p_i \sum_j P_{kj} G_{kj}}{T_1},$$

or, noting that  $G_{ij} = cX_{ij} + (1 - c) Y_{ij}$ ,

$$\begin{aligned} X'_{ik} = & \frac{1/2[cX_k + (1 - c) Y_k][\sum_j P_{ij}(cX_{ij} + (1 - c) Y_{ij})]}{T_1} \\ & + \frac{1/2[cX_i + (1 - c) Y_i][\sum_j P_{kj}(cX_{kj} + (1 - c) Y_{kj})]}{T_1}. \end{aligned} \quad (1)$$

This is equivalent to

$$2T_1 X'_{ik} = c(X_k Z_i + X_i Z_k) + (1 - c)(Y_k Z_i + Y_i Z_k).$$

Similarly, the recursion equation for  $Y_{ij}$  is

$$\begin{aligned} Y'_{ik} = & \frac{1/2[cX_k + (1 - c) Y_k][\sum_j (1 - P_{ij})(cX_{ij} + (1 - c) Y_{ij})]}{T_{II}} \\ & + \frac{1/2[cX_i + (1 - c) Y_i][\sum_j (1 - P_{kj})(cX_{kj} + (1 - c) Y_{kj})]}{T_{II}} \\ = & \frac{[cX_k + (1 - c) Y_k][cX_i + (1 - c) Y_i] - T_1 X'_{ik}}{T_{II}}, \end{aligned} \quad (2)$$

where

$$T_{II} = \sum_i \sum_j (1 - P_{ij})(cX_{ij} + (1 - c) Y_{ij}) = 1 - T_1.$$

Recursion equations for gene frequencies are obtained by summing (1) and (2) over  $k$  to yield

$$2T_1 X'_i = c[Z_i + X_i T_1] + (1 - c)[Z_i + Y_i T_1]$$

or

$$X'_i = Z_i/2T_1 + 1/2[cX_i + (1 - c) Y_i] \quad (3a)$$

and

$$Y'_i = \frac{[cX_i + (1 - c) Y_i] - T_1 X'_i}{(1 - T_1)}. \quad (3b)$$

### 3. ANALYSIS OF THE MODEL

At equilibrium the genotype frequencies in the mating pool,  $G_{ij}$ , are in Hardy-Weinberg proportions (see Appendix). It is thus sufficient to determine the conditions under which gene frequencies are at equilibrium in order to characterize the evolutionary equilibria of the model.

At equilibrium,  $X'_i = X_i$  and  $Y'_i = Y_i$ . Substituting these equivalents into (3b) yields

$$(c - T_1) X_i = (c - T_1) Y_i, \quad (4)$$

$$X_i = 1/2[Z_i/T_1 + cX_i + (1 - c) Y_i],$$

and

$$Z_i = T_1[(2 - c) X_i - (1 - c) Y_i].$$

Equation (4) implies two classes of equilibrium exist. One, corresponding to the condition  $c - T_1 = 0$  or  $c = T_1$ , is designated asymmetric because gene frequencies are not equal in the two habitats. The condition  $c = T_1$  implies that survivorship in the two habitats is equal at these equilibria since it implies that the proportion of all offspring placed in habitat I is equal to the proportion of the total mating pool that develops in habitat I. The condition  $c = T_1$  also implies that the set of asymmetric equilibria form an  $n - 1$  dimensional surface in the gene frequency space  $p_1 \times p_2 \times \cdots \times p_n$  (Fig. 1). A second class of equilibria corresponds to the condition  $X_i = Y_i$  for all  $i$  and is designated symmetric because gene frequencies are equal in the two habitats.

In the two-allele case, asymmetric equilibria are always locally stable to perturbation of allele frequencies, whereas symmetric equilibria may be

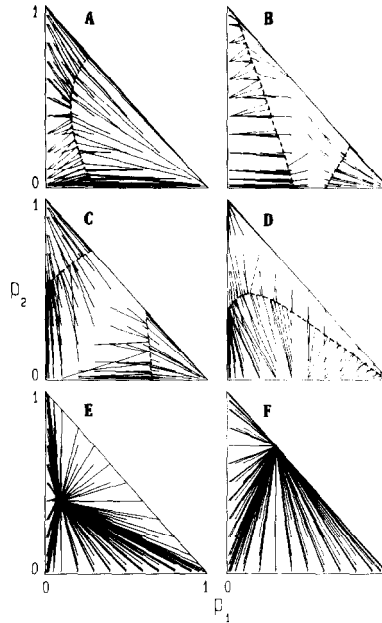


FIG. 1. Examples of equilibria and their stability properties for a locus with three alleles. In all cases depicted the axes are gene frequencies of alleles  $A_1$  and  $A_2$ . Broken lines are asymmetric equilibria. Solid lines connect initial and final (on line of equilibrium or equilibrium point in all cases) gene frequencies for a particular run. They cross only because the true trajectories are not plotted. For all cases portrayed the fecundities were equal. The value of  $c$  was 0.5 for all cases except *D*, for which  $c = 0.4$ . (A) One globally stable line of equilibrium (symmetric). The preference ( $P$ ) values for this run were 0.893, 0.853, 0.178, 0.820, 0.563, and 0.146 for genotypes  $A_1A_1$ ,  $A_1A_2$ ,  $A_2A_2$ ,  $A_1A_3$ ,  $A_2A_3$ , and  $A_3A_3$ , respectively. (B) Two locally stable lines of equilibrium (asymmetric) with an intervening line of unstable equilibrium (symmetric). Preference values are 0.776, 0.052, 0.554, 0.191, 0.840, and 0.797. (C) Similar to (B). Preference values are 0.120, 0.881, 0.261, 0.784, and 0.804. (D) A single locally stable line of equilibrium (asymmetric), and a locally stable equilibrium at  $p_2 = 1.0$ . Preference values are 0.389, 0.155, 0.405, 0.897, 0.281, and 0.547. (E) A globally stable symmetric equilibrium. Preferences are 0.872, 0.612, 0.673, 0.355, 0.354, and 0.652. (F) Globally stable two-allele symmetric equilibrium. Allele  $A_3$  is actively excluded because it fails the criterion of Eq. (7). Preferences are 0.010, 0.494, 0.281, 0.287, 0.277, 0.129.

stable or unstable (Rausher, 1984). For more than two alleles, stability criteria have not yet been determined analytically. However, extensive numerical iterations (see below) suggest that asymmetric equilibria involving three or more alleles are locally stable in the following sense: perturbation of gene frequencies away from the surface of equilibrium points represented by the equation  $c = T_1$  leads to subsequent return of gene frequencies to a point on that surface. Moreover, the smaller the perturbation, the closer the new equilibrium frequencies will be to the pre-perturbation frequencies (Fig. 1).

*Evolutionary Genetic Stability of Asymmetric Equilibria*

In this section we consider the conditions under which  $n$ -allele equilibria are resistant to invasion by an arbitrary new allele, which Eshel and Feldman (1982, 1984) have termed conditions of evolutionary genetic stability. Assume that a new allele  $A_{n+1}$  appears at low frequency. Let  $\varepsilon_i = X_{i,n+1}$  and  $\delta_i = Y_{i,n+1}$  represent half the frequencies of the heterozygote  $A_i A_{n+1}$  in habitats I and II, respectively. Furthermore, let  $\varepsilon = \sum_i \varepsilon_i$  and  $\delta = \sum_i \delta_i$  be the frequencies of  $A_{n+1}$  in the two habitats. Then from (1) and (2) one obtains, ignoring quadratic terms involving  $A_{n+1} A_{n+1}$ ,

$$\varepsilon'_i = \frac{[\sum_j c\varepsilon_j + (1-c)\delta_j] \hat{Z}_i + \hat{p}_i [\sum_j P_{j,n+1} [c\varepsilon_j + (1-c)\delta_j]]}{2T_1} \quad (5)$$

and

$$\delta'_i = \frac{[\sum_j c\varepsilon_j + (1-c)\delta_j](\hat{p}_i - \hat{Z}_i) + \hat{p}_i [\sum_j (1 - P_{j,n+1}) [c\varepsilon_j + (1-c)\delta_j]]}{2(1 - T_1)} \quad (6)$$

for  $i = 1, 2, \dots, n$ .

The new allele will increase in frequency if the absolute value of the largest eigenvalue of the Jacobian of the system (5) and (6) is greater than 1 and decrease if it is less than 1. The Jacobian of this system near an  $n$ -allele asymmetric equilibrium is given in Table I (see Appendix). Its largest eigenvalue equals 1 and all other eigenvalues equal 0 (see Appendix).

This result suggests that an asymmetric equilibrium is neutrally stable to any invading allele. Numerical iterations of equations (3) were performed to determine whether this inference is valid.

A trial consisted of first choosing  $P_{11}$ ,  $P_{12}$ , and  $P_{22}$  randomly, subject to the constraint that  $P_{12}$  lie between  $P_{11}$  and  $P_{22}$  to ensure the existence of a two-allele asymmetric equilibrium. Gene and genotype frequencies at this equilibrium were then calculated using the formula on p. 599 of Rausher (1984) and the Hardy-Weinberg law. Finally, a third allele with values of  $P_{13}$ ,  $P_{23}$ , and  $P_{33}$  chosen randomly from the interval  $[0, 1]$  was then introduced at a frequency of 0.005 and the frequencies at succeeding generations were calculated until a new equilibrium was reached. For each two-allele equilibrium, three trials with different third alleles were performed. A total of 1350 trials were run, 270 at each of five values of  $c$ : 0.5, 0.4, 0.3, 0.2, and 0.1. A trial was considered to have reached an equilibrium when the sum of the absolute values of the differences in genotype frequencies over 10 generations was less than 0.000006.

In every trial gene frequencies converged to values lying on the line

$c = T_1$ . The total change in gene frequency of the new allele was always small, averaging 0.00113 (average of  $|\Delta p|$ ), with a standard deviation of 0.00156. It thus appears that upon introduction of a third allele, deterministic forces cause the population to converge toward a three-allele asymmetric equilibrium, regardless of the  $P$ -values of the third allele.

Because even in large populations random processes will perturb frequencies from this new three-allele equilibrium, further iterations were performed to determine the fate of the new allele upon perturbation. In each of three hundred trials using a value of  $c = 0.5$ ,  $P_{11}$ ,  $P_{12}$ , and  $P_{22}$  were chosen randomly, genotype frequencies were set to those at a two-allele asymmetric equilibrium and a third allele with randomly chosen  $P$ -values was introduced at low frequency. The recursion equations were iterated until convergence occurred. Gene frequencies were then perturbed by adding random numbers over the interval  $[0, 0.2]$  to the frequencies of genotypes involving the third allele. Upon normalization of frequencies, the recursions were iterated again until convergence occurred. For each trial, five such perturbations were performed.

In all trials, gene frequencies reconverged to values lying on the line  $c = T_1$  without elimination of any alleles, though the post-perturbation frequencies were in general different from the pre-perturbation frequencies. Four representative trials are shown in Fig. 2.

These results suggest that the surface  $c = T_1$  acts as an attractor for gene frequencies. Further iterations confirm this suggestion. In these iterations,

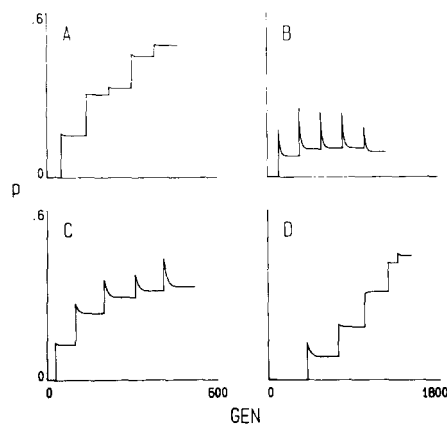


FIG. 2. Response of gene frequency ( $p$ ) of newly-introduced allele to perturbation ( $c = 0.5$ ; see text for details). Abcissa is generation. Vertical lines represent upward perturbation of gene frequency. (A) Preference values: 0.809, 0.158, 0.414, 0.911, 0.156, 0.526. (B) Preference values: 0.915, 0.846, 0.1419, 0.801, 0.772, 0.875. (C) Preference values: 0.052, 0.647, 0.996, 0.775, 0.508, 0.648. (D) Preference values: 0.451, 0.461, 0.696, 0.080, 0.480, 0.960.

the trajectory from each of 120 starting gene frequency combinations to convergence was determined. The starting frequencies were chosen systematically to cover all areas spanned by combinations of  $p_1$ ,  $p_2$  and  $p_3$ .

Of 135 trials (45 trials each with  $c = 0.5, 0.4$ , and  $0.3$ ) performed with randomly chosen  $P$  values, 112 (83%) were characterized by one or two three-allele asymmetric equilibria. In all cases, these equilibria were locally attractive (see Figs. 1a-d, for examples). Of the remaining 23 trials that lacked asymmetric equilibria, 3 were characterized by a globally stable three-allele symmetric equilibrium (see Fig. 1e) and 20 were characterized by the existence of one or more locally stable two-allele symmetric equilibria (Fig. 1f).

The main conclusion arising from this numerical work is that asymmetric equilibria exhibit a neutral stability to invasion by any other allele. The stability is neutral in the sense that neither the novel allele nor any of the original alleles is actively eliminated, nor do any alleles increase greatly in frequency. The only changes in gene frequencies that occur are readjustments that bring frequencies to values that satisfy the condition  $c = T_1$ . Since the surface defined by  $c = T_1$  includes the asymmetric equilibrium to which the novel allele is introduced, gene frequencies immediately after introduction of the novel allele will not lie far from that surface and the readjustments required will cause only small changes in gene frequencies.

#### *Evolutionary Genetic Stability of Symmetric Equilibria*

For a symmetric equilibrium, gene frequencies are equal in the two habitats and the Jacobian of (5), (6) is given in Table II. This matrix has  $2n - 1$  zero eigenvalues and one eigenvalue given by

$$\frac{1}{2} + \frac{1}{2} \sum_i \hat{X}_i [cP_i/\hat{T}_1 + (1-c)(1-P_i)/(1-\hat{T}_1)] - \lambda = 0$$

(see Appendix; note that all terms in the above equation are positive, so that the eigenvalue is also positive). If we let  $N = \sum_i X_i P_i$ , then the condition for evolutionary genetic stability,  $\lambda < 1$ , is

$$(c - \hat{T}_1)(\hat{N} - \hat{T}_1) > 0. \quad (7)$$

In other words, the new allele will invade if and only if the average preference of the heterozygotes involving the new allele ( $N$ ) deviates less in the same direction or at all in the opposite direction from the optimum value  $c: 1 - c$  than the current ratio of offspring placed in the two habitats by all females,  $\hat{T}_1: 1 - \hat{T}_1$ . Under these conditions, an increase in the

frequency of the new allele should bring the system closer toward the condition  $c = T_I$ .

Although this stability analysis gives the conditions under which a new allele can invade a symmetric equilibrium, it does not reveal what the new equilibrium will be. To obtain some indication of the properties of the new equilibrium, we performed additional iterations. For each trial,  $P_{11}$ ,  $P_{12}$ , and  $P_{13}$  were chosen randomly subject to the constraint that a stable two-allele symmetric equilibrium exists. Genotype frequencies were then set to those at that equilibrium and a third allele with randomly chosen  $P$ -values (subject to the constraint in (7)) was introduced at low frequency. Equations (3) were then iterated until convergence occurred. A total of 1350 trials were performed, 270 each at  $c = 0.5, 0.4, 0.3, 0.2,$  and  $0.1$ .

These trials can be divided into four classes. In 55.9% of the trials, gene frequencies converged toward a three-allele asymmetric equilibrium characterized by  $c = T_I$ . In 26.2% of the trials, gene frequencies converged toward a three-allele symmetric equilibrium. In 15.9% of the trials, one of the original alleles was eliminated and gene frequencies converged toward a new two-allele symmetric equilibrium. Finally, in 1.9% of the trials, both original alleles were eliminated as the new allele went to fixation. In all cases, however, the proportions of offspring placed in the two habitats was closer to the optimum ratio  $c : 1 - c$  than they were at the original two-allele equilibrium. In other words, in all cases the system evolved toward the optimal placement of offspring.

#### *Genotypes with Unequal Fecundities*

The previous analysis assumes that genotypes with different preferences have the same fecundity. Although this assumption may often be valid (Rausher, 1984), fecundities may differ if preference for the rarer habitat decreases oviposition rate and hence total number of eggs laid during a female's lifetime.

A biologically interesting special case of unequal fecundities is when fecundities are inversely proportional to total search time, as when searching for one type of habitat precludes searching for others (e.g., Rausher, 1978, 1980; Stanton, 1982). One way to model this situation is to assume that, on average, an individual searches for a time  $S_I$  prior to ovipositing in a patch of habitat I and for a time  $S_{II}$  before ovipositing in a patch of habitat II (Rausher, 1984). The mean search time per egg laid is then, for genotype  $ij$ ,

$$S_{ij} = P_{ij} S_I + (1 - P_{ij}) S_{II} = S_I [P_{ij} + (1 - P_{ij}) \phi],$$

where  $\phi = S_{II}/S_I$ , i.e., the parental search time investment in an egg laid in habitat II expressed in units of search time for an egg laid in habitat I. If all

genotypes live on average a fixed amount of time,  $L$ , then the number of eggs laid is simply  $F_{ij} = L/S_{ij}$  or

$$1/F_{ij} = S_I [P_{ij} + (1 - P_{ij}) \phi] / L = [P_{ij} + (1 - P_{ij}) \phi] / s, \quad (8)$$

where  $s = L/S_I$ . The recursion equations (1) and (2) are then modified by substituting  $P_{ij} F_{ij}$  for  $P_{ij}$  and  $(1 - P_{ij}) F_{ij}$  for  $(1 - P_{ij})$ .

Analysis of this case proceeds as before. Equilibria satisfy the condition

$$X_i(cs - T_I) = Y_i(cs - T_I)$$

(see Appendix). From this it is apparent that there are again two kinds of equilibria: asymmetric, corresponding to the condition

$$cs = \hat{T}_I, \quad (9)$$

and symmetric, corresponding to the condition

$$\hat{X}_i = \hat{Y}_i.$$

Asymmetric equilibria are characterized by the relationship

$$\tau \equiv T_I / (T_I + T_{II}) = c\phi / [c\phi + (1 - c)], \quad (10)$$

where

$$T_I = \sum_i \sum_j P_{ij} F_{ij} [cX_{ij} + (1 - c) Y_{ij}] \quad (11a)$$

$$T_{II} = \sum_i \sum_j (1 - P_{ij}) F_{ij} [cX_{ij} + (1 - c) Y_{ij}]. \quad (11b)$$

This relationship implies that parental search-time investment is equal in the two habitats at asymmetric equilibria (Rausher, 1984). It is derived by noting that (8) and (11) imply

$$T_I + \phi T_{II} = s. \quad (12)$$

Together, (9) and (12) imply

$$c\phi / (1 - c) = T_I / T_{II},$$

from which (10) follows. At symmetric equilibria, the difference between  $\tau$  and  $c\phi / [c\phi + (1 - c)]$  is the smallest possible given the genetic constraints of the system (Rausher, 1984), which is another way of saying that the difference between total parental investment in the two habitats is minimized given the genetic constraints.

Analysis of stability to invasion proceeds as before and yields the result that there is one non-zero eigenvalue for the matrix describing the dynamics of a new allele introduced at an  $n$ -allele equilibrium. This eigenvalue is

$$\lambda = \frac{1}{2} + c \left[ \sum_i \hat{p}_i P_i F_i \right] / 2\hat{T}_I + (1-c) \left[ \sum_i \hat{p}_i (1-P_i) F_i \right], \quad (13)$$

regardless of whether the equilibrium is asymmetric or symmetric.

Consider first the stability of an asymmetric equilibrium to invasion. Together, (10) and (12) imply that

$$c/T_I = 1/s, \quad (14a)$$

$$(1-c)/(1-T_{II}) = \phi/s. \quad (14b)$$

Substituting these into (13) and simplifying yields  $\lambda = 1$ . Consequently, as in the case of equal fecundities, a linear stability analysis does not permit a definite conclusion about stability to invasion by a new allele.

Additional numerical work indicates that in this case of unequal fecundities, asymmetric equilibria exhibit neutral stability to invasion. In a set of 1350 trials, in which a new allele was introduced to a two-allele equilibrium, the system converged to a new 3-allele equilibrium at which (10) and (14) were true, with very little change in gene frequencies. Perturbation of this new equilibrium was followed by convergence to a new 3-allele equilibrium also satisfying (10) and (14). Finally, a set of 135 runs indicated that the line given by (10) is locally attractive whenever it exists. It thus appears that when an arbitrary allele is introduced at an  $n$ -allele asymmetric equilibrium, the system quickly reaches a new  $(n+1)$ -allele asymmetric equilibrium. This in turn implies that (10), and hence equal parental investment in the two habitats, is an evolutionary stable state.

Next consider introduction of a new allele to a symmetric equilibrium. From (13), instability occurs if and only if

$$\frac{1}{2} + csN/2T_I + (1-c)s(1-N)/2T_{II} > 1, \quad (15)$$

where  $N = \sum_i \hat{p}_i P_i / [P_i + (1-P_i)\phi]$ . Since (12) is still valid,  $T_{II} = (s - T_I)/\phi$ , and this, when substituted into (15) and simplified, yields

$$(cs - T_I)(Ns - T_I) > 0. \quad (16)$$

This expression, which is completely analogous to (7), says that a symmetric equilibrium is unstable to invasion by a new allele if and only if  $Ns$ , the average number of offspring placed in habitat I by heterozygote females

carrying the new allele, is closer to the optimal value  $cs$  than is the average number of offspring placed in that habitat by the population as a whole at the original symmetric equilibrium.

A set of 1350 numerical iterations equations indicated that introduction of alleles satisfying (16) resulted in an increase in the frequency of the new allele until a new equilibrium was reached. In every case, the value of  $\tau$  at the new equilibrium was closer to the optimum given by (10) than was true at the original equilibrium and parental investment in the two habitats was more equal.

#### 4. DISCUSSION

The results presented here and earlier (Rausher, 1984; see also Slatkin, 1978) indicate that when population size is regulated independently in two habitats, habitat preference will evolve toward a state of equal survivorship, or, when fecundities of different preference genotypes differ, toward equal parental investment in the two habitats. An intuitive justification for this result can be obtained by considering the case of equal fecundities for all genotypes. At an asymmetric equilibrium, the fitness of all females is equal, since all have the same probability of survival, fecundity, and expected offspring survivorship. Perturbation of gene frequencies from this equilibrium state increases the number of offspring placed in one habitat and decreases the number placed in the other. Since the total number (or proportion) of adults emerging from each habitat is fixed by independent regulation, offspring survivorship is lower in the first habitat than in the second. Consequently, the fitness of genotypes placing a large proportion of their offspring in the first habitat will be lower than that of genotypes placing a smaller proportion of their progeny in that habitat. The latter genotypes will increase in frequency, and the overall proportion of offspring placed in the first habitat will decrease back toward the original equilibrium.

This heuristic argument can also be used to explain the stability properties of equilibria challenged by new alleles. Introduction of a new allele to an asymmetric equilibrium perturbs the population slightly from equal survivorship in the two habitats because individuals carrying new alleles do not (except with infinitesimally small probability) place progeny in the two habitats in equilibrium proportions,  $c:1-c$ . Overall survivorship in one habitat thereby increases, while that in the second decreases. Genotypes placing a greater proportion of their progeny in the first habitat will then be favored and gene frequencies will change until a new asymmetric equilibrium is reached. Even if the preferences ( $P$ -values) of the new allele deviate very far from the "optimal" allocation proportions,  $c:1-c$ , the

allele will not be eliminated because near an asymmetric equilibrium there will be at least one other genotype whose preferences deviate in the same direction from the optimal proportions. Selection will decrease the frequency of this genotype as well as that of genotypes carrying the "bad" new allele and a new equilibrium will be reached before the new allele (or any allele) is eliminated.

At a symmetric equilibrium, by contrast, survivorship is not equal in the two habitats, the fitnesses of all genotypes are not equal, and genetic variation is maintained by a type of heterosis. There exist alleles (those satisfying Eq. (7)) that, at least at low frequency, cause females to place offspring in the two habitats in proportions that deviate less from the optimal  $c:1-c$  ratio than the population does. In effect, these females place their offspring in less-crowded habitats. The survivorship of those offspring is thus greater than that of the average individual in the population and hence the fitness of those females is greater than that of the average female. Selection thus favors an initial increase in the frequency of new alleles satisfying (7), which is accompanied by a decrease in the deviation from the  $c:1-c$  ratio of the population's allocation of progeny to the two habitats. Depending on the preferences of the various genotypes, the population may actually achieve the optimal ratio by reaching an asymmetric equilibrium. The numerical results presented here indicate that once an asymmetric equilibrium, and hence a state of equal survivorship, is reached, further evolution may substitute new alleles and alter the frequencies of preference phenotypes, but will not alter the proportions of offspring placed in each habitat by the population as a whole. Equalization of survivorship thus represents an evolutionary stable state.

#### *Genetic Variation at Equilibrium*

With two alleles, the conditions for maintenance of genetic variation for preference are unrestrictive (Rausher, 1984). Only if the preferences ( $P$ -values) of both homozygote genotypes deviate in the same direction from the ratio  $c\phi:1-c$  and the preference of the heterozygote deviates more in the same direction than that of at least one homozygote will fixation occur.

The present analysis indicates that if mutation can produce a large number of alleles at a locus affecting habitat preference, genetic variation at that locus is virtually inevitable. Consider first a population at an asymmetric equilibrium. That population is already genetically variable. Introduction of a new allele leads to a new equilibrium with one additional allele. However, perturbations (including those of sampling in finite populations) can cause gene frequencies to move off the surface of equilibrium. In general, deterministic forces will return gene frequencies to a different point on that surface. A series of perturbations will thus cause gene frequencies to

wander through the equilibrium surface in a more or less random fashion. Eventually gene frequencies will reach a boundary of the equilibrium surface, corresponding to loss of one or more alleles.

The mean number of alleles present over long periods will probably reflect a balance between the rate at which new alleles are added by mutation and the rate at which they are eliminated by perturbations. A precise determination of the expected number of alleles once this balance has been achieved is beyond the scope of this paper. Nevertheless, by analogy with genetic drift (Kimura and Ohta, 1971), it seems likely that large populations will harbor a larger effective number of alleles than will small populations. In any event, once an asymmetric equilibrium has been reached, selection will prevent the number of alleles from being reduced to less than two by maintaining gene frequencies near the surface of equilibrium, at all points of which there are at least two alleles with non-trivial frequencies (see Fig. 1).

Next consider a population at a two-allele symmetric equilibrium. The iterations presented here suggest that in most cases (>98% of trials), introduction of a new allele either leads to an increase (>82%) or to no net change (>15.9%) in the number of alleles at equilibrium, and hence to maintenance of or an increase in genetic variation. Rarely (<2% of trials), however, a stable, polymorphic symmetric equilibrium can be invaded by a new allele,  $A_i$ , that becomes fixed. This new allele can subsequently be replaced by another new allele,  $A_j$ , which in turn will become fixed as long as  $P_{jj}:1-P_{jj}$  deviates less in the same direction from  $c\phi:1-c$  than do  $P_{ii}:1-P_{ii}$  and  $P_{ij}:1-P_{ij}$  (see Rausher, 1984). This process of "leap-frogging" toward the optimal ratio  $c\phi:1-c$  can continue as long as alleles with the appropriate preferences exist. However, as the optimal ratio is approached, the chances that a newly arising allele,  $A_k$ , will be such that  $P_{kk}:1-P_{kk}$  deviates in the same direction from the optimal ratio as does  $P_{jj}:1-P_{jj}$  will tend to become smaller than the chance that preferences of the new allele will deviate in the opposite direction. Once the latter situation occurs, the population will evolve to a two-allele asymmetric equilibrium. Thus, while invasion of a symmetric equilibrium can occasionally lead to elimination of genetic variability, such elimination will be transitory. Eventually the population will reach a polymorphic, asymmetric equilibrium, at which considerable genetic variation for preference is expected, as described above.

Several recent investigations have indicated that many herbivorous insects are genetically variable for habitat (host) preference (Tabashnik *et al.*, 1981; Wasserman and Futuyma, 1981; Carson and Ohta, 1981; Tavormina, 1982; Sokolowski, 1982, 1985; Sokolowski *et al.*, 1984; Jaenike and Grimaldi, 1983; Lofdahl, 1984). The model described here provides an explanation for this variability and also makes the testable prediction that

the ratio of parental investment in the two hosts should be in the ratio  $c\phi/1-c$ . Whether this explanation and prediction are valid will depend on the appropriateness of the model's assumption that population size is regulated independently on different hosts. Some field evidence suggests this may be the case in mushroom-feeding and cactophytic *Drosophila* that also exhibit genetic variation for habitat preference (Fellows and Heed, 1972; Mangan, 1982; Jaenike and Grimaldi, 1983; Grimaldi and Jaenike, 1984; Lofdahl, 1984), but for other insects too little is known about population regulation to allow any definitive conclusions on the validity of the model's main assumption.

## APPENDIX

*Hardy-Weinberg Frequencies at Equilibrium*

In this section of the appendix I demonstrate that whenever the population is at equilibrium, the genotype frequencies in the mating pool are in Hardy-Weinberg equilibrium.

First define  $V_{ij}$  and  $W_{ij}$  as  $\frac{1}{2}$  the genotype frequencies of  $A_iA_j$  in habitats I and II, respectively after offspring have grown up to be adults but before dispersal. Define  $V_{ii}$  and  $W_{ii}$  as the analogous genotype frequencies of  $A_iA_i$ . Then by the assumption that habitats I and II contribute fractions  $c$  and  $1-c$  to the mating pool, one has

$$G'_{ij} = cV_{ij} + (1-c)W_{ij}. \quad (\text{A1})$$

Since mortality is random with respect to habitats,

$$V_{ij} = X_{ij} \quad (\text{A2a})$$

and

$$W_{ij} = Y_{ij} \quad (\text{A2b})$$

Finally, the frequencies  $X_{ij}$  and  $Y_{ij}$  can be expressed in terms of  $G_{ij}$ ,  $p_i$ , and  $p_j$  as

$$X_{ij} = \frac{1}{2} \left[ \frac{p_i \sum_k G_{jk} P_{jk} + p_j \sum_k G_{ik} P_{ik}}{T_I} \right], \quad (\text{A3a})$$

$$Y_{ij} = \frac{1}{2} \left[ \frac{p_i \sum_k G_{jk}(1-P_{jk}) + p_j \sum_k G_{ik}(1-P_{ik})}{T_{II}} \right]. \quad (\text{A3b})$$

Substituting (A2) and (A3) into (A1) then yields the recursion equation for the genotype frequencies in the mating pool:

$$\begin{aligned} G'_{ij} &= \frac{1}{2}p_i \left[ \frac{c \sum_k G_{jk} P_{jk}}{T_1} + \frac{(1-c) \sum_k G_{jk} (1-P_{jk})}{T_{II}} \right] \\ &\quad + \frac{1}{2}p_j \left[ \frac{c \sum_k G_{ik} P_{ik}}{T_1} + \frac{(1-c) \sum_k G_{ik} (1-P_{ik})}{T_{II}} \right] \\ &= \frac{1}{2}p_i \tilde{p}_j + \frac{1}{2}p_j \tilde{p}_i, \end{aligned} \quad (\text{A4})$$

where  $\tilde{p}_i$  and  $\tilde{p}_j$  symbolize the expressions in brackets. By a similar argument it can be shown that the recursion equation for gene frequency in the mating pool is given by

$$\begin{aligned} p'_i &= \frac{cp_i}{2} + \frac{c[\sum_k G_{ik} P_{ik}]}{2T_1} + \frac{(1-c)p_i}{2} + \frac{(1-c)[\sum_k G_{ik} (1-P_{ik})]}{2(1-T_1)} \\ &= \frac{1}{2}p_i + \frac{1}{2}\tilde{p}_i. \end{aligned} \quad (\text{A5})$$

Whenever genotype frequencies are in equilibrium, then gene frequencies are necessarily in equilibrium and  $p'_i = p_i$ . Equation (A5) then reduces to

$$p_i = \tilde{p}_i$$

and (A4) then reduces to

$$G'_{ij} = p_i p_j.$$

These are the Hardy-Weinberg frequencies.

#### *Evolutionary Genetic Stability*

In this section of the appendix we derive the matrices in Tables I and II and determine the eigenvalues of those matrices. First, differentiating (4) and (5) yields

$$\frac{\partial e_i}{\partial \varepsilon_j} = \frac{c\hat{Z}_i + c\hat{p}_i P_j}{2\hat{T}_1}, \quad (\text{A6})$$

$$\frac{\partial e_i}{\partial \delta_j} = \frac{(1-c)}{c} \frac{\partial e_i}{\partial \varepsilon_j}, \quad (\text{A7})$$

$$\frac{\partial \delta_i}{\partial \delta_j} = \frac{(1-c)(\hat{p}_i - \hat{Z}_i) + (1-c)\hat{p}_i(1-P_j)}{2(1-\hat{T}_1)}, \quad (\text{A8})$$

$$\frac{\partial \delta_i}{\partial \varepsilon_j} = \frac{c}{(1-c)} \frac{\partial \delta_i}{\partial \delta_j}, \quad (\text{A9})$$

where  $P_j$  is used as a short abbreviation for  $P_{j,n+1}$ .

TABLE I  
The Jacobian **A** of Eqs. (5) and (6) Evaluated at Asymmetric Equilibrium

$\hat{T}_1 \hat{X}_1 - \frac{1}{2} \hat{p}_1(c - P_1)$	$\dots$	$\hat{T}_1 \hat{X}_1 - \frac{1}{2} \hat{p}_1(c - P_n)$	$\dots$	$\frac{(1-c)}{c} [\hat{T}_1 \hat{X}_1 - \frac{1}{2} \hat{p}_1(c - P_1)]$	$\dots$	$\frac{(1-c)}{c} [\hat{T}_1 \hat{X}_1 - \frac{1}{2} \hat{p}_1(c - P_n)]$
$\vdots$		$\vdots$		$\vdots$		$\vdots$
$\hat{T}_1 \hat{X}_n - \frac{1}{2} \hat{p}_n(c - P_1)$	$\dots$	$\hat{T}_1 \hat{X}_n - \frac{1}{2} \hat{p}_n(c - P_n)$	$\dots$	$\frac{(1-c)}{c} [\hat{T}_1 \hat{X}_n - \frac{1}{2} \hat{p}_n(c - P_1)]$	$\dots$	$\frac{(1-c)}{c} [\hat{T}_1 \hat{X}_n - \frac{1}{2} \hat{p}_n(c - P_n)]$
$\frac{c}{(1-c)} [\hat{p}_1 - \hat{T}_1 \hat{X}_1 + \frac{1}{2} \hat{p}_1(c - P_1)]$	$\dots$	$\frac{c}{(1-c)} [\hat{p}_1 - \hat{T}_1 \hat{X}_1 + \frac{1}{2} \hat{p}_1(c - P_n)]$	$\dots$	$\hat{p}_1 - \hat{T}_1 \hat{X}_1 + \frac{1}{2} \hat{p}_1(c - P_1)$	$\dots$	$\hat{p}_1 - \hat{T}_1 \hat{X}_1 + \frac{1}{2} \hat{p}_1(c - P_n)$
$\vdots$		$\vdots$		$\vdots$		$\vdots$
$\frac{c}{(1-c)} [\hat{p}_n - \hat{T}_1 \hat{X}_n + \frac{1}{2} \hat{p}_n(c - P_1)]$	$\dots$	$\frac{c}{(1-c)} [\hat{p}_n - \hat{T}_1 \hat{X}_n + \frac{1}{2} \hat{p}_n(c - P_n)]$	$\dots$	$\hat{p}_n - \hat{T}_1 \hat{X}_n + \frac{1}{2} \hat{p}_n(c - P_1)$	$\dots$	$\hat{p}_n - \hat{T}_1 \hat{X}_n + \frac{1}{2} \hat{p}_n(c - P_n)$

TABLE II  
The Jacobian **A** of Eqs. (5) and (6) Evaluated at Symmetric Equilibrium

$[c\dot{X}_v/2][1+P_v/\dot{r}_1]$	$\cdots$	$[c\dot{X}_v/2][1+P_m/\dot{r}_1]$	$\cdots$	$[(1-c)\dot{X}_v/2][1+P_v/\dot{r}_1]$	$\cdots$	$[(1-c)\dot{X}_v/2][1+P_m/\dot{r}_1]$
$\vdots$		$\vdots$		$\vdots$		$\vdots$
$[c\dot{X}_m/2][1+P_v/\dot{r}_1]$	$\cdots$	$[c\dot{X}_m/2][1+P_m/\dot{r}_1]$	$\cdots$	$[(1-c)\dot{X}_m/2][1+P_v/\dot{r}_1]$	$\cdots$	$[(1-c)\dot{X}_m/2][1+P_m/\dot{r}_1]$
$[c\dot{X}_v/2][1+(1-P_1)/(1-\dot{r}_1)]$	$\cdots$	$[c\dot{X}_v/2][1+(1-P_n)/(1-\dot{r}_1)]$	$\cdots$	$[(1-c)\dot{X}_v/2][1+(1-P_1)/(1-\dot{r}_1)]$	$\cdots$	$[(1-c)\dot{X}_v/2][1+(1-P_n)/(1-\dot{r}_1)]$
$\vdots$		$\vdots$		$\vdots$		$\vdots$
$[c\dot{X}_m/2][1+(1-P_1)/(1-\dot{r}_1)]$	$\cdots$	$[c\dot{X}_m/2][1+(1-P_n)/(1-\dot{r}_1)]$	$\cdots$	$[(1-c)\dot{X}_m/2][1+(1-P_1)/(1-\dot{r}_1)]$	$\cdots$	$[(1-c)\dot{X}_m/2][1+(1-P_n)/(1-\dot{r}_1)]$





valuation of the determinant gives

$$0 = (-\lambda)^{2n-1} \hat{X}^{n-1} \left[ \frac{1}{2} + \frac{1}{2} \sum_i X_i (cP_{ij}/\hat{T}_1 + (1-c)(1-P_i)/(1-\hat{T}_1)) - \lambda \right]. \quad (\text{A22})$$

There are thus  $2n-1$  zero eigenvalues and one eigenvalue equal to the expression in brackets. Letting  $N = \sum_i X_i P_i$ , and recognizing that the equilibrium is unstable to invasion if and only if the largest eigenvalue is greater than 1, one obtains Eq. (18) as the conditions for instability.

### *Unequal Fecundities*

In this section, we show that equilibria satisfy the condition

$$X_i(cs - T_1) = Y_i(cs - T_1) \quad (\text{A23})$$

when fecundities are given by (8).

Summing the genotype recursion equations analogous to (1) and (2) over  $k$  and evaluating at equilibrium yields

$$\hat{X}_i = \frac{\sum_j P_{ij} F_{ij} \hat{G}_{ij}}{2\hat{T}_1} + \frac{\hat{p}_i}{2}, \quad (\text{A24a})$$

$$\hat{Y}_i = \frac{\sum_j (1-P_{ij}) F_{ij} \hat{G}_{ij}}{2\hat{T}_{11}} + \frac{\hat{p}_i}{2}, \quad (\text{A24b})$$

where

$$\hat{G}_{ij} = c\hat{X}_{ij} + (1-c)\hat{Y}_{ij}.$$

Isolating the  $\hat{p}_i/2$ , equating (A24a) and (A24b), and substituting  $(s - T_1)$  for  $T_{11}$  (Eq. (12)) produces

$$\begin{aligned} 2\hat{T}_1(s - \hat{T}_1) \hat{X}_i - s \sum_j P_{ij} F_{ij} \hat{G}_{ij} + \hat{T}_1 \sum_j [P_{ij} + (1 - P_{ij})] F_{ij} \hat{G}_{ij} \\ = 2\hat{T}_1(s - \hat{T}_1) \hat{Y}_i. \end{aligned} \quad (\text{A25})$$

But from (A24a),

$$\sum_i P_{ij} F_{ij} \hat{G}_{ij} = \hat{T}_1(2\hat{X}_i - \hat{p}_i). \quad (\text{A26})$$

Substituting this and (8) into (A25) and simplifying then yields (A23).

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