DENSITY-DEPENDENT SELECTION INCORPORATING INTRASPECIFIC COMPETITION. II. A DIPLOID MODEL

MARJORIE A. ASMUSSEN

Department of Mathematics and Department of Molecular and Population Genetics, University of Georgia, Athens, Georgia 30602

Manuscript received May 6, 1982
Revised copy accepted September 27, 1982

ABSTRACT

A diploid model is introduced and analyzed in which intraspecific competition is incorporated within the context of density-regulated selection. It is assumed that each genotype has a unique carrying capacity corresponding to the equilibrium population size when only that type is present. Each genotypic fitness at a single diallelic autosomal locus is a decreasing function of a distinctive effective population size perceived as a result of intraspecific competition. The resulting fitnesses are both density and frequency dependent with selective advantage determined by a balance between genotypic carrying capacity and sensitivity to intraspecific competition. A major finding is that intergenotypic interactions may allow genetic variation to be more easily maintained than in the corresponding model of purely density-dependent selection. In addition, numerical study confirms the possible existence of multiple interior equilibria and that neither overdominance in fitness nor carrying capacity is necessary for stability. The magnitude of the equilibrium population size and optimization principles are also discussed.

ABUNDANT experimental evidence confirms that the operation of selection may be significantly influenced by the size and genetic composition of a population. Although density- and frequency-dependent interactions may each singly be an important factor in the maintenance of genetic variation, it may be commoner for these two phenomena to act together. Indeed, numerous population studies have found that both the kind and numbers of genotypes in a population jointly affect the genotypic fitnesses (e.g., BIRCH 1955, 1960; LEWONTIN 1955; SOKAL and KARTEN 1964; SMOUSE and KOSUDA 1977; TOŠIĆ and AYALA 1981).

The first theoretical treatment of this type of system was given by CLARKE (1972) who considered a completely dominant, diallelic diploid locus in which the selective value of each phenotype depended upon both the total population size and the relative frequencies of the two types. Subsequently, MATESSI and JAYAKAR (1976) and CHRISTIANSEN and LOESCHcke (1980) determined equilibrium properties for special cases of a multi-allelic diploid model with genotypic fitnesses analogous to the growth rates in the classical LOTKA-VOLTERRA competition model. Some general evolutionary principles, applicable to any diploid system in which fitnesses are both frequency and density dependent, have also
now been obtained (NAGYLAKI 1979; SLATKIN 1979). Additional details and references on experimental and theoretical studies of density- and frequency-dependent selection can be found in ASMUSSEN (1983).

In the first paper in the present series (ASMUSSEN 1983), a class of density-dependent haploid selection models was introduced and analyzed in which intergenotypic interactions were explicitly incorporated, thereby adding frequency dependence. Selective advantage in this system is determined by a balance between genotypic carrying capacity and sensitivity to density pressures from the various genotypes. When the density effect of one individual upon another depends upon their respective genotypes, genetic variation may be maintained at a stable interior equilibrium, unlike the corresponding haploid model of purely density-regulated selection. These theoretical findings further confirm experimental evidence of the importance of intraspecific interactions in determining the evolutionary history of a population.

Here, an analogous class of diploid models is presented, which includes as special cases the models of CLARKE (1972) and MATESSI and JAYAKAR (1976). The aim is, again, to determine the conditions under which genetic variation is maintained and to interpret them in terms of the explicit biological parameters of the model. A complete equilibrium analysis is given for the case of complete dominance, which is qualitatively similar to the haploid model with two phenotypes (ASMUSSEN 1983). The investigation of the general model addresses conditions for a protected polymorphism and the issues raised by MATESSI and JAYAKAR concerning the existence of (1) multiple interior equilibria and (2) optimization principles. The conclusions are compared and contrasted with those from the haploid model, as well as the corresponding diploid models, of purely frequency- or density-dependent selection.

THE MODEL

Here a class of models is derived for selection at a single diallelic autosomal locus in a diploid organism with discrete generations. Joint density- and frequency-dependent interactions are taken into account as per ASMUSSEN (1983), with the density effect of an individual of genotype $j$ upon an individual of genotype $i$ weighted by an intraspecific competition coefficient $a_{ij}$. The three genotypes $A_1A_1, A_1A_2, A_2A_2$ are denoted here by the indices 1, 2, 3, respectively.

Let $p_t$ be the frequency of $A_1$, and $N_t$ be the total population size among newly formed zygotes in generation $t$. The zygotes are then subject to viability selection. The surviving adults mate at random with respect to the A locus, all matings having equal fertilities. We assume that the net fitnesses in generation $t$ can be written as

\[
\begin{align*}
\text{Genotype} & \quad \text{Fitness} \\
A_1A_1 & \quad w_1^{(t)} = w_1[p_{1}^2N_t + \alpha_{12}2p_1(1-p_1)N_t + \alpha_{13}(1-p_1)^2N_t] \\
A_1A_2 & \quad w_2^{(t)} = w_2[p_{2}^2N_t + \alpha_{23}2p_2(1-p_2)N_t + \alpha_{23}(1-p_2)^2N_t] \\
A_2A_2 & \quad w_3^{(t)} = w_3[p_{3}^2N_t + \alpha_{32}2p_3(1-p_3)N_t + \alpha_{33}(1-p_3)^2N_t]
\end{align*}
\]
where each $w_i$ is a nonnegative, strictly decreasing, continuously differentiable function on $[0, \infty)$ such that $w_i^{-1}(1) = L_i > 0$. In the analysis that follows the dependence of the genotypic fitnesses upon the total density and genetic composition of the population will frequently be denoted by the compact notation $w_i^{(t)} = w_i(p_t, N_t)$.

Changes in the gene frequency and total population size are governed by the transition equations

\[ p_{t+1} = p_t \left[ p_t w_1(p_t, N_t) + (1 - p_t) w_2(p_t, N_t) \right] / \bar{w}_t \]

\[ N_{t+1} = N_t \bar{w}_t \]

where $\bar{w}_t = \bar{w}(p_t, N_t) = p_t w_1(p_t, N_t) + 2p_t(1 - p_t) w_2(p_t, N_t) + (1 - p_t)^2 w_3(p_t, N_t)$ is the mean fitness of the population in generation $t$.

The fitness formulation (1) is reminiscent of the species interactions in the classic LOTKA-VOLTERRA competition equations (STROBECK 1973) and the genotypic interactions in the frequency-dependent selection model of COCKERHAM et al. (1972). One way to view (1) is that the fitness of an individual of genotype $i$ in generation $t$ is a decreasing function of the effective population size $E_i^{(t)}$ it perceives, where

\[ E_i^{(t)} = [\alpha_{i1} p_t^2 + \alpha_{i2} 2p_t(1 - p_t) + \alpha_{i3}(1 - p_t)^2]N_t \quad i = 1, 2, 3. \]

The class of models (equations 1 to 2) includes those of MATESSI and JAYAKAR (1976) with $w_i(E_i) = r(b - E_i)$, and CLARKE (1972) with $w_i(E_i) = k_i w_i/(k_i + E_i)$ and complete dominance ($w_1 = w_2$ and $E_1 = E_2$).

It should also be noted that if $\alpha_{ij} = \alpha_i \quad i, j = 1, 2, 3$ then (1)–(2) reduces modulo a slight change of notation to the model of purely density-regulated selection in which the genotypic fitnesses are strictly decreasing functions of the total population size (e.g., ASMUSSEN and FELDMAN 1977). To facilitate comparison between the present model and the latter purely density-dependent system, the equations (1)–(2) can be used with the parameters $L_i = w_i^{-1}(1)$ to associate a carrying capacity $K_i$ with each genotype $i$. For instance, the carrying capacity of the homozygote $A_A A_A$ is naturally defined as the equilibrium population size corresponding to fixation for $A_A$, which gives the relations $K_i = L_i/\alpha_{ii}$ $i = 1, 3$. By analogy, the carrying capacity of the heterozygote is defined by $K_2 = L_2/\alpha_{32}$. Note that, when the intraspecific competition coefficients are normalized so that $\alpha_{ii} = 1 \quad i = 1, 2, 3$, the two parameters are identical, namely $K_i = L_i, i = 1, 2, 3$.

The general model given by (1)–(2) is quite complicated and difficult to analyze. The case of complete dominance is considered first, for which a complete equilibrium analysis is possible.

**COMPLETE DOMINANCE**

Suppose that allele $A_2$ is completely dominant to $A_1$ so that $A_1 A_1$ individuals are identical with $A_2 A_2$ individuals in terms of their fitness and interaction parameters. There are then two distinct phenotypes in the population with fitnesses in generation $t$
\[ A_1A_1: w_i^{(\theta)} = w_1[\alpha_{11} p_i^2 N_i + \alpha_{12}(1 - p_i^2)N_i] \]
\[ A_2 - w_2^{(\theta)} = w_2[\alpha_{21} p_i^2 N_i + \alpha_{22}(1 - p_i^2)N_i] \]

where \( w_i^{(\theta)}(1) = L_i = \alpha_{ii} K_i > 0 \) for \( i = 1, 2 \). The transformation (2) applies with \( \bar{w}_i = p_i^2 w_i^{(\theta)} + (1 - p_i^2) w_0^{(\theta)} \). This system is mathematically similar to the haploid case with two genotypes considered previously (ASMUSSEN 1983). The two situations are not equivalent, however, because there are still three distinct genotypes involved in genetic transmission in the diploid model (2)-(3). Nonetheless, the qualitative conclusions are much the same. For instance, as for the haploid case, fixation for \( A_i \) with \( \bar{N} = K_i \) is locally stable if, and only if, (1) \( \alpha_{ii} K_i > \alpha_{ij} K_j \) for the other phenotype \( j \neq i \), and (2) \(-2 < L_i \bar{w}_i'(L_i) < 0\).

From (2) and (3), an interior equilibrium \((\hat{\rho}, \hat{N})\) with \( 0 < \hat{\rho} < 1 \) and \( \hat{N} > 0 \) corresponds to the intersection of the two level curves \( w_1(p, N) = 1 = w_2(p, N) \) in \( \{(p, N): 0 < p < 1 \) and \( N > 0\} \). This gives a unique interior equilibrium

\[ \hat{\rho} = \sqrt{\frac{\alpha_{ii}(K_i - \alpha_{ii} K_j)}{\alpha_{ii}(K_i - \alpha_{ii} K_j) + \alpha_{ii}(\alpha_{ii} K_j - \alpha_{ii} K_i)}} \quad (4a) \]
\[ \hat{N} = \frac{\alpha_{ii}(K_i - \alpha_{ii} K_j) + \alpha_{ii}(\alpha_{ii} K_j - \alpha_{ii} K_i)}{\alpha_{ii} K_i - \alpha_{ii} K_j} \quad (4b) \]

which is admissible if and only if either

\[ \alpha_{ii} K_i > \alpha_{ii} K_j \quad \text{and} \quad \alpha_{ii} K_j > \alpha_{ii} K_i \quad (5a) \]

or

\[ \alpha_{ii} K_i < \alpha_{ii} K_j \quad \text{and} \quad \alpha_{ii} K_j < \alpha_{ii} K_i. \quad (5b) \]

The fact that \( w_1(\hat{\rho}, \hat{N}) = w_2(\hat{\rho}, \hat{N}) = 1 \) illustrates the general result of SLATKIN (1979) that the fitnesses of all phenotypic classes will be equal at a polymorphic equilibrium when the number of phenotypic classes does not exceed the number of alleles. Note that the equilibrium values (4) and existence conditions (5) are precisely those predicted from the corresponding haploid model (ASMUSSEN 1983) if the frequency of haplotype 1 is \( p_1^2 \). A local stability analysis for (2)-(3) in the neighborhood of (4) is, however, more difficult than the haploid case. The details appear in the APPENDIX where it is shown that a set of necessary and sufficient conditions for the existence of a locally stable interior equilibrium is given by

\[ \alpha_{ii} K_i > \alpha_{ii} K_j \quad \text{and} \quad \alpha_{ii} K_j > \alpha_{ii} K_i, \quad (6a) \]
\[ 2 + (B_1 + \hat{\rho}^2)L_1 w_1'(L_1)[2 + (B_2 + 1 - \hat{\rho}^2)L_2 w_2'(L_2)] \]
\[ - (1 - \hat{\rho}^2 - B_1)(\hat{\rho}^2 - B_2)L_1 w_1'(L_1)L_2 w_2'(L_2) > 0, \quad (6b) \]

and

\[ 4 + (B_1 + \hat{\rho}^2)L_1 w_1'(L_1) + (B_2 + 1 - \hat{\rho}^2)L_2 w_2'(L_2) > 0, \quad (6c) \]

where

\[ B_1 = 2\hat{\rho}^2(1 - \hat{\rho})N_1(\alpha_{ii} - \alpha_{ii})/L_1 \quad \text{and} \quad B_2 = 2\hat{\rho}^2(1 - \hat{\rho})N_2(\alpha_{ii} - \alpha_{ii})/L_2. \]
Although the conditions (6b) and (6c) are more complicated than the corresponding haploid criteria (ASMUSSEN 1983), they share many important qualitative features. First, (6) is always satisfied if
\[ \alpha_{11} K_1 > \alpha_{12} K_2, \quad \alpha_{22} K_2 > \alpha_{21} K_1, \]  
(7a)
and
\[ -2 < L_i w'(L_i) < 0 \quad i = 1, 2. \]  
(7b)
As in the haploid model, (7) is, therefore, sufficient for the existence of a locally stable interior equilibrium. Second, if \( L_1 w'_1(L_1) = L_2 w'_2(L_2) \), (7) constitutes both necessary and sufficient conditions. This holds, for instance, when all genotypes have a common birth rate \( r_i = r \quad i = 1, 2 \) in the exponential form \( w_i(E_i) = \exp[r_i(1 - E_i/L_i)] \) and the linear logistic form \( w_i(E_i) = 1 + r_i - r_i E_i/L_i \) for which \( L_i w'_i(L_i) = -r_i \), and the hyperbolic form \( w_i(E_i) = (1 + r_i)/(1 + r_i E_i/L_i) \) for which \( L_i w'_i(L_i) = -r_i/(1 + r_i) \). Third, if \( L_1 w'_1(L_1) \neq L_2 w'_2(L_2) \), (7b) is not in general necessary for stability of (4); if both \( L_i w'_i(L_i) < -2 \quad i = 1, 2 \), however, none of the joint equilibria are locally stable and regular and chaotic cycling can arise. As a final similarity to the haploid model, both fixation equilibria are unstable whenever there is a locally stable interior equilibrium, and the interior equilibrium (4) exists but is unstable whenever both fixation equilibria are stable.

The actual local stability eigenvalues for the haploid and diploid models differ, however, even when \( L_1 w'_1(L_1) = L_2 w'_2(L_2) \). Because the sign of the eigenvalues varies with the exact fitness forms and parameter values, the relative asymptotic rates of approach to the two interior equilibria (when stable) also varies for the two models. Moreover, although satisfaction of the haploid stability criteria may imply the satisfaction of the diploid criteria (6), for other parameter values the converse may hold. This suggests that there may be cases in which the haploid model has a locally stable interior equilibrium but the diploid model does not, or vice versa.

This analysis applies to any fitnesses of the form (3), including the dominant linear logistic case considered by MATESSI and JAYAKAR (1976), and also CLARKE's (1972) hyperbolic model. Since the present results strengthen those for the latter model, an augmented analysis is included here. With \( p \) the frequency of the recessive allele, CLARKE's fitnesses
\[ w_1^{(i)} = \frac{k_2 \omega_2}{k_2 + N_i[\omega_2 p_i^2 + \beta \omega_1 (1 - p_i^2)]} \quad w_2^{(i)} = \frac{k_1 \omega_1}{k_1 + N_i[\alpha \omega_2 p_i^2 + \omega_1 (1 - p_i^2)]} \]  
(8)
(where \( \omega_i > 1 \quad i = 1, 2 \)) are of the form (3) with \( L_1 = k_2(\omega_2 - 1), L_2 = k_1(\omega_1 - 1), L_1 w'_1(L_1) = \omega_2^2 - 1, L_2 w'_2(L_2) = \omega_1^2 - 1, \alpha_{11} = \omega_2, \alpha_{12} = \beta \omega_1, \alpha_{21} = \alpha \omega_2, \) and \( \alpha_{22} = \omega_1 \). CLARKE concluded that the interior equilibrium (4) is locally stable under (8) if \( 0 < \alpha < 1, 0 < \beta < 1, \) and
\[ \alpha < \frac{k_1(\omega_1 - 1)}{k_2(\omega_2 - 1)} \quad \text{and} \quad \beta < \frac{k_2(\omega_2 - 1)}{k_1(\omega_1 - 1)}. \]  
(9)
The criteria (6) applied to the fitnesses (8) show, however, that the condition (9) alone is both necessary and sufficient for stability. Similarly, CLARKE's statement
that any nontrivial equilibrium for the fitnesses \( \mathbf{8} \) is stable holds only under the restriction that \( 0 < \alpha, \beta < 1 \); otherwise, whenever both inequalities in \( \mathbf{9} \) are reversed [so that \( \mathbf{5b} \) holds], the interior equilibrium \( \mathbf{4} \) exists but is unstable.

As a final technical comment, CLARKE's stability analysis is somewhat misleading in that the LYAPUNOV criterion he invoked,

\[
\left( \frac{\partial \Delta p}{\partial p} \right) \left( \frac{\partial \Delta N}{\partial N} \right) > \left( \frac{\partial \Delta p}{\partial p} \right) \left( \frac{\partial \Delta N}{\partial N} \right) \quad \text{and} \quad \left( \frac{\partial \Delta p}{\partial p} \right) + \left( \frac{\partial \Delta N}{\partial N} \right) < 0,
\]

fails as a general stability criterion. Although necessary, the two conditions are not sufficient for local stability in a discrete time model, for they do not preclude an unstable equilibrium due to a (real) local stability eigenvalue less than \(-1\) or a complex eigenvalue with modulus greater than unity. It should be pointed out that these issues were considered by CLARKE (personal communication) even though not explicitly mentioned in his analysis. Nonetheless, because these may be common misconceptions, an outline of the standard local stability analysis is given in the APPENDIX.

When \( \alpha_{ij} = \alpha_i, i, j = 1, 2 \) the general system \( \mathbf{2} \)-(\mathbf{3}) reduces to a purely density-dependent model for which no interior equilibrium is possible with complete dominance (e.g., ASMUSSEN and FELDMAN, 1977). This analysis shows, however, that when intraspecific competition is taken into account and the net density pressure between individuals of unlike genotypes is weaker than that between individuals of like genotype (i.e., \( \alpha_{12} a_{21} < \alpha_{11} a_{22} \)), it is possible to have a stable polymorphic equilibrium in gene frequency and population size. This result is the first indication that intraspecific competition may be an important factor in the maintenance of genetic variation within a density-regulated diploid population.

THE GENERAL MODEL: CONDITIONS FOR A PROTECTED POLYMORPHISM

The general model (\mathbf{1})-(\mathbf{2}) without dominance is much more difficult to analyze. Preliminary insight can be gained from the conditions under which both alleles are protected from loss. This is the case if and only if both fixation equilibria \((p = 1, N = K_1)\) and \((p = 0, N = K_3)\) are unstable. By making a linear approximation to the recursion system \(2\) in the neighborhood of each of these boundary states, the desired conditions can be obtained. In particular, the fixation equilibrium \((p = 1, N = K_1)\) is locally stable if and only if

\[
\alpha_{21} K_1 > \alpha_{22} K_2 \quad \text{(10a)}
\]

and

\[
-2 < L_1 w'_1(L_1) < 0. \quad \text{(10b)}
\]

Similarly, the equilibrium \((p = 0, N = K_3)\) is locally stable if and only if

\[
\alpha_{23} K_3 > \alpha_{22} K_2 \quad \text{(11a)}
\]

and

\[
-2 < L_3 w'_3(L_3) < 0. \quad \text{(11b)}
\]
The extinction line \( \{(p, N) : 0 \leq p \leq 1, N = 0\} \) is always unstable because under the assumptions on the fitness functions, \( w_i(p, 0) > 1 \) for all \( 0 \leq p \leq 1 \) \( i = 1, 2, 3 \). Consequently, it follows from (10) and (11) that both alleles will be maintained in the population if

\[
\alpha_{21} K_1 < \alpha_{22} K_2 \quad \text{and} \quad \alpha_{33} K_3 < \alpha_{22} K_2. \tag{12}
\]

The conditions (12) can also be viewed as sufficient for the increase of each allele when rare. When \( \alpha_{ij} = \alpha_i, i, j = 1, 2, 3 \), (12) reduces to the familiar requirement of overdominance in carrying capacity. When intraspecific competition is taken into account, the protection conditions take on a different character. As is the case with complete dominance, selective advantage is then determined by a balance between carrying capacity and sensitivity to density pressures, through competition with individuals of other genotypes. For example, a new rare allele may not be able to enter the population even though the resulting heterozygote has the largest carrying capacity if the heterozygote is more adversely affected by density pressures from the homozygotes than the homozygotes are themselves. In particular, \( A_2 \) cannot successfully invade a population fixed for \( A_1 \) when \( K_1 < K_2 \) and \( -2 < L_1 w'_i(L_1) < 0 \) if \( \alpha_{21} > \alpha_{22} \) and \( \alpha_{21} K_1 > \alpha_{22} K_2 \). On the other hand, if \( \alpha_{21} < \alpha_{22} \) or \( \alpha_{23} < \alpha_{22} \), invasion by a new allele may be possible even though the resulting heterozygote has a lower carrying capacity than the original homozygotes. This would be the case if the heterozygote is sufficiently insensitive to competitive density effects from the homozygotes. In this sense, it can be easier for a new allele to successfully enter the population when density pressures resulting from competition between individuals of unlike genotypes are weaker than those between like individuals.

**INTERIOR EQUILIBRIA**

Due to the complexity of the general model (1)–(2), a full equilibrium analysis has not been possible. Certain additional insights can be obtained, however, from further analytic investigations. As for the purely density-dependent model (e.g., Asmussen 1979a) a joint interior equilibrium in \( p \) and \( N \) corresponds to the intersection of the two level curves,

\[
v_1(p, N) = pw_1(p, N) + (1 - p)w_2(p, N) = 1 \tag{13a}
\]

and

\[
v_2(p, N) = pw_2(p, N) + (1 - p)w_3(p, N) = 1, \tag{13b}
\]

on which the marginal fitnesses of the two alleles are one. For general fitnesses of the form (1), the conditions (13) require the solution of a complicated pair of simultaneous equations in the two variables \( (p, N) \). Paralleling the case of complete dominance, there is a unique solution satisfying \( w_i(\hat{p}, \hat{N}) = 1, i = 1, 2, 3 \) given by

\[
\hat{p} = \sqrt{\frac{\Delta_i}{\Delta_1 + \Delta_2 + \Delta_3}} \quad \text{and} \quad \hat{N} = \frac{\Delta_1 + \Delta_2 + \Delta_3}{\Delta},
\]
where $\Delta = [\alpha_{ij}]$ is the determinant of the matrix of intraspecific competition coefficients, and for $i = 1, 2, 3 \Delta_i$ is the determinant of the matrix obtained after replacing the $i$th column of $\alpha = [\alpha_{ij}]$ by the column vector $(\alpha_{11}K_1, \alpha_{22}K_2, \alpha_{33}K_3)^T$. This determines an admissible interior equilibrium if and only if each determinant $\Delta_i, i = 1, 2, 3$ has the same (nonzero) sign and $|\Delta_2| = 2 \sqrt{\Delta_1\Delta_3}$. These conditions are very restrictive and, consequently, such a solution is not expected to be of general applicability.

Since all other interior equilibria satisfy $w_i(\hat{p}, \hat{N}) \neq 1$ all $i = 1, 2, 3$, it will not usually be possible to obtain explicit equilibrium solutions from (13) for the general model (1)-(2). By considering the nature of the two level curves (13), however, it is possible to derive sufficient conditions for the existence of an interior equilibrium. Since the fitness functions $w_i$ are continuously differentiable monotonic functions, the implicit function theorem assures us that each of the equations $v_i(p, N) = 1 = v_2(p, N)$ determines $N$ as a function of $p$. In particular, for $i = 1, 2 v_i(p, N) = 1$ determines $N = N_i(p)$ as a continuously differentiable function of $p$ for all gene frequencies in $[0, 1]$. By inspection of the intercepts of each (implicit) function $N_i(p)$ at the boundaries, $p = 0$ and $p = 1$, it is clear from continuity that there will be at least one interior equilibrium when either

$$\alpha_{21}K_1 < \alpha_{22}K_2 \quad \text{and} \quad \alpha_{23}K_3 < \alpha_{22}K_2$$

(15a)

or

$$\alpha_{21}K_1 > \alpha_{22}K_2 \quad \text{and} \quad \alpha_{23}K_3 > \alpha_{22}K_2.$$  

(15b)

These criteria show that, when the density pressures between individuals depend on their respective genotypes, a polymorphic equilibrium may occur even when the heterozygote's carrying capacity is equal or intermediate to those of the two homozygotes. Furthermore, a comparison with (12) reveals that the conditions for a protected polymorphism are sufficient to guarantee the existence of an interior equilibrium. From (10) and (11) there will also always be at least one interior equilibrium when both fixation states are stable. Determining whether or not such an equilibrium is stable is a more difficult issue. When all intergenotypic interactions are of the same magnitude, as in the purely density-regulated model, the maintenance of both alleles at a stable equilibrium requires that the heterozygote be overdominant in carrying capacity. For the general model, simple stability conditions for interior equilibria are not readily derived analytically.

The exact number of equilibria is also not readily determined for general fitnesses of the form (1). When $\alpha_{ij} = \alpha_{i}, i, j = 1, 2, 3$ the two functions $N = N_i(p)$ representing the two level curves in (13) are monotonic functions of $p$ and intersect at most once, yielding an (unique) interior equilibrium if and only if there is overdominance ($K_2 > K_1, K_3$) or underdominance ($K_2 < K_1, K_3$) in carrying capacity (Asmussen and Feldman 1977). When genotypes differ in their intraspecific competitive effects, the level curves are no longer necessarily monotonic, and it is conceivable that there may be multiple intersections in $(0, 1)$ and, hence, as suggested by Mateassi and Jayakar (1976), multiple interior equilibria,
as has been found in the corresponding model of purely frequency-dependent interactions (Cockerham et al. 1972). This would result in a profound change in population dynamics as compared with the purely density-regulated system. The potential is most evident for the simple linear logistic fitmesses

\[ w_i(\tilde{a}_iN) = 1 + r_i - r_i\tilde{a}_iN/L_i \quad i = 1, 2, 3 \]  

where

\[ \tilde{a}_i = \alpha_{i3}p^2 + \alpha_{i2}2p(1 - p) + \alpha_{i3}(1 - p)^2 \quad i = 1, 2, 3. \]  

Then (13) determines a quartic polynomial in the gene frequency \( p \), which reduces to a cubic equation when all three genotypes have a common birth rate \( r_i = r \). From (2b) the equilibrium population size corresponding to a given polymorphic equilibrium gene frequency \( \delta \) is given by

\[ \hat{N} = \frac{p^2r_1 + 2\hat{p}(1 - \hat{p})r_2 + (1 - \hat{p})^2r_3}{p^2\tilde{a}_1r_1/K_1 + 2\hat{p}(1 - \hat{p})\tilde{a}_2r_2/K_2 + (1 - \hat{p})^2\tilde{a}_3r_3/K_3} \]

These fitnesses can, therefore, have up to four interior equilibria.

**Numerical Investigations**

The existence, multiplicity and stability of interior equilibria were investigated by performing numerical iterations of the system (1)-(2) in which the three genotypes had either exponential fitmesses

\[ w_i(\tilde{a}_iN) = e^{a_i(1 - \tilde{a}_iN/L_i)} \quad i = 1, 2, 3 \]  

or hyperbolic fitmesses

\[ w_i(\tilde{a}_iN) = \frac{1 + r_i}{1 + r_i\tilde{a}_iN/L_i} \quad i = 1, 2, 3 \]  

where \( \tilde{a}_i \) is defined in (17). Attention was restricted to symmetric competition matrices satisfying \( a_{ij} = a_{ji} \) and \( \alpha_{ij} \leq 1 = \alpha_{ii} \) all \( i, j = 1, 2, 3 \) and fitness functions that allowed a stable population size regulating mechanism (i.e., \(-2 < L_iw'_i(L_i) < 0 \) \( i = 1, 2, 3 \)). Within each category, as determined by how many fixation equilibria are stable, the actual number of interior equilibria found depended on whether the genotypic carrying capacities were overdominant (i.e., \( K_2 > K_1, K_3 \)), intermediate (i.e., \( K_1 < K_2 < K_3 \) or \( K_3 < K_2 < K_1 \)) or underdominant (i.e., \( K_2 < K_1, K_3 \)). The results for each of the three categories are summarized by the representative numerical examples in Table 1. (Note that the assumption \( \alpha_{ij} \leq \alpha_{ii} \) limits the possible relative orders of the genotypic carrying capacities in the cases of one or two stable fixation equilibria.)

Only stable interior equilibria were detectable by numerical interations of (1)-(2). The results, however, motivate certain speculations about the full equilibrium structure. For example, the lack of a stable interior equilibrium in examples with both fixation states stable (together with the analysis of the last section) suggests that in these cases there is a unique interior equilibrium that is unstable.
TABLE 1

Stable interior equilibria (β,  NONINFRINGEMENT) for the exponential (18) and hyperbolic (19) fitnesses with birth rate parameters r₁ = 1.0, r₂ = 0.75, r₃ = 0.5 and symmetric intraspecific competition coefficients satisfying α₁₁ = α₁₂ = α₃₃ = 1

<table>
<thead>
<tr>
<th>K₁</th>
<th>K₂</th>
<th>K₃</th>
<th>α₁₂</th>
<th>α₁₃</th>
<th>α₂₃</th>
<th>Exponential fitnesses</th>
<th>Hyperbolic fitnesses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both fixation equilibria unstable:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O: 70</td>
<td>100</td>
<td>85</td>
<td>0.75</td>
<td>0.50</td>
<td>0.65</td>
<td>(0.3445, 114.2383)</td>
<td>(0.3532, 114.1737)</td>
</tr>
<tr>
<td>I: 70</td>
<td>80</td>
<td>100</td>
<td>0.75</td>
<td>0.50</td>
<td>0.65</td>
<td>(0.1492, 110.4423)</td>
<td>(0.1462, 110.3821)</td>
</tr>
<tr>
<td>U: 125</td>
<td>100</td>
<td>140</td>
<td>0.75</td>
<td>0.50</td>
<td>0.65</td>
<td>(0.1011, 148.7734)</td>
<td>(0.0811, 148.3073)</td>
</tr>
<tr>
<td>&amp;</td>
<td>&amp;</td>
<td>&amp;</td>
<td>&amp;</td>
<td>&amp;</td>
<td>&amp;</td>
<td>(0.8965, 131.3227)</td>
<td>(0.8616, 132.9719)</td>
</tr>
</tbody>
</table>

-One fixation equilibrium stable (A₂):
| I: 70 | 80 | 100 | 0.75 | 0.50 | 0.90 | None | None |
| U: 125 | 100 | 140 | 0.75 | 0.50 | 0.80 | (0.9044, 130.8789) | (0.8639, 131.8680) |

-Both fixation equilibria stable:
| U: 125 | 100 | 140 | 0.90 | 0.50 | 0.80 | None | None |

The examples are grouped first according to the number of locally stable fixation equilibria and then as to whether the genotypic carrying capacities are overdominant (O), intermediate (I), or underdominant (U).

In general, unstable interior equilibria are expected to exist to separate the domains of attraction of each pair of stable equilibria, with a pattern similar to that found in the analogous model of purely frequency-dependent interactions (Cockerham et al. 1972).

The number of stable interior equilibria found ranged from zero to two (see Table 1), with the maximum achieved only when both fixation equilibria were unstable and the genotypic carrying capacities underdominant. The numerical analysis, therefore, confirms the possible existence of multiple interior equilibria when the density pressures of one individual upon another depend on their respective genotypes. The examples with both fixation states unstable also reveal the important fact that the population size at a stable interior equilibrium can exceed the maximum genotypic carrying capacity when intraspecific competition between like genotypes is stronger than that between unlike genotypes.

The equilibrium structures for the exponential and hyperbolic models always agreed, except for one fundamental difference. It is possible to have no stable equilibria in the exponential case when the birth rates become too large. In this case cycling in pᵣ and Nᵣ is observed, as under the purely density-dependent model (Asmusson 1979b). The linear logistic fitnesses (16) were not used extensively because of their potential negativity problems (Asmusson and Feldman 1977). Numerical iterations of (2) for these fitnesses under the particular parameter sets in Table 1, however, produced the same qualitative results as found for the exponential and hyperbolic fitnesses. Furthermore, in each case numerical solutions of (13) with the linear logistic fitnesses (16) confirmed the conjectures made about the total number of interior equilibria by revealing an unstable interior equilibrium in the third, fifth and sixth examples given in Table 1.
INTERGENOTYPIC COMPETITION

OVERDOMINANCE PRINCIPLES

The results point out that the existence and stability conditions for a polymorphic equilibrium can be significantly different when intraspecific competition is taken into account in a density-dependent framework. From (13), the genotypic fitnesses may all be equal at an interior equilibrium, with \( w_1(\hat{p}, \hat{N}) = 1 \) for \( i = 1, 2, 3 \) as in the completely dominant system (3). Otherwise, the fitnesses at a polymorphic equilibrium are everdominant with \( w_1(\hat{p}, \hat{N}), w_3(\hat{p}, \hat{N}) < 1 < w_2(\hat{p}, \hat{N}) \), in which case

\[
\frac{\alpha_1K_1}{\hat{a}_1}, \frac{\alpha_3K_3}{\hat{a}_3} < \hat{N} < \frac{\alpha_2K_2}{\hat{a}_2}, \tag{20a}
\]

or underdominant with \( w_2(\hat{p}, \hat{N}) < 1 < w_1(\hat{p}, \hat{N}), w_3(\hat{p}, \hat{N}) \), so that

\[
\frac{\alpha_2K_2}{\hat{a}_2} < \hat{N} < \frac{\alpha_1K_1}{\hat{a}_1}, \frac{\alpha_3K_3}{\hat{a}_3}. \tag{20b}
\]

When \( \alpha_2 = \alpha_i \) \( i = 1, 2, 3 \) and selection is purely density regulated, stability of an interior equilibrium requires that the heterozygote have maximum fitness at the equilibrium (ASMUSSEN 1979a). This is equivalent to (20a) which reduces to the familiar condition of overdominance in carrying capacity when fitnesses are strictly decreasing functions of the total population size (ASMUSSEN and FELDMAN 1977). Both of these principles are violated when the density pressure of one individual upon another depends on their respective genotypes; the numerical examples with exponential and hyperbolic fitnesses given in Table 1 shows that a stable interior equilibrium is possible with the heterozygote inferior in fitness and inferior or intermediate in carrying capacity.

We have seen that the relative order of the three genotypic carrying capacities appears to determine the actual number of equilibria within each of the categories based on the number of stable fixation equilibria. These same examples suggest that the relative order of the carrying capacities also determines whether the heterozygote has the lowest or highest fitness at a stable interior equilibrium. In all of the cases in which the heterozygote had the largest carrying capacity, forcing both boundaries to be unstable, the heterozygote had the largest fitness at the unique stable interior equilibrium and (20a) held. When the heterozygote was intermediate or inferior to both homozygotes in carrying capacity, the heterozygote always had the lowest fitness at all stable interior equilibria and (20b) held.

OPTIMIZATION PRINCIPLES

Constant viability selection at a single autosomal locus always acts to maximize the mean fitness in the population. This is not the case for models of purely density-dependent selection and holds only for highly symmetric cases of frequency-dependent selection (e.g., COCKERHAM and BURROWS 1971; COCKERHAM et al. 1972). An important optimization principle does hold for purely density-regulated fitnesses which are strictly decreasing functions of the total population size; the equilibrium population size is always maximized at a stable
equilibrium (ASMUSSEN and FELDMAN 1977; GINZBURG 1977; NAGYLAKI 1979). It is readily shown that this is not necessarily the case for either the present diploid or corresponding haploid model (ASMUSSEN 1983) that incorporate intraspecific competition.

From a study of linear logistic fitnesses, MATESSEI and JAYAKAR (1976) suggested that natural selection in the context of density dependent intraspecific competition tends instead to reduce the average competition,

$$\tilde{\alpha} = p^2 \tilde{\alpha}_1 + 2p(1-p)\tilde{\alpha}_2 + (1-p)^2 \tilde{\alpha}_3,$$

perceived by the population. Their model assumed that all three genotypes had a common birth rate and carrying capacity parameter, as well as symmetric competition coefficients. For the general model of complete dominance (3) and the multi-allelic haploid model (ASMUSSEN 1983), the average competition will be minimized by selection when the genotypes have a common fitness parameter $L_i = L$ and symmetric intraspecific competition coefficients ($\alpha_{ij} = \alpha_{ji}$). This is not usually the case, however, for either the diploid model (1) or the analogous haploid model. Indeed, if $\alpha_{ij} < \alpha_{ii} = 1$ so that competition between individuals of different genotypes is weaker than that between like individuals, natural selection will always cause the average competition to eventually increase (to one) if fixation is approached from an interior point. These results show that, when genotypes differ both in their carrying capacities and their sensitivity to competition from other genotypes, neither the equilibrium population size nor the average intraspecific competition will generally be optimized. Instead, selection may strike a balance between these two optima, much as selective advantage is determined by a balance between the genotypic carrying capacities and their sensitivity to density pressures from other individuals.

**DISCUSSION**

Biological evidence suggests that genotypic fitnesses may often depend jointly upon both the kinds and numbers of individuals present. This may occur because genotypes differ in their sensitivity to intraspecific density pressures from competition with other genotypes within the same population. In the first paper of this series (ASMUSSEN 1983), a class of density-regulated haploid models was introduced and analyzed that take such interactions into account. Here an analogous class of diploid models was developed in which the density effect of an individual of genotype $j$ upon an individual of genotype $i$ is weighted by an intraspecific competition coefficient, $\alpha_{ij}$. The formulation is reminiscent of the species interactions in the classical LOTKA-VOLTERRA competition system. The fitness of a given genotype is assumed to be a decreasing function of the effective population size it perceives as a result of competitive interactions with the other individuals in the population. As a result, the genotypic fitnesses are functions of both the size and genetic composition of the population.

A significant discovery from numerical examples with exponential, hyperbolic and linear logistic fitnesses is that the population size at a stable interior equilibrium may exceed all three genotypic carrying capacities and, hence, is larger than any possible under purely density-regulated selection. This arises
when density pressures between individuals of different genotypes are weaker than those between like genotypes, causing the effective population size perceived by each genotype to be less than the absolute total number. As a consequence, the environment is able to support a larger sized population than when all intergenotypic interactions are of equal strength. In general, as for arbitrary regimes that are both frequency and density dependent (Slatkin 1979), the equilibrium population size is no longer maximized by selection. Neither is the average intraspecific size minimized except in very special symmetric situations. Instead, selection may be expected to strike a balance between these two optimizing trends.

The single most important conclusion from these investigations is that intergenotypic interactions strongly influence the genetic structure of a population. With intraspecific competition incorporated in a density-dependent framework, selective advantage is determined by a balance between genotypic carrying capacity and sensitivity to density pressures from interactions with other genotypes. Analysis shows that it can be easier for a new allele to enter a population if competition between individuals of different genotypes is weaker than that between like individuals. The conditions for the maintenance of two alleles through a protected polymorphism indicate, moreover, that genetic variation is more apt to be preserved when genotypes differ both in their carrying capacities and in their response to intraspecific competition.

Numerical and analytic investigations show that the increased potential for genetic polymorphism found with the incorporation of intergenotypic interactions is due to an equilibrium structure closely paralleling that of purely frequency-dependent models (e.g., Haldane and Jayakar 1963; Clarke and O'Donald 1964; Cockerham et al. 1972). For instance, unlike the corresponding model of purely density-regulated selection, there may be a stable interior equilibrium even though the heterozygote is not superior in carrying capacity and even though fixation for one or both alleles is stable. Both alleles may even be maintained under a completely dominant selection regime if the net competitive effect between individuals of unlike genotypes is weaker than that between individuals of like genotypes. Furthermore, multiple stable interior equilibria may exist under the general model (1)-(2) without dominance. Numerical examples with exponential, hyperbolic and linear logistic fitnesses produced up to three interior equilibria with up to two locally stable. In cases characterized by the number of stable fixation states, the exact number of polymorphic equilibria appears to depend upon the relative order of the genotypic carrying capacities.

Another feature of the present model, which is true of frequency-dependent but contrasts with purely density-regulated systems, is that the heterozygote does not always have the highest fitness at a stable interior equilibrium. In the only cases in which we would expect a stable polymorphic equilibrium on the basis of the corresponding density-dependent model, namely, those with overdominance in carrying capacity, the usual condition of heterozygote superiority in fitness held at the equilibria. In the present system stable interior equilibria may exist at which the heterozygote instead has the lowest fitness as well as
under complete dominance. The former only (and always) occurred in numerical examples in which the heterozygote was inferior or intermediate in carrying capacity. It is interesting that these are precisely those cases peculiar to the intraspecific competition model and for which genetic variation could not be maintained at a stable equilibrium with purely density-dependent selection. Unlike some frequency-dependent models (e.g., Wright 1955), however, it is not possible to maintain genetic variation at an equilibrium where the heterozygote is intermediate in fitness between the two homozygotes.

Together the results from the diploid and analogous haploid model (Asmusen 1983) show that the likelihood of genetic polymorphism is greatly increased when intraspecific competition is incorporated within a density-regulated framework. The findings confirm that the joint presence of density- and frequency-dependent interactions commonly found in actual populations (e.g., Lewontin 1955; Smouse and Kosuda 1977) may well be an important factor for the maintenance of genetic variation.

Research supported in part by National Science Foundation grants DEB 78-09397 and DEB 82-00664. The author has been advised that an independent investigation of this model with emphasis on the linear logistic fitness functions will soon be published by her colleagues, Wyatt Anderson and Jonathan Arnold.

**LITERATURE CITED**


Matessi, C. and S. D. Jayakar, 1976 Models of density-frequency dependent selection for the
intergenotypic competition


Corresponding editor: B. S. Weir

APPENDIX: LOCAL STABILITY ANALYSIS

The conditions for local stability of a joint equilibrium \( \mathbf{x} = (\mathbf{x}_1, \mathbf{x}_2, \ldots, \mathbf{x}_n) \) of the system of \( n \) independent equations

\[
\mathbf{x}_{i+1}^{(r+1)} = f_i(x_1^{(r)}, x_2^{(r)}, \ldots, x_n^{(r)}) \quad i = 1, 2, \ldots, n
\]

are that the \( n \) eigenvalues of the matrix

\[
\left\{ \frac{\partial f_i}{\partial x_j} (\mathbf{x}) \right\}_{i,j = 1}^n
\]

all have magnitude less than unity. For a joint gene frequency-population size equilibrium \((\mathbf{p}, N)\) of the two dimensional system

\[
\begin{align*}
\mathbf{p}_{i+1} &= f_i(p_i, N_i) \\
N_{i+1} &= f_d(p_i, N_i)
\end{align*}
\]

this reduces to the requirement that both roots of the quadratic equation

\[
f(A) = A^2 - A\lambda + B = 0
\]

lie in \((-1, 1)\) if both are real, or have modulus less than one if both are complex, where

\[
A = \frac{\partial f_1}{\partial p} + \frac{\partial f_2}{\partial N} \quad \text{and} \quad B = \frac{\partial f_1}{\partial p} \frac{\partial f_2}{\partial N} - \frac{\partial f_2}{\partial p} \frac{\partial f_1}{\partial N}
\]

are evaluated at the joint equilibrium \((\mathbf{p}, N)\).

From the properties of quadratic functions it follows that the equilibrium \((\mathbf{p}, N)\) is locally stable if and only if either

\[
(1) \quad A^2 - 4B \geq 0, \quad f(1) = 1 - A + B > 0, \quad f(-1) = 1 + A + B > 0, \quad \text{and} \quad -2 < A < 2; \quad (A3)
\]

or

\[
(2) \quad A^2 - 4B < 0 \quad \text{and} \quad B < 1. \quad (A4)
\]

Although the stability criteria (A3)-(A4) are easily specified, they are difficult to interpret when \( A \) and \( B \) are complicated functions of the various parameters of the model. The stability of an interior equilibrium \((\mathbf{p}, N)\) of the general diploid model (1)-(2) is a case in point.
When $A_2$ is completely dominant to $A_1$ as in (3), however, a complete analysis is possible. For an interior equilibrium (4) satisfying (5a) or (5b) the coefficients of the characteristic equation (A1)-(A2) are

$$A = 2 + (B_1 + \beta \delta) L_1 w_1' (L_1) + (B_2 + 1 - \beta \delta) L_2 w_2' (L_2)$$

(A5)

$$B = 1 + (B_1 + \beta \delta) L_1 w_1' (L_1) + (B_2 + 1 - \beta \delta) L_2 w_2' (L_2) + (B_2 + B_2) L_1 w_1' (L_1) L_2 w_2' (L_2)$$

(A6)

where

$$B_1 = 2 \beta \delta (1 - \pi) \tilde{N}(a_{11} - a_{12}) / L_1$$

and

$$B_2 = 2 \beta \delta (1 - \pi) \tilde{N}(a_{22} - a_{22}) / L_2.$$

The model assumes that $w_i'(L_i) < 0$ for $i = 1, 2$ and $a_{ij} \geq 0$ for $i, j = 1, 2$. The analysis hinges on the set of inequalities

$$- \beta \delta < B_1 < 1 - \beta \delta \quad \text{(A7)}$$

$$- (1 - \beta \delta) < B_2 < \beta \delta \quad \text{(A8)}$$

which follow from the equilibrium relations $L_i = \tilde{N} [a_{1i} \beta \delta + a_{2i} (1 - \beta \delta)] / L_i$ when $a_{ij} \geq 0$.

As in the haploid model (ASMUSSEN 1983), both eigenvalues are real since (A7)-(A8) show that the discriminant,

$$A^2 - 4B = [(B_1 + \beta \delta) L_1 w_1'(L_1) - (B_2 + 1 - \beta \delta) L_2 w_2'(L_2)]^2 + 4(1 - \beta \delta - B_1) (\beta \delta - B_2) L_1 w_1'(L_1) L_2 w_2'(L_2),$$

is always positive under the assumptions of the model. Applying the stability criterion (A3), we find $f(1) = 2 \beta \delta (1 - \pi) \tilde{N} (a_{11} a_{22} - a_{12} a_{22}) w_1'(L_1) w_2'(L_2)$ is positive if and only if $a_{12} a_{21} < a_{11} a_{22}$. Comparison with the existence conditions (5) reveals that (5a) is necessary for stability; under (5b) the interior equilibrium (4) is automatically unstable because then $f(1) < 0$. Next, the inequalities (A7)-(A8) applied to (A5) show that we always have $A < 2$. The remaining criteria, $f(-1) > 0$ and $A > -2$ are satisfied if and only if (6b) and (6c) hold, respectively. It follows that the conditions (6a)-(6c) are necessary and sufficient for the existence of a locally stable interior equilibrium. Furthermore, the relations (A7)-(A8) show that (6a) together with $-2 < L_i w_i'(L_i) < 0$ for $i = 1, 2$ are sufficient for stability. When $L_i w_i'(L_i) = L_i w_i'(L_i)$ the latter set of conditions is both necessary and sufficient for then the two eigenvalues are given explicitly by

$$\lambda_1 = 1 + 2 \beta \delta (1 - \pi) \tilde{N} (a_{11} a_{22} - a_{12} a_{22}) L_1 w_1'(L_1) / L_1 L_2$$

$$\lambda_2 = 1 + L_1 w_1'(L_1).$$

(A9)