DENSITY-DEPENDENT POPULATION GROWTH AND NATURAL SELECTION IN FOOD-LIMITED ENVIRONMENTS: THE *DROSOPHILA* MODEL

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The theory of density-dependent natural selection is perhaps the most successful union of population genetics and ecology. This union has had a long history. The evolutionary consequences of different types of population-size regulation were discussed by Dobzhansky (1950), and population-genetics models in which fitness depends on total population size were developed by Wright (1960). However, MacArthur and Wilson (1967) were the first to explore systematically the consequences of evolution at extreme densities and to derive a theoretical framework for life-history evolution.

The heuristic arguments of MacArthur and Wilson were formalized in a number of studies in which population size and allele frequencies at a single locus were explicitly modeled (Anderson 1971; Charlesworth 1971; King and Anderson 1971; Roughgarden 1971; Clarke 1972). The most common approach in these modeling efforts was to assume that the fitnesses of alternative genotypes were linearly decreasing functions of total population size. The slope and intercept of these linear functions were determined by the parameters r and K, which varied between genotypes. The models predict that for populations kept at low densities, well below carrying capacity, natural selection favors genotypes with the highest value of r; hereafter, this is called r-selection. In populations at carrying capacity, genotypes with the highest value of K are favored; this is called K-selection. Clearly, if the genotype with the highest r also has the highest K, then the outcome of evolution does not depend on the population's ecology. However, if there exists a trade-off between r and K traits, then the outcome of evolution depends critically on population ecology. In populations kept continually at low densities, high-r and low-K genotypes will predominate because of the assumed trade-off, whereas the opposite will be true in populations close to saturation densities.

If this theoretical framework is correct, then three predictions follow. Populations that have evolved in low- and high-density environments should be differentiated with respect to density-dependent rates of population growth. The low-density population should have higher rates of population growth at low densities relative to the high-density population; just the opposite should be true at high

population densities. These predictions, which are most closely tied to the formal theory of r- and K-selection, have been tested only twice. Results contrary to expectations were observed in *Escherichia coli* (Luckinbill 1978), but *Drosophila melanogaster* (Mueller and Ayala 1981a) yielded consistent results.

The formal theory of density-dependent natural selection has been associated, unfortunately, with an expansive verbal theory (Pianka 1970; review in Stearns 1977). The verbal theory attempts to identify phenotypes that are causally related to r and K and then use the predictions concerning the evolution of these parameters to predict the evolution of these correlated phenotypes. Thus, the theory of r- and K-selection is considered by many to make specific predictions concerning the evolution of phenotypes such as body size, age-specific survival and fecundity, and developmental time (Taylor and Condra 1980; Barclay and Gregory 1981; Luckinbill 1984).

Much of the logic behind the verbal theory is found to be faulty upon closer examination. For instance, the verbal theory predicts that K-selection should favor an iteroparous life history and r-selection should favor a semelparous life history. Yet, this prediction is at odds with some formal models of selection in age-structured populations, in which natural selection at carrying capacity is predicted to favor increases in fecundity and survival at earlier ages (Charlesworth 1980, p. 211). Models that lead to the evolution of different age-specific life histories require high juvenile survival relative to adult survival or vice versa, rather than the presence or absence of density-dependent population regulation (Murphy 1968; Charnov and Schaffer 1973). Whether natural selection favors high fecundity and low juvenile survival or low fecundity and high juvenile survival depends on which life stage is subject to density-dependent regulation, rather than on the mere presence or absence of density dependence (Iwasa and Teramoto 1980).

Given these substantial problems with the verbal theory of r- and K-selection, it is reasonable to ask if the formal theories that assume logistic fitness functions can be used to predict the evolution of phenotypes other than density-dependent rates of population growth. The answer is probably no. To develop a theory that accounts for the evolution of body size or competitive ability, models with the relevant ecological phenomena must be developed. The drawback of such a modeling process is that the relevant ecological details probably differ between taxa.

It has become increasingly clear that very general models of population dynamics, like the logistic model, are inappropriate for certain types of organisms. For instance, many populations of sessile marine invertebrates are limited by free space on which larvae may settle. Qualitatively different models are needed to describe the dynamics of such populations (Roughgarden et al. 1985). Likewise, the dynamics of many plant populations require models that account for the competitive effects of close neighbors (Pacala and Silander 1985). Attempts to model density-dependent natural selection in variable environments with the logistic and related models have yielded odd results. For instance, depending on the model of population dynamics used, natural selection could cause a population to become more stable or less stable (Turelli and Petry 1980; Mueller and Ayala

1981*b*). Christiansen (1984) has argued that evolutionary models should use explanatory models of population dynamics rather than phenomenological models such as the logistic. These explanatory models attempt to incorporate important events in the life history of an individual.

In light of these considerations, the present paper develops a model of population dynamics and evolution in food-limited environments by taking into account many details of *Drosophila* ecology. These models may be applicable to insects and animals other than *Drosophila*; however, since the ultimate goal is to test the theoretical predictions with data from *Drosophila*, the discussion focuses on this genus, particularly *D. melanogaster*.

In addition to limiting available food, crowding larvae and adult *Drosophila* has a variety of consequences, such as increased concentrations of wastes (Botella et al. 1985) and limitation of suitable pupation sites (Chiang and Hodson 1950; De Souza et al. 1970; Mueller and Sweet 1986). This study concentrates on the effects of food limitation since a great deal is known about the effects of food on fitness components, and experimentally, food is a variable that is relatively easy to control.

THE ECOLOGICAL RECURSION

The properties of density-dependent population growth in food-limited environments are investigated first. Where possible, the ecological components of this model are illustrated by observations of laboratory populations of *Drosophila melanogaster*. The origin and maintenance of these populations is described elsewhere (Mueller and Ayala 1981*a*). The details of the collection of the experimental results will be included in a later paper describing tests of the present theory.

Viability

The effects of food availability on viability, or the survival from newly hatched larva to adult, has been studied in painstaking detail by Bakker (1961). Models of viability in food-limited environments consistent with Bakker's results have been developed by de Jong (1976) and Nunney (1983). Nunney's model, which accurately describes his own data on the viability of different genotypes and sexes of *Drosophila melanogaster*, is adopted in its entirety; it will also be described fully because it is a crucial component of the total model.

Assume a population that reproduces and has resources renewed at discrete intervals. Any excess food at the end of a generation is assumed lost. Let n_t be the number of eggs present at time t. Let the fraction of eggs that hatch to become young larvae and compete for food be V. If the total amount of food available for the Vn_t larvae is constant, say B mg, then each larva receives an average of B/Vn_t mg of food. As a result of environmental or perhaps genetic differences between individuals, the actual amount of food consumed by a single individual may be more or less than the average available to the population. In fact, let the distribution of food consumed by individuals follow a normal distribution with mean B/Vn_t and variance $(B/Vn_t)^2\sigma^2$, as shown in figure 1. Individuals must consume at least m
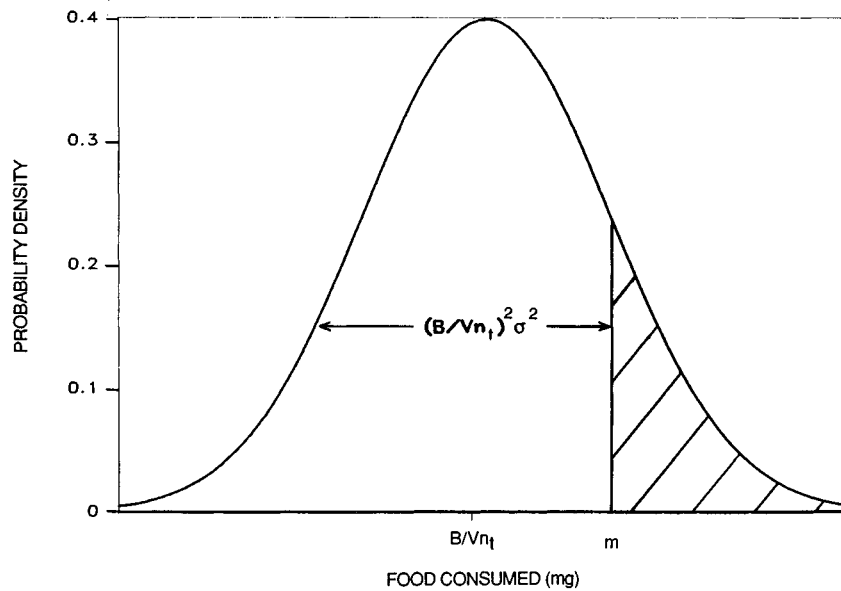
Fig. 1.—The distribution of food consumed by Vn_t larvae in an environment with B mg of food. The density-independent viability of eggs is V. Only larvae that consume more than m mg of food survive.

mg of food to pupate successfully. Thus, the viability of the Vn_t larvae is proportional to the hatched area of the curve.

Transform food consumed into standard units, y, by subtracting the mean and dividing by the standard deviation in figure 1. Then, the viability, $W(Vn_t)$, of young larvae is given by

$$W(Vn_t) = \int_x^\infty \phi(y)\,dy, \tag{1}$$

where $x = [(mVn_t/B) - 1]/\sigma$, and $\phi(y)$ is the unit-normal density function, $(2\pi)^{-1/2}\exp(-y^2/2)$.

The proportion of 100 newly hatched first-instar larvae that survive to become adults has been determined at a variety of food levels. The techniques used in these experiments are similar to those of Nunney (1983). Results from experiments on one population carried out on three separate occasions are shown in figure 2. Predicted viabilities from equation (1) for three different values of m are also shown. The parameter values used were chosen as biologically reasonable and do not represent least-squares estimates from the data in figure 2. The figure illustrates that equation (1) is sufficiently flexible to describe empirical results accurately. Decreasing the value of m increases viability, especially when food is in short supply. When food is abundant, nearly all larvae survive, except those that die from density-independent factors accounted for by V, and decreasing m has negligible effects on viability.
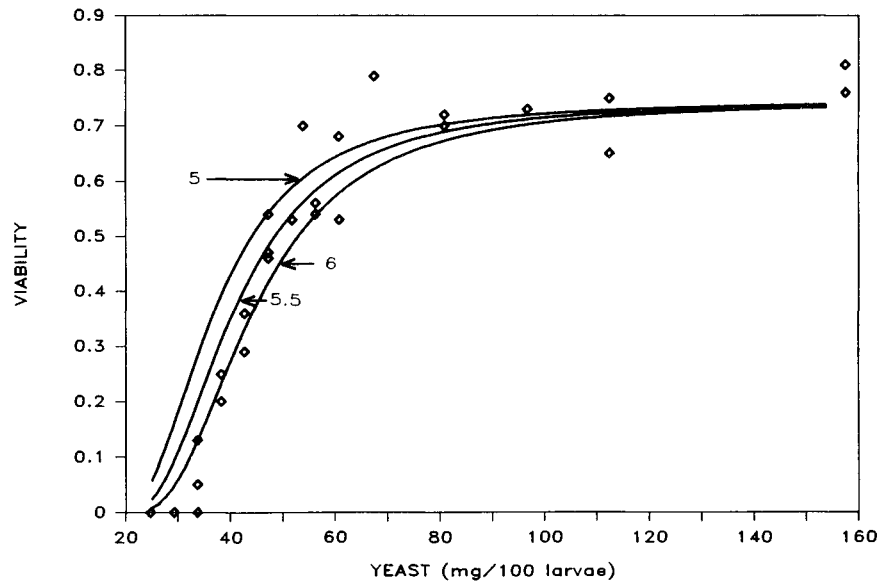
Fig. 2.—Each diamond represents the fraction of 100 first-instar larvae that survived to become adults at various food levels. The data are for a $K - 1$ population (described in Mueller and Sweet 1986). The curves are predicted viabilities from equation (1) for $V = 0.75$, $\sigma = 0.35$, and three values of m in units of 10^{-4} g.

Larval Competition and Female Fecundity

Increased competition among larvae results in a reduction in the size of the emerging adults, as well as in a reduction in viability (Marks 1982). This reduced size is presumably due to the limitation of available food (Bakker 1961). This effect is important to the dynamics of a population (Prout and McChesney 1985), since smaller females lay fewer eggs (Chiang and Hodson 1950; Robertson 1957; Mueller 1987).

The relationship between female size and food consumed is readily apparent from studies in which food level is carefully controlled (Bakker 1961). Figure 3 shows the average thorax lengths of females emerging from the same experiments reported in figure 2. It is evident from figures 2 and 3 that the effects of limited food on female size are expressed over a greater food range than are the effects on viability. Thus, when 100 larvae are provided ≥ 70 mg of yeast, viability remains unaffected (see fig. 2) but female size continues to increase substantially.

Assume that the function $s(b)$ describes the size of adult females that have consumed b mg of food. Although all larvae that consume more than m mg of food survive, it is evident from figure 1 that they do not all consume the same amount of food. Thus, there should be considerable variation in female size when food is limiting. The average size of the emerging females, $\bar{s}(Vn_t)$, can be predicted using the distribution of food consumed by surviving larvae as

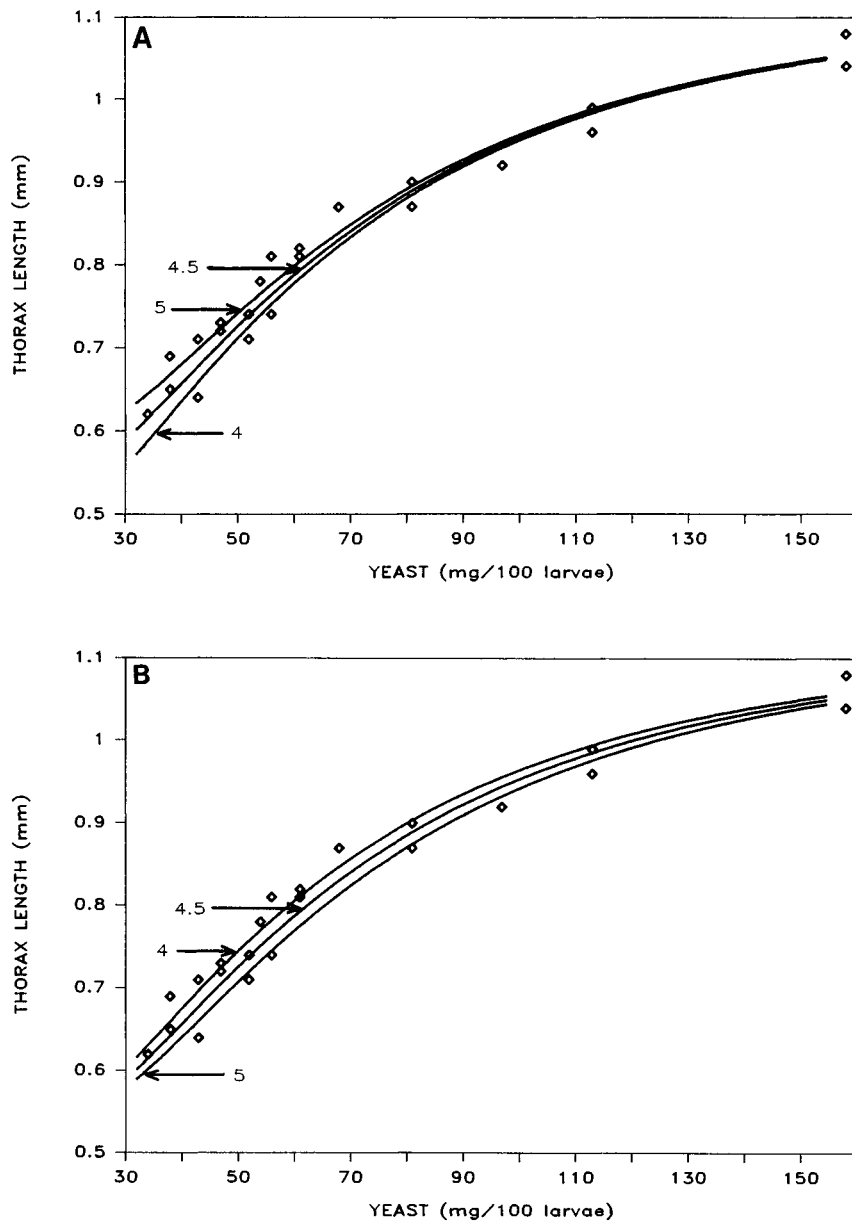$$\bar{s}(Vn_t) = W^{-1}(Vn_t) \int_x^\infty s[B(\sigma y + 1)/Vn_t]\phi(y)dy. \tag{2}$$

Fig. 3.—Each diamond represents the average thorax length of adult females from the experiment described in figure 2: A, model 1; B, model 2. The curves represent the predictions from equation (2) with $s(b) = a_0 + a_1\{1 - \exp[-a_2(b - m)]\}$, where $a_0 = 0.5$, $a_1 = 0.623$, $a_2 = 1700$, and the remaining constants are as in figure 2. Curves are shown with various values of m (units of 10^{-4} g).

One question, for which no empirical evidence exists, concerns the relationship between the minimum amount of food necessary for successful pupation, m, and female size. I have decided to model this relationship in two different ways, although it is not unreasonable to assume that the actual behavior of *Drosophila* populations lies somewhere in between. In the first case, called model 1, assume that all populations have the same $s(b)$ function, and the ability to pupate successfully on less food is thus accomplished by reducing the size of the resulting adult (see fig. 4A). Model 2 assumes that the minimum adult size is the same for all populations. Thus, populations that require less food to pupate successfully do so by processing food more efficiently. The $s(b)$ curves for populations with different m's in model 2 are just rigid translations of a single curve (see fig. 4B). In figure 3A, the three curves are the predicted average sizes of females from equation (2) and model 1 for the same three values of m used in figure 2. The particular function used in figure 3 is actually a modification of the functional response of a predator exhibiting satiation (Roughgarden 1979, p. 443). This function reaches a minimum adult size when the food consumption is m and asymptotically approaches a maximum adult size. It appears to give an adequate description of the empirical data and has been used in all numerical work in this study. Figure 3B shows the predicted values for model 2. In figure 3A the bottommost curve is due to the smallest value of m, whereas in figure 3B it is due to the largest. The increased viability that results when m is reduced is due to the survival of very small flies in model 1. Thus, the average size of surviving females actually decreases with decreasing m even though viability increases (see fig. 3A). Model 2 behaves quite differently. Viability still increases with decreasing m, but average female size increases (see fig. 3B). This occurs because, under model 2, decreasing m implies increasing efficiency; thus, even though the amounts of food consumed by each female remain unchanged, populations with smaller m turn that food into additional biomass.

To complete this section, a function is needed to translate female size into fecundity: $f[s(b)]$. The mean fecundity of all females, $\bar{f}(Vn_t)$, is given by

$$\bar{f}(Vn_t) = W^{-1}(Vn_t) \int_x^\infty f\{s[B(\sigma y + 1)/Vn_t]\}\phi(y)\,dy. \tag{3}$$

Figure 5 shows the total number of eggs laid during the first week of life as a function of female size for the $K - 1$ population. The curve drawn in figure 5 is the least-squares approximation for a line through the data transformed by taking the log of the egg numbers and the thorax length. These data were collected using single male-female pairs in 8-dram vials that were transferred daily. Even the smallest females (thorax length, 0.68 mm) can lay about 20 eggs per day. Such high fecundity is incompatible with population stability in these models, and hence the effects exerted by adults on female fecundity (see next section) play an important role in population stability.

Adult Density and Female Fecundity

A number of studies have documented dramatic declines in female fecundity with increasing adult density (Pearl 1932; Bodenheimer 1938; Robertson and Sang
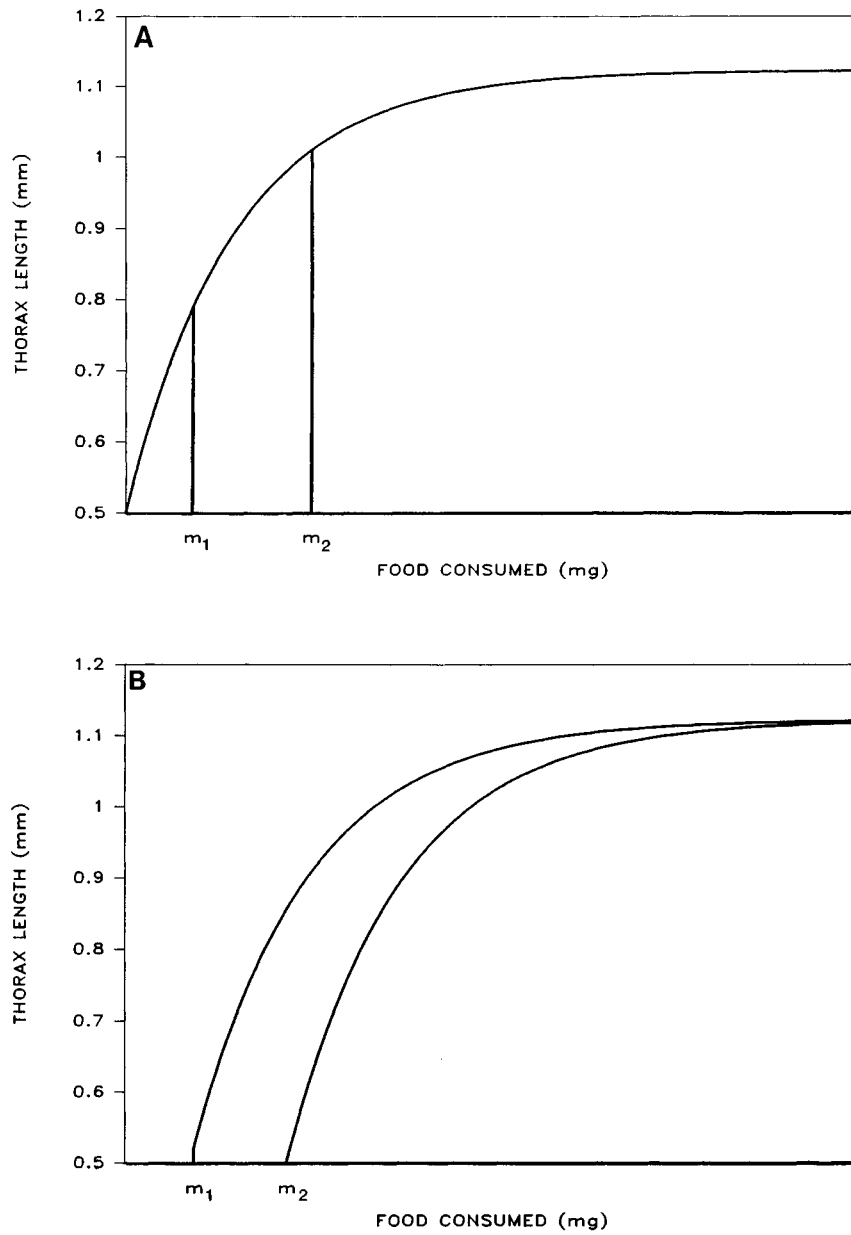
Fig. 4.—Two possible relationships between minimum food and minimum adult size: in A, model 1, smaller values of m imply that larvae are able to pupate at a smaller size; in B, model 2, the minimum adult size is constant, and smaller m is achieved by greater larval efficiency.
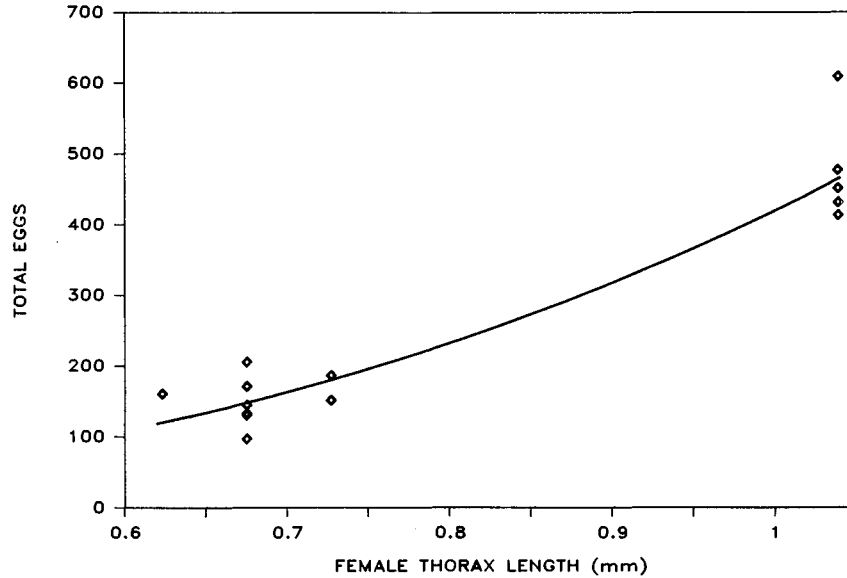
FIG. 5.—Total number of eggs laid during the first week of adult life by females from the
$K - 1$ population as a function of thorax length. The curve is predicted from the relationship
ln(egg number) = 6.041 + 2.644 ln(thorax length).

1944; Chiang and Hodson 1950). A consistent observation has been that female
fecundity declines most rapidly at very low adult densities, whereas at moderate
to high densities changes in female fecundity are almost imperceptible (Prout and
McChesney 1985; Mueller 1986). Even this slight density dependence at high
densities can have substantial consequences for population stability and hence
should not be ignored (Mueller 1986). Let $G(N_t)$ reflect the effects of adult density,
N_t, on female fecundity. By convention, let $G(2) = 1$. In the numerical work that
follows, a one-parameter hyperbolic function will be used: $G(N_t) = (1 + 2a)/(1 +
aN_t)$. In figure 6, relative fecundity is shown as a function of adult density. An
important feature of this function is the rapid decline in fecundity at relatively low
densities.

The Recursion for Egg Numbers

Results from the previous sections can now be used to construct a first-order
nonlinear difference equation for the number of eggs at time $t + 1$:

$$n_{t+1} = \frac{1}{2}G(N_t) \int_x^\infty f\{s[B(\sigma y + 1)/Vn_t]\}\phi(y)\,dy\,Vn_t, \tag{4}$$

where adult numbers at time t are given by

$$N_t = W(Vn_t)Vn_t. \tag{5}$$

Equation (4) is multiplied by $\frac{1}{2}$ since only half the adult population contributes
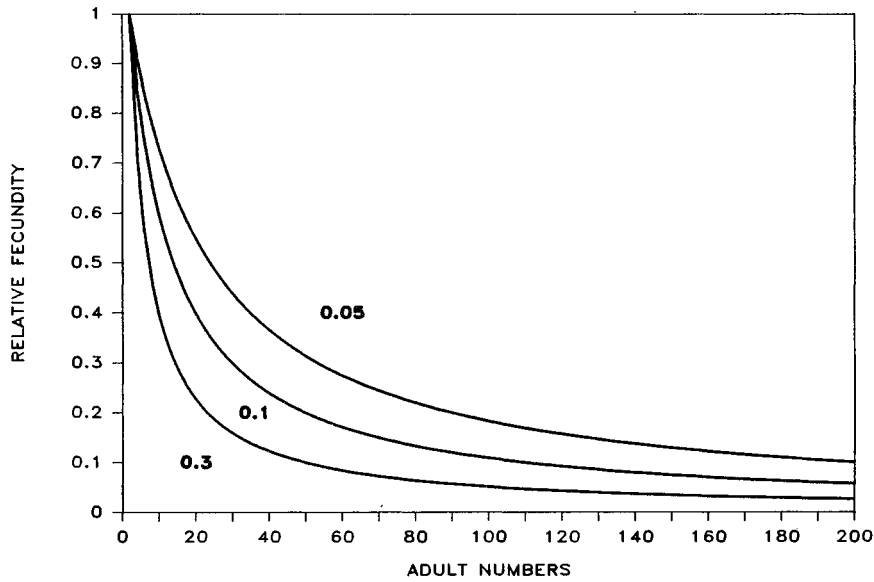eggs to the next generation.

Fig. 6.—Relative fecundity as a function of adult density predicted by the hyperbolic model for three different values of the parameter a.

The equilibrium number of eggs in the population, $\hat{n}$, may be defined implicitly as

$$\hat{n} = \tfrac{1}{2}G[W(V\hat{n})V\hat{n}] \int_{\hat{x}}^{\infty} f\{s[B(\sigma y + 1)/V\hat{n}]\}\phi(y)\,dy\,V\hat{n}, \tag{6}$$

where $\hat{x} = (mV\hat{n}/B - 1)/\sigma$. The stability of the equilibrium requires that $|\lambda| < 1$, where

$$\lambda = 1 + \hat{n}G'(\hat{n})/G(\hat{n}) + \hat{n}VG(\hat{n})H'(\hat{n})/2,$$

$$H'(\hat{n}) = \int_{\hat{x}}^{\infty} f'\{s[B(\sigma y + 1)/V\hat{n}]\}\phi(y)\,dy - f\{s[B(\sigma\hat{x} + 1)/V\hat{n}]\}\phi(\hat{x})mV/B\sigma,$$

and prime denotes differentiation with respect to n_t. It has proved difficult to derive a simplified expression for $\hat{n}$ and λ even when a simple approximation to the normal density function such as $1 + \cos(x)$ ($-\pi \le x \le \pi$) is used. Thus, discussion will be limited to results obtained numerically from equation (4).

All numerical work was programmed in Pascal and used double-precision real variables with about 15 significant digits. The integral in equation (1) was approximated with a polynomial expansion (Johnson and Kotz 1970, p. 55, eq. 27). Evaluation of equation (3) was performed by Romberg integration (Phillips and Taylor 1973, p. 136). The implementation of Romberg integration used here successfully integrated the unit-normal density function with an accuracy of more than 9 significant digits.

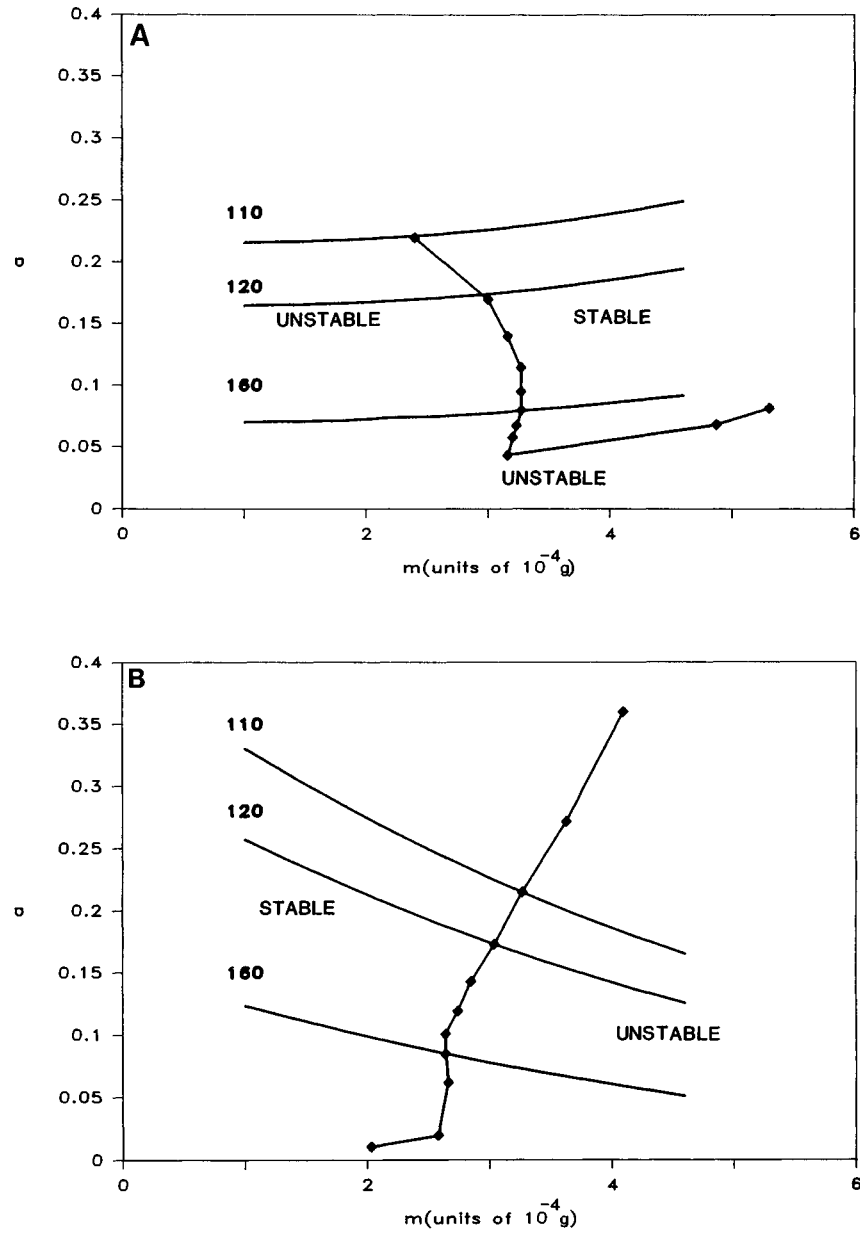For the numerical results reported, the functions of food versus size in figure 3

Fig. 7.—Equilibrium-egg-number isoclines: *A*, model 1; *B*, model 2. The points connected by a line separate the region with stable equilibrium points from the region with cycles or chaos.

and of size versus fecundity in figure 5 have been used. The effects of adults on fecundity have been modeled with the one-parameter hyperbolic function described previously. The two parameters that have the greatest effects on the equilibrium population size and stability are m and a. In figure 7, the isoclines of equilibrium egg number are shown for models 1 and 2. For both models, decreasing a increases the equilibrium egg numbers (see fig. 7). Smaller values of a imply less adult density dependence on female fecundity. When $a = 0$, female fecundity is independent of adult density. Consequently, more eggs should be laid by a given adult population with decreasing values of a.

As m decreases, a greater fraction of the larval population survives. Such decreases in m should increase the adult equilibrium population size: this result is observed (not shown here). The effects of decreasing m on equilibrium egg numbers is more complicated. When m is decreased in model 1, the adult population size increases; yet, the average size of females decreases. Thus, their fecundity is, on the average, less. The larger adult population size reinforces this effect by further decreasing the fecundity of females. The result is decreasing equilibrium egg numbers with decreasing values of m (see fig. 7A).

The results of decreasing m on equilibrium egg numbers are quite different for model 2. Adult population size is larger because of smaller values of m, and the females are also larger and more fecund (see fig. 3B). Thus, the decline in fecundity that results from a large adult population size is more than compensated for by the increased efficiency and size of the adults. Consequently, in figure 7B the equilibrium egg numbers increase with decreasing m (a held constant).

Figure 7 shows points in the a-m plane, which separates populations with stable equilibria from those with unstable equilibria. It has not been verified that lines joining these points also separate stable from unstable combinations of a and m. When the equilibrium (6) is unstable, the population may exhibit a variety of behaviors, depending on the parameter values, which have been described for other nonlinear difference equations (Roughgarden 1979, chap. 18). These include low-order cycles and behavior indistinguishable from random environmental noise. For both models, some level of adult regulation of female fecundity is clearly necessary for stable population dynamics (see fig. 7) because of the high fecundity of a single, small female. The dependence of population stability on m is quite different for the two models. Both models become unstable if m is very large. Recall that, when m is large, larval survival is low and the adult population size is therefore small. However, the females in the adult population will be rather large and the adult effects on fecundity reduced because of the small adult population size. As adult numbers decline, the slope of the hyperbolic function regulating fecundity increases dramatically (see fig. 6). If the adult numbers become too small, because m has become too large, the regulation of adult fecundity becomes sensitive to small changes in population size and ultimately causes the population to become unstable.

Very small values of m cause instability in model 1 but not in model 2. As m decreases, larval survival increases and the adult population size increases. Examination of figure 6 shows the hyperbolic fecundity function to be flat at high

densities. Consequently, population stability should not be affected by adult density-dependent effects on female fecundity. In figure 3A, one can see that at low food levels per larva (typical of an equilibrium population), the slope of the average size function increases with decreasing m. This is not the case for model 2 (see fig. 3B), where these lines are parallel. Thus, at low values of m under model 1, female size and hence fecundity is sensitive to small changes in egg numbers and ultimately will destabilize the population if m is made sufficiently small.

There are clearly large regions of parameter space, in figure 7, that predict stability under model 1 and instability under model 2, and vice versa. It thus seems reasonable to expect that, if the parameter m is allowed to evolve, the outcome of evolution on population stability may differ drastically between models 1 and 2.

NATURAL SELECTION

The consequences of natural selection at high population density are examined next by allowing genetic variation in competitive ability and the parameter m. In the preceding section, each population was assumed to consist of a single competitive type. Assume here that individuals may differ in their ability to consume limited food because of genotypic differences (although sex may also influence relative competitive ability; Nunney 1983).

If there are n competitive types in a population, let α_i be the competitive ability of type i ($i = 1, \ldots, n$), which occurs with frequency g_i, and $\Sigma g_i = 1$. If the mean competitive ability of individuals in the population is $\bar{\alpha} = \Sigma \alpha_i g_i$, then the average amount of food consumed by type-i individuals is $B\alpha_i/Vn_t\bar{\alpha}$. Clearly, if $\alpha_i > \bar{\alpha}$, then type-i individuals consume more food than the average individual; the converse is true if $\alpha_i < \bar{\alpha}$. When the competitive types are simply different genotypes in Hardy-Weinberg equilibrium, their frequencies may be obtained from the vector of allele frequencies, $\mathbf{p}_t$. Allowing for different competitive types in the population means that both fecundity and viability depend on density and frequency. This dependence is emphasized by rewriting these functions, $\bar{f}(Vn_t, \mathbf{p}_t)$ and $W(Vn_t, \mathbf{p}_t)$.

It is clear from this formulation that populations monomorphic for different competitive types have the same equilibrium population size as defined by equation (6).

Evolution of α

The evolution of competitive ability will be studied by assuming that a single locus with two alleles controls variation in α as summarized by the first four rows of table 1. With such variation, the egg recursion is modified to

$$n_{t+1} = \frac{1}{2}G(N_t)\bar{F}(Vn_t, \mathbf{p}_t)\bar{W}(Vn_t, \mathbf{p}_t)Vn_t. \tag{7}$$

If we suppress the arguments of the fecundity and viability functions, then the mean fecundity is $\bar{F} = \Sigma\Sigma_{ij}p_ip_j\bar{f}_{ij}$, and the mean viability is $\bar{W} = \Sigma\Sigma_{ij}p_ip_jW_{ij}$. The genotypic values for fecundity and viability for heterozygotes are given by

$$W_{12} = \int_{x_{12}}^{\infty} \phi(y)\,dy, \tag{8}$$

TABLE 1

Population-Genetics Models

Genotypic Values	Genotype		
	A_1A_1	A_1A_2	A_2A_2
Frequency	p_1^2	$2p_1p_2$	p_2^2
Viability	$W_{11}(Vn_t, \mathbf{p}_t)$	$W_{12}(Vn_t, \mathbf{p}_t)$	$W_{22}(Vn_t, \mathbf{p}_t)$
Fecundity	$\bar{f}_{11}(Vn_t, \mathbf{p}_t)$	$\bar{f}_{12}(Vn_t, \mathbf{p}_t)$	$\bar{f}_{22}(Vn_t, \mathbf{p}_t)$
Relative competitive ability	α_{11}	α_{12}	α_{22}
Minimum food required	m_{11}	m_{12}	m_{22}

where $x_{12} = (mVn_t\bar{\alpha}/B\alpha_{12} - 1)/\sigma$ and

$$\bar{f}_{12} = W_{12}^{-1} \int_{x_{12}}^{\infty} f\{s[B\alpha_{12}(\sigma y + 1)/Vn_t\bar{\alpha}]\}\phi(y)\,dy\,; \tag{9}$$

fecundity and viability for homozygotes are analogous to equations (8) and (9). Allele-frequency dynamics are governed by the recursion

$$p_1' = p_1[(WF)_1/\overline{WF} + W_1/\overline{W}]/2, \tag{10}$$

where $W_1 = \Sigma p_i W_{1i}$, $(WF)_1 = \Sigma p_i W_{1i}\bar{f}_{1i}$, and $\overline{WF} = \Sigma p_i(WF)_i$. The right-hand side of equation (10) may be interpreted as the current allele frequency times the relative marginal fitness of the A_1 allele averaged over both sexes. This equation is a special case of the model of fertility and viability selection of Nagylaki (1987, eq. 76).

To study the evolution of competitive ability we examine the stability of boundary equilibria. Specifically, assume that $p_2 = 1$ and that the population is at a stable equilibrium, $\hat{n}$. We seek conditions that permit the increase of A_1 alleles when they are rare. Under these conditions the linearized dynamics of the A_1 allele are given by

$$p_1' = (p_1 \hat{W}_{12}/2\hat{W}_{22})[(\overset{\circ}{f}_{12}/\overset{\circ}{f}_{22}) + 1], \tag{11}$$

where $\hat{W}_{ij}, \overset{\circ}{f}_{ij}$ are the equilibrium survivorship and fecundity functions evaluated at $n = \hat{n}$ and $p_2 \simeq 1$. From equation (11), the condition for A_1 to increase when rare is

$$\hat{W}_{12}\overset{\circ}{f}_{12} + \hat{W}_{12}\overset{\circ}{f}_{22} > 2\hat{W}_{22}\overset{\circ}{f}_{22}. \tag{12}$$

If $\alpha_{12} > \alpha_{22}$, then making use of equations (8) and (9) and the equivalent equations for homozygotes shows that $\hat{W}_{12} > \hat{W}_{22}$ and $\hat{W}_{12}\overset{\circ}{f}_{12} > \hat{W}_{22}\overset{\circ}{f}_{22}$, which ensure that condition (12) holds. Thus, natural selection favors increased competitive ability (increased values of α), and overdominance ($\alpha_{12} > \alpha_{11}, \alpha_{22}$) ensures a protected polymorphism (Prout 1968).

From inspection of equations (8) and (9) it is clear that the intensity of selection for increased α increases with population size. However, comments from the

preceding section imply that a population that becomes fixed for a superior competition genotype will have its carrying capacity unchanged. In addition, absolute viability will remain unchanged. Thus, although α is under strong selection pressure at high population density, it has no effect on the equilibrium numbers of eggs or adults. Similar results have been noted previously (Slatkin 1979; Anderson and Arnold 1983; Asmussen 1983).

The complicated dependence of $\hat{W}_{ij}$ and $\hat{f}_{ij}$ on allele frequencies precludes an explicit derivation of equilibrium allele frequencies. A characterization of an equilibrium that is related to single-locus viability theory can be derived. First, assume that there exists an equilibrium population size, $\hat{n}$, and allele frequency, $\hat{p}_1$. Let $\hat{W}_{ij}, \hat{f}_{ij}$ be the genotypic viabilities and fecundities evaluated at the equilibrium. Now, let $\bar{W}_{ij} = \hat{W}_{ij}/\hat{W}$ and $(\bar{W}F)_{ij} = \hat{W}_{ij}\hat{f}_{ij}/\hat{W}\bar{F}$, where $\hat{W} = \Sigma\Sigma_{ij}p_ip_j\hat{W}_{ij}$. Finally, for each genotype define $\psi_{ij} = (\bar{W}F)_{ij} + \bar{W}_{ij}$, where ψ_{ij} is the sum of the relative fitnesses of males and females of genotype A_iA_j. An equilibrium for equation (10) will be

$$\hat{p}_1 = (\psi_{12} - \psi_{22})/(2\psi_{12} - \psi_{11} - \psi_{22}),$$ (13)

where the ψ's play the role of viabilities in single-locus viability models. The equilibrium (13) will be called viability-analogous after similar terminology introduced by Uyenoyama et al. (1981).

When there is only genetic variation in the α's and $\alpha_{11} = \alpha_{22}$, then the ψ_{ij}'s in equilibrium (13) can be replaced by α_{ij}'s yielding the equilibrium $\hat{p}_1 = 0.5$ (see the Appendix). When $\alpha_{11} \neq \alpha_{12}$, the equilibrium is expected to be "close" to

$$\hat{p}_1 = (\alpha_{11}\alpha_{12} - \alpha_{11}\alpha_{22})/(\alpha_{11}\alpha_{12} + \alpha_{22}\alpha_{12} - 2\alpha_{11}\alpha_{22}).$$ (14)

Numerical results have shown that the true equilibrium is always greater than equation (14) although, as explained in the Appendix, this may not always be the case.

The gross effects of competition on viability and female size can be seen in figures 8 and 9. Figure 8 shows the viability of F_1 progeny from three populations that have been kept at low density for 130 generations. When larvae from this F_1 population compete with a stock homozygous for the white (w) allele, their viability declines dramatically at low food levels (most-intense competition). Likewise in figure 9, the size of females from this same population is shown in the presence and absence of the white-eyed competitors. At low food levels, female size decreases noticeably when competitors are present. At high food levels, viability and female size are relatively unaffected by the presence of competitors.

Evolution of 'm'

Now assume that the genetic variants in table 1 differ according to the minimum amount of food required for pupation only, as shown in row 5 of table 1. As before, consider first a population fixed for the A_2 allele and derive conditions that allow A_1 to increase when rare. Equation (12) is still the correct inequality for predicting an initial increase of A_1, although W_{ij} and $\bar{f}_{ij}$ will be different because of the assumption that genetic variation is present in the m's and not in α.

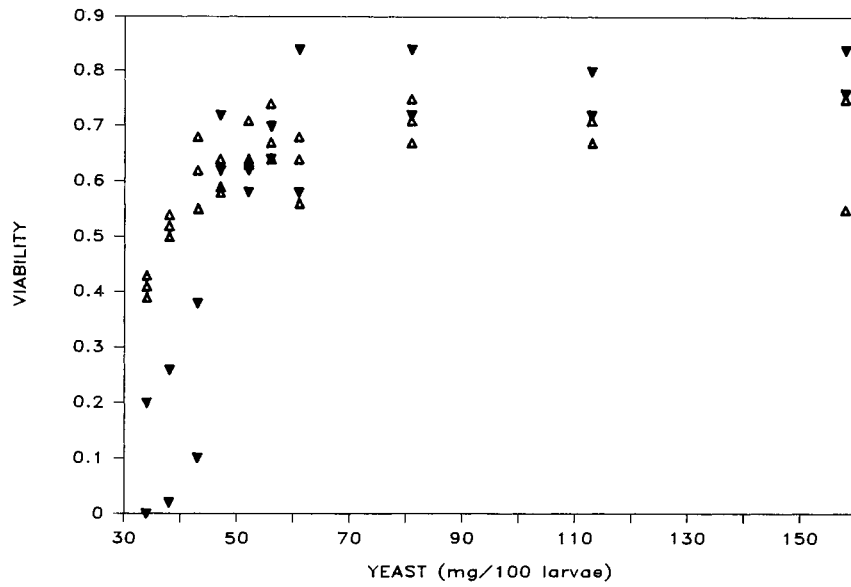For both models 1 and 2, the A_1 allele increases when rare if $m_{12} < m_{22}$.

Fig. 8.—Viability as presented in figure 2 for the r-F₁ population (described in Mueller and Sweet 1986). *Open triangles*, only r-F_1 larvae; *solid triangles*, half of the larvae were r-F_1, and half white-eyed larvae.
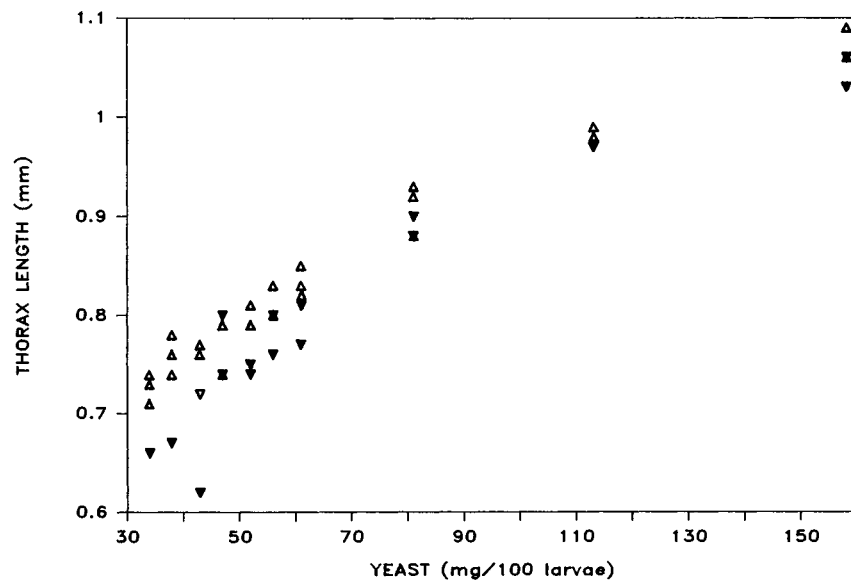

Fig. 9.—The average size of females from the experiment illustrated in figure 8. Symbols as in figure 8.

Overdominance yields a protected polymorphism. Thus, natural selection at high population density favors decreasing m, which has the effect of increasing viability in models 1 and 2. However, as a population evolves a lower m in model 1, the average size of a female should decline. This is because all the additional survivors are larvae that have consumed very little food and are correspondingly smaller (see fig. 3A). Model 2 predicts quite different results for female size. In model 2 all females with smaller values of m are also more efficient at turning food into biomass. The result is that average female size increases as evolution decreases m.

Distinguishing between models 1 and 2 is an empirical matter. These results do highlight the earlier comments concerning the faulty logic of the verbal theory, which predicts that natural selection should favor increased body size at high population densities. Clearly, the ecological details of how viability, fecundity, and body size interact with population density must be specified before the course of evolution can be reasonably evaluated.

Decreasing m via natural selection has a variety of effects on equilibrium egg numbers and stability. In both models, equilibrium adult numbers are expected to increase with decreasing m. Under model 1, however, the equilibrium number of eggs may actually decrease. In addition, a population that has a stable carrying capacity may become unstable if m becomes sufficiently small under model 1.

With only genetic variation in m and with $m_{11} = m_{22}$, the ψ_{ij}'s in equation (13) can be replaced by m_{ij} to yield

$$p_1^* = (m_{22} - m_{12})/(m_{11} + m_{22} - 2m_{12}),$$

which is 0.5 in this example. When the homozygotes have unequal m's, then the equilibrium (13) should be close to p_1^*. Furthermore, if $m_{11} > m_{22} > m_{12}$, then $\hat{p}_1 < p_1^*$; and if $m_{22} > m_{11} > m_{12}$, then $\hat{p}_1 > p_1^*$ (see the Appendix). The magnitude of the differences between $\hat{p}_1$ and p_1^* depend on the numerical details of the problem.

The Joint Evolution of α and 'm'

The evolution of both α and m is studied by allowing the single locus in table 1 to have pleiotropic effects on both these parameters. The novel feature of this model is the possibility of antagonistic pleiotropic effects. Such an assumption would require genotypes that are superior competitors to be inefficient and need large amounts of food to pupate successfully. The boundary problem of interest, then, is to derive conditions for the increase of A_1 when $m_{12} < m_{22}$ and $\alpha_{12} < \alpha_{22}$ or when $m_{12} > m_{22}$ and $\alpha_{12} > \alpha_{22}$. To ensure that $\hat{W}_{12} > \hat{W}_{22}$, it is necessary that $\hat{x}_{12} < \hat{x}_{22}$, which is satisfied if

$$m_{12}/m_{22} < \alpha_{12}/\alpha_{22} \tag{15}$$

for both models 1 and 2. However, for the initial increase of A_1, the additional requirement that $\hat{W}_{12}\hat{f}_{12} > \hat{W}_{22}\hat{f}_{22}$ must be satisfied. For model 1, this implies

$$\int_{\hat{x}_{12}}^{\infty} f\{s[B\alpha_{12}(\sigma y + 1)/V\hat{n}\alpha_{22}]\}\phi(y)dy > \int_{\hat{x}_{22}}^{\infty} f\{s[B(\sigma y + 1)/V\hat{n}]\}\phi(y)dy; \tag{16}$$

TABLE 2

Stability of the A_2 Fixation

Genotypic Parameters	Genotype			Change in A_1	
	A_1A_1	A_1A_2	A_2A_2	Increase	Decrease
α	α_x	α_x	1.1		
m	10^{-4}	2×10^{-4}	3×10^{-4}		
Evolution according to					
Model 1				$\alpha_x \geq 1.08$	$\alpha_x \leq 1.06$
Model 2				$\alpha_x \geq 0.95$	$\alpha_x \leq 0.92$

Note.—The initial frequency of A_1 was 0.01 and $a = 0.3$. All other parameter values are the same as in figure 3.

and for model 2,

$$\int_{\hat{x}_{12}}^{\infty} f\{s[B\alpha_{12}(\sigma y + 1)/V\hat{n}\alpha_{22} + (m_{22} - m_{12})]\}\phi(y)\,dy$$

$$> \int_{\hat{x}_{22}}^{\infty} f\{s[B(\sigma y + 1)/V\hat{n}]\}\phi(y)\,dy. \tag{17}$$

For model 2 it is evident that even if $\hat{x}_{12} < \hat{x}_{22}$, the inequality (17) may not be satisfied. To evaluate inequality (17) the size function would have to be specified. The same problem arises for model 1 when $\alpha_{12} < \alpha_{22}$ and $m_{12} < m_{22}$. A sufficient condition for the increase of A_1 with model 1 is inequality (15) and $m_{12} > m_{22}$. This does not turn out to be a very useful condition in many instances. The sorts of trade-offs in m and α necessary for initial increase or polymorphism are quite different for model 1 and model 2 because changes in m produce larger fitness differences in model 2 than 1. This effect is illustrated in table 2. In this example, a population is nearly fixed for the A_2A_2 homozygote, which is a superior competitor but inferior with respect to m. The value of α_{12} that allows the increase of A_1 alleles is then approximated from numerical iteration of equation (10). It is clear from table 2 that model 1 requires a much higher value of α_{12} than does model 2, since the fitness advantage of a low m in model 1 is not nearly as great as in model 2.

Table 3 shows the equilibrium values of viability, fecundity, and allele frequencies for models 1 and 2. These examples illustrate that a polymorphism described by equation (13) may be maintained without overdominance in m or α. Genotypes must, however, show some trade-off in their competitive ability and minimum food requirement. Another characteristic of the equilibria shown in table 3 is that each population maintains substantial additive genetic variation for m and α. Populations characterized by such polymorphisms could be expected to respond rapidly to changing environments in which the fitness effects of m and α were weighted differently than in the present model.

TABLE 3

Polymorphism Resulting from Antagonistic Pleiotropy

Parameter Values	Model		Parameter Values	Model	
	1	2		1	2
n	100	109	p_1	0.237	0.693
α_{11}	1.05	0.85	W_{11}	0.993	0.992
α_{12}	1.08	1.05	W_{12}	0.984	0.984
α_{22}	1.10	1.10	W_{22}	0.964	0.967
m_{11}	10^{-4}	10^{-4}	F_{11}	2.59	2.64
m_{12}	2×10^{-4}	2×10^{-4}	F_{12}	2.69	2.80
m_{22}	3×10^{-4}	3×10^{-4}	F_{22}	2.79	2.63

Note.—All other parameter values are the same as in table 2.

DISCUSSION

The model developed here describes density-dependent effects on the viability and fecundity of individuals. Density affects fecundity in two ways: through competition for food and adult size (hence, the fecundity of females is affected), and by modulating the number of interactions between adults. Natural selection in such environments favors the increase in competitive ability for food although such changes do not result in permanent changes in the equilibrium number of eggs or adults.

Natural selection also favors genotypes that can successfully pupate on less food. In one model, this is achieved by larvae eating less and pupating at a smaller size. In the second model, larvae pupate on less food by becoming more efficient at using the available resources. No single prediction emerges from this theory concerning the evolution of adult size at high densities. This result is contrary to the standard predictions of the verbal theory of r- and K-selection (Pianka 1970; Southwood 1976).

Models 1 and 2 make very different and perhaps extreme assumptions about the pleiotropic consequences of alleles that affect the parameter m. In model 1 there is a "cost" of increased survival. This cost is extracted in female fecundity and is accomplished by extrapolating the existing relationships between food consumed and size and between size and fecundity. In model 2 there is no cost to reductions in m. Other assumptions could have been explored. The cost could have been less extreme than assumed in model 1, or the reduction in fecundity could have been accomplished without a reduction in size. The predictions concerning the evolution of adult size would probably continue to be sensitive to these other possible assumptions. Without strong empirical evidence favoring any particular assumption, we have not pursued these additional possibilities.

The existence of genetic variation with pleiotropic effects on m and α is suggested by empirical studies. *Drosophila melanogaster* larvae may become better competitors for food by increasing the number of cephalopharyngeal retractions made per minute while feeding (Burnet et al. 1977). Larvae that are fast feeders

pass food more quickly (Burnet et al. 1977) and are more active (Sewell et al. 1975) than slow feeders. Thus, it is possible that a correlated response to increased α is increased metabolic rate and reduced efficiency of assimilation (as a result of the faster ingestion rate; Scribner and Slansky 1981). These correlated responses would almost certainly lead to increased values of m.

Gurney and Nisbet (1985) have developed continuous-time, density-dependent models of population growth that explicitly incorporate larval competition and its effects on adult fecundity. Because the present models differ from Gurney and Nisbet's in a number of minor and major details (presence or absence of adult effects on fecundity, individual variation in food consumed), comparison is difficult. The present models have the advantage of being more congruent with the ecology of laboratory *Drosophila* populations.

At this point, it is worth summarizing what is known about density-dependent natural selection in *Drosophila*. My colleagues and I have undertaken a long-term study of six replicate populations: three kept at low population density, called *r*-selected, and three kept at high densities, called *K*-selected. After eight generations, the populations had become differentiated with respect to density-dependent rates of population growth (Mueller and Ayala 1981*a*). This observation was consistent with predictions of the formal models of density-dependent natural selection (see, e.g., Roughgarden 1971). These differences in rates of population growth also imply that the carrying capacity of adult *K*-selected flies is greater than the carrying capacity of *r*-selected flies (contrary to the claims of Stearns 1984). Additional work has shown that under crowded larval conditions, the *K*-selected populations survive better and produce larger adults than the *r*-selected populations (T. Bierbaum, Mueller, and F. Ayala, unpubl. results). It now seems clear that part of the increased larval survival of the *K* populations can be attributed to the evolution of increased pupation height (Mueller and Sweet 1986). However, the behavioral differences in pupation-site choice cannot explain the differences in adult size in crowded cultures.

Clearly, evolution consistent with model 2 could offer a plausible explanation for the size and viability differences observed in the empirical studies. Testing these ideas requires collecting information on survival and size as a function of food level similar to the study of Nunney (1983). In addition, the appropriate experiments could also yield estimates of α.

SUMMARY

The action of density-dependent population growth is modeled through the effects of limited food. Scramble competition for food affects viability and adult size, which are correlated with the fecundity of females. Adult effects on fecundity are also explicitly modeled. In the two submodels considered, changes in the minimum amount of food necessary for successful pupation lead to (1) changes in the minimum size of an adult with no change in overall efficiency or (2) constant minimum size but changes in the efficiency of food use. The resulting population dynamics of the two submodels are qualitatively different. For both submodels,

population stability requires some degree of adult effects on female fecundity for parameter values typical of *Drosophila*.

When genetic variation is present for competitive ability and minimum food required, natural selection at equilibrium population size favors increasing competitive ability and decreasing the minimum food requirement. Evolutionary changes in the competitive ability of a population do not affect equilibrium population size. Decreases in the minimum food requirements typically increase the equilibrium adult population size but have variable effects on equilibrium egg numbers, depending on the submodel examined. Biological evidence suggests that competitive ability and minimum food requirements may be positively correlated. Genetic models with this antagonistic pleiotropy can maintain allelic variation without overdominance in either character. Furthermore, contrary to established verbal theory, there is no consistent prediction concerning the evolution of average body size. An advantage of this theory is that parameters of interest may be easily estimated in laboratory populations of *Drosophila*.

APPENDIX

This appendix derives some simplified expressions for equation (13).
Evaluation of equation (13) for model 1 and genetic variation only in m yields

$$\hat{p}_1 = \frac{\int_{\hat{x}_{12}}^{\hat{x}_{22}} \phi(y)\,dy + \int_{\hat{x}_{12}}^{\hat{x}_{22}} f(y)\phi(y)\,dy}{\int_{\hat{x}_{12}}^{\hat{x}_{22}} \phi(y)\,dy + \int_{\hat{x}_{12}}^{\hat{x}_{22}} f(y)\phi(y)\,dy + \int_{\hat{x}_{12}}^{\hat{x}_{11}} \phi(y)\,dy + \int_{\hat{x}_{12}}^{\hat{x}_{11}} f(y)\phi(y)\,dy}. \tag{A1}$$

Repeated application of the mean-value theorem and some algebra yields

$$\hat{p}_1 = \frac{(m_{22} - m_{12})[Q(W) + \phi(W')]}{(m_{22} - m_{12})[Q(W) + \phi(W')] + (m_{11} - m_{12})[Q(Z) + \phi(Z')]}, \tag{A2}$$

where $f(y)\phi(y) = Q(y)$, $\hat{x}_{12} \le (W, W') \le \hat{x}_{22}$, and $\hat{x}_{12} \le (Z, Z') \le \hat{x}_{11}$. If $m_{11} = m_{22}$, then $W = Z$, $W' = Z'$, and the equilibrium allele frequency is given by

$$p_1^* = (m_{22} - m_{12})/(m_{11} + m_{22} - 2m_{12}). \tag{A3}$$

However, if $m_{11} > m_{22}$, then $Q(Z) + \phi(Z') > Q(W) + \phi(W')$ and $\hat{p}_1 < p_1^*$. Similar arguments require that, when $m_{11} < m_{22}$, $\hat{p}_1 > p_1^*$.

When genetic variation exists in the m's and model 2 applies, the analysis is somewhat more complicated. If the fecundity function is replaced by Taylor's polynomial plus remainder (Phillips and Taylor 1973, p. 38), then the equilibrium allele frequency is

$$\hat{p}_1 = (m_{22} - m_{12})(A + A')/[(m_{22} - m_{12})(A + A') + (m_{11} - m_{12})(L + L')], \tag{A4}$$

where

$$A = Q(W) + \phi(W'),$$

$$L = Q(Z) + \phi(Z'),$$

$$A' = B\sigma \int_{\hat{x}_{12}}^{\infty} f'\{s[B(\sigma y + 1)/V\hat{n} + \zeta_{22}]\}\phi(y)\,dy \Big/ V\hat{n},$$

$$L' = B\sigma \int_{\hat{x}_{12}}^{\infty} f'\{s[B(\sigma y + 1)/V\hat{n} + \zeta_{11}]\}\phi(y)\,dy \Big/ V\hat{n},$$

$f'(\cdot)$ denotes differentiation with respect to $m_{ii} - m_{12}$, and $0 < \zeta_{ii} < m_{ii} - m_{12}$. Certainly, when $m_{11} = m_{22}$, equation (A4) equals p_1^*. When $m_{11} > m_{22}$, there is no guarantee that $\zeta_{11} > \zeta_{22}$; but for m_{11} sufficiently close to m_{22}, then $\zeta_{11} \approx \zeta_{22}$, in which case $\hat{p}_i < p_i^*$, as before.

When genetic variation exists in α, the equilibrium p_i reduces to

$$\hat{p}_1 = A(\alpha_{22}^{-1} - \alpha_{12}^{-1})/[A(\alpha_{22}^{-1} - \alpha_{12}^{-1}) + L(\alpha_{11}^{-1} + \alpha_{12}^{-1})]. \tag{A5}$$

A and L are complicated functions and they are equal when $\alpha_{11} = \alpha_{22}$. However, inequalities such as $\alpha_{11} = \alpha_{22}$ are insufficient for evaluating the relative magnitudes of A and L, without specifying the specific fecundity function and additional parameter values.

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