

PLANT POPULATION ECOLOGY

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10. GENETIC INFLUENCES ON THE DISTRIBUTION AND ABUNDANCE OF PLANTS

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SUMMARY

- 1 The frequent observation of genetic differentiation amongst ecologically contrasting populations has led to the inference that the abundance and distribution of organisms has a 'genetic component'.
- 2 This chapter uses theoretical models to explore the conditions under which the genetic component may or may not affect (a) population distribution along an environmental gradient, and (b) numerical abundance within a heterogeneous environment.
- 3 Under the logistic model of population growth the intrinsic rate of population increase, r , influences population size if the population is genetically polymorphic, if density dependence acts only during one phase of the life-cycle, or if birth-rates and death-rates are affected separately by density-dependent versus independent factors.
- 4 To understand empirically the effects of genetic differentiation on abundance there is a need to assess the forces of population regulation on contrasting genotypes by using reciprocal transplant experiments at a range of densities.
- 5 In a heterogeneous environment, intrinsic growth-rate, carrying capacity, and the fraction of individuals entering each sub-habitat determine both gene frequency and population size.
- 6 If population parameters in one sub-habitat are genetically correlated with those in a second sub-habitat, then strong evolutionary forces in one sub-habitat may dominate responses in the other. This may have large consequences for numerical abundance, with the possibility that evolutionary change results in a decline in overall population size.

INTRODUCTION

It has become a commonplace observation that plant populations from diverse habitats and different geographical regions are genetically differen-

tiated, and form 'ecotypes' adapted to those regions. Recently abundant evidence has also been gathered for adaptive microdifferentiation over very short distances. Thus it appears that genetic differentiation is in some measure responsible for the habitat and geographical range of a species. The idea that the distribution and abundance of a species has a genetic component is important because it not only brings into question the use of 'the species' (or its Latin binomial) as an ecologically useful unit (Harper 1982) but also extends our inquiry on the determinants of species distribution beyond simple proximal physiological properties of species as sufficient explanations, into a consideration of forces responsible for evolutionary limits to those properties (Pickett 1976; Antonovics 1976a).

While in extreme cases the role of genetic differentiation in determining species distribution cannot be denied (such as metal tolerance on toxic waste spoil, or where geographically diverse ecotypes die when reciprocally transplanted to alien sites), it is important to remember that although differential survival and reproduction may lead to adaptive genetic change, such change may not necessarily affect population size. Indeed, the result of adaptation may be a *decrease* rather than increase in population size (as in cases where selection favours larger body size or larger territories). In order to gain a full understanding of how genetic change may affect distribution and abundance of a species we need to explore models that simultaneously consider genetic and numerical change, and to use these models as a framework for empirical studies. Only in this way will we gain a realistic sense of 'where population genetics begins and ecology ends'. The relatively recent fusion of population genetics and population ecology theory has given us a basis from which to approach this question (Anderson 1971; Charlesworth & Giesel 1972; Roughgarden 1972). This chapter explores the conditions under which genetic change will affect population distribution along a gradient and numerical abundance within a heterogeneous environment. It also aims to use particular examples to illustrate both the dynamical nature of gene frequency/population size interactions, as well as the great need for experiments which depart from simple demonstrations of 'adaptive differentiation'.

DISTRIBUTION ALONG AN ENVIRONMENTAL GRADIENT

A simplistic scenario

Consider a continuous environmental gradient along which there are a number of discrete populations (Fig. 10.1). Along this gradient, as the environment becomes 'harsher', birth-rates decline and death-rates increase.

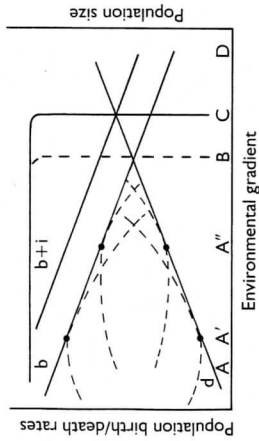


FIG. 10.1. Schematic diagram of demographic processes along an environmental gradient. b = birth-rate, d = death-rate, i = net immigration-rate. Curved lines associated with positions A' and A'' along the environmental gradient represent values of b and d for genotypes sampled from these positions and transplanted to other positions along the gradient. The horizontal line shows the predicted population size assuming continuous population growth and equal density dependent regulation along the gradient. Beyond C population size is zero. See text for further explanation.

There is also some immigration and emigration, giving a positive net migration rate. Then the population will increase in regions A–C, but decline in regions C–D. Within A–C, the region B–C would have negative population growth if it were not for immigration from less harsh regions, circumstances referred to previously as a 'demographically marginal' population (Antonovics 1976b). Given that we have continuous population growth which follows the logistic equation, $dN/dt = rN(1-N/K)$, the population would be expected to reach some carrying capacity, K . In this simple deterministic situation, at equilibrium, the abundance of a species *within* the distribution range of the species (A–C) will be determined solely by K (i.e. forces of density-dependent regulation) while the distributional range of the species will be determined by the intrinsic growth rate $r > 0$, i.e. where birth- and immigration-rates exceed death-rates. Actual distributional range will be affected if genotypes can be produced that have a greater birth-rate and/or lower death-rate, in the regions just beyond C.

Elsewhere, (Antonovics 1976b) I have emphasized that there are a substantial number of evolutionary forces that may retard the evolution of populations at the margins of their ecological ranges. When considered singly, the limits imposed by gene flow, small population size, coevolutionary responses of associated species, or by difficulty of generating adaptive character combinations may not be absolute. But combinations of these forces may result in relatively static species boundaries. Thus at an ecotone between a field and woodland, we could infer that evolutionary response of *Anthoxanthum* to the woodland habitat was probably limited by the difficulty of evolving unique character combinations given a small population size and

large amounts of gene flow from the field habitat where *Anthoxanthum* was extremely abundant and vigorous (Antonovics 1976b; Grant & Antonovics 1978).

The idea that population size of a species within the distributional range will only be affected if the genotypes vary in response to density-dependent factors has been termed Haldane's Dictum (Haldane 1953; Charlesworth 1971). A corollary of this dictum is that even if the region A-C contains differentiated populations, unless they are differentiated with regard to response to density-dependent factors, this genetic differentiation will have no effect on population size. Therefore, imagine sampling two populations A' & A'' (see Fig. 10.1) and performing a reciprocal transplant experiment. Individuals of population A' when transplanted to A'' will have a lower birth-rate and higher death-rate (i.e. will appear 'maladapted' to region A''). However as long as their birth-rate still exceeds the death-rate, there will be no effect of this 'maladaptation' on population size. Therefore a simple demonstration of ecotypic differentiation is by itself insufficient proof that genomics affects either abundance or distribution. However Haldane's Dictum requires qualification.

Determinants of abundance within a homogeneous population

Haldane's Dictum applies to situations where there is 'perfect' logistic density-dependent regulation. Given continuous population growth and instantaneous population regulation (i.e. $dN/dt = rN(K-N)/K$) or discrete population growth and birth and death processes being determined solely by numbers at the start of each time interval (i.e. $N_{t+1} = N_t(1 + r(K-N)/K)$) it has been repeatedly shown (Anderson 1971; Charlesworth 1971; Roughgarden 1971) that K , the carrying capacity (or density response) and not r , the intrinsic rate of population increase determines equilibrium population size.

However, if the population is genetically polymorphic for carrying capacity (e.g. there is heterozygote advantage in K or some other mechanism maintains genetic variance in K) then r will have an effect on equilibrium population size (Charlesworth 1971). This is illustrated in Fig. 10.2, a case where increasing r of one of the genotypes results in an increase of the frequency of that genotype as well as of equilibrium population size.

Another circumstance where r will impact on population size is where density dependence acts only during one phase of the life-cycle, the other components being density independent (Charlesworth 1971; Prout 1980). Consider the life-history to consist of discrete phases, such that numbers can be assessed at times t , $t + 1/2$, and $t + 1$. A complete cycle of iteration is $t = 1$. Let N_t represent the number of seeds. Consider now that regulation is by

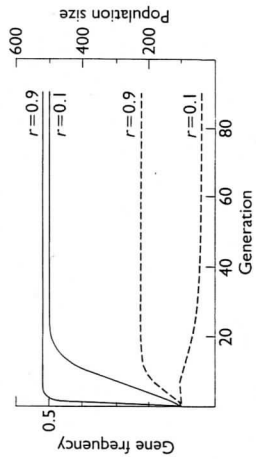


FIG. 10.2. Effect of r on population size (solid line) and gene frequency of A (dashed line) in a population genetically polymorphic for K . ($K_{AA} = 300$, $K_{AB} = 600$, $K_{BB} = 500$).

density dependence during the seedling phase, i.e. interval $t \rightarrow t + 1/2$, and seed production after this is density independent. Then (after Charlesworth 1971):

$$N_{t+1/2} = N_t(1 + r(K - N_t)/K) \quad (1)$$

and

$$N_{t+1} = N_{t+1/2}c \quad (2)$$

where c = probability of surviving from $t \rightarrow t + 1/2$, this probability being density independent. Substituting (1) into (2) we obtain at equilibrium:

$$N_t = K(cr + c - 1)/cr \quad (3)$$

Alternatively, if seedlings die in a density-independent manner in the interval $t \rightarrow t + 1/2$, following which there is density-dependent regulation in the adult phase ($t + 1/2 \rightarrow t + 1$); then:

$$N_{t+1/2} = N_t c \quad (4)$$

Logistic density dependence in the next time interval results in

$$N_{t+1} = N_{t+1/2} \cdot (1 + r(K - N_{t+1/2})/K) \quad (5)$$

Substituting (1) into (2) we obtain at equilibrium

$$N_t = K(cr + c - 1)/c^2 r \quad (6)$$

When r is large, and/or $c \approx 1$, then density-independent mortality will have no effect. However, when r is small, as near a population boundary, density-independent processes may have an important effect on abundance. A similar conclusion was reached by Watkinson (1985) using the reciprocal yield law of density dependence. However, in his formulation, r , (or $r = \ln \lambda$) and

equilibrium population size are functionally dependent. It is therefore not clear whether the effects he demonstrates are due to the fact that r and K are confounded in his model. An expectation from these models is that genetic change in response to density-independent mortality factors is unlikely to affect population size at the centre of a species range, but may well do so at the margins, where r is expected to be small. However its exact effect will depend also on the stage of the life-cycle at which there is density-independent mortality.

Clearly other models of population growth may lead to different conclusions. Thus in the logistic formulation of population growth, r , the intrinsic rate of population increase is clearly a compound of both age-specific survival and fecundity. It is assumed in this model that the density-dependent term $(1 - K/N)$ acts on a single parameter (r) that does not distinguish births and deaths. If however we distinguish between survival and fecundity, and only one (say fecundity) is subject to density dependence, then:

$$\frac{dN}{dt} = (b(K - N)/K - d)N$$

and at equilibrium:

$$N = K(b - d)/b$$

In this formulation, equilibrium population size is a function of K as well as of $r (= b - d)$. This point has been illustrated graphically by Watkinson (1985).

How do these results relate to empirical data on population differentiation? Normally we examine genetic differentiation among populations using spaced plants in a 'common garden' or reciprocal transplant experiment. In these situations, fitness will be estimated on spaced plants and will therefore measure r (or some component of performance under low density). However, as we have shown above, such estimates may have little relation to equilibrium population size. Only if there is lethality in alien environments is it (reasonably) safe to conclude that genetic differentiation will affect abundance. These considerations illustrate that in order to understand how genetic factors affect population abundance and distribution we need rather special information. It is not sufficient to demonstrate ecotype formation using a common garden, nor is it sufficient to perform a 'realistic' reciprocal transplant experiment to estimate fitness in 'home' versus 'alien' habitats. Instead such transplant experiments should be done under a range of densities, at a series of stages throughout the life-cycle, to assess (1) whether the 'home' genotype has a different predicted equilibrium size from the

'alien' population and (2) whether causative links can be established between traits (physiological, morphological, etc.) differentiating the populations, and the relative roles of these traits in the responses to the ecotypes to density-dependent and density-independent factors.

As far as we know, reciprocal transplant experiments under a range of densities have only been carried out in one study (Davy & Smith 1985; Davy & Smith 1988). In this study, when *Salicornia* was reciprocally sown into upper and lower salt marsh at low density, home populations did much better than alien populations. However, the predicted equilibrium densities (where seed output per seed sown = 1) were quite similar in the lower marsh, but very different in the upper marsh (Davy & Smith 1985). In this case there was adaptive differentiation (in that home populations outperformed alien populations) but in only one site (upper marsh) would the adaptation clearly result in greater abundance. At the other site (lower marsh) genetic differentiation seemed to have little impact on abundance or distribution, in that less well adapted upper marsh plants would attain similar population densities.

Other studies have investigated the individual 'ingredients' of such density dependence. For example, Watkinson & Harper (1978), clearly demonstrated the interaction between density dependent and independent factors in regulating populations of *Vulpia*; realistic transplant experiments (carried out in the field) with demographic accounting of fates of individuals have become more common (Rauscher & Feeney 1980; Antonovics & Primack 1982; McGraw & Antonovics 1983); and genetic differences in density response have been shown by Shaw (1986) within natural populations of *Salvia lyrata*. However, we have almost no data that can provide even a tentative answer to the question of whether the 'genetic component' does or does not generally influence population size. The situation becomes more intriguing as we increase the realism of our models. We will consider briefly the effects of competitive interactions, stochastic events, and then the effects of environmental heterogeneity.

Models which demonstrate that in a uniform habitat the genotype with the highest carrying capacity will 'win', are predicated on the assumption that all genotypes contribute equally to the density effect. It may be more realistic to consider that genotypes also compete (i.e. density effects on genotypes, by genotypes vary). In two-component mixtures, it can be shown that abundance is a function of competition coefficients and carrying capacities (Pielou 1969). However, curiously and somewhat counter-intuitively, in three-component mixtures abundance also becomes a function of the intrinsic rates of increase (Strobeck 1973). And, where the competing 'components' (be they genotypes or species) show spatial sub-structuring,

dispersal becomes an added component of coexistence and abundance (Pacala 1986).

Stochastic events may be on a spatial or temporal scale. If populations along an environmental gradient are subject to random reductions in size (but the frequency or intensity of such reductions do not vary along the gradient) then because of the different rates of population growth there will be different rates of 'rebound' from such perturbations. The rates of rebound will be slower in marginal situations where population growth rates are low. The result is that stochastic events (of equal magnitude and frequency) will result in a gradual decrease in species abundance towards the margin. If these populations are thus held below carrying capacity, selection will favour those genotypes with high intrinsic growth-rates. The observation that marginal low density populations have genotypes with higher allocation to reproduction was made by Grant & Antonovics (1978).

The perturbations may be so great and/or suitable sites for population establishment so infrequent that the most critical events determining the abundance of a species along a gradient are population establishment and extinction rates (Carter and Prince 1981). In such instances, demographic processes at a within-population level may be relatively unimportant, and overshadowed by long-term probabilities of propagules arriving at suitable sites, or populations going extinct due to environmental change (Carter & Prince 1981, 1988). The ability of a species to extend its distribution at a boundary may then become a function of 'transmission rates' of the propagules, i.e. of genetic variation in dispersal ability and establishment. However, if there are trade-offs between, say, dispersal ability and competitive ability (e.g. seed size versus seed number), then there will be selection in opposite directions at the within- and among-population levels. Individual selection will oppose group selection, so retarding expansion of a species range at the boundary. Evidence for such forces acting at a within species level has been obtained by Olivieri & Gouyon (1985) who showed that individuals of *Carduus* established early in succession have smaller seeds than individuals of the same species sampled later in succession.

ABUNDANCE IN A HETEROGENEOUS ENVIRONMENT

The frequent demonstration of genetic microdifferentiation within plant populations raises the question of whether such microdifferentiation is an important component of population abundance. In a heterogeneous environment, will genetic differentiation act to increase 'global' population size or not? Is evidence of microdifferentiation also evidence that the 'genetic

component' is important in determining abundance? Until recently, most models of genetic microdifferentiation have made specific assumptions regarding population size, namely that either population size in each microsite was independently regulated and that the number emerging from each microsite was constant (e.g. Levene 1953; Dickinson & Antonovics 1973), or that the population numbers emerging from each site were determined solely by the fitnesses of the component genotypes and that the population was regulated at some global level. These have been termed 'soft-selection' or 'constant adult number' model and the 'hard-selection' or 'constant zygote number' model, respectively (Karlin 1982; Hedrick 1986). More recently, models have been developed where population regulation within each niche is not absolute, but depends on the action of density dependent responses (Arnold & Anderson 1983). The following sections employ the model of Arnold & Anderson (1983) to illustrate some of the potential interactions between genetic variation and population size in heterogeneous environments. The model assumes there are two niches and that in each niche the genotypes *AA*, *Aa*, and *aa* are independently regulated by a discrete version of the logistic equation (Roughgarden 1971). The fitness of the *i*th genotype (where *i* and *j* represent alleles) in a given niche is:

$$W_{ij} = 1 + r_{ij}(K_{ij} - N)/K_{ij}$$

where $1 + r_{ij} = \lambda_{ij}$ or the fitness of the *i*th genotype at zero density; K_{ij} = carrying capacity of *i*th genotype; and N = total population size (N_{ij} times the fraction of the population that enters the niche). The total population size in the niche after selection is then the sum of the fitnesses of the genotypes times the numbers of each genotype entering that habitat (as zygotes). In this model positive density dependence may arise either when $K_{ij} < N$ or $r_{ij} < 0$. To avoid this unrealistic situation, we modify the recursion equations used in the simulations such that when $K_{ij} < N$ population size declined independently of r_{ij} (i.e. $w_{ij} = K_{ij}/N$) and such that when $r_{ij} < 0$, there was negative density dependence by setting $w_{ij} = \lambda_{ij}(K_{ij} - N)/K_{ij}$; if w_{ij} was < 0 , then w_{ij} was set = 0. Following selection and density regulation individuals mated at random and the genotypes were then distributed equally among the two niches. Further details for setting up the basic recursions can be found in Arnold & Anderson (1983).

Initially, consider a population consisting of only one genotype say *AA*, which can only survive in niche 1, but which is lethal in niche 2. This genotype will have a particular r_{ij} and a particular K_{AA} . It will produce propagules, but half of them will be 'lost' (by virtue of emigration into the other niche). In this case r_{AA} will have to be greater than 1, otherwise the population is more than halved every generation and declines. As r_{AA}

increases, population size increases to a plateau. Since emigration is equivalent to density independent mortality, this illustrates the interaction, alluded to earlier, of density independent mortality with r_{AA} in determining population size.

If an allelic mutation (*B*) now arises that permits the species to invade the alternative niche, not surprisingly overall population size increases (Fig. 10.3a). The condition for such a mutation to invade has been determined by Arnold and Anderson (1983) and is a function of r_{ij} , K_{ij} , and the fraction entering each sub-habitat. In these situations therefore, both r and K will contribute to genotypic spread. Moreover a number of rather counter-intuitive situations may pertain. In the example in Fig. 10.3, genotype *BB*, because $r_{BB} < 1$, would not be able to maintain itself in niche 2 without the presence of *AA* in niche 1. Although it has a positive population growth within niche 2, its progeny would emigrate to niche 1, so resulting in a net population decline. However heterozygotes are formed which show positive population growth in both niches. The mutant *B* could even have a negative population growth in niche 2 ($r_{BB} < 0$) yet still be maintained in the population (Fig. 10.3b). In this case, immigration from niche 1 results in a positive growth rate in niche 2, so permitting *BB* to persist.

The situation can be even more extreme. One can show that there exist conditions under which neither *AA*, nor *aa* by themselves can persist in a heterogeneous habitat but a polymorphic population can (Fig. 10.4). This is because interbreeding among the two genotypes produces heterozygotes that contribute to population growth in both niches even though there is no

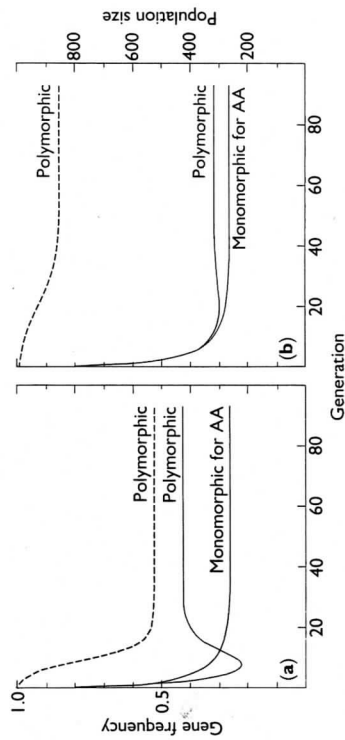


FIG. 10.3. Effect of a mutant *B*, permitting invasion of a second niche, on overall population size (solid line) and overall gene frequency of *A* (dashed line). (a) Niche 1: $K_{AA} = 800$, $K_{AB} = 400$, $K_{BB} = 0$; $r_{AA} = 1.2$, $r_{AB} = 0.6$; $r_{BB} = -1$. Niche 2: $K_{AA} = 0$; $K_{AB} = 300$, $K_{BB} = 600$; $r_{AA} = -1$, $r_{AB} = 0.3$, $r_{BB} = 0.6$. (b) Mutant *B*, has a negative population growth in niche 2. Parameters for niche 1, as in (a); niche 2: $K_{AA} = 0$, $K_{AB} = 4000$, $K_{BB} = 8000$; $r_{AB} = -0.1$, $r_{BB} = -0.05$. Line labelled 'monomorphic' shows size of population fixed for *A*.

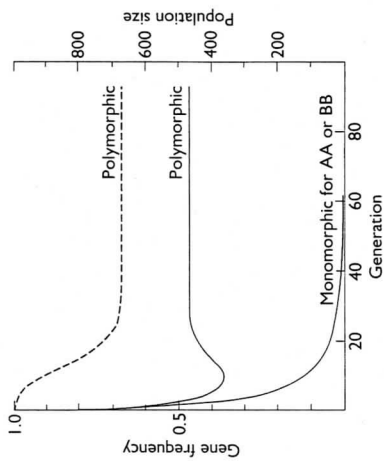


FIG. 10.4. Illustration of a situation where populations fixed for either *A* or *B* alleles will go extinct, but populations that are genetically polymorphic will show finite population size; niche 1: $K_{AA} = 800$, $K_{AB} = 400$, $K_{BB} = 0$; $r_{AA} = 0.9$, $r_{AB} = 0.5$, $r_{BB} = -1$, niche 2: $K_{AA} = 300$, $K_{BB} = 600$; $r_{AA} = -1$, $r_{AB} = 0.5$, $r_{BB} = 0.9$.

heterozygote advantage *per se* within any one niche. Therefore in this case the ability to invade a heterogeneous habitat will be conditional on the presence of genetic variation. Conversely, we can conclude that individuals which reproduce sexually will be able to invade such heterogeneous habitats, whereas asexual individuals, unless they are heterozygous, will not be able to do so. However, whether this can be a persistent force for the evolutionary maintenance of sexual reproduction is not clear—thus in the above examples asexual heterozygotes would persist in the population, albeit at a lower carrying capacity than in the mixed population. It may therefore be a mechanism whereby asexual reproduction preserves more flexible, 'generalist genotypes'.

QUANTITATIVE GENETIC MODELS OF EVOLUTION IN HETEROGENEOUS ENVIRONMENTS

The above considerations illustrate that the r - K dichotomy has been overstated. In nearly all the examples, population abundance is determined by both the intrinsic rate of increase and the density response (as reflected by carrying capacity). Clearly the forces of selection on these two parameters will vary with the particular ecological situation under consideration. The responses to such selection will in turn depend on the genetic variances and covariances among the quantitative traits that underly demographic parameters of populations (Khan, Antonovics & Bradshaw 1976; Shaw 1986).

Ultimately, it is adaptation in these quantitative traits that, through the life-history parameters of the population, determines the spatial pattern of abundance in plant populations. Quantitative genetic models of evolution are a useful complement to the single-locus models that we have been discussing because they permit the study of aspects of adaptation that are not addressed by the more classical population genetic models. Using a quantitative genetic approach, we will see that genetic constraints on adaptation to a patchy environment can also affect the abundance of plants in each sub-habitat.

In heterogeneous environments, the response to selection on characters that determine the intrinsic rate of population increase or density response may not be independent in the different subhabitats. If we think of a character expressed in two environments as two character states, then a non-zero genetic correlation between the states is caused by common gene effects expressed in each environment. In one-locus models with two alleles, the genetic correlation between the character states expressed in two environments can only be $+1$. However, in quantitative genetic models, this correlation can take any value between $+1$ and -1 , thus permitting study of the full range of evolutionary possibilities. Complete details of a quantitative genetic model for the dynamics of adaptation to a spatially patchy environment can be found in Via & Lande (1985). Here, we describe this model briefly in order to show that unfavourable genetic correlations between character states expressed in different environments not only constrain the rate of adaptation, but also may dramatically affect population abundance in each sub-habitat. Although the model is currently restricted to density-independent selection, we feel that it still provides some insight into the link between adaptation and population abundance.

Consider a situation in which a random-mating population is found in an environment with two sub-habitat types. Population regulation is global, such that each sub-habitat contributes to the mating pool in proportion to its frequency and the mean fitness of individuals selected there. Each individual experiences only one niche. In each sub-habitat, there is variation in a character that influences the intrinsic rate of population increase in that environment. Let the phenotypic value of this character in the environment be z_i , with a phenotypic variance of $P_{ii} = G_{ii} + E_{ii}$, where G_{ii} and E_{ii} are, respectively, the genetic and microenvironmental variances in the character state expressed in the i sub-habitat. Then, G_{ij} is the genetic covariance between the character states that are expressed in each niche (see Via & Lande 1985 for a more complete explanation of terms and assumptions).

Within each sub-habitat, assume that the character state that influences the demographic parameters is under stabilizing selection possibly toward

different optima in each niche (Fig. 10.5). The strength of selection is determined by the width of the fitness function [$w(z_i)$] and the distance of the mean phenotype from the optimum ($\bar{z}_i - \theta$).

In this framework, the evolution of adaptation to the heterogeneous environment is the product of the genetic covariance matrix for the character states expressed in each environment and the vector of selective forces acting directly on each character state (Via & Lande 1985, equation 9). The overall level of adaptation is expressed by the mean fitness $\bar{W} = \sum q_i W_i$, where q_i is the fraction of the population that experiences the i subhabitat, and \bar{W}_i is the mean fitness of individuals selected there. Because $\bar{W} = N(t+1)/N(t) = e^r$, the expressions for the evolution of adaptation of the mean phenotype that are written in terms of genetic parameters and selective forces can be linked to population growth and numerical abundance.

Via & Lande (1985) showed that the genetic covariance between the character states expressed in each sub-habitat can produce significant constraints on adaptation. Genetic correlations cause a lack of independence between adaptation to the various sub-habitats. When the direction of selection on the character states expressed in each sub-habitat differ, an evolutionary increase in adaptation to one sub-habitat can result in a

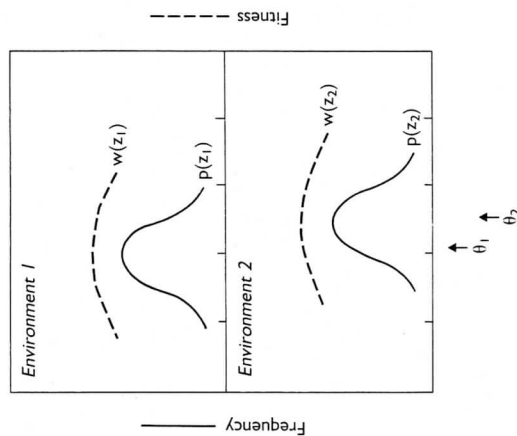


FIG. 10.5. Diagram illustrating the phenotypic distribution, $p(z_i)$, of a character z_i , in each of two environments, different phenotypic optima (θ_1, θ_2) in each environment, and the fitness functions, $w(z_i)$, for stabilizing selection on those phenotypes in the two environments. The x-axis represents the phenotypic value (z).

correlated loss of adaptation to other sub-habitats. This maladaptation leads to decreases in population abundance in some sub-habitats. In this model, the rate of adaptation to the i th niche depends on (1) the difference between the phenotypic mean and the optimum phenotype, (2) the width of the fitness function, (3) the frequency of each niche, and (4) the magnitude and signs of the genetic variances and covariances of the character states. In general, when constraining genetic covariances lead to loss of adaptation in one or more of the niches, it will be the one(s) in which evolution is occurring most slowly due to asymmetries in any of the four aspects just discussed.

The effects of genetic correlations across environment on mean fitness (and thus on e') will be shown for a two-environment case when one environment is rare ($q_2 = 0.3$), and when there is moderate genetic variance and weak selection in each environment. If the character states expressed in the two environments are uncorrelated (Fig. 10.6a), then mean fitness in both sub-habitats increases fairly rapidly. However, if the character states in the two sub-habitats are selected in the same direction but are negatively genetically correlated (in Fig. 10.6b), then the mean fitness in the rare environment can decrease dramatically as a correlated response to adaptation in the more common environment where evolution is occurring more rapidly. In this example, mean fitness in the rare environment drops to such a low state that it is effectively a population sink, slowing the rate of overall population growth ($\bar{W} = e'$) considerably compared to what is attained in the absence of the constraining genetic correlation.

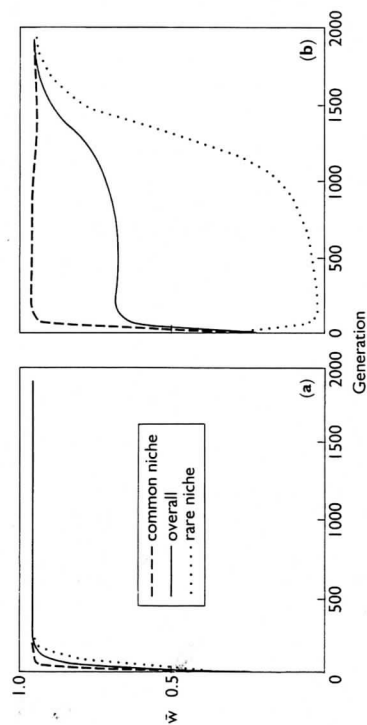


FIG. 10.6. Effects of genetic correlation across two environments in a quantitative character on the mean fitness (\bar{W}) both within each sub-habitat. Phenotypic optima are $\theta_1 = 1.5$, $\theta_2 = 2.7$. Relative frequencies of environments 1 and 2 are 0.3 and 0.7. (a) Genetic correlation between character states in different environments is zero, so that adaptation can proceed independently in each niche. (b) Genetic correlation across environments is -0.78 .

These examples show that even for the density-independent case, the abundance of plants in different sub-habitats can critically depend on the genetic structure of plant populations. Although we have not yet integrated models of joint evolution of population parameters with models of density-dependent population regulation in each of the subhabitats, the potential consequences of such evolution can be illustrated by some examples. Thus consider that λ_{AA} is initially 1.25 (i.e. $r_{AA} = 0.25$) in both habitats; it then evolves to 4.75 in niche 2, but this evolution of high λ in niche 2 is accompanied by a decline in λ to 0.25 in niche 1. This approximates the situation shown in Fig. 10.6. Although the intrinsic rate of population increase (λ or r) averaged over both habitats is increasing, the overall population size declines (Fig. 10.7). This is because in niche 2, the population is more severely limited by its carrying capacity and so the net increase in λ in niche 2 does not fully compensate for the decrease in λ in niche 1. Therefore in heterogeneous habitats, net increases in r do not necessarily result in increased population size.

While the above example represents a quantum shift in the population parameters, we can study this shift in a more continuous way, by incrementing r in the two habitats in each generation. When we do this, large shifts in population size may occur (Fig. 10.8a) sometimes in a very abrupt manner. This is because in the models, λ below 1 (i.e. negative r) leads to shifts in patterns of density dependence, so resulting in substantial shifts in population dynamics. Thus in Fig. 10.8a, the model specification is such that when r becomes negative then if the population is above carrying capacity, density effects result in zero population growth. However, change this assumption, and consider that when r becomes negative, then there is no density

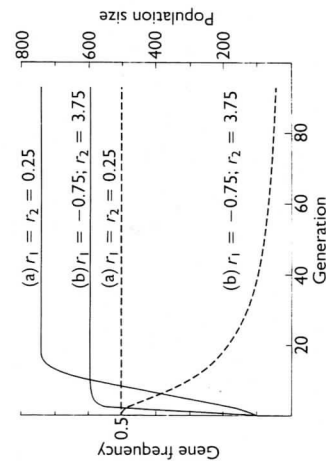


FIG. 10.7. Effect of a difference of r on population size and gene frequency; niche 1: $K_{AA} = 800$, $K_{AB} = 800$, $K_{BB} = 700$; niche 2: $K_{AA} = 700$, $K_{AB} = 800$, $K_{BB} = 800$. (a) r in both niches $= 0.25$ (b) in niche 1, $r = -0.75$; in niche 2, $r = 3.75$.

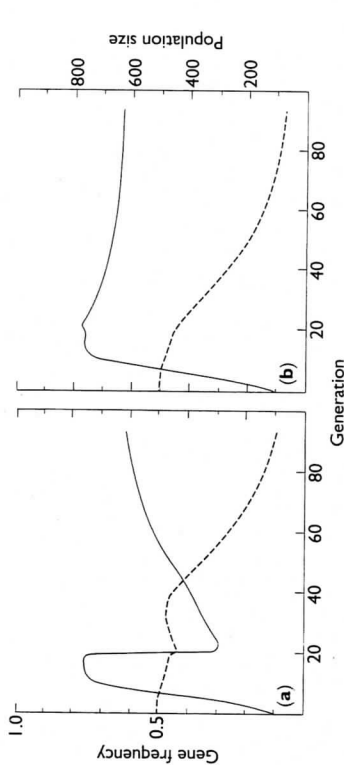


FIG. 10.8. Effect of a gradual change in r in the two niches on population size and gene frequency. Parameters as in Fig. 10.7, except that r changes such that linearly over 100 generations, in niche 1 $0.25 \rightarrow 0.75$, and in niche 2 from $0.25 \rightarrow 3.75$. (a) Density dependence in r logistic when $r > 0$. When $r < 0$, $r = -1$ when $N > K$, and $r = r(N/K - 1)$ when $N < K$. (b) Density dependence in r logistic when $r > 0$. When $r < 0$, no density dependence occurs.

regulation at all. The result is that now populations size increases (Fig. 10.8b), sometimes abruptly so. Unfortunately, it is not clear to what extent these abrupt changes are model dependent, because given the mathematical form of the model, it is difficult to maintain a functional form of the density dependence for negative values of r similar to that for positive values.

Nevertheless, these models illustrate how populations may possibly go extinct by virtue of 'over-specialization'. Thus consider a population in an area, a small proportion of which is a sub-habitat that has a very high carrying capacity, but low population growth-rate, and another much larger portion of that area which is a sub-habitat that has a low growth-rate, but also a low carrying capacity. Then in the small sub-habitat there will be strong selection to increase r . If this is negatively correlated with r in the abundant habitat, then the population will evolve as before, decreasing in the larger area, and increasing in the smaller area. However it is possible that while this is occurring, total population size actually decreases (Fig. 10.9). This is because most of the migrants enter the abundant sub-habitat, where they now have a low r , while the increased r in the rare habitat is insufficient to maintain the population in view of the large amount of emigration to the more abundant habitat. (As r continues to increase in the rare habitat, the population eventually 'recovers', but in the meantime, it has gone through a very severe bottleneck.) It is actually rather unlikely, given the unequal amount of gene flow, that selection in the rarer habitat would be sufficient to result in negatively correlated responses in the more common habitat. However, the example illustrates how relatively simple scenarios can be

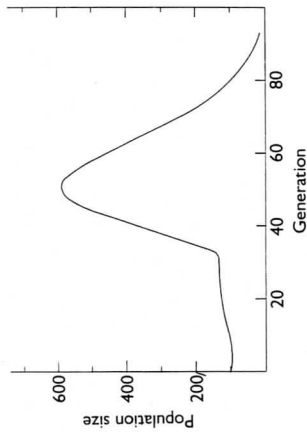


FIG. 10.9. Effect of a gradual change in r in the two niches on population size; niche 1: $K = 200$, initial $r = 0.4$; niche 2: $K = 800$, initial $r = -0.5$; r is decremented by 0.01 in niche 1 and increased by 0.05 in niche 2 each generation. From the mating pool, a fraction 0.85 enters niche 1, and a fraction 0.15 enters niche 2.

constructed which show dramatic effects of genetic change on population abundance. Assessment of whether these scenarios are at all realistic will require considerably more empirical evidence than is currently available.

CONCLUSION

The models presented in this chapter have been used to illustrate the various ways in which genetic variation in population parameters can affect numerical abundance. Examples have been chosen to emphasize that a basic knowledge of mechanisms limiting population size are essential before one can begin to translate any experimental observation of genetic differentiation into a statement about the numerical effects of such differentiation. Because knowledge of regulatory mechanisms is unavailable for any natural plant population (with few exceptions), analytical experimental approaches should involve adjusting densities of not only extant populations, but also of reciprocally transplanted populations. Input-output studies of contrasting genotypes sown at a range of densities into natural habitats can be used to predict equilibrium population sizes in the simplest situation of spatially separated, yet ecotypically differentiated populations. In more complex situations, such as heterogeneous environments, analysis of the component genetic and ecological factors determining abundance may prove difficult or impossible.

Nevertheless, other approaches are feasible. These approaches do not attempt to analyse the component phenomena, but simply ask about rates of population growth and spread given different input levels of genetic variance. Input levels of genetic variance can be controlled initially by varying the

number of founders (but then matters become confounded with inbreeding) or by varying the genotypic diversity of the founders. Alternatively, 'natural' seed dispersal could be replaced by dispersal from manipulated seed sources. These seed sources could be totally genetically uniform, as from crosses between different doubled haploids, or relatively less so, as with seed derived from open pollinated plants. Such experiments would give us a much clearer idea of whether genetics does or does not make a difference to population abundance. However, we know of no such studies.

There is also a need to develop parallel theoretical studies which incorporate model assumptions that are more realistic than those used here. In particular, there is serious lack of data on levels of genetic variation and covariation in natural populations with regard to the population parameters discussed here. Such data provide a firmer basis for translating field experiments into realistic expectations. Controversy about the importance of the 'genetic factor' in abundance and distribution will remain in the realm of belief till recent theories integrating population ecology and genetics are translated into experimental analyses.

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