PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

Frequency Dependence and Competition [and Discussion]

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Phil. Trans. R. Soc. Lond. B 1988 **319**, 587-600
doi: 10.1098/rstb.1988.0067

Phil. Trans. R. Soc. Lond. B **319**, 587–600 (1988)
Printed in Great Britain

Frequency dependence and competition

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Intraspecific competition implies interaction among the individuals of a population, so natural selection on genotypic variation in characters related to the competition will necessarily be frequency dependent. Intraspecific antagonistic competition exhibits properties similar to other behavioural interactions between individuals. In exploitative intraspecific competition the interactions among individuals are less direct. Exploitation modifies the abundance of the various limiting resources according to the use of these resources by the individual members of the population. The amount of resource available to an individual is therefore a function of the phenotypes present in the population, through their density and frequency.

Introduction

Natural selection orginates from differences in the performance of individuals in the environment where they live. The description of natural selection therefore proceeds by depicting the relation between the individual and its environment, the physical environment as well as the biotic environment made up of other living organisms. The process of natural selection is therefore perceived as an ecological process, and the classification of ecological processes provides obvious categories of possible causes for natural selection. Other views of selection in the synthetic theory of evolution are suggested by the indirect nature of biological inheritance, but here it suffices to address the ecological classification, which refers to existing phenotypic variation as material for natural selection.

Natural selection depends on the particular environment in which the population of interest lives, so selective forces shaped by the biotic environment vary with the characteristics of this environment. Individual fitness therefore becomes a function of the size and the phenotypic composition of the populations with which the individual coexists. A most important component of the biotic environment of an individual is the population of conspecific individuals, so individual fitness depends on the size and composition of its own species population. In population genetical models the influence of the biotic environment is incorporated by letting the individual fitness depend on the density of the species and on the frequency of the various phenotypes under consideration. Density-dependent selection and frequency-dependent selection therefore express the extension of classical models of population genetics to the situation where the effects of intraspecific interactions or interactions between species are also taken into account. The distinction between the two types of environmental influence on individual fitness is often purely technical, as selection dependent on the composition of a population usually varies with the density of the population.

Interspecific and intraspecific interactions are qualitatively different as causes of natural selection. Three fundamentally different types of interaction are considered in ecology, namely competition, mutualism and predator–prey (or host–parasite) interactions. Seen as interspecific

interactions, their influence on natural selection and evolution in a given species may be judged, on the basis of the classical models of constant fitnesses, to provide reasonable short-term estimates under simplified circumstances. This simplification is, of course, a reflection of the fact that natural selection and evolution occur within a population. Changes in a given species only amount to changes in the environment of other species. Even though this may alter the evolution of the other species, and therefore change the environment of the original species, it all happens in the future. Long-term results have in any case to take account of the concurrent evolution in the interacting species. For any particular species these models, strictly speaking, describe its evolution and the development of its biotic environment. Therefore, it is not surprising that conclusions from the comprehensive models are adequately predicted by classical models of population genetics.

The description of intraspecific interactions, on the other hand, is simply an integral part of the description of natural selection. In general, variation in the phenotypes related to intraspecific interactions gives rise to natural selection with individual fitnesses that are inherently frequency- and density dependent. Of the three fundamental types of ecological interactions the most relevant here are intraspecific competition and intraspecific mutualism. I will be concerned mainly with the effects of competition, but competition and mutualism share some evolutionary properties. The interaction of an individual with other individuals has an effect both on the individual concerned and on other individuals, but the survival and well being of the individual concerned need not depend on the effect it has on the other individuals. The benefit or the harm done to the individuals may have no influence on the fitness of the individual concerned. Indeed such actions may often be detrimental to the individual, as exemplified by Haldane's (1932) problem of the evolution of socially valuable, but individually disadvantageous, characters. In sedentary organisms the effect of interference competition between individuals is simpler, in that the detrimental effect of the interaction on one individual may be equivalent to the advantageous effect on the other individual, as for instance in competition for space among barnacles.

Intraspecific competition may also be less direct, where the individual fitness is influenced by the presence of other individuals through their modification of the environment. In motile organisms this may occur as exploitative competition, where the individuals use a limited resource like, for instance, food. The availability of food is then influenced by the consumption of the individuals in the population. Thus, the population modifies the environment, reflecting the average individual consumption, and the consumption of the average individual is determined by the composition of the population. The environmental modification, and therefore the individual performance, is a function of the frequency and density of the various phenotypes in the population.

Intraspecific competition

The simplest model of intraspecific competition is the logistic model of Verhulst (1838) and Pearl & Reed (1920). This model originated as a modification of the Malthusian growth model, so that the expected number of offspring per individual decreases linearly with increasing population density in order to reflect competition for limiting resources. As in the Malthusian model, all individuals in the logistic model are considered identical. All individuals are affected equally by an increase in population density, and the measure of population density is equally affected by each individual. Models introducing heritable variation in the

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

competitive effects of various phenotypes were introduced and analysed by Kostitzin (1936, 1938; see Scudo & Ziegler 1976, 1978), who considered models of natural selection based on the Lotka–Volterra models for interspecific competition. These were models of frequency-dependent selection and he argued, for instance, that in a haploid species polymorphism may be maintained in characters related to the exploitation of resources. Kostitzin's models were 'continuous-time' models in the tradition of Fisher (1930) and Norton (1928). After this founding work in theoretical evolutionary ecology little happened until the study by MacArthur (1962), who had little to say about frequency-dependent effects. Levin (1971) and Smouse (1976) studied the frequency-dependent effects in more detail, using models of inter- and intraspecific competition closely related to those of Kostitzin.

Contemporary developments in theoretical evolutionary ecology are based on discrete-time models, which simplify the handling of the population genetic aspects of the evolutionary process (Anderson 1971; Roughgarden 1971; Clarke 1972). These are models of density-dependent selection, but the model of Clarke also incorporates variation in characters related to intraspecific competition. The model of Anderson and Roughgarden is a discrete-time analogue of the logistic model, but Clarke's model, also called the hyperbolic model, is more directly related to the logistic (Leslie 1957). The extension of Clarke's model to a discrete-time logistic was introduced by Poulsen (1979). Poulsen's model extends the discrete models by recognizing that the genetical simplification only needs discrete breeding and non-overlapping generations. It describes competition and survival in continuous time.

The Poulsen model for population regulation considers a homogeneous population with non-overlapping generations, and it monitors population size immediately before reproduction. Let x denote the population size at this stage in a given generation. Assume that these x individuals each produce B offspring, so the initial population size in the offspring generation is $u(0) = Bx$. The number of offspring at age t is $u(t)$, and the individuals are assumed to mature after a fixed time of development, say T. Immediately before reproduction the population size of the offspring generation is $x' = u(T)$. During development the population experiences mortality due to density-independent and density-dependent causes, so the population size changes according to

$$\frac{\mathrm{d}u}{\mathrm{d}t} = -(d+cu)\,u, \tag{1}$$

where the constants d and c are the density-independent death rate and the coefficient of the density-dependent death rate respectively. In this model the probability of survival in a low-density population is approximately $D = \mathrm{e}^{-dT}$, so the population multiplies by the factor DB per generation when the population size is small. The recurrence equation for the population size can be written:

$$x' = \frac{DBx}{1+\gamma(1-D)\,Bx}, \tag{2}$$

where $\gamma = c/d$. The population size therefore grows according to the logistic model, in that the population sizes follow an integral curve of the continuous-time logistic model at discrete time intervals. If the density-independent growth factor DB is larger than unity, that is, if the population grows at low densities, then the population size converges to a globally stable equilibrium given by

$$x = \frac{DB-1}{\gamma(1-D)\,B}. \tag{3}$$

The evolutionary model considered by Clarke (1972) may be viewed as basing its fitness definition on (2), but given the present specification in terms of primary fitness parameters, the rates of birth and death, the formulation of a model of natural selection can be made more directly (Poulsen 1979). Consider an autosomal locus with two alleles, A and a, in a panmictic population. Assign to the genotypes AA, Aa and aa the indices 1, 2 and 3, respectively, and let x_i, $i = 1, 2, 3$, denote the number of the three genotypes in the population in a given generation. The sum $x = x_1 + x_2 + x_3$ is the total population size. The parameters of the model may then be assigned for each genotype, so, for instance, genotype i will have the density independent death rate d_i, $i = 1, 2, 3$. For simplicity we will assume that all genotypes have the same fecundity B, as fecundity selection and sexual selection may give rise to special kinds of frequency-dependent selection (see, for example, Feldman *et al.* 1983; O'Donald 1980). The frequency of allele A in this population is $p = (2x_1 + x_2)/(2x)$, and that of allele a is $q = (2x_3 + x_2)/(2x)$, so with random mating and equal fecundity the number of offspring of the various genotypes becomes

$$\begin{bmatrix} u_1(0) \\ u_2(0) \\ u_3(0) \end{bmatrix} = \begin{bmatrix} p^2Bx \\ 2pqBx \\ q^2Bx \end{bmatrix}. \tag{4}$$

These offspring suffer a genotype-dependent mortality during development, so the change in u_i, $i = 1, 2, 3$, is given by the equation

$$\frac{\mathrm{d}u_i}{\mathrm{d}t} = -(d_i + c_{i1}u_1 + c_{i2}u_2 + c_{i3}u_3)\,u_i, \tag{5}$$

where the density-dependent death rate coefficient c_{ij}, $j = 1, 2, 3$, describes the mortality inflicted on genotype i by the presence of an individual of genotype j. After development the three genotypes exist in the numbers $x_i' = u_i(T)$, $i = 1, 2, 3$.

Equation (5) has the same correspondence to equation (1) as the Lotka–Volterra equations for interspecific competition have to the logistic equation, so (5) is the discrete-time parallel to Kostitzin's equations. The density-dependent death rate coefficients of (5) describe the effects of intraspecific competition, and the result may be selection on the genotypic variation that depends both on the density and on the composition of the population.

Not every matrix $(c_{ij})_{i,j=1,2,3}$, however, produces density- and composition-dependent selection. If all density-dependent death rate coefficients are equal, then selection becomes density independent, but the same happens if only c_{ij} is independent of i, that is, $c_{ij} = g_j$ for all $i, j = 1, 2, 3$. In this case (5) integrates to the (implicit) equation

$$x_i' = u_i(T) = u_i(0)\,D_i\,G[u_1(0), u_2(0), u_3(0)], \tag{6}$$

where

$$G[u_1(0), u_2(0), u_3(0)] = \exp\left[-\int_0^T (g_1u_1 + g_2u_2 + g_3u_3)\,\mathrm{d}t\right] \tag{7}$$

and $D_i = \exp(-d_i\,T)$. As the factor G in (6) does not depend on i, it does not produce selection, so the gene frequency in the offspring generation becomes

$$p' = \frac{p(pD_1 + qD_2)}{p^2D_1 + 2pqD_2 + q^2D_3}, \tag{8}$$

which is the ordinary recurrence equation for constant fitnesses.

Therefore, intraspecific competition with variation in the impact of the presence of a given individual on other individuals does not produce either density dependence or frequency dependence in the selection coefficients unless concurrent variation in the susceptibility of the individuals occurs. The result is frequency-dependent population regulation, so changes in population size during evolution need not bear any immediate relation to the increase in mean survival of the population (Poulsen 1979). In particular, a genotype that differs from other genotypes by being either more altruistic or more antagonistic will not be distinguished by selection unless it at the same time differs in its susceptibility to the behaviour of the other individuals or in aspects of its density-independent fitness. This conclusion pertains to the panmictic situation considered here, but both behavioural characteristics may be affected by kin selection under appropriate circumstances.

If the density-dependent coefficients of death rate within each of the equations given by (5) are equal, that is, if c_{ij} is independent of j so $c_{ij} = f_i$ for all $i, j = 1, 2, 3$, then selection becomes density dependent. In this case (5) integrates to the (implicit) equation

$$x_i' = u_i(T) = u_i(0)\, D_i F(u)^{f_i},$$

where $u(t) = u_1(t) + u_2(t) + u_3(t)$ and

$$F(u) = \exp\left[-\int_0^T u(t)\,\mathrm{d}t\right]. \tag{10}$$

This factor F only depends on the density of the population during development, so in this sense selection is purely density dependent. However, the variation during development in the total density, u, is determined by the variation in the density of each genotype, u_i, $i = 1, 2, 3$, so the variation in u depends on the initial composition of the population, that is, F is frequency-dependent. The frequency dependence, however, is weak, and the model has a property that parallels the purely density-dependent fitness models (Roughgarden 1976), namely that the average population size, $F(u)$, increases after an initial period during evolution (Iwasa & Teramoto 1980), a property that is not characteristic of other frequency-dependent selection models (Cockerham *et al.* 1972).

Symmetrical competition

The Poulsen model is very detailed, and its analysis in more complicated situations is difficult. For the further analysis of intraspecific competition it has proved convenient to use the Anderson–Roughgarden model which, in its 'difference equation' formulation, refers more directly and simply to the differential equations of continuous time Lotka–Volterra models. As a model for population regulation it is given by the recurrence equation

$$x' = x[1 + V(K - x)]. \tag{11}$$

Unlike the Poulsen model this uses the heuristic parametrization of Gause (1934), in terms of the equilibrium population size, K, also called the carrying capacity, and a proportionality parameter V that provides the growth factor at low population densities as approximately $1 + VK$. The model has the unfortunate property that the factor $1 + V(K - x)$ becomes negative for x sufficiently large, and this happens inevitably if V is sufficiently large. In addition the model involves a time-lag in the density response, so for moderately large values of V cyclic or

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

chaotic behaviour is expected. Therefore, the model should be viewed as an approximation to the continuous-time logistic model, and V should be assumed to be small (Christiansen & Fenchel 1977).

Equation (11) immediately suggests the form of the individual fitness values in an evolutionary model, in that the growth factor $W = 1 + V(K - x)$ plays the role of fitness in classical population genetics. This procedure was first suggested by Wright (1960) and later used extensively, commencing with the work of Anderson (1971), Roughgarden (1971) and Clarke (1972). This way of specifying density-dependent genotypic fitnesses leads to the general result that selection on the variation at one locus in a panmictic population maximizes the equilibrium population size in the population (Roughgarden 1976). This result is reminiscent of MacArthur's (1962) result in continuously breeding populations, and it parallels the fitness maximization principle for constant genotypic fitnesses (Fisher 1930; Kingman 1961).

The extension of the Anderson–Roughgarden model to interspecific competition was made by Roughgarden (1972, 1974, 1976) and to intraspecific competition by Matessi & Jayakar (1976). Leon (1974) extended Clarke's model to interspecific competition. With the same genetic model as in the previous section intraspecific competition provides the genotypic fitnesses

$$W_i = 1 + V[K_i - x(p^2\gamma_{i1} + 2pq\gamma_{i2} + q^2\gamma_{i3})], \tag{12}$$

which are clearly density- and frequency dependent (Matessi & Jayakar 1976; Christiansen & Fenchel 1977). The parameters γ_{ij}, $i, j = 1, 2, 3$, are the intraspecific competition coefficients, and they describe the decrease in the fitness value of genotype i due to the presence of an individual of genotype j. The K parameters have a significance that parallels the K parameter in Equation (11), but here the equilibrium population size in a monomorphic AA population is K_1/γ_{11}.

The fitness maximization principle for constant selection and the similar maximization principle for purely density-dependent selection both break down when frequency dependence is allowed. However, Matessi & Jayakar (1976, 1980, 1981) analysed the situation of symmetrical competition, where $\gamma_{ij} = \gamma_{ji}$ for $i, j = 1, 2, 3$, and found that the maximization principle for density-dependent selection may be extended. The quantity that is maximized by selection may be written as K_0^2/γ_{00}, where

$$K_0 = p^2K_1 + 2pqK_2 + q^2K_3 \tag{13}$$

is the average carrying capacity in the population and

$$\gamma_{00} = p^2\gamma_{10} + 2pq\gamma_{20} + q^2\gamma_{30} \tag{14}$$

is the average competition felt by an individual in the population, which here is given in terms of the average competition felt by an individual of genotype i:

$$\gamma_{i0} = p^2\gamma_{i1} + 2pq\gamma_{i2} + q^2\gamma_{i3}. \tag{15}$$

If selection is purely density dependent, so $\gamma_{ij} = 1$ for $i, j = 1, 2, 3$, then selection maximizes the average carrying capacity K_0 of the population (Anderson 1971; Charlesworth 1971; Roughgarden 1976).

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

In terms of intraspecific competition the result is equally straightforward, which may be seen by considering the case $K_1 = K_2 = K_3$. The maximization principle then says that selection minimizes γ_{00}, the average competition felt by an individual in the population (Matessi & Jayakar 1976). Thus, the maximization of K_0^2/γ_{00} can be interpreted as a balance between the maximization of the carrying capacity and the minimization of intraspecific competition.

Exploitative competition

The general principles derived for selection by intraspecific competition can be applied to more particular situations, where the competitive interactions are understood and can be related to observable phenotypic variation in the population. The exploitative competition model of MacArthur & Levins (1967) and Levins (1968) which relates the consumption of food to the size of trophic organs is a good basis for a more detailed analysis of intraspecific competition.

The MacArthur–Levins model has been used as a basis for the description of competition for food among *Anolis* lizards (Schoener & Gorman 1968; Roughgarden 1983) and among *Hydrobia* mudsnails (Fenchel 1975; Fenchel & Kofoed 1976). *Hydrobia* feeds on microscopic algae (mainly diatoms) at the surface of marine sediments and a snail ingests food and other particles with a distribution that depends on its size. The mean particle size ingested is proportional to the size of the snail. On a logarithmic scale the distribution of particle sizes for an individual snail is approximately normally distributed with a variance independent of the size of the snail (Fenchel 1975). The grazing snails inflict a mortality on the algae, and this predation may be described as a depression in the standing crop of each algal species, since their generation time is short compared to the snail predator (Fenchel & Kofoed 1976; Christiansen & Fenchel 1977). The community of algal species, as a simplification of the model, is finally described as a continuum of sizes of food particles.

The MacArthur–Levins model of resource exploitation is specified in terms of the resource spectrum $S(\rho)$ that describes the abundance of resources of quality ρ (size of the algal cell) in the absence of predators (the snails), and the utilization function of an individual predator $U(\rho)$ that specifies the rate with which the individual consumes resources of quality ρ. With x individuals all having the same utilization function the resource is grazed down to the level $R(\rho) = S(\rho) - xU(\rho)$, and each individual is ingesting resources at the rate $\int U(\rho)\, R(\rho)\, \mathrm{d}\rho$. This formulation of resource dynamics produces a model for the population dynamics by making individual fitness dependent on the amount of ingested resources (see Christiansen & Fenchel 1977).

The model becomes particularly simple when the resource abundance spectrum and the utilization functions are proportional to Gaussian distributions. For simplicity let the resource spectrum, S, have the mean zero and the variance σ^2. The utilization functions are determined by the individual mean resource quality utilized and by the variance in resource utilization by the individual. In *Hydrobia* this variance was homogeneous among individuals, and in the following argument such homogeneity will be assumed for the model population. The individual means vary in the *Hydrobia* snails, and the variance in utilization between individuals is typically about a sixteenth of the variance within individuals (Fenchel 1975), so the total variance in utilization by the snail population is dominated by the variation in resource use of the individual snail.

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

Suppose our two-allele autosomal locus influences a character related to the utilization of the resources, and suppose the three genotypes, AA, Aa and aa, on the average utilize resources with mean value of D_1, D_2 and D_3. This average use can be described by a genotypic utilization function, U_i, $i = 1, 2, 3$, with mean D_i and variance W^2, say. This variance in genotypic use is larger than the individual utilization variance because it includes the variation among individuals within the particular genotype. This added variance is the environmental variance and the genotypic variance at other loci influencing the resource utilization. Making the simplifying assumption that the genotypic utilization functions are Gaussian, the present model provides fitnesses of the form (12) where

$$K_i = \exp\left[-\frac{D_i^2}{2(\sigma^2+W^2)}\right] \tag{16}$$

and

$$\gamma_{ij} = \exp\left[-\frac{(D_i-D_j)^2}{4W^2}\right] \tag{17}$$

for $i, j = 1, 2, 3$ (Christiansen & Fenchel 1977; Christiansen & Loeschcke 1980*a*). The carrying capacity parameter (16) depends on the distance of the mean utilization of the genotype from the mode in the resource abundance spectrum. The intraspecific competition coefficient (17) between two genotypes depends on the distance between the mean utilizations of the genotypes, so the competition is symmetric. The two distances are, however, evaluated with respect to different scales. The carrying capacity parameter is scaled with respect to the sum of the resource variance and the utilization variance, whereas the competition coefficient only depends on the utilization variance. This difference in scaling is given by the parameter $\kappa^2 = \sigma^2/W^2$ that expresses the width of the resource spectrum in relation to the width of the genotypic utilization.

Consider a simplified genetic model of a locus where the genes contribute additively to the genotypic effects, that is, $D_1 = 2d_A$, $D_2 = d_A + d_a$ and $D_3 = 2d_a$, where d_A and d_a are the effects of the alleles. The intraspecific competition then maintains polymorphism when

$$(\kappa^2-1)\,(d_A-d_a)\,[d_A-d_a(\kappa^2+7)/(\kappa^2-1)] > 0 \tag{18}$$

and

$$(\kappa^2-1)\,(d_a-d_A)\,[d_a-d_A(\kappa^2+7)/(\kappa^2-1)] > 0, \tag{19}$$

otherwise the population becomes monomorphic AA if (18) is fulfilled and monomorphic aa if (19) is fulfilled (Christiansen & Loeschcke 1980*a*). In addition, the polymorphic equilibrium is globally stable whenever it exists. Thus, if the utilization of the genotypes are fairly close, that is d_A and d_a are close in value, then either condition (18) or condition (19) is violated, and the population will become monomorphic for the genotype closest to the resource optimum. This occurs, for instance, when all genotypic utilization functions are far from, and to the same side of, the resource optimum, and the utilization spectrum of the population converges towards the resource optimum. On the other hand, if the utilization spectra of the homozygotes are situated nearly symmetrically around the resource optimum (that is, d_A and d_a are numerically close in value, but of opposite signs) then conditions (18) and (19) are fulfilled, and the population will remain polymorphic. The magnitude of κ is important for the qualitative properties of the condition for polymorphism. A wide resource spectrum, $\kappa > 1$, implies that polymorphism prevails whenever d_A and d_a are of opposite signs, and polymorphism is possible also when d_A and d_a are of the same sign. A narrow resource spectrum, $\kappa < 1$, implies that polymorphism

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

never persists when d_A and d_a are of the same sign, and polymorphism only is possible when d_A and d_a are of opposite signs.

The equilibrium gene frequency is the solution to the equation

$$\frac{pK_1+qK_2}{p\gamma_{10}+q\gamma_{20}}=\frac{pK_2+qK_3}{p\gamma_{20}+q\gamma_{30}}. \tag{20}$$

The model is most reasonable when viewed as an atom of a quantitative inheritance model, so it is reasonable to assume that the difference between the allele effects d_A and d_a is small. In this case the polymorphic equilibrium only exists if both allele effects are small, and then the equilibrium is unique and can be found as

$$p=\frac{(\kappa^2-1)\,d_A-(\kappa^2+7)\,d_a}{2(\kappa^2+3)\,(d_A-d_a)}. \tag{21}$$

This equilibrium is stable (K_0/γ_{00} is maximized).

The genotypic fitnesses at the gene frequency equilibrium is found from equation (12) by using the equilibrium equation (20):

$$\left.\begin{aligned} W_1&=1-(q/p)\,s_2,\\ W_2&=1+s_2\\ \text{and}\quad W_3&=1-(p/q)\,s_2, \end{aligned}\right\} \tag{22}$$

where s_2 equals $V(K_2\gamma_{00}-K_0\gamma_{20})/\gamma_{00}$ evaluated at equilibrium. If, for instance, the utilization functions of the genotypes are symmetric around the resource optimum, that is $d_A=-d_a$, then the equilibrium gene frequencies are both $\frac{1}{2}$ and s_2 is negative when $\kappa>1$ and positive when $\kappa<1$. Thus, for a wide resource spectrum, $\kappa>1$, the hetrozygote will appear as having a lower fitness than the homozygotes, whereas for a narrow resource spectrum, $\kappa<1$, the opposite will be the case, that is, over-dominance in fitness will prevail. As another example let $d_a=0$, so the genotype aa is at the resource optimum, then a polymorphic equilibrium exists for a wide resource spectrum, $\kappa>1$, and the equilibrium gene frequency of allele A is $p=(\kappa^2-1)/[2(\kappa^2+3)]$. Now s_2 is negative when $\kappa>3.45$ and positive when $1<\kappa<3.45$. Again for a sufficiently wide resource spectrum the heterozygote will appear as having a lower fitness than the homozygotes, whereas overdominance in fitness will prevail only for a narrower resource spectrum.

The variation of the genotypic fitnesses (12) can be studied in the case of small allele effects by assuming that the population size is at the equilibrium corresponding to the prevailing gene frequency, i.e. we assume that $W_0=1$ or the equilibrium population size, $x(p)$ say, is given by $x(p)=K_0/\gamma_{00}$. Again considering the symmetric case where $d_A=-d_a$ we have that for $p=0$ the fitnesses are $W_1=1+VK(1-\gamma^4)$, $W_2=1+V(1-K\gamma)$ and $W_3=1$, respectively, where $W_1>W_2>W_3$. Thus, the least-frequent type has the highest fitness, and the dominating type in the population has the lowest fitness. The genuine variation in fitness values is, however, very hard to observe in natural or experimental populations, because the variation is reminiscent of the spurious variation in estimated fitness values that one would expect when the breeding components of selection are incompletely described by the data (Prout 1965; Christiansen *et al.* 1977; Christiansen 1984*a*).

The two-allele model with additive effects extends readily to a multiple-allele model, but the present analysis suffices as a description of a polymorphic population, in that at most two alleles

may segregate at any polymorphic equilibrium unless the allele effects are very large (Christiansen & Loeschcke 1980*a*). The introduction of a new allele into a population at a polymorphic equilibrium will have different consequences depending on whether the resource spectrum is wide, $\kappa > 1$, or narrow, $\kappa < 1$. With a wide resource spectrum a new allele A' will increase in the population when rare, when its effect, $d_{A'}$ say, lies outside the interval bounded by d_a and d_A, that is, for $d_a < d_A$ say, A' will increase if $d_{A'} < d_a$ or $d_A < d_{A'}$. After the initial increase of a new allele the population will settle on a new polymorphic equilibrium segregating two alleles (A and A', when $d_{A'} < d_a$, and a and A', when $d_A < d_{A'}$). With a narrow resource spectrum ($\kappa < 1$) a new rare allele A' will increase in frequency, when its effect lies inside the interval bounded by d_a and d_A that is, A' will increase if $d_a < d_{A'} < d_A$ (again assuming $d_a < d_A$), and the increase of A' will either result in a two-allele polymorphism segregating A' or in monomorphism for the genotype $A'A'$. Thus, selection due to intraspecific exploitative competition will be phenotypically diversifying in an environment with a wide resource spectrum. However, this increase in the variance among individuals may initially happen at the same time as genetic polymorphism arises, but if genetic polymorphism at a locus already exists, then the phenotypic diversification is not followed by a genetic diversification at that locus. The tendency towards phenotypic stabilization in an environment with a narrow resource spectrum will eventually lead to monomorphism given sufficient genetic variation. A discussion of these effects within the framework of a quantitative genetic model is given by Slatkin (1979).

The principle of a maximum of two alleles at a polymorphic equilibrium has the immediate corollary that closely linked polymorphic loci that influence a character involved in intraspecific competition will show a high degree of linkage disequilibrium (Loeschcke & Christiansen 1984). The model may be extended to cover multi-dimensional resources with very similar results (Christiansen & Loeschcke 1987). With additive gene effects the maximum number of alleles at a polymorphic equilibrium equals one plus the dimension of the resource quality description, and selection will have a diversifying tendency if the resource spectrum is wide in all dimensions and a stabilizing tendency if it is narrow in all dimensions. At equilibrium the average fitness of a given allele will be less than the fitness of the corresponding homozygote when the resource spectrum is wide in all dimensions and conversely it will be less if the resource spectrum is narrow in all dimensions.

Another route of generalization is to relax the assumption of additive gene effects and allow for dominance (Christiansen & Loeschcke 1980*b*; Christiansen 1984*b*). The introduction of dominance does not fundamentally alter the basic conclusions, except for the global stability of the polymorphic equilibrium. With dominance our genetic model becomes $D_1 = 2d_A$, $D_2 = d_A + d_a - h(d_A - d_a)$ and $D_3 = 2d_a$, where d_A and d_a are the allele effects and h describes the effect of dominance. If we restrict attention to the case of a wide resource spectrum, $\kappa > 1$, and assume that $|d_a| < d_A$ (no loss of generality), then a globally stable polymorphism prevails in the additive model, $h = 0$, when $d_a/d_A < (\kappa^2 - 1)/(\kappa^2 + 7)$, and otherwise monomorphism aa ensues. For complete dominance, $h = -1$ or 1, a globally stable polymorphism exists when $d_a/d_A < (\kappa^2 - 1)/(\kappa^2 + 3)$, and otherwise the monomorphic aa equilibrium is globally stable. Thus complete dominance and no dominance give very similar results.

For intermediate dominance, $-1 < h < 1$, a globally stable polymorphism exists when

$$\frac{d_a}{d_A} < \frac{(1-h)(\kappa^2-1)}{(\kappa^2+7)-h(\kappa^2-1)}. \tag{23}$$

For $h = 0$ and for $h = -1$ this condition coincides with the previously stated conditions, but for $h = 1$ it is different. The monomorphic *aa* equilibrium is locally stable when condition (23) is not fulfilled and $h \neq 1$, but for $h = 1$ it is unstable also when $0 \leqslant d_a/d_A < (\kappa^2-1)/(\kappa^2+3)$. This discontinuity in the conditions is caused by the simultaneous existence of the locally stable monomorphic equilibrium and a locally stable polymorphic equilibrium for an interval of d_a/d_A values for intermediate dominance and high values of the dominance parameter, that is, for $(\kappa^2+3)/(3\kappa^2+1) < h < 1$. Thus a high degree of dominance by the allele that places the homozygote closest to the optimum may cause the final outcome of selection to depend on the initial state. For parameters with suitable values, a population initially *AA* will end up polymorphic if allele *a* is introduced as rare, whereas a population initially *aa* will remain monomorphic if *A* is introduced as rare. Similar behaviour of models with absolute dominance and without dominance therefore provides no guide to the behaviour of models with intermediate dominance.

The results of this section all rely on the assumption that the utilization functions of the genotypes are Gaussian. This, however, is not a crucial assumption as any 'bell-shaped' distribution would provide a very similar structure of the intraspecific competition coefficients (May 1974). However, a qualitatively different shape, as for instance the very 'pointed' shape used by Roughgarden (1972), yields very different results, and the limitation to two alleles for a one-dimensional resource spectrum is replaced by the possibility of a large number of alleles. The mechanism of competition, as provided by the MacArthur–Levins model, is considerably more important. Even though the model seems quite robust, it is limited by the description of rather passive competition for a renewable food resource. Competition for space, a rather fixed supply of food, or any resource that an individual can occupy is not well described by the model, and evolutionarily interesting ecological models exist for these phenomena (Schoener 1976; Shigesada *et al.* 1979; Abrams 1986). Competition for space often occurs during a sedentary phase that allows individual by individual interactions, so the evolution of antagonistic interactions become feasible.

Interspecific competition

Interspecific exploitative competition is felt by the individual in the population through the depletion of resources, just like intraspecific competition. The influence on individual fitness due to interspecific exploitative competition therefore adds to the influence of intraspecific competition. Interspecific competition may be viewed as setting the stage where intraspecific competition acts, in that it forms the shape of the resource spectrum. The evolutionary consequences for a species that competes with other species in a MacArthur–Levins model with Gaussian utilization functions and a Gaussian resource spectrum are very close to the results for intraspecific competition with the optimum and the variance of the resource spectrum determined by the other species (Roughgarden 1976; Fenchel & Christiansen 1977; Christiansen & Loeschcke 1980*a*; Loeschcke 1984*a*, *b*). The shape of the spectrum of resources available to a species under interspecific competition is not Gaussian, but the results for a Gaussian resource hold with a good approximation (Loeschcke 1984*a*, *b*). It is rather unimportant for the evolution of a given species that the density of competitors depends on its phenotypic composition, even though it is very important for the dynamics of the guild of competitors (Loeschcke 1985).

These results are part of a general principle for symmetrical competition. Symmetrical

interspecific competition show the same conformity to intraspecific competition as interspecific exploitative competition shows to intraspecific exploitative competition in the MacArthur–Levins model. Matessi & Jayakar (1980, 1981) showed that evolution driven by natural selection due to symmetrical interspecific and intraspecific competition maximizes the quantity K_{00}^2/γ_{0000}, where K_{ij} is the carrying capacity parameter of genotype j in species i, γ_{ijkl} is the competition coefficient between genotype j in species i and genotype l in species k, and the index zero signifies the population average (see (13), (14) and (15)). Thus the joint evolution of a guild of competitors tends to maximize the average carrying-capacity parameter of each species and at the same time minimize the average competition in the population.

Asymmetrical interspecific competition may or may not have an influence on the evolution of a community of competitors. In general, evolution driven exclusively by competition will proceed so as to minimize the competition pressure felt by the average individual of a species (Roughgarden 1979). As with intraspecific competition it is conceivable that variation in phenotypes connected to interspecific competition is neutral with respect to natural selection because the differences between phenotypes is only in their effect on other individuals. Thus the evolution of interspecific antagonistic interactions between motile organisms poses problems that parallel those of intraspecific interactions, in that the price for costly interactions should in some way be conveyed to individuals related to the individual concerned. This could be achieved by the interspecific interaction being based on altruism within family groups, but it is also possible within groups of less-related individuals (Slatkin & Wilson 1979). In any case these dual-level individual interactions give rise to a kind of frequency-dependence in individual fitnesses that is best handled within the study of evolution of animal behaviour, and this kind of frequency-dependence is very different from that produced by the rather passive interaction between individuals and their environment.

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Discussion

J. Antonovics (*Botany Department, Duke University, U.S.A.*). Could more than two alleles be maintained in the population if the reserve spectrum was bimodal, trimodal, etc. rather than Gaussian as in Dr Christiansen's model?

F. B. Christiansen. The limit of two alleles pertains to the situation of small additive effects of the alleles on the phenotype. The result is reminiscent of the limiting similarity results for similar models of interspecific exploitative competition, and it holds for values of the allele effects up to about the standard deviation of the utilization function (Christiansen & Loeschcke 1980*a*).

With small allele effects the assumption on the functional form of the resource spectrum is immaterial. The resource spectrum determines the carrying-capacity parameter through a convolution with the utilization function, that is the carrying-capacity parameter is proportional to the average resource abundance picked by the genotype. Thus a small difference in utilization mode will correspond to a small difference in carrying-capacity parameter for all reasonable resource spectra, and this is exactly the property used in the arguments of Christiansen & Loeschcke (1980*a*). The limit of two alleles is therefore independent of the particular form of the resource spectrum.

The condition for increase of a rare allele A' introduced into a population at a stable equilibrium segregating alleles A and a also holds for a general resource spectrum. The only change is that the characterization of whether a particular resource is narrow or wide is not simply a function of the variance in the resource spectrum.

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