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QUANTITATIVE GENETICS AND POPULATION DYNAMICS

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Abstract.—This study examines the dynamics of a competition and a host-parasite model in which the interactions are determined by quantitative characters. Both models are extensions of one-dimensional difference equations that can exhibit complicated dynamics. Compared to these basic models, the phenotypic variability given by the quantitative characters reduces the size of the density fluctuations in asexual populations. With sexual reproduction, which is described by modeling the genetics of the quantitative character explicitly with many haploid loci that determine the character additively, this reduction in fitness variance is magnified. Moreover, quantitative genetics can induce simple dynamics. For example, the sexual population can have a two-cycle when the asexual system is chaotic. This paper discusses the consequences for the evolution of sex. The higher mean growth rate implied by the lower fitness variance in sexual populations is an advantage that can overcome a twofold intrinsic growth rate of asexuals. The advantage is bigger when the asexual population contains only a subset of the phenotypes present in the sexual population, which conforms with the tangled bank theory for the evolution of sex and shows that tangled bank effects also occur in host-parasite systems. The results suggest that explicitly describing the genetics of a quantitative character leads to more flexible models than the usual assumption of normal character distributions, and therefore to a better understanding of the character's impact on population dynamics.

Key words.—Chaos, evolution of sex, population dynamics, quantitative genetics.

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Population dynamics lies at the core of ecological and evolutionary theory. It shapes the structure of communities and generates evolutionary processes, determining persistence and stability, invasion and extinction. The importance of knowing the dynamic behavior of populations is reflected in the amount of interest drawn to complex dynamics since chaos was introduced to ecology by May (1974, 1976). May's models were one-dimensional and set in discrete time, but since then chaos has been found in many multidimensional systems with continuous time as well (e.g., Gilpin 1979; Takeuchi and Adachi 1983; Hastings and Powell 1991; Vandermeer 1993). However, the ubiquity of complex dynamics in ecological models contrasts with the scarce evidence for chaos in natural populations. Influential papers showing that natural systems tend to have simple dynamics include Hassell et al. (1976) and Thomas et al. (1980). However, even if a real population exhibits chaos, such complex dynamics would be difficult to detect, and some authors suggest that chaos occurs more often than commonly believed (Schaffer 1984; Schaffer and Kot 1986; Hastings et al. 1993). A convincing example of chaos is the dynamics of measles in many cities (Olson et al. 1988; Sugihara and May 1990).

In an attempt to solve this controversy one can look for general mechanisms that tend to prevent or enhance complex population dynamics. For example, the dynamics of a metapopulation can be very different from those of its constituent local populations (Gilpin and Hanski 1991), and the mixing of interactions through dispersal between local populations can stabilize the whole system (Hastings 1993; Stone 1993; Doebeli 1995a). A mix of interactions also occurs among different phenotypes, and one line of work examines the influence of phenotypic variability and population genetics on the dynamics of populations (Begon and Wall 1987; Koella 1988; Saloniemi 1993; Doebeli and Koella 1994; Hastings and Harrison 1994; Doebeli 1995b). In particular, it has been argued by Doebeli and Koella (1994) that sexual reproduction, with segregation at one locus, simplifies the dynamics.

In this paper, I study the effect of a continuously varying character on population dynamics. I extend a nonlinear, one-dimensional model by assuming that the strength of the interactions between two individuals depends on the relative value of the character. The genetics are modeled explicitly by many haploid loci with two alleles that determine the character additively. I describe a simple method to obtain a model for quantitative genetics that keeps track of the frequencies of single phenotypes.

The key questions in quantitative genetics concern patterns of variation and the maintenance of heritability for quantitative characters (Barton and Turelli 1989). When population dynamics enter the picture, they are usually a tool rather than the goal of study. Thus the model I use here originates in the competition model used by Slatkin (1980) to analyze ecological character displacement. However, instead of modeling the genetics explicitly, Slatkin made the one assumption that is almost ubiquitous in quantitative genetics: he assumed that the distribution of the continuous character is normal in each generation. Although convenient for his purposes, this assumption implies severe restrictions on the dynamics of the population and always leads to stability (Slatkin 1980). The main purpose of this paper is to examine the dynamic consequences of dropping the assumption of normality and instead introducing explicit quantitative genetics.

To achieve some generality, I analyze two different models, the first a model for competition, the second for host-parasite interactions. In both cases, I first compare the dynamics of the basic homogenous model with the dynamics of a phenotypically variable asexual population. The demographic parameters of the single phenotypes in this population are the same as those in the basic model, and the phenotypes vary in their value of the quantitative character determining the interactions. I then compare these results with the dy-

namics of phenotypically variable populations in which reproduction is sexual, modeled by quantitative genetics.

Differences in population dynamics can generate evolutionary change. Therefore, I compare the sexual and asexual populations in an evolutionary context. In a pioneering study, Hamilton (1980, 1982) observed that sex reduces the fitness variance in frequency-dependent host-parasite systems, which leads to a higher mean growth rate (Gillespie 1977). This can imply a competitive advantage of sex large enough to overcome the classical twofold growth rate of asexuals. May and Anderson (1983) replied that this advantage is lost with density-dependence, but Doebeli and Koella (1994) argued that there is an advantage even with density-dependence if enough phenotypic variability is present. This is confirmed here and extended from host-parasite systems to purely competitive interactions.

The advantage of sex is enhanced if the asexual population contains only a subset of the phenotypes present in the sexual population, because sex eases competition from asexuals by producing phenotypes that are not present among asexuals. This conforms with the tangled bank theory for the evolution of sex (Bell 1982). In the host-parasite system there is apparent competition for phenotype space, which also induces tangled bank effects. Thus the present models connect the tangled bank theory and Hamilton's ideas. The results suggest that describing the genetics explicitly instead of assuming normal character distributions leads to models that are less rigid and give more insights about the effect of quantitative characters on population dynamics.

The Quantitative Genetic Model

Let us begin with models for the dynamics of populations with discrete generations, in which all individuals are assumed to be equal:

$$N_{t+1} = N_t \cdot f(N_t). \tag{1}$$

Here N_t is the density of the population at time t, and $f(N)$ is the reproductive output per individual if the density is N, i.e., $f(N)$ is the fitness function. This model is extended to a population with many phenotypes given by a quantitative character. Then the fitness function of each phenotype depends, in general, on its own as well as on the density of the other phenotypes. Suppose k denotes a quantitative character that can take on values in a finite interval $[0, c]$. Let $p_t(k)$ be the frequency distribution of the phenotypes at time t. Let $n_t(k)$ be the density distribution of the phenotypes at time t. Then the total density N_t of the population is

$$N_t = \int_0^c n_t(k)\, dk, \tag{2}$$

and we have

$$n_t(k) = N_t \cdot p_t(k). \tag{3}$$

The fitness functions of the phenotypes now depend on n_t, i.e., on the distribution of all phenotypes. If reproduction is asexual and the phenotypes breed true, and if f^k denotes the fitness function of phenotype k, the dynamics of the system are described by

$$n_{t+1}(k) = n_t(k) \cdot f^k(n_t). \tag{4}$$

This equation determines the density distribution at time $t + 1$, $n_{t+1}(k)$, as a function of the distribution at time t. For example, it the fitness of a phenotype only depends on its own density, then f^k only depends on the value of n_t at k, and we get for each phenotype the original model (1) back:

$$n_{t+1}(k) = n_t(k) \cdot f^k(n_t(k)). \tag{5}$$

In this case the phenotypes are independent of each other, and the dynamics of the whole population is the linear superposition of the dynamics of the single phenotypes, i.e., no coupling between phenotypes occurs. In general, f^k will depend on a range of values of n_t, i.e., on the densities of other phenotypes as well. As we will see, such coupling can change the dynamics quite drastically.

To incorporate population genetics, I assume the simplest possible model, in which the quantitative character k is determined additively by many haploid loci, each with two alleles. For simplicity, I assume that the upper boundary c for the character values is an integer, that there are c loci, and that the effects of the two alleles at each locus are 0 and 1. These assumptions do not imply any loss of generality, since appropriately rescaling the allelic effects and linearly transforming the interval [0,c] always reduces the problem to such a situation. Since individuals are haploid, their genetic structure is described by a string of 0s and 1s of length c, corresponding to which allele is present at each locus, and I assume that the phenotype of an individual is given by the number of 1s in its genetic string. Thus, if an individual has the 0-allele at $c - k$ loci and the 1-allele at k loci, its character value is k. This means that there is no environmental variance in the phenotype. There are three reasons for this assumption.

First, my goal is to see the effect of quantitative genetics on the dynamics of the population with as few confounding factors as possible. Second, it will be clear from the definition of the fitness function of the phenotypes that the results obtained without environmental variance will remain qualitatively the same if a small amount of environmental variance is introduced. Small environmental variances were used in the models of Slatkin (1980), Taper and Case (1985) and Koella (1988), on which the present model is based. Third, there is no conceptual difficulty in introducing environmental variance in the model, but it slows down the computer simulations substantially.

Phenotypes thus range from 0, displayed by individuals having the 0-allele at each locus, to c, which corresponds to having the 1-allele at each locus. The dynamics of such a population still depend on the fitness functions f^k, which determine the contribution of phenotype k to the gamete pool. However, in a sexual population selection, given by the f^k, is followed by random mating. Compared to the asexual model, this corresponds to an intermediate step of "reshuffling" that determines the frequency distributions of the phenotypes in the next generation. To see the effect of mating, we have to determine the phenotypic distribution of the offspring, given the phenotypes of the parents. For this the basic assumption is free recombination between loci, so that the allele of an offspring at a particular locus has probability 0.5 to come from either parent. This is a common assumption for

population dynamic models in quantitative genetics (Lande 1976; Slatkin 1980; Taper and Case 1985), but it is of course at best an approximation of the real genetics of a quantitative character. However, without this assumption it would be difficult to keep track numerically of explicit genetics with many loci.

Suppose then that the phenotypes of the parents are i and j, so that one parent has i 1s and $c - i$ 0s in the genome, whereas the other has j 1s and $c - j$ 0s. The number of loci at which both parents have the 1-allele is defined as the overlap o. It is easy to see that

$$\max(0, i + j - c) \leq o \leq \min(i, j), \tag{6}$$

see Figure 1. From the o overlap loci, the offspring inherits the 1-allele. Also, there are $i + j - o$ loci at which one parent has the 1-allele and the other one has the 0-allele. At these loci the offspring has either one of the alleles with probability 0.5, since I assume free recombination between loci. At the remaining loci both parents have the 0-allele, hence so does the offspring. It follows that the phenotype of the offspring lies in the interval

$$[o, i + j - o], \tag{7}$$

and that the phenotype distribution in this interval is binomial. Therefore, if p_{ij}^o denotes the phenotype frequency distribution of offspring having parents with phenotypes i and j and with overlap o, we have

$$p_{ij}^o(k) = \begin{cases} \binom{i + j - o}{k - o} \cdot (0.5)^{i+j-o} & \text{for } k \in [o, i + j - o] \\ 0 & \text{otherwise} \end{cases}. \tag{8}$$

To determine the frequency distribution p_{ij} for the offspring phenotype of parents i and j, we have to take the sum of the distributions determined by the overlaps, weighted by the probability that a particular overlap occurs:

$$p_{ij} = \sum_{\text{all overlaps}} \Pr(\text{overlap} = o) \cdot p_{ij}^o. \tag{9}$$

By equation (6), o lies in the interval $[\max(0, i + j - c), \min(i, j)]$ and it is easy to determine the probability for each overlap, given i and j. Equation (9) means that the phenotype distribution p_{ij} of the offspring is a weighted sum of nested binomial distributions (Fig. 1). However, for computational convenience I only used the outermost of these nested distributions in the numerical simulations, that is, I used the approximation

$$p_{ij} \sim p_{ij}^{o_{\min}}, \tag{10}$$

where $o_{\min} = \max(0, i + j - c)$ is the minimal possible overlap. This approximation assigns slightly higher probabilities to extreme offspring phenotypes, and slightly lower probabilities to common phenotypes (Fig. 1). It saves a lot of computer time, and a few numerical experiments showed that the quality of the results of the next two sections does not change when the correct equation (9) is used instead of the approximation (10). An indication of how good this approximation is given below in Figure 2.

We can now determine the dynamics of the sexual population. Let $p_t(k)$ and $n_t(k)$ be the frequency and density distributions of the phenotypes at time t. To describe the dynamics we have to give a recursive formula for $p_{t+1}(k)$ and $n_{t+1}(k)$, and for this it is enough to know $p_{t+1}(k)$ and the total density N_{t+1} by equation (3). Let f^k be the fitness function of phenotype k as before. Then

$$\bar{w}_t = \sum_{k=0}^{c} p_t(k) \cdot f^k(n_t) \tag{11}$$

is the mean fitness of the population at time t, and clearly the total density at time $t + 1$ is

$$N_{t+1} = N_t \cdot \bar{w}_t. \tag{12}$$

Figure 1a

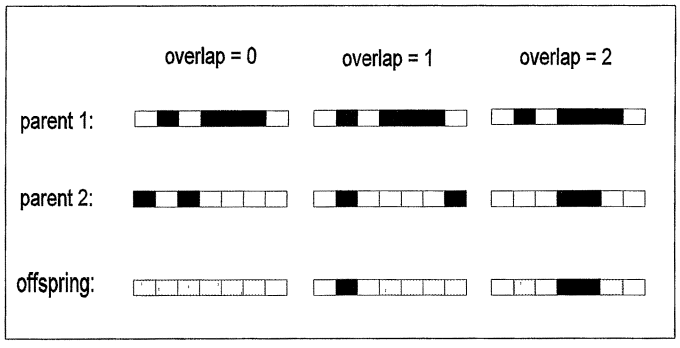

Figure 1b

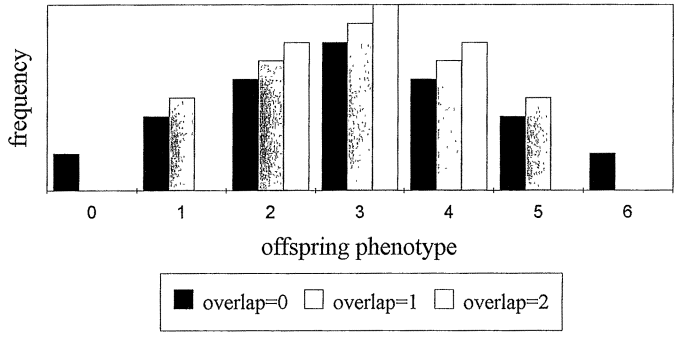

FIG. 1. Schematic description of the quantitative genetic model. 1a shows how different overlaps of the parent genomes lead to different distributions of the offspring phenotype. The distributions are shown in 1b. In this example, the genomes have seven loci. At each locus, the 0-allele is shown as a white and the 1-allele as a black rectangle. In the offspring genome, a grey rectangle indicates that the chance of having either allele at the corresponding locus is 0.5, because the parents have different alleles at this locus. The number of black rectangles is the minimal, the sum of black and grey rectangles the maximal possible phenotype value. The probability of having a particular phenotype within this range is given by binomial distributions. In this example, the overlap in the parents is either 0, 1, or 2. For different overlaps, the number of black and grey rectangles in the offspring genome varies, leading to the different distributions shown schematically in 1b. The sum of these distributions, weighted by the probability that the corresponding overlap occurs, determines the total distribution of offspring phenotypes. For the numerical simulations, I only used the distribution corresponding to the minimal possible overlap, 0 in this example, giving the widest range of phenotypes. This distribution is indicated by dark shading in 1b.

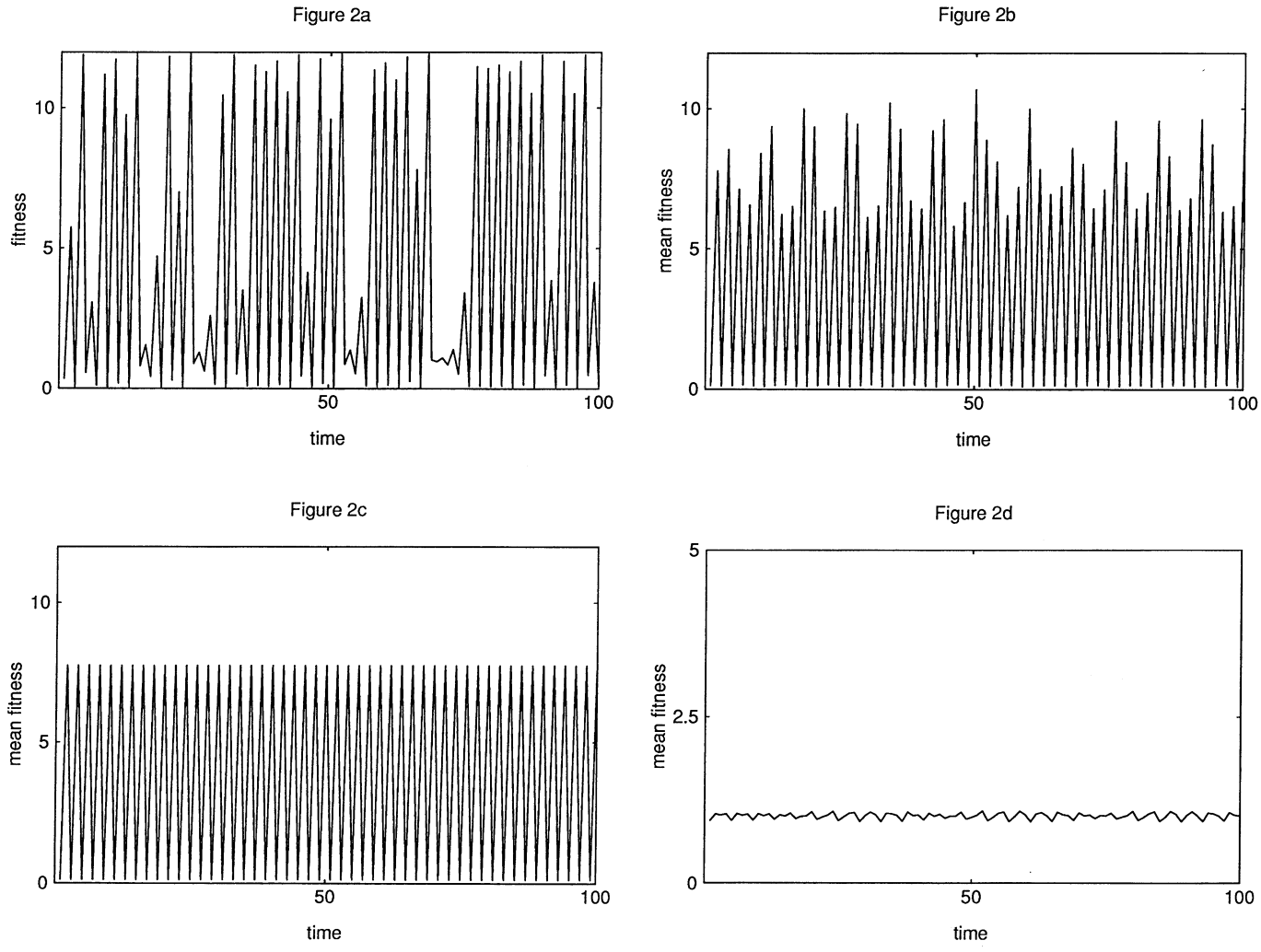

FIG. 2. Dynamics of the competition model: (a) Basic model (15) without phenotypic variability; (b) and (d) Asexual population with quantitative phenotypic variability; (c) Sexual population with quantitative genetics. The figures show the mean fitness, defined as N_{t+1}/N_t, where N_t is the total population density at time t, in successive generations. The basic model exhibits chaotic dynamics for the chosen parameters (2a). With the same parameter values for the single phenotypes and an intermediate niche width σ_β, the quantitatively variable asexual population also exhibits chaos, but with smaller fitness fluctuations (2b), and the sexual population moves on a simple two-cycle (2c) with still smaller fluctuations. This behavior is typical for a wide range of parameters: the quantitative character reduces fitness variance; this effect is magnified when reproduction is sexual, and sex leads to simpler dynamics. For some parameter combinations, phase locking can occur between the phenotypes in the asexual population (2d), such that their fluctuations cancel each other, and the total density remains almost constant over time. In this case, the asexual system exhibits simpler dynamics than the sexual system. Phase locking depends on the initial conditions, and the corresponding attractor seems to be small. It cannot occur in sexual systems because of recombination. The parameter values for Figures 2a, 2b, and 2c: $c = 40$, such that there were 40 loci in the genetic model; $\lambda = 12$, $d = 2$ (note that a choice of d determines b once λ is given), $a(k) \equiv a = 0.1$ and $\sigma_\beta^2 = 27$; for Figure 2d: $c = 30$, $\lambda = 7$, $d = 2.5$, $a(k) \equiv 0.1$ and $\sigma_\beta^2 = 6$; note that the value of d in 2d implies chaotic and highly fluctuating dynamics in the basic model (15).

Selection takes place before reproduction, and the distribution q_t in the gamete pool at time t is given by

$$q_t(k) = \frac{p_t(k) \cdot f^k(n_t)}{\bar{w}_t}, \qquad 0 \le k \le c. \tag{13}$$

Here $q_t(k)$ is the frequency of gametes coming from parents with phenotype k. The frequency distribution of the phenotypes in the next generation is now given as the sum of the offspring distributions p_{ij} coming from parents i and j, eq. (9) respectively eq. (10), weighted by the frequency of matings between i and j, i.e., by the factor $q_t(i) \cdot q_t(j)$, where q_t is given by eq. (13). Thus

$$p_{t+1} = \sum_{i,j=0}^{c} q_t(i) \cdot q_t(j) \cdot p_{ij}. \tag{14}$$

Equations (12) and (14) describe the dynamics of the sexual population, once the fitness functions f^k are determined.

The quantitative genetics in the model are described by the phenotype distributions p_{ij} of the offspring from parents with phenotypes i and j, equation (9). This formula was derived under the assumption of free recombination between haploid loci. In principle, one could derive analogous equations for more complicated genetic assumptions such as diploidy and linkage, but I have not explored these possibilities.

In the next two sections I describe results obtained numerically using equations (11)–(15) in two different population dynamic settings, in which the fitness functions are determined by different mechanisms. In the first, the quantitative character influences the competition between phenotypes, and in the second it determines the susceptibility to parasites. In both cases the fitness functions are nonlinear, so that the basic model (1) without phenotypic variation can exhibit complex dynamics. The results describe how quantitative phenotypic variability can affect these dynamics in both asexual and sexual populations.

Results

The Competition Model

For competition, the fitness function for the basic model (1), first used by Maynard Smith and Slatkin (1973), has the form

$$f(N) = \frac{\lambda}{1 + (aN)^b}. \tag{15}$$

The parameter $\lambda > 1$ is the intrinsic growth rate of the population. Depending on the assumptions about the competitive process that leads to density-dependence, the parameter b reflects different types of competitive interaction (e.g. Hassell 1975; Schoener 1976). The parameter a measures how well the individuals can cope with the environment. It influences the equilibrium density N^*, which can be thought of as the carrying capacity of the population. It is defined as the density at which the fitness of the population is 1:

$$f(N^*) = 1, \tag{16}$$

hence,

$$N^* = \frac{(\lambda - 1)^{1/b}}{a}. \tag{17}$$

Model (15) was considered by Bellows (1981) to be the most generally applicable one-dimensional ecological model. If perturbed away from the equilibrium N^*, its dynamic behavior is determined by the derivative at this point, more precisely by the modulus

$$d = \left|\frac{df}{dN}(N^*)\right| = \left|1 - b\frac{\lambda - 1}{\lambda}\right|. \tag{18}$$

Note that the parameter a does not occur in the expression for d, hence does not influence the dynamic behavior of the system. If $d < 1$, N^* is stable, with perturbed densities returning either exponentially or with damped oscillations to the equilibrium. This stability is lost when $d > 1$. As d increases, the systems displays the familiar bifurcation behavior (May and Oster 1976), going from a stable two-cycle to a stable four-cycle and, more generally, to a stable 2^n-cycle before it reaches chaos at a value of d $\sim$ 1.7.

To extend this model to a population with a continuously varying character $k \in [0, c]$, we must specify how the fitness function of phenotype k, f^k, depends on k and on the density distribution of the phenotypes, $n_t(k)$, at time t, taking into account that the strength of competition between two phenotypes depends on how similar they are. Following Slatkin (1980), the fitness of phenotype k at time t is

$$f^k(n_t) = \frac{\lambda}{1 + \left\{a(k)\cdot\int_0^c n_t(l)\beta(k, l)\, dl\right\}^b}. \tag{19}$$

The function $\beta(k, l)$ measures the strength of competition between individuals of phenotypes k and l, so that the weighted sum

$$\int_0^c n_t(l)\beta(k, l)\, dl \tag{20}$$

is the density that an individual of phenotype k experiences under competition in a population with phenotype distribution n_t. In this paper I use the following form for this competition function:

$$\beta(k, l) = \exp\left[\frac{-(k - l)^2}{2\sigma_\beta^2}\right], \tag{21}$$

where the parameter σ_β is the within phenotype niche width. Note that the function $\beta(k, l)$ only depends on the relative position of two phenotypes, that is, only on the difference $k - l$, and not on their absolute character values. Competition is strongest between similar phenotypes. Although the choice of this function conforms with existing literature (e.g., Slatkin 1980; Taper and Case 1985; Koella 1988), it has the caveat of representing density-independent competition coefficients. It has been argued that density-dependent competition coefficients are more realistic (Abrams 1980), but I have not explored this complication of the model.

It is implicitly assumed in the model that all phenotypes have the same growth rate λ and experience the same type of competition, given by b. This implies that all phenotypes have the same dynamic behavior when alone. The parameter $a(k)$ depends in general on the phenotype, reflecting an effect of the phenotype on the ability to cope with the environment. Recall that, for fixed λ and b, the parameter a determines the equilibrium density, equation (17). If the basic model (15) is extended to a model for competition between two species, the one with the higher equilibrium density is the stronger competitor (Doebeli 1995c). One then typically assumes that extreme phenotypes near the edges of the interval $[0, c]$ have a lower equilibrium density, i.e., a higher a, due for example to a lower ability to use the available resources (Slatkin 1980). This implies a competitive disadvantage for extreme phenotypes. However, for most of my results I assumed that $a(k)$ is a constant. I did this to keep the model as simple as possible and to see the effects of the quantitative character alone, without any additional effects such as differential resource use. It is straightforward to model a nonconstant $a(k)$ that takes on higher values at the edges of $[0, c]$ and has a minimum in the middle. With such $a(k)$ the results do not change qualitatively, and some indication of the quantitative change is given below. The changes are generally as one would predict, knowing that a higher a-value implies a competitive disadvantage for extreme phenotypes.

Once the parameters λ, b and σ_β, as well as the function $a(k)$ are specified, the fitness functions f^k, equation (20), are

used to determine the dynamics of asexual and sexual populations by iterating equation (4), respectively equations (12)–(14).

Asexual Reproduction

For numerical simulations the system has to be made discrete. For this I assume, in analogy to the sexual model, that the upper boundary of the character interval is an integer, and I consider only integer phenotypes in $[0, c]$. For large c this gives a good approximation to the continuous model.

If the niche width σ_β is large ($\sigma_\beta^2 > c$), competition is intense, and most of the phenotypes in the asexual population go extinct. This leads to a spiked distribution n_i. In the extreme case, only the phenotypes 0 and c survive. Then the system effectively describes competition between two populations. In such systems competition can lead to simple dynamics when both populations would have complex dynamics when alone, as was shown by Hassell and Comins (1976). Similar phenomena can be observed here. However, I am more interested in niche widths that allow all phenotypes to coexist. For such σ_β, the general effect of the continuous character is to decrease the size of the fluctuations in the system, as compared to the basic model (15). When making this comparison, it is assumed that the parameters in the basic model are the same as those for the single phenotypes in the phenotypically variable population. In principle, to account for the wider resource distribution for the phenotypically variable population, one should assume a lower value of the parameter a, that is, a higher carrying capacity, in the homogenous model. However, as mentioned above, the parameter a has no influence on the dynamics. Rescaling it can therefore be omitted, since I am only interested in the dynamic properties of the systems. What is important is that the basic, homogenous model has the same dynamics as would be displayed by a single phenotype of the variable population, which is achieved by assuming equal parameter values.

The main difference between the two models is that the variance in the mean fitness $\overline{w}_t$ of the phenotypically variable population is smaller (Fig. 2). Thus the continuous character stabilizes the system in the intuitive sense of decreasing its density fluctuations. This is a general phenomenon and happens for all parameters. In addition, the dynamics can change qualitatively, and the phenotypically variable population can exhibit periodic behavior when the basic model (15) is chaotic. However, this reduction in complexity is confined to a small region in parameter space. It only happens if the the dynamics of the single phenotypes, given by d in equation (18), are not too chaotic, that is, if d is not too large, and only for a small range of intermediate niche widths σ_β. Moreover, the corresponding simple attractor is not global and coexists with chaotic attractors. Thus it depends on the initial conditions whether the system displays the simple dynamics. But even if it moves on the chaotic attractors, its fitness variance is lower than that in the basic model (15). Another phenomenon that occurs is phase locking: for some niche widths σ_β, the variation in fitness can become very small, so that the total density remains almost constant. In this state the fluctuations of the single phenotypes tend to cancel each other because the interaction width given by σ_β has reached a critical value. An example is shown in Figure 2d. Like complexity reduction, phase locking only occurs for a small region in parameter space, and it depends on the initial conditions in the system. In summary, the main effect of quantitative phenotypic variability in the asexual population is a decrease in the size of the density fluctuations as compared to the basic model without variability.

Figure 3a

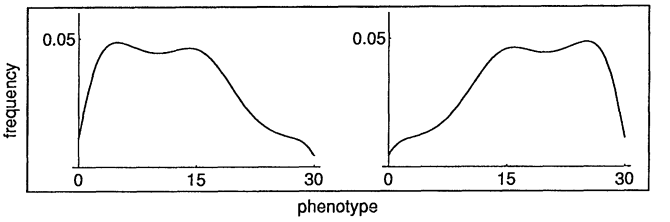

Figure 3b

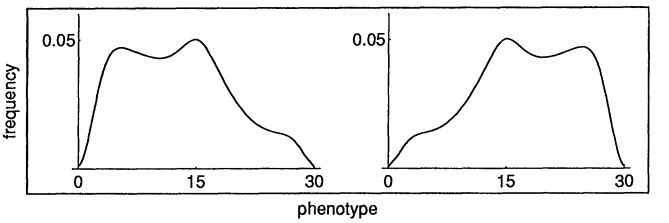

FIG. 3. Fluctuating phenotype distributions when sexual reproduction induces a stable equilibrium for the total population size. At the equilibrium, the phenotypes alternate between the two distributions shown. In 3a, the approximation (10) was used in the quantitative genetic model. Using the exact formula (9) yields qualitatively the same result (3b), but intermediate phenotypes are slightly more common, as expected. The parameter values for the figure were: $c = 30$, $\lambda = 10$, $d = 1.2$, $a(k) \equiv 0.1$ and $\sigma_\beta^2 = 8$. The total density of an asexual population with the same parameters moves on a two-cycle.

Sexual Reproduction

With sexual reproduction no phenotypes go extinct even with large niche widths σ_β, because they are constantly recreated through random mating. Therefore, the dynamics of the sexual system can be more complex than that of the asexual population if niche widths are very large. However, with niche widths for which the asexual population can maintain all the phenotypes, the stabilizing effect of the continuous character is magnified in the sexual population. In general, the fluctuations are smaller than in the asexual population. Moreover, the sexual population tends to have simpler dynamics. Two examples are shown in Figures 2 and 3. In Figure 2, the sexual population moves on a two-cycle, whereas the asexual population is chaotic. In Figure 3, sex changes the dynamics from a two-cycle to a stable equilibrium. However, the system "remembers" the two-cycle: at the equilibrium for the total density, the frequency distribution of the phenotypes is cycling with period 2, and the density of each phenotype moves on a two-cycle, whereas the total density remains constant. The phenomenon that phenotype frequencies have nontrivial dynamics when the total population size

is at a stable equilibrium occurs for all parameter values for which the basic model has complex dynamics that are reduced to a stable equilibrium by quantitative genetics. What is also shown in Figure 3 is an example of the effect of using the exact formula (9) instead of the approximation (10) in the genetic model. Qualitatively, the result is the same, but there are small quantitative differences in the cycling phenotype distributions.

In general, sex reduces the fitness variance as compared to the asexual system for a wide range of parameters. In fact, the higher the complexity d and the intrinsic growth rate λ in the system, the more pronounced is the effect. Increasing either one of these parameters increases the size of the fluctuations in the basic model (15) (Doebeli 1995c). It follows that the stabilizing effect of sex is enhanced with more complex dynamics of the single phenotypes.

With sex, the reduction in dynamic complexity happens for a much larger region in parameter space than without sex. Even for quite complicated dynamic behavior of the single phenotypes, sex induces simple dynamics for intermediate niche widths σ_β. Moreover, these simple dynamics are robust: they seem to be globally attracting and do not depend on initial conditions. This confirms an observation made in Doebeli (1995b) for a different genetic system, namely that attractors in sexual populations have larger basins of attraction than those in asexual systems, which reflects a greater tendency for asexual models to have multiple attractors.

The fact that it is really the quantitative variability that makes the dynamics more stable can be seen by comparing models with different numbers of loci. Figure 4 shows the dynamics of the sexual system for fixed parameters b, d, and a for the single phenotypes, but for variable lengths c of the character interval. To make the comparison, the niche width σ_β has been adjusted in each case to a fixed proportion of c. Although the basic model (15) exhibits chaos for the given parameter values, the sexual model with 10 loci has a stable cycle of high order. Increasing the number of loci reduces the period of the stable cycle as well as its amplitude. Thus the phenotypically more variable systems are dynamically more stable.

The reduction of fitness variance and dynamic complexity does not happen for the parameters for which phase locking with almost constant densities occurs in the asexual population. Because of recombination, locked phases are not possible in the sexual system. In general, sexual reproduction tends to maintain normal character distributions: intermediate characters are more common than extreme characters. However, the distributions can have more than one maximum, and they can change over time (Fig. 3). The simpler the dynamics of the system, the closer the character distributions are to being normal and constant. However, even when the total density reaches a stable equilibrium, the explicit modeling of the quantitative genetics can lead to nonequilibrium dynamics for the genetic structure, as is shown in Figure 3.

Competition between Sexuals and Asexuals

Reducing the variance in the mean fitness increases the geometric mean growth rate of a population (Gillespie 1977). This is the basis of Hamilton's theory for the evolution of

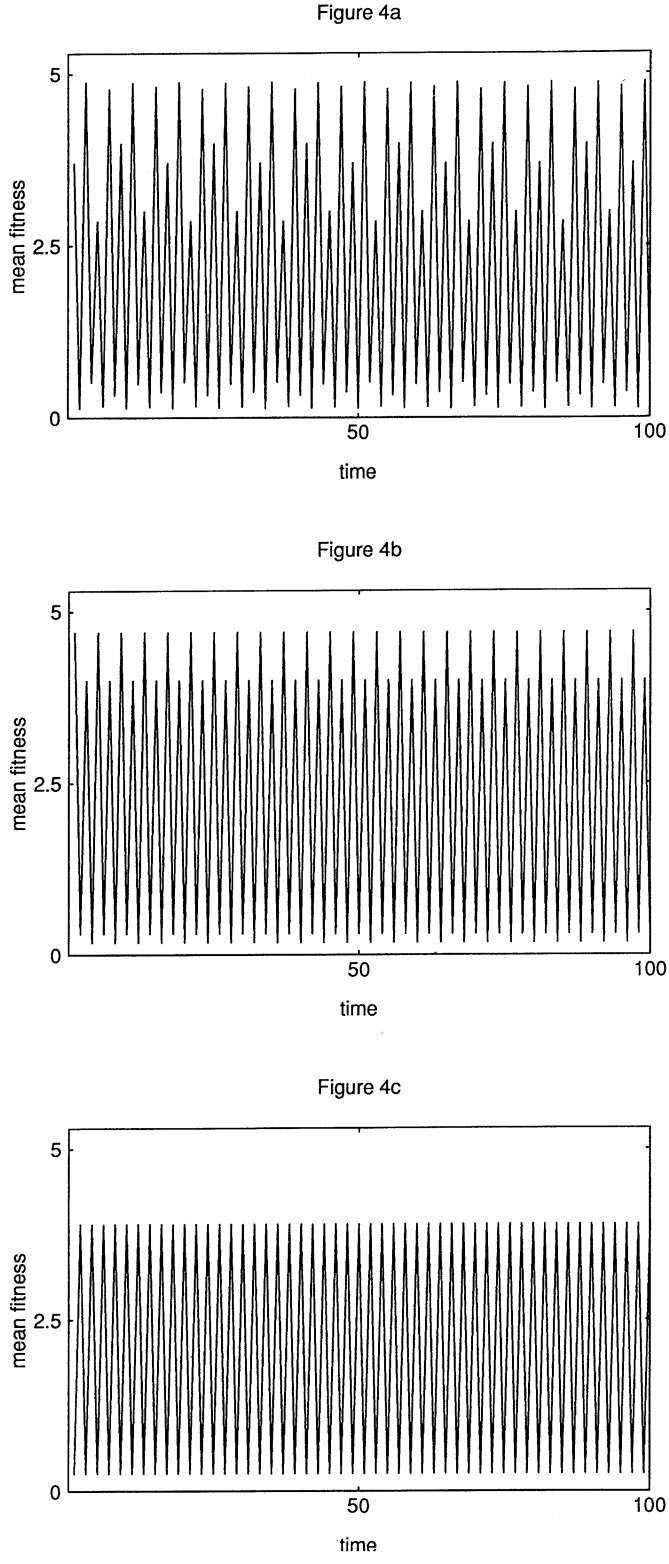

FIG. 4. Effect of increasing the number of loci in the sexual system. With 10 loci ($c = 10$, 4a), the system moves on a stable cycle of order 16. With increasing number of loci, the period of the cycle is reduced to four ($c = 20$, 4b), and finally to two ($c = 70$, 4c), whereas the amplitude of the fluctuations also becomes smaller. The parameters for the figures were $\lambda = 5$, $d = 2$ and $a(k) \equiv 0.1$. In each case, the niche width σ_β^2 was set at a fixed proportion of 0.75 of the number of loci c.

sex in host-parasite systems (Hamilton 1980, 1982). In his models, sex reduces fitness variance, which enables the sexuals to outcompete the asexuals despite their classical twofold advantage of not having to invest in male function (Maynard Smith 1978). In the present situation, competition between a sexual and an asexual population is modeled by assuming that the fitness functions of the phenotypes depend on their joint distribution in the two populations. Thus I assumed that the competitive impact of sexuals on asexuals is the same as that of asexuals on asexuals and vice versa. In particular, sexuals and asexuals have the same niche width. Furthermore, I assumed that the asexuals have twice the growth rate of the sexuals, but that the single phenotypes have the same equilibrium density and the same dynamic complexity, given by d in equation (18), in both populations. This is the traditional way of modeling the twofold advantage of asexuals (Hamilton 1980; May and Anderson 1983), and it implies an intrinsic advantage for the asexuals in the present model. This can be seen by considering the basic model (15). If this model is extended to model competition between two populations with the same equilibrium densities and the same dynamic complexity, then the one with the higher intrinsic growth rate is the stronger competitor and drives the other to extinction (Doebeli 1995c). A twofold disadvantage of sexuals is a "worst case" scenario, because in reality, the disadvantage may be reduced by mechanisms like unequal sex ratios, sexual dimorphism or parental care (Maynard Smith 1978). However, as mentioned before, a higher growth rate also leads to larger fluctuations in the system (without changing its dynamics qualitatively). Therefore, the sexual advantage of having a lower fitness variance than the asexuals is enhanced by having only half their growth rate. On the other hand, recombination leads to another disadvantage for the sexuals. Because recombination tends to produce many intermediate phenotypes, a sexual population cannot match the uniform resource distribution as well as the corresponding asexual population. This leads to a lower carrying capacity of the sexuals. The effect can be seen when comparing populations exhibiting a stable equilibrium. Then the equilibrium population size of the asexuals is larger than that of the sexuals, which suggests that the carrying capacity is also larger for the asexuals when the equilibrium is unstable and the dynamics are complex. As already mentioned, higher carrying capacities have no effect on the dynamics displayed by the system, but they imply a competitive advantage. This is an example where recombination leads to a decrease in a fitness component, which is reminiscent of the work of Abrams et al. (1993), who show that sexual populations undergoing frequency-dependent selection can get trapped at trait values that minimize fitness.

In summary, when competing against a corresponding asexual population, the sexuals have the disadvantage of having half the growth rate and a lower carrying capacity than the asexuals, but they have the advantage of a lower variance in fitness, which could enable them to coexist with the asexuals. Moreover, coexistence should be more likely for high d and λ, for which the stabilizing effect of sex is larger, as was explained above.

Numerical simulations confirmed these predictions. For low d and λ, the asexuals outcompete the sexuals and drive them to extinction (Fig. 5). Increasing either one of these parameters increases the fluctuations in the asexual system and the advantage of sex due to its stabilizing effect on the dynamics (Fig. 5). As a consequence, the sexuals can coexist with the asexuals. Coexistence can lead to complicated dynamics, in which crashes and outbursts of the proportion of sexuals alternate irregularly.

Because of phase locking in the asexual system, one would expect that the asexuals would drive the sexuals to extinction even for high d and λ if the niche width is chosen appropriately. However, phase locking is a rather delicate phenomenon, and the corresponding basin of attraction for the dynamics seems to be small. Thus phase locking is not robust against perturbations such as the presence of a sexual population, and I have never observed that phase-locked asexual populations could withstand invasion of sexuals if such invasion was possible for nearby parameter values for which there was no phase locking in the asexuals.

It is worth mentioning that the advantage of sex tends to increase when the resource distribution is unimodal rather than uniform, at least when niche widths are small. The reason is that the sexuals can match a unimodal resource distribution much better than a uniform distribution, whereas the difference is small for asexuals. Therefore, unimodal distributions reduce the sexual disadvantage of having lower carrying capacities. Another way to favor sexuals in this model is to increase the number of loci, i.e., the range of phenotypes. This enhances the stabilizing effect of sex (Fig. 4), whereas it again does not seem to affect the dynamics of asexual populations very much. Thus larger numbers of loci increase the sexual advantage of having a lower fitness variance.

The competitive advantage of sex due to lower fitness variance is also more prominent when only a subset of all possible phenotypes is present in the asexual population (Fig. 5). This conforms with the tangled bank theory for the evolution of sex (Bell 1982), according to which a sexual population has a broader niche width and can therefore exploit the environment more efficiently than a phenotypically more uniform asexual population. The asexuals cannot avoid competition of sexuals with the same phenotypes, but the sexuals can avoid the asexuals partly by producing phenotypes that are not present in the asexual population. In turn, these phenotypes replenish those that compete most strongly with asexuals. This puts the asexuals at a disadvantage. Numerical simulations show that whenever a sexual population can coexist with an asexual population in which all phenotypes are present, then the proportion of sexuals increases if a few of the asexual phenotypes are missing. It is also possible that sexuals can only coexist with an asexual population that lacks some of the phenotypes (Fig. 5d), so that only a tangled bank allows coexistence. The present model thus connects the tangled bank theory for the evolution of sex with Hamilton's theory, in which sex reduces fitness variance. Although Hamilton found this mechanism in host-parasite systems, it has been shown here to work in competition models as well. On the other hand, we will see in the next section that tangled bank effects can also occur with host-parasite interactions.

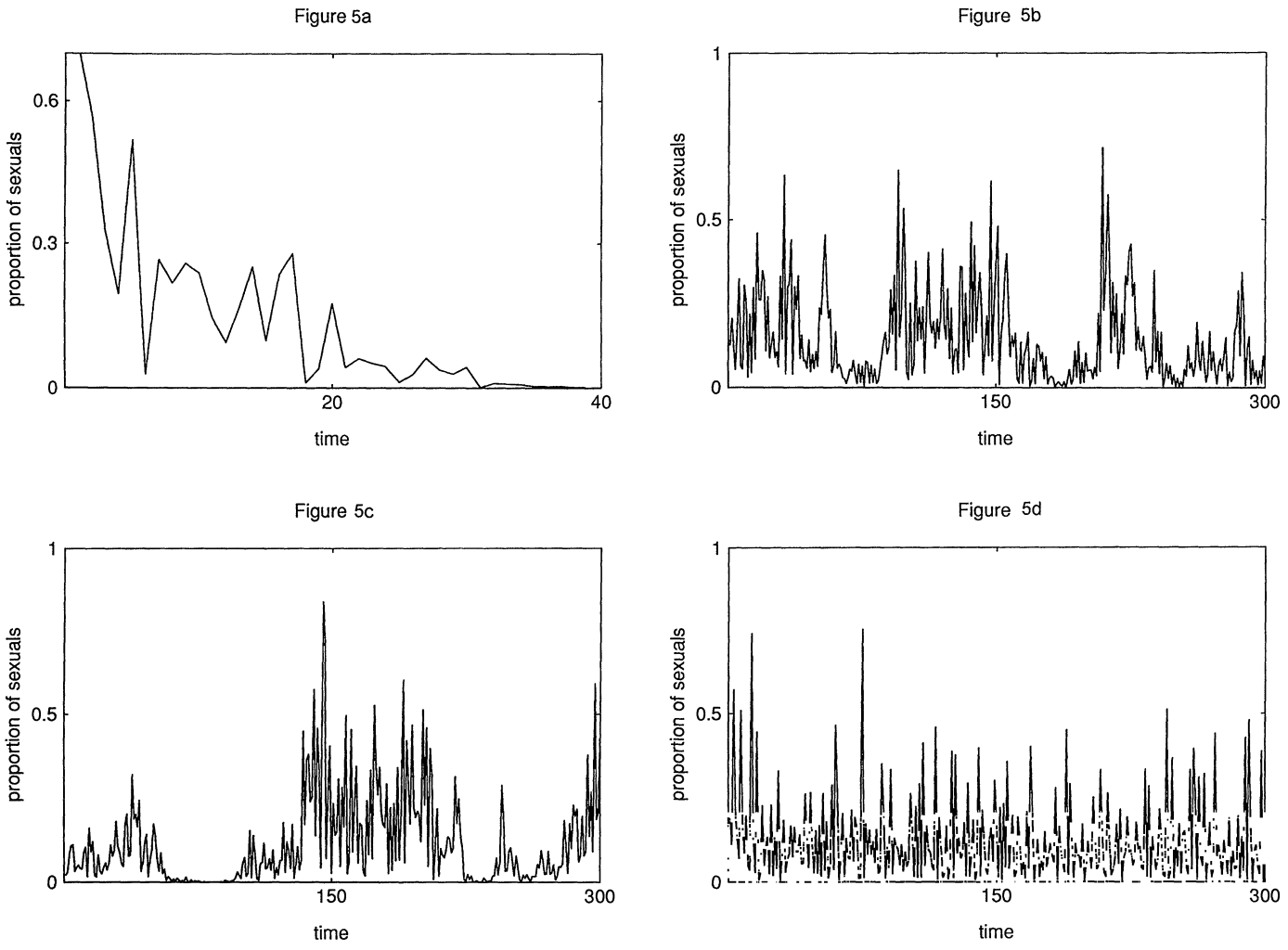

FIG. 5. Competition between sexuals and asexuals. The figures show the proportion of sexuals in the total population as a function of time. In 5a, the advantage of the sexuals gained from having a lower fitness variance is not enough to avoid extinction. Increasing the fluctuations in the asexual system increases the advantage of sex. Fluctuations can be magnified by increasing the intrinsic growth rate λ (5b) or the dynamic complexity d (5c). In both cases, the sexuals are now able to coexist with the asexuals. Coexistence can lead to complicated dynamics, in which crashes and outbursts of the sexuals alternate irregularly. Figure 5d is the same as 5a, but now the asexual population contains fewer phenotypes than the sexual. This tangles the bank for the asexuals and again allows the sexuals to coexist. The parameters for the figures were $c = 30$, $a(k) \equiv 0.1$ and $\sigma_\beta^2 = 7$ for all; $\lambda = 10$ and $d = 2.5$ in 5a; $\lambda = 30$ and $d = 2.5$ in 5b; $\lambda = 10$ and $d = 3.3$ in 5c; $\lambda = 10$ and $d = 2.5$ in 5d (same as 5a), but the 10 outer phenotypes (i.e., phenotypes 0, 1, 2, 3, 4 and 26, 27, 28, 29, 30) are missing in the asexual population.

The Host-Parasite Model

Here the basic model was introduced by May and Anderson (1983) and describes a host population whose density is regulated by a parasite. The parasite spreads in each generation of the host before reproductive age is reached, and a fraction $I(N)$ of the host population N is killed. Thus, if $\lambda > 1$ denotes the intrinsic growth rate of the host, the fitness function is

$$f(N) = \lambda(1 - I(N)). \tag{22}$$

May and Anderson (1983) derived the density-dependent fraction $I(N)$ from epidemiological considerations as the solution of the equation

$$1 - I = \exp\left[-\frac{I \cdot N}{N_T}\right]. \tag{23}$$

N_T is the threshold density of the host: if $N < N_T$, the only solution to equation (23) is $I = 0$, i.e., the parasite cannot spread. May (1985) showed that a host population with fitness given by equation (22) exhibits chaotic dynamics for all growth rates $\lambda > 1$, regardless of the size of the threshold density N_T.

Although this dynamic property is certainly not a common feature of host-parasite models, there are many examples in nature where parasites regulate host populations (e.g., Pacala and Hassell 1991), and theoretical models of such interactions typically can have very complicated dynamics. More common forms of such models are derived from the Nicholson-Bailey equations (e.g., Holt and Hassell 1993), but the model used here has the advantage that only the host species is explicitly described, and that the impact of the parasites is

derived using the epidemiological rather than phenomenological Kermack-McKendrick differential equations (May and Anderson 1983).

May and Anderson (1983) extended the model with population genetics based on gene-for-gene interactions between the host and the parasite. Then each host genotype, respectively the corresponding phenotype, is susceptible to a unique parasite, and the fitness of each genotype is given by equations (22) and (23) independently of the density of the other genotypes. May and Anderson (1983) considered one locus with two alleles and dominance, and Doebeli and Koella (1994) studied systems with one locus and two or three alleles without dominance, as well as systems with two haploid loci.

Here I assume that the host-parasite interaction is determined by a host character that is quantitative in the sense that it is additively determined by many haploid loci according to the quantitative genetic model. I also assumed that the character k takes on discrete values in the interval $[0, c]$, and that the corresponding phenotypes are susceptible to a unique parasite. This generalizes the gene-for-gene principle to an additively determined character. The metaphor in the gene-for-gene model is that of key and keyhole, in which a parasite can only attack that host genotype to which it has the right key. Here the keyholes are the phenotypes given by the character value, and a particular parasite can attack a phenotype if the latter has the right number of 1-alleles, regardless of where these alleles occur in the genome. Thus the interactions with the parasites are based on additive matching. This is the natural extension of gene-for-gene interactions to quantitative genetics.

Then the phenotypic fitness functions f^k only depend on the density of phenotype k. Thus, if n_t is the density distribution of the phenotypes as usual, these fitness functions are given by

$$f^k(n_t) = \lambda[1 - I(n_t(k))], \tag{24}$$

where $I(n_t(k))$ is the solution of

$$1 - I = \exp\left[-\frac{I \cdot n_t(k)}{N_T}\right]. \tag{25}$$

I assume that all phenotypes have the same growth rate λ in the absence of the parasites, and the same threshold density N_T. To be a bit more realistic, one can assume that the specificity of the parasites is not complete. Then the phenotype k-specific parasite can also attack other phenotypes with similar character values, although less efficiently. This implies that f^k also depends on the densities of other phenotypes, and could be modeled by

$$f^k(n_t) = \lambda\left[1 - I\left(\sum_{l=0}^{c} n_t(l) \cdot \beta(k, l)\right)\right], \tag{26}$$

where $\beta(k, l)$ is a function that has its maximum value of 1 at $k = l$, for example

$$\beta(k, l) = \exp\left[\frac{-(k - l)^2}{2\sigma_\beta^2}\right]. \tag{27}$$

The parameter σ_β is then a measure for the specificity of the parasites. However, reasonable choices of σ_β do not change the conclusions, and to see the pure effects of the genetics in the model I report the results obtained from equations (24) and (25), i.e., with complete parasite specificity.

Population Dynamics

For the numerical simulations I used the fitness function (24) in a discretized version of equation (4) for the asexual population and in equations (12)–(14) for the sexual population. The asexual population consists of $c + 1$ uncoupled phenotypes, each of which exhibits the same dynamic behavior as the basic model (22). Thus its dynamics are chaotic for all growth rates λ. However, sensitive dependence on initial conditions of the dynamics of the single phenotypes leads to a thorough mixing of their density fluctuations. Therefore, the phenotypically variable population has a much lower fitness variance than a single phenotype (Fig. 6). This phenomenon is independent of initial conditions. Thus phenotypic variability again has a stabilizing effect on the dynamics, because it induces a lower fitness variance. With sexual reproduction the system exhibits simple dynamics in form of a stable equilibrium or a two-cycle for low to intermediate growth rates (Fig. 6). For higher growth rates, when the dynamics become more complex, the fitness variance in the sexual population is again lower than in the asexual population. Similar phenomena were observed in Doebeli and Koella (1994), where we noted that increased phenotypic variability strengthens the stabilizing effect of sex. This is confirmed here, as the dynamics with the quantitative character are much simpler for a wide range of growth rates than the dynamics of the systems we studied in Doebeli and Koella (1994).

Competition between Sexuals and Asexuals

May and Anderson (1983) studied competition between an asexual population with two phenotypes whose fitness functions were given by equation (22), and a sexual population with two phenotypes that were susceptible to the same parasites, but had only half the growth rate of the asexuals. Contrary to Hamilton's (1980) pioneering study, they showed that the asexuals always drove the sexuals to extinction. However, in Doebeli and Koella (1994) we argued that the advantage of sex could be restored with greater phenotypic variability, at least for high growth rates. This is confirmed and extended here.

To model competition, I assumed that the parasites attack sexuals and asexuals equally. Consequently, the fitness function of a phenotype depends on the sum of its densities in the sexual and asexual populations. This leads to an apparent competition for phenotype space between sexuals and asexuals. As in the competition model of the last section, the fitness of a phenotype is inversely density-dependent, but here the reason is the parasites. Consequently, the fitness of a phenotype decreases if the same phenotype is also present in a coexisting population. Holt and Lawton (1993) have proposed that apparent competition for enemy free space between host species may often lead to competitive exclusion and may thus play an important role in structuring host species communities. Here the apparent competition is between phenotypes, and the question is whether genetic coupling between them confers an advantage over breeding true. To

Figure 6a

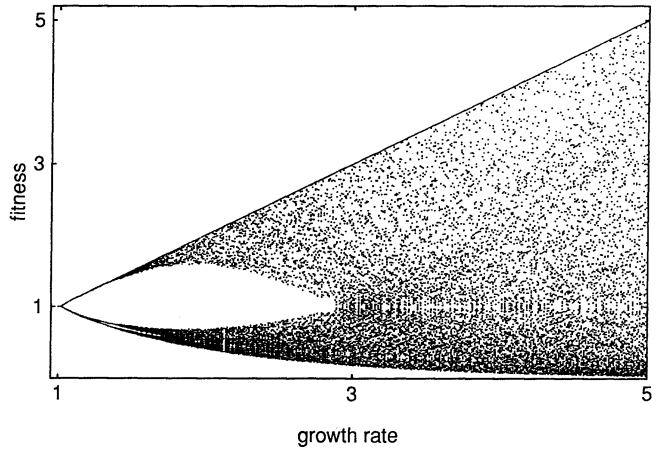

Figure 6b

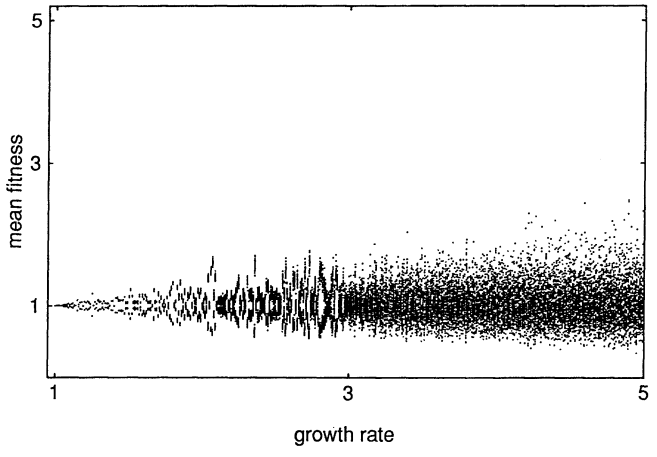

Figure 6c

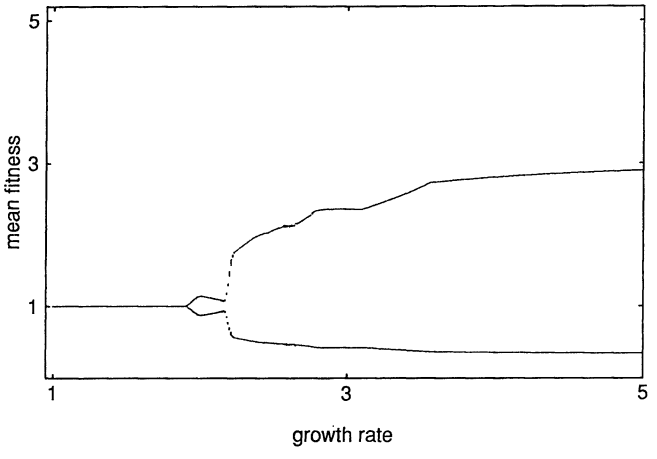

FIG. 6. Time distribution of the mean fitness in the host-parasite system for a range of intrinsic growth rates λ. Mean fitness is defined as N_{t+1}/N_t, where N_t is the total population density at time t. Even though the basic model (22) stays close to a two-cycle for small λ (6a), it is chaotic for all growth rates (May 1985), as is the asexual system with phenotypic variability (6b). However, the latter has a much lower fitness variance because of asynchrony in the fluctuations of the single phenotypes. The sexual system (6c) has simple dynamics for the growth rates shown. For $\lambda \leq 2$, it has a stable equilibrium, and for $2 \leq \lambda \leq 7$ it moves on a two-cycle. For higher λ the dynamics become more complex. For these growth rates (not shown), the fitness variance is lower than in the asexual population. For all figures, the threshold density N_T, which has no effect on the dynamics, was set = 1. The different systems were first iterated long enough to eliminate transient behavior, and then the mean fitness was plotted for many generations. This was done for discrete values of λ in the interval [1, 5] with stepsize 0.01. The number of phenotypes was 20, i.e., $c = 19$.

model competition I again assumed that the asexuals have twice the growth rate of the sexuals.

With the quantitative character determining the interactions, the asexuals cannot outcompete the sexuals even when intrinsic growth rates are low (Fig. 7a). Again, this seems to be due to lower fitness variance in the sexual population. The asexual advantage of having twice the growth rate of the sexuals also implies a disadvantage, because higher growth rates induce more fluctuations and a larger fitness variance. The point is that with quantitative phenotypic variability, the balance tips in favor of the sexuals when growth rates are still low.

In addition, a tangled bank phenomenon can be observed if the asexual population contains only part of all possible phenotypes. Then the sexuals often drive the asexuals to extinction (Fig. 7c). Since the degree of parasitism of a phenotype increases if the same phenotype is also present in the other population, the sexual population can produce phenotypes that are less heavily parasitized because the corresponding phenotypes are missing in the asexual population. In contrast, every asexual phenotype is also present in the sexual population, which decreases their fitness. This parasitic tangled bank offsets the asexual advantage of having twice the growth rate of the sexuals.

DISCUSSION

The competition model and the host-parasite model both display more stable dynamics when a quantitative character determines the interactions. When reproduction is asexual, phenotypic variability reduces the variance in the mean fitness of the populations, and it can change the dynamics qualitatively. Sexual reproduction, modeled by explicit quantitative genetics, magnifies these effects. It decreases the fitness variance further and lowers the dynamic complexity of the system, generally leading to simpler dynamics, for example from chaos to a two-cycle.

Since May (1974, 1976) showed that very simple ecological models can have very complicated dynamics, there has been a debate about how often such dynamics occur in natural populations. Although the early studies of May and others focused on models set in discrete time, it has later been shown that complex dynamics are a likely outcome in models with continuous time if three or more species interact (Gilpin 1979; Takeuchi and Adachi 1983; Hastings and Powell 1991; Vandermeer 1993). This lead some authors to propose that chaos should be the rule rather than the exception (Schaffer

Figure 7a

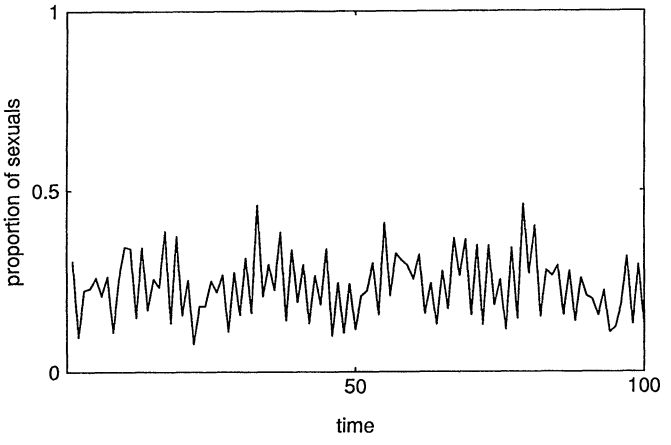

Figure 7b

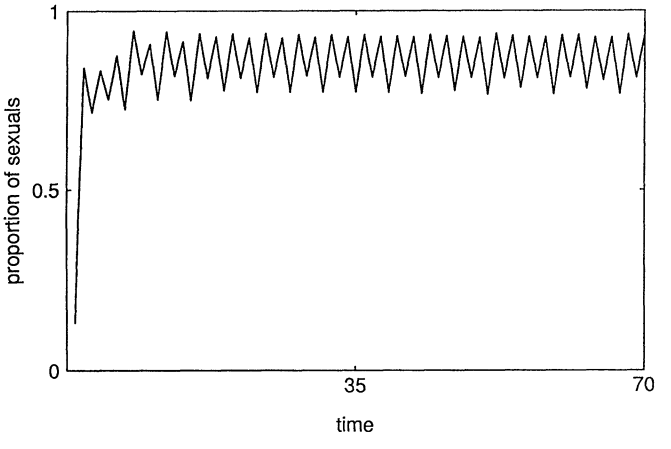

Figure 7c

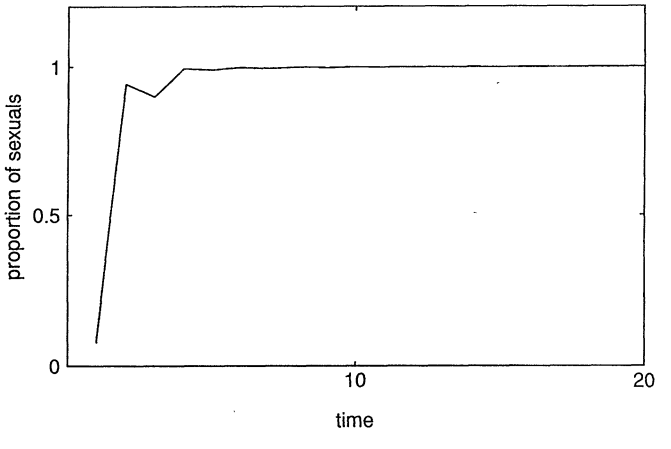

FIG. 7. Competition between sexual and asexual host populations. The figures show the proportion of sexuals in the total population. The asexuals have twice the growth rate of the sexuals, which is set = 5. Even for this low growth rate, the sexuals can coexist with the asexuals, because sexuals with growth rate 5 have a much lower fitness variance than asexuals with growth rate 10. In fact, with the given phenotypic variability, $c = 30$, the sexuals can resist competition from asexuals for all growth rates $\gtrsim 2.5$, and they drive the asexuals to extinction for growth rates $\gtrsim 30$. What the figures show is the effect of the parasitic tangled bank described in the text. When all 31 phenotypes are present in the asexual population (7a), the average proportion of sexuals is quite low. It increases (7b) when the eight outer phenotypes are missing in the asexual population (i.e., the phenotypes 0, 1, 2, 3 and 27, 28, 29, 30). Note that the sexuals start to impose their two-cycle on the dynamics of the system. When the 22 outer phenotypes are missing (i.e., the asexual population contains only the phenotypes 11, . . . , 19), the sexuals drive the asexuals to extinction (7c): the impact of apparent competition on the asexuals becomes too large. All threshold densities were 1 for the figures.

1984; Schaffer and Kot 1986; Olson et al. 1988; Hastings et al. 1993). However, the real systems where it has been demonstrated are few (e.g., Sugihara and May 1990), and there are quite a few empirical studies suggesting that complex dynamics are rare in natural populations (Hassell et al. 1976; Thomas et al. 1980; Mueller and Ayala 1981; Philippi et al. 1987). Even though some of these data have been reanalyzed with new methods that lead to different conclusions (Turchin and Taylor 1992), the traditional view is still that of a stable world in which fluctuations are mainly caused by environmental stochasticity (e.g., Berryman and Millstein 1989). Thomas et al. (1980) suggested that group selection could lead to simple dynamics by eliminating highly fluctuating populations through stochastic effects after population crashes. But chaotic dynamics do not necessarily lead to very low population densities (Hastings and Powell 1991), and more detailed studies on the evolution of stability parameters in models based on individual selection have yielded ambiguous results (Heckel and Roughgarden 1980; Turelli and Petry 1980; Mueller and Ayala 1981; Hansen 1992; Ferrière and Clobert 1992; Gatto 1993; Ferrière and Gatto 1993; Doebeli 1993, 1995c).

One approach to the question of what types of population dynamics should prevail is to look for general mechanisms that favor one type over another. The comparison between metapopulation dynamics and the dynamics of the constituent local populations is one example of this (Gilpin and Hanski 1991), and it has been shown that dispersal between local populations can greatly simplify the dynamics (McCallum 1992; Hassell et al. 1992; Hastings 1993; Stone 1993; Doebeli 1995a; but see Bascompte and Solé 1994). These studies suggest that mixing and asynchrony of interactions lead to simpler dynamics. Since segregation and recombination induce mixing, they can be expected to have stabilizing effects. Indeed, we have shown in Doebeli and Koella (1994) that segregation at a fitness locus simplifies population dynamics, and we conjectured that this effect should be larger with more phenotypic variability. It is shown here that this is true if the phenotypic variability is given by a quantitative character that is determined additively by many loci: the more variable a population, the more stable its dynamics (Fig. 4). Overall, the results indicate that quantitative variation and sexual reproduction both simplify population dynamics. That quantitative variability can have stabilizing effects on ecological dynamics has been noted by Frank, who also studied host-parasite systems with quantitative characters (Frank 1993, 1994). However, the stabilizing effect occurred in models in

which the character determined the degree of resistance to one parasite, rather than susceptibility to different types of parasites (Frank 1994). Also, there was no genetics in the form of recombination or segregation in these models. Nevertheless, it is encouraging that different forms of quantitative variation seem to have similar general effects.

The models I used for sexual populations describe one sex only, which corresponds to assuming that the dynamics of the population are driven by that sex, independently of the abundance of the other sex. Caswell and Weeks (1986) have shown that two-sex models can have much larger fluctuations, and that their dynamics can be more complicated than those of one-sex models. It is an open question how much of the stabilizing effect of sex would remain if the models used here were extended to include two sexes.

If group selection favors simple dynamics, it should also favor sex, which has a stabilizing effect. But the reduction in fitness variance should also imply an advantage of sex based on individual selection (Gillespie 1977). This was the basis of Hamilton's (1980) theory for the evolution of sex in host-parasite systems, but his conclusions were partly refuted by May and Anderson (1983), who used the epidemiological and density-dependent fitness function (22) instead of Hamilton's phenomenological and frequency-dependent fitness. However, with this model May and Anderson (1983) only studied populations with two phenotypes, and we conjectured in Doebeli and Koella (1994) that the advantage of sex should be restored with greater phenotypic variability. This is confirmed here (Fig. 7). Moreover, the theory is not restricted to host-parasite systems, as the reduction of fitness variance also favors sex in the competition model (Fig. 5).

If the fitness of a phenotype depends on the density of phenotypes that are similar to it, sex can be advantageous by spreading out the phenotype distribution. Thus, if a sexual population is competing against an asexual population that contains only part of the sexual phenotypes, it can avoid competition by producing phenotypes that are not present among the asexuals. This is the tangled bank theory for the evolution of sex (Bell 1982), and it works in both models considered here (Figs. 5, 7). Koella (1988) came to similar conclusions using Slatkin's competition model. However, he only considered the case when the asexual population consists of a single phenotype.

Traditionally, a tangled bank is the consequence of competition for resources. The present results show that it can also occur in host-parasite interactions when the parasites are phenotype-specific. Then the fitness of a phenotype is reduced if the same phenotype is also present in another population, which leads to an apparent competition for phenotype space. This is analogous to the competition for enemy free space in insect host communities that was described by Holt and Lawton (1993). Under this apparent competition, the asexuals are at a disadvantage because they breed true. A possible consequence of this parasitic tangled bank is to prevent invasion of an asexual phenotype into a sexual population: if the corresponding sexual phenotype is abundant, the fitness of the asexual phenotype is low despite its initial rarity. Howard and Lively (1994) studied invasion of asexuals into a sexual population and concluded that for intermediate to low degrees of parasitism, the advantage of sex due to evading parasites by producing rare phenotypes is not enough to prevent invasion, because the asexuals are initially also rare and therefore not heavily parasitized. It is straightforward to define the analogues of the parameters used by Howard and Lively (1994) for the present model by taking time averages. Then a different picture emerges: the parasitic tangled bank prevents invasion of asexual phenotypes even for very low degrees of parasitism, and no appeal to the mutation accumulation theory is necessary as in Howard and Lively's (1994) model.

The models I used here are conceptually based on Slatkin's (1980) quantitative genetic model for population dynamics. In his model, the genetics are described implicitly by assuming a normal character distribution at the beginning of each generation. Selection then acts on the mean and the variance of this distribution. One consequence of this is a drastic reduction in the dynamic complexity of the system: it always has a stable equilibrium (Slatkin 1980). Similar observations have been made by Saloniemi (1993) for a Lotka-Volterra predator-prey system. Modeling quantitative genetics with normal character distributions broke the neutral stability of the original model without genetics, and either lead to extinction of both species or to coexistence with a stable equilibrium. Thus, even though Abrams (1992) gave an example where quantitative genetic models with normal character distributions produced population cycles, it seems that normality is in general too rigorous an assumption if the whole range of population dynamic behavior is the focus of interest. An alternative approach is to model the genetics of a quantitative character explicitly. This makes the models less rigid and has consequences even when the dynamics remain simple and the total density reaches an equilibrium, at which the genetic structure can still have nonequilibrium dynamics (Fig. 3). This indicates that modeling the genetics of a quantitative character in more detail reveals insights about the character's impact on population dynamics not gained under the usual assumption of normal character distributions. It will be interesting to see the effect of explicit genetics in other population dynamic models, for example in models for ecological character displacement, or in predator-prey systems that are based on quantitative characters.

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