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Genetic Variability in Sensitivity to Population Density Affects the Dynamics of Simple Ecological Models

Michael Doebeli

Zoology Institute, University of Basel, Rheinsprung 9, CH-4051 Basel, Switzerland
E-mail: doebeli@ubaclu.unibas.ch

and

Gerdien de Jong

Population Genetics Group, Department of Plant Ecology and Evolutionary Biology, University of Utrecht, Padualaan 8, NL-3584 CH Utrecht, The Netherlands
E-mail: G.deJong@boev.biol.ruu.nl

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Many 1-dimensional discrete time ecological models contain a sensitivity parameter that does not affect the dynamic complexity of these models. We show that genetic variability in this parameter can have a strong effect on population dynamics. We incorporate ecological dynamics in two different population genetic models with one locus and two alleles. The first is the classical model of a randomly mating population in Hardy–Weinberg equilibrium, and the second is a model of differential selection in males and females. In populations in Hardy–Weinberg equilibrium, variability in the sensitivity parameter can be maintained by overdominance. In this case, the dynamics of the polymorphic population tend to be much simpler than those of monomorphic populations. In the model with different selection in males and females, polymorphisms can be maintained in various ways, e.g., by opposing directional selection in males and females. Polymorphism in the sensitivity parameter tends to simplify population dynamics in the model with different selection in males and females as well. A number of interesting dynamic effects can be observed, e.g., multiple attractors with complicated basins of attraction. Then the final state of the system after a successful invasion by mutant alleles may depend on the mutation rate and on the distribution of mutational steps. In addition, there are situations in which genetic variability destabilizes a stable population dynamic equilibrium in the monomorphic model. There is an analogy between genetic variability and variability imposed by the environment. If differences in sensitivity are caused by the environment, dynamic effects similar to those in the genetic models can be observed. In addition, source-sink structures that are known to occur in spatially structured models can be seen in the genetic model if one of the genotypes is inviable. The results suggest that combining ecological and population genetic models can lead to a number of new insights. More work is needed, e.g., with fertility models, in which fitnesses are not assigned to individuals, but to mating pairs.

Key Words: **population dynamics; population genetics; polymorphism; chaos; stability; differential selection in males and females.**

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1. INTRODUCTION

Ecological models can have complicated dynamics (May 1976). For example, chaotic dynamics are predicted in single species models of populations with discrete generations if the intrinsic growth rate is high and if competition is strong (Bellows 1981). Such models might lead to the expectation that chaos should be common in natural populations. This seems to be at odds with existing empirical data (Hassell *et al.* 1976, Thomas *et al.* 1980, Mueller and Ayala 1981, Bellows 1981, Philippi *et al.* 1987; but see Turchin and Taylor 1992, Hastings. *et al.* 1993). The apparent scarcity of chaotic dynamics in natural populations has generated interest in the mechanisms and conditions that stabilize population dynamics. Phenomena as diverse as demographic stochasticity, migration, sex, evolution, and genetic variability have been considered. For example, Smith and Mead (1980) argued that stochastic demographic events blur the deterministic details of complex dynamics enough to reduce complexity in real populations. A different sort of blurring of interactions occurs through dispersal in metapopulations, and it has been argued that the dynamics of metapopulations tend to be simpler than those of the constituent local populations (Hastings 1993, Stone 1993, Ruxton 1994, Doebeli 1995a). Sexual reproduction also leads to mixing of interactions and can reduce the tendency to chaos in predator–prey, in host–parasite, and in competition models (Doebeli and Koella 1994, Doebeli 1995b, 1996), even if only part of the population reproduces sexually (Ruxton 1995). And even though selection may favour more chaotic phenotypes in some models (Ferrière and Fox 1996), the evolution of demographic parameters in 1-dimensional difference equation models typically leads to simple population dynamics (Doebeli and Koella 1995).

Models including both population dynamics and genetic dynamics of allele frequencies can be used to investigate the consequences of genetic variability within a population on its ecological dynamics. For example, Asmussen (1979) found that overdominance in carrying capacity delayed the onset of cyclical or chaotic behaviour in the Ricker equation, and Doebeli (1996) argued that increased variability in a quantitative character determining ecological interactions simplifies population dynamics. In this paper we describe another scenario in which genetic variability has a strong effect on population dynamics.

We consider density-dependent fitness functions $f(N)$. Without genetic or phenotypic variability, these functions yield dynamic models for the density of populations with discrete generations of the form $N_{t+1} = f(N_t) \cdot N_t$, where N_t is the population size at time t. Typically, the density N enters the function f only as a product aN, where a is a scaling parameter measuring the sensitivity to population size. As is well known, the dynamics of the ecological model are determined by the slope at the equilibrium (May and Oster 1976). In all the 1-dimensional ecological models appearing in the literature (Bellows 1981), the scaling parameter a has no effect on this slope, and hence the scaling parameter does not influence the dynamic complexity of these models. Nevertheless, in this paper we show that genetic variability in the scaling parameter can have very strong effects on the dynamics of a population.

In Section 2, we describe the basic two-dimensional model for a population with density-dependent fitness functions and genetic variability at one locus with two alleles and random mating. We consider the case where variability in the scaling parameter is maintained by heterozygote advantage, and we show that in this case the dynamics of the population generally tend to be much simpler than in the corresponding 1-dimensional models. In Section 3, we extend the genetic model to include differential selection in males and females. Various mechanisms lead to polymorphim in the resulting 3-dimensional model, and we again concentrate on the effects of genetic variability in the scaling parameter on the ecological dynamics of the system. While the general result that variability in this parameter simplifies population dynamics still holds, there are some interesting differences, including a case where genetic variability destabilizes the system. In addition, multiple attractors with complicated basins of attraction can occur, which can lead to a sensitive dependence of the final state of the system on mutation rates and chance events.

Finally, in Section 4 we discuss more generally how polymorphisms can be maintained by constraining relations between the demographic parameters in the system. With differential selection in males and females, constraints can maintain variability even if one of the phenotypes present is not viable and would go extinct when alone. This phenotype represents a genetic sink, and the polymorphism generates a source-sink structure in the population. As in metapopulation models, such a source-sink structure can stabilise the dynamics of the population, which again shows that genetic variability can affect ecological dynamics.

2. THE 2-DIMENSIONAL MODEL

In this section, we derive the basic results for the simplest genetic model with density-dependent selection.

As fitness function we use Maynard Smith and Slatkin's (1973) equation

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

In the monomorphic case without genetics, this fitness function leads to the recurrence equation

$$\begin{aligned} N_{t+1} &= f(N_t) \cdot N_t \\ &= \frac{\lambda N_t}{1 + (aN_t)^b} \end{aligned} \tag{2}$$

for the population density N_t at time t. The parameter λ in this model describes the intrinsic growth rate of the species, i.e., the growth rate in the absence of density dependence. The parameter b describes the type of competition that leads to density dependence. Often a high b is associated with scramble competition, and a low $b \sim 1$ with contest competition (but see Schoener 1976). Finally, the parameter a scales the impact of the population density on individual fitness. As is well known, model (2) can exhibit a wide variety of dynamics, including stable equilibria and chaos (May and Oster 1976). The complexity of the dynamics is determined by the slope of Eq. (2) at the equilibrium N^* of (2), which is defined by $f(N^*) = 1$, hence

$$N^* = \frac{(\lambda - 1)^{1/b}}{a}. \tag{3}$$

The slope c of (2) at N^* is

$$c = 1 - b\frac{\lambda - 1}{\lambda}. \tag{4}$$

As the modulus $|c|$ increases above 1, the system exhibits the period-doubling route to chaos (May and Oster 1976). Note that the scaling parameter a only influences the equilibrium density N^*, but not the complexity c.

We extend model (2) to a diploid population with genetic variability at one locus with two alleles A and B by interpreting the fitness function (1) as genotypic fitness. Thus we have three density dependent fitness functions $f_1(N)$, $f_2(N)$, and $f_3(N)$, where $1 = AA$, $2 = AB$, and $3 = BB$. For the simplest genetic model one assumes that females control population size, that male gametes occur in the same frequencies as female gametes, and that the population is in Hardy–Weinberg equilibrium (Roughgarden 1979). We also assume that the fitness of each genotype depends only on the total density N_t of the population, i.e., on the sum of the densities of the three genotypes. This leads to a 2-dimensional system with one recurrence equation for the population size and one for the frequency p_t of allele A at time t:

$$\begin{aligned} N_{t+1} &= N_t \cdot \bar{w}(N_t) \\ p_{t+1} &= p_t \cdot \frac{p_t f_1(N_t) + (1 - p_t)\, f_2(N_t)}{\bar{w}(N_t)}. \end{aligned} \tag{5}$$

Here

$$\bar{w}(N_t) = p_t^2 f_1(N_t) + 2p_t(1 - p_t)\, f_2(N_t) + (1 - p_t)^2\, f_3(N_t) \tag{6}$$

is the mean fitness at time t. In the sequel, we are interested in the case where the two homozygotes have equal fitness, and where heterozygote advantage maintains a polymorphism in the system. We will assume that this advantage is expressed in a different scaling parameter a in the heterozygote fitness function f_2. It is clear that whatever the density N, a smaller value of a will always lead to a higher value of the basic fitness function $f(N)$, Eq. (1), i.e., that $\partial f / \partial a(N) > 0$ for all N. Therefore, we will assume that the heterozygote fitness function has a lower scaling parameter, $a_2 < a_1 = a_3$, but the same intrinsic growth rate λ and the same competition parameter b as the two homozygotes. Thus, at any point in time the fitness of the two homozygotes is the same and is lower than the fitness of the heterozygotes. Therefore, the gene frequency will equilibrate at 1/2, and we want to determine the dynamic properties of the total population density N_t.

For the calculations regarding the dynamic complexity we assume that p_t is at its equilibrium value of 1/2. It is then enough to consider the derivative of the first equation in system (5) with respect to N and to evaluate this derivative at the equilibrium solution $\hat{N}$ of the system. The derivative is given by

$$\bar{w}(N) + N \cdot \frac{\partial \bar{w}}{\partial N}(N). \tag{7}$$

At the equilibrium N we have $\bar{w}(\hat{N}) = 1$, and using $p_t = 1/2$ and $f_1 = f_3$ in Eq. (6) for $\bar{w}$, we get from Eq. (7) the complexity of this system:

$$\tilde{c} = \bar{w}(\hat{N}) + \hat{N} \cdot \frac{\partial \bar{w}}{\partial N}(\hat{N}) = 1 + \hat{N} \cdot \left[\frac{1}{2}\frac{\partial f_1}{\partial N}(\hat{N}) + \frac{1}{2}\frac{\partial f_2}{\partial N}(\hat{N}) \right]. \tag{8}$$

(In fact, under the given assumptions $\tilde{c}$ is the dominant eigenvalue of the Jacobian of system (5) at the equilibrium.) To calculate $\hat{N}$ we use $\bar{w}(\hat{N}) = 1$, and since $f_1 = f_3$ and $p_t = 1/2$, this leads to

$$\tfrac{1}{2} f_1(\hat{N}) + \tfrac{1}{2} f_2(\hat{N}) = 1. \qquad (9)$$

Substituting the expressions for the fitness functions yields

$$\frac{\lambda}{1+(a_1\hat{N})^b} + \frac{\lambda}{1+(a_2\hat{N})^b} = 2, \qquad (10)$$

where $a_2 < a_1$ by assumption (recall that λ and b are the same for homozygotes and heterozygotes). This equation can be solved analytically for $\hat{N}$, and the result can be substituted into Eq. (8). In this way, the complexity $\tilde{c}$ becomes a function $\tilde{c}(a_1, a_2, \lambda, b)$ of the four parameters in the model. Note that if the homozygotes and heterozygotes are equal, $a_1 = a_2$, the system reduces to the 1-dimensional case (2), hence $\tilde{c}(a, a, \lambda, b) = c = 1 - b(\lambda - 1)/\lambda$ is the complexity given by (4). In general, however, the scaling parameters of the different genotypes do not cancel out. Thus, in contrast to the

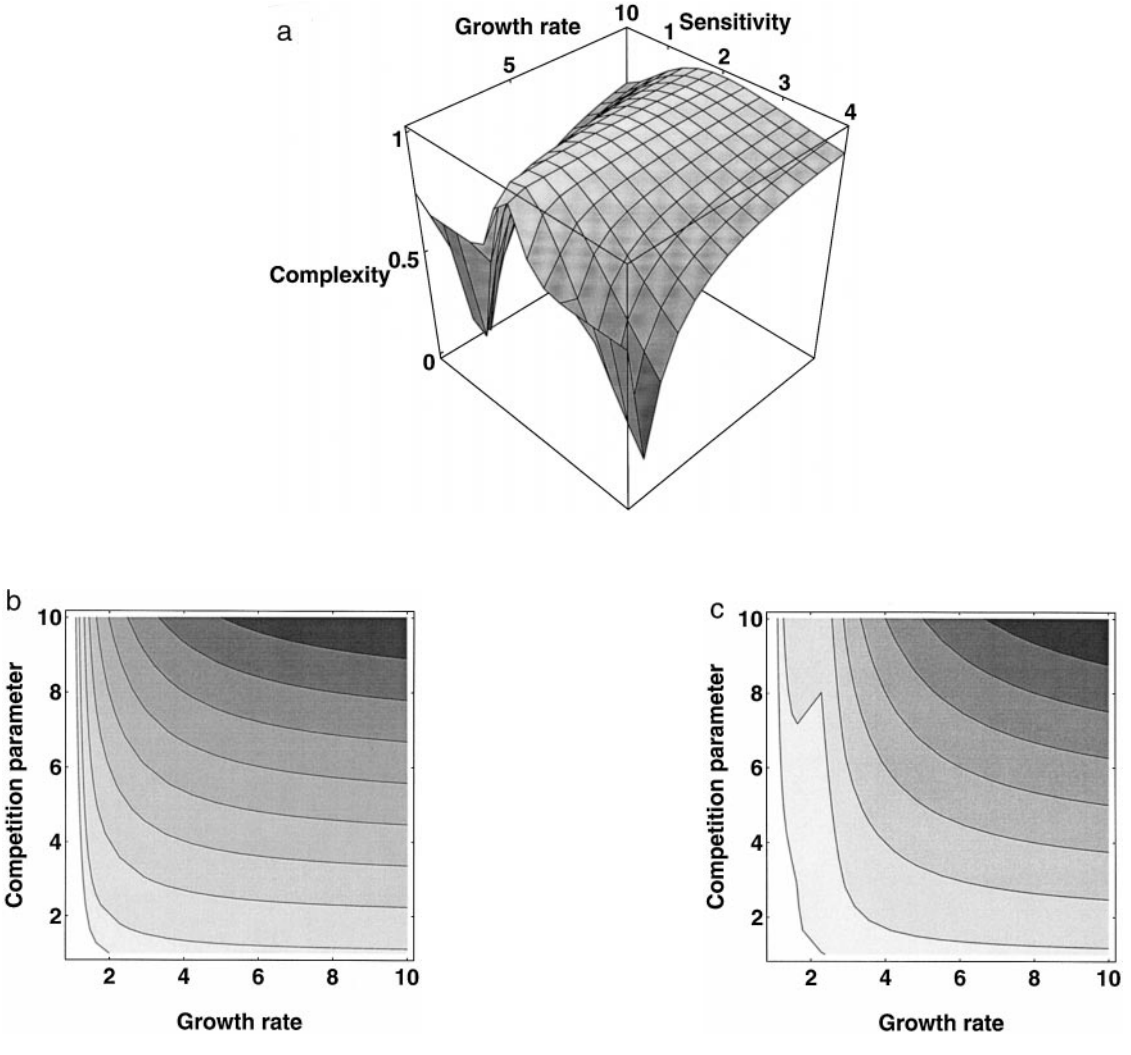

FIG. 1. Effects of variability in the scaling parameter on the dynamic complexity $\tilde{c}$, Eq. (8). In (a) $|\tilde{c}|$ is shown as a function of the growth rate λ (equal for all genotypes) and of the sensitivity parameter a_2 in the heterozygote. The sensitivity of the homozygotes was set to $a_1 = a_3 = 2$. For each value of λ, the parameter b (equal for all genotypes) was chosen so that the complexity in the basic model (2) was -1. $|\tilde{c}|$ is maximal (and equal to 1) for $a_2 = 2$, and decreases as $|a_2 - a_1|$ increases. The effect is strongest for intermediate growth rates. Note that polymorphisms only occur if $a_2 < a_1$, i.e., with heterozygote advantage. In (b) and (c), isoclines of the complexity in the monomorphic system and in the polymorphic system are shown. The curves correspond to values of c, Eq. (4), and of $\tilde{c}$, Eq. (8), of -0.5, -1, -2, -3, etc. The sensitivity parameters in the genetic model were $a_1 = a_3 = 0.1$ and $a_2 = 0.02$.

1-dimensional system, the scaling parameters influence the dynamics of the genetically variable population.

The way in which the dynamics change compared to the 1-dimensional system can be seen by taking the derivative of $\tilde{c}$ with respect to the scaling parameter a_2 of the heterozygote and evaluating it at the limiting value $a_2 = a_1$. Note that $\tilde{c}$ is always <1, as can be seen from Eq. (8), and hence non-equilibrium dynamics with $|\tilde{c}| > 1$ only occur if $\tilde{c}$ is negative. It can be shown analytically that $\tilde{c}$ actually has a minimum at $a_1 = a_2$. It immediately follows that, compared to the 1-dimensional system, heterozygote advantage in the scaling parameter (i.e., assuming $a_2 < a_1$) decreases the modulus $|\tilde{c}|$ and hence generally leads to simpler dynamics. (Strictly speaking, if the slope at the equilibrium in the 1-dimensional model is positive, i.e., if $\tilde{c}(a, a) > 0$, then its minimality implies that the complexity $|\tilde{c}|$ is larger in the polymorphic system. However, in both systems the complexity will then be a number between 0 and 1, and hence both systems will exhibit Beverton–Holt type stability with monotonic damping back to the equilibrium after a perturbation.) Thus, the maximal complexity occurs when the 2-dimensional system reduces to the 1-dimensional case.

The analytical proof of this result consists of taking derivatives and comparing various rather complicated terms, and no essential insights can be gained from it. We therefore omit this proof and instead illustrate the phenomenon graphically. Figure 1a shows $|\tilde{c}|$, given by Eq. (8), as a function of a_2 and λ for fixed a_1 and b. Clearly, $|\tilde{c}|$ is maximal when $a_2 = a_1$. Moreover, numerical calculations strongly suggest that the maximum of $|\tilde{c}|$ at $a_1 = a_2$ is global. Figure 1a also indicates that the simplifying effect of heterozygote advantage in the scaling parameter depends on the growth rate λ: the maximality of $|\tilde{c}|$ at $a_1 = a_2$ is most pronounced, and hence any deviation $a_2 < a_1$ most effective, for a range of small to intermediate growth rates. The range for which the effect is biggest depends on the parameter b. Another way of visualizing the dynamic effects is to consider isoclines of the complexity as a function of b and λ. Figure 1b shows the isoclines for the 1-dimensional model, and Fig. 1c for the genetic model with overdominance in the scaling parameter a. Clearly, the isoclines are more spaced out in the latter case, which corresponds to the generally simpler dynamics in this model. The isocline pictures reveal in more detail how the simplifying effect depends on b and λ.

The mathematical reason for the dynamic effects observed can be seen by considering next generation maps for the total population size, i.e., by plotting N_{t+1} in the first equation in system (5) as a function of N_t under the assumption that $p_t = 1/2$ in Eq. (6). In contrast to the corresponding map for the 1-dimensional model, Eq. (2), this map typically has two maxima (Fig. 2). At first thought this seems to be counterintuitive, since one would expect increasing densities to monotonically worsen the conditions and hence to lead to a monotonic decrease in the next generation map after a maximum has been reached. However, this need not be the case in polymorphic populations, in which different types have different density dependent fitness functions and hence different next generation maps when alone. In the situation shown in Fig. 2, the first maximum of the next generation map of the total population occurs near the maximum in the next generation map of the homozygotes. However, by assumption the total population consists of equal amounts of homozygotes and heterozygotes, and since the next generation map of the heterozygotes is still increasing when that of the homozygotes has its maximum, there is a second peak for the total population near the density for which the next generation map of the heterozygotes has its maximum. If the equilibrium for the total population size, i.e., the point at which the next generation map for the total population intersects with the 45 degree line, lies in the shallow region between the two maxima, then the modulus of the slope of this map at the equilibrium is

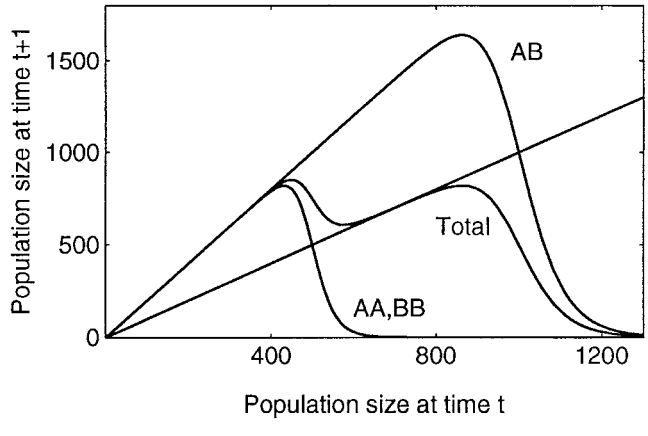

FIG. 2. Next generation maps in the genetic system. All three genotypes have one humped next generation maps, but the map for the total population, which consists of equal amounts of heterozygotes and homozygotes, has two peaks due to the difference between the genotypes. The straight line is the line $N_{t+1} = N_t$, and the intersection of this line with the next generation map determines the equilibrium population size. In the example shown, the slope of the next generation map for the total population is positive at the equilibrium, hence the system exhibits monotonic damping of Beverton–Holt type when perturbed away from the equilibrium. This occurs despite highly chaotic dynamics that would results from the next generation maps of each of the three genotypes. Parameters for the figure were $\lambda = 2$ and $b = 20$ for all genotypes, $a_1 = a_3 = 0.002$ and $a_2 = 0.001$.

likely to be small, and hence simple dynamics will occur. The point to remember is that next generation maps for polymorphic populations need not necessarily be one-humped and that this can affect the dynamics of such populations.

Of course, one would like to know how big the simplifying effect is in terms of the fluctuations in the system, i.e., in terms of the corresponding time series. Figure 3 shows a rather striking example. For fixed values of $a_1 > a_2$ and of λ, we plotted bifurcation diagrams with the complexity $|c|$, Eq. (4), of the basic model (2) as bifurcation parameter. Figure 3a shows the 1-dimensional system (2), while Fig. 3b depicts the dynamic behaviour of system (5) with heterozygote advantage in the scaling parameter. (To produce this figure, we no longer assumed that the frequency of both alleles is 1/2 and instead simulated the full system (5). Nevertheless, the allele frequencies always quickly equilibrated at 1/2.) The

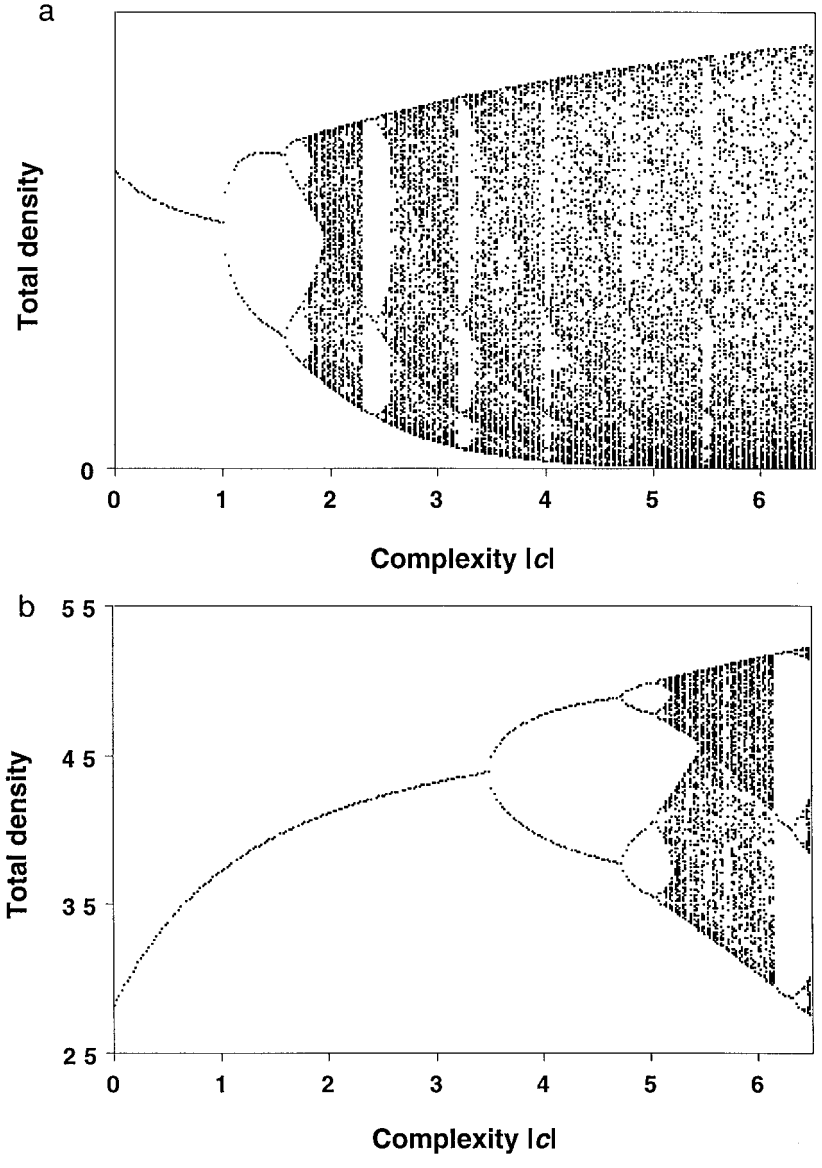

FIG. 3. Bifurcation diagrams for the dynamics of systems (2) (a) and system (5) (b) with the complexity $|c|$ in the basic model (2) as bifurcation parameter. The growth rate was $\lambda = 2.8$ for both panels. For 260 equally spaced values of c, the parameter b was calculated from Eq. (4). For each set of parameters, the systems were run for 1000 generations to remove transient effects, and then the total population size in the next 100 generations was plotted in the vertical direction (in system (5) the gene frequency p_t always equilibated at 1/2). In the genetic system we set $a_2 = 0.02$ and $a_1 = a_3 = 0.1$. The latter value was also used in the monomorphic model. b and λ were given as above and were the same for all genotypes.

simplifying effect is rather large: whereas the period-doubling route to chaos in the 1-dimensional system starts at $|c| = 1$ and chaos occurs for values of $|c|$ above ca. 1.8, the onset of 2-cyclic dynamics is delayed in the genetically variable system, in which chaos occurs only for $|c|$-values above ca. 5.2. For high as well as for very low values of λ the dynamic effect of genetic variability in the scaling parameter is less pronounced.

3. DIFFERENT FITNESS IN MALES AND FEMALES: THE 3-DIMENSIONAL MODEL

In this section, we show that genetic variability in the scaling parameter can have an effect on population dynamics not only with heterozygote advantage, but also with different selection in males and females. If males and females have different fitness functions, the population will in general not be in Hardy–Weinberg equilibrium. Therefore, we will need a recurrence equation for each genotype. We still assume that females control the population size by the number of eggs they produce, and we will assume a 50 : 50 sex ratio at all times. Thus, if $N_{1,t}$, $N_{2,t}$, and $N_{3,t}$ are the densities of females of genotypes AA, AB, and BB at time t, then the total number of individuals in the population at time t is

$$N_t = 2(N_{1,t} + N_{2,t} + N_{3,t}). \tag{11}$$

The total number in the next generation will then be

$$N_{t+1} = N_{1,t} f_1(N_t) + N_{2,t} f_2(N_t) + N_{3,t} f_3(N_t), \tag{12}$$

where, as before, f_1, f_2, and f_3 are the fitness functions of females of genotype AA, AB, and BB. The proportion p_t of female gametes of type A produced in generation t is

$$p_t = \frac{N_{1,t} f_1(N_t) + (1/2)\, N_{2,t} f_2(N_t)}{N_{t+1}}. \tag{13}$$

If selection in males and females were the same, this would also be the proportion of male gametes of type A, and under the assumption of random mating, zygotes would then from in Hardy–Weinberg proportions, which would lead to system (5). Here, however, we assume that male gamete frequencies are different from female gamete frequencies. To model this we introduce male fitness functions $m_1(N)$, $m_2(N)$, and $m_3(N)$ of the same general type as the basic fitness function (1) in Section 2. As mentioned, we still assume that female fitness controls population size (Eq. (12)), so that we will only be interested in male gamete frequencies, but not in male gamete numbers. Therefore, we will set the intrinsic growth rate in the male fitness functions equal to 1.

Since we assume a 50 : 50 sex ratio, i.e., that the number of males of the three genotypes in generation t are also given by $N_{1,t}$, $N_{2,t}$, and $N_{3,t}$, it follows that the frequency q_t of male gametes of type A at time t is

$$q_t = \frac{2N_{1,t} m_1(N_t) + N_{2,t} m_2(N_t)}{2N_{1,t} m_1(N_t) + 2N_{2,t} m_2(N_t) + 2N_{3,t} m_3(N_t)} \tag{14}$$

(where N_t is given by Eq. (11)). With random mating, and with the frequency p_t of female gametes of type A given by (13) and the total density N_{t+1} given by (12), the densities of females of the three genotypes in generation $t+1$ will then be

$$\begin{aligned} N_{1,t+1} &= \tfrac{1}{2} p_t q_t N_{t+1} \\ N_{2,t+1} &= \tfrac{1}{2}[\, p_t(1-q_t) + (1-p_t)\, q_t\,]\, N_{t+1} \\ N_{3,t+1} &= \tfrac{1}{2}(1-p_t)(1-q_t)\, N_{t+1}. \end{aligned} \tag{15}$$

(For example, $p_t q_t N_{t+1}$ is the total density of genotype AA, but only half of the individuals are females, hence $N_{t+1} = p_t q_t N_{t+1}/2$.) This is the 3-dimensional system for the three (female) genotypes that we promised at the beginning of this section. Without density-dependence, i.e., with constant fitness values f_1, f_2, f_3, m_1, m_2, m_3, it reduces to a 2-dimensional system whose equilibria and dynamic behaviour have been the subject of considerable interest (Kidwell *et al.* 1977, Selgrade and Ziehe 1987, Szucs 1991). It is known that the 2-dimensional system has only point attractors (Szucs 1991).

We are still interested in the effect of genetic variability in the scaling parameter that occurs in the various fitness functions. However, instead of heterozygote advantage as in the last section, we now consider the case in which selection among the genotypes with respect to the scaling parameter is directional, but of opposite signs in males and females. Thus we assume (for example) that, in females, the fitness function of genotype AA has the lowest and that of genotype BB the highest scaling parameter, while the reverse holds for the male fitness functions. In systems with constant fitness values rather than density dependent fitness functions, it is known that opposite directional selection in males and females can lead to polymorphisms (Kidwell 1977, Hartl and Clark 1989). Numerical simulations show that the same is true in system (15): with opposite directional selection on the scaling parameter in males and females, polymorphisms can easily be maintained in the population.

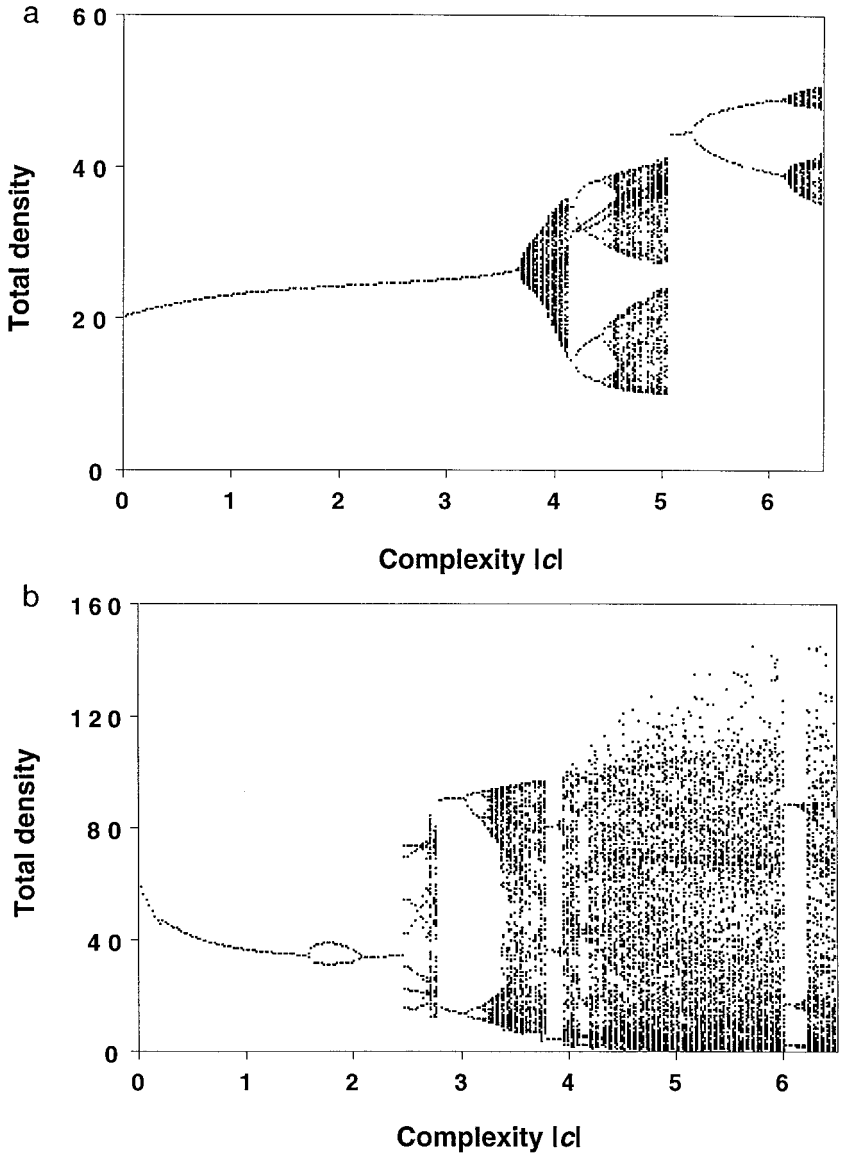

FIG. 4. Bifurcation diagrams for model (15). The same procedure as for Fig. 3 was used. For a given value of the complexity c in the basic model (2), we calculated the competition parameter b for the females from Eq. (4), using the effective growth rates of the females. The effective growth rate is half the nominal growth rate, because males do not contribute to population size (see text). In (a), the nominal growth rate in the females was set to $\lambda = 5.6$, which amounts to the same effective growth rate of 2.8 as in Fig. 3. In (b) we set $\lambda = 16$, i.e., an effective growth rate of 8. In (c), we set $\lambda = 2.3$, i.e., an effective growth rate of 1.15. For the sensitivity parameters, we set $a_1 = 0.1$, $a_2 = 0.05$, and $a_3 = 0.02$ in the females, and $a_1 = 0.02$, $a_2 = 0.05$, and $a_3 = 0.1$ in the males for all the panels. The competition parameter for all male genotypes males was $b = 4.5$ in (a) and (b), and $b = 50$ in (c).

For the remainder of this section we assume that opposite directional selection on the scaling parameter in males and females maintains a polymorphism in the population, and we address the question of how the ensuing genetic variability in the scaling parameter affects the population dynamics. However, there are other mechanisms than opposite directional selection in males and females than can lead to polymorphisms in system (15). Some of these will be discussed in the next section.

Similar to the scenarios considered in the last section, where variability in the scaling parameter was maintained by heterozygote advantage, variability in the scaling parameter that is maintained by opposite directional selection in males and females has a strong effect on the complexity of the dynamic behavior of

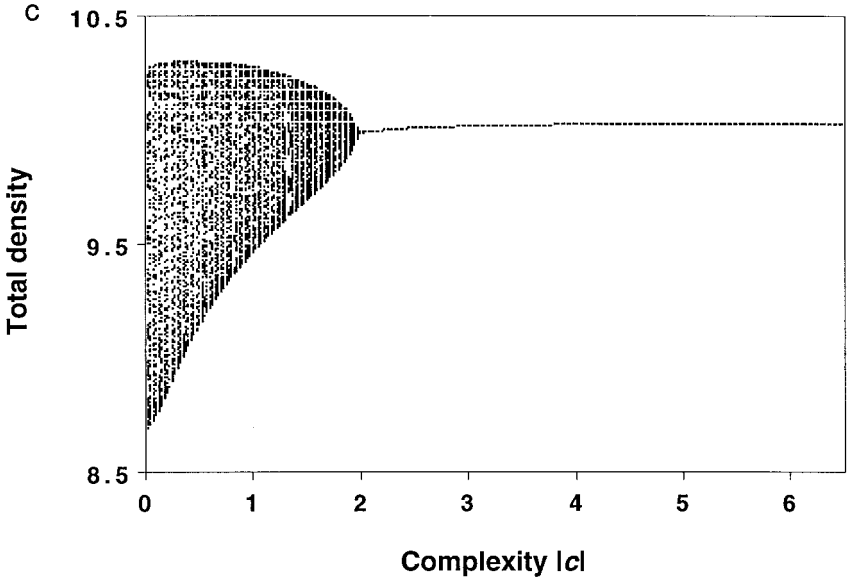

FIG. 4—*Continued*

system (15): over a wide range of parameters, the system exhibits much simpler dynamics than the corresponding monomorphic populations. However, there are some significant differences between the effects of maintaining polymorphism by heterozygote advantage and maintaining polymorphism by opposite directional selection in males and females. First of all, in the latter case the population is in general out of Hardy–Weinberg equilibrium, the disequilibrium being larger for greater strengths of directional selection in the scaling parameter. Note that to compare the dynamics of the simple system (5) with those of the more complicated system (15), we have to assume that the growth rate of females in (15) is twice the growth rate in model (5), because half of all offspring are males which by assumption do not contribute to population size. Thus the effective growth rate is half of the nominal growth rate appearing in system (15). (This is reflected in the factor 1/2 that appears in Eqs. (15).) Figure 4 shows some of the main differences between the 2-dimensional system (5) and the 3-dimensional system (15). For the intermediate growth rates for which the effect is strongest in the simpler system (5), stabilization also occurs in system (15) (Fig. 4a, cf. Fig. 3b). Note, however, that the system does not follow the period-doubling route to chaos anymore. In addition, in system (15) simple dynamics can be obtained even for high growth rates (Fig. 4b). For such growth rates only small effects are visible in system (5).

With different selection in males and females, the simplifying effect of variability in the scaling parameter occurs also for very low growth rates (Fig. 4c), again in contrast to system (5), in which the effect is much smaller for low growth rates. However, for a small range of parameters for which the original 1-dimensional system (2) (and hence also system (5)) has a stable equilibrium, system (15) can have complicated dynamics (Fig. 4c, left margin). This happens if the competition parameter b in the male fitness functions is very high. Recall that the competition parameter in the males has no effect on population dynamics, because the population size is controlled by the females. However, the demographic parameters in males do have an effect on gene frequency dynamics, hence this result shows that a strong destabilizing effect on gene frequency dynamics (mediated here by strong competition in males) can lead to a destabilization of the population dynamics. An example of this phenomenon is shown in Fig. 5. In Fig. 5a, the total population density is plotted against time, and Fig. 5b shows the time series of the frequency of allele A in males and females after selection in each generation. The values in successive generations are joined by lines. The system exhibits interesting dynamics, in which extended periods of population growth alternate with extended periods of population decline. Note that this is not the usual type of cyclic dynamics in discrete systems, in which the system jumps up and down in successive generations. In fact, it appears that the dynamics shown in the figure exhibit sensitive dependence on initial conditions and hence are chaotic.

Finally, we note that besides the trivial states with only homozygotes of one type present, system (15) can

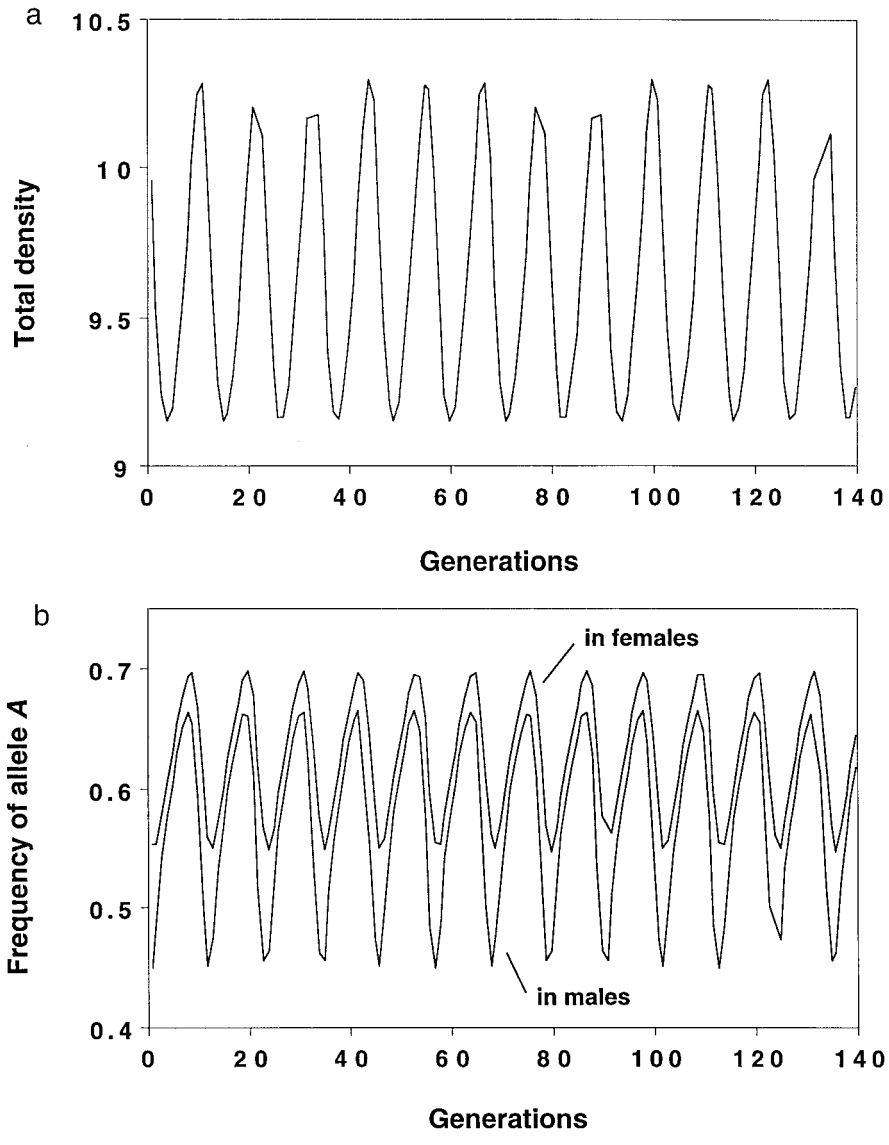

FIG. 5. Dynamics corresponding to the point $c = -0.5$ in Fig. 4c (i.e., to a value of $b = 11.5$ for the females). In (a) is shown the total population size as a function of time, and in (b) is shown the frequency of allele A in males and females. (Values in successive generations are joined by a continuous line to improve presentation.) Note that the frequency dynamics are not symmetric around 0.5 because of the different values of the competition parameter b in males and females.

have multiple attractors, in contrast to system (5) with heterozygote advantage. This is again reminiscent of the simpler systems with constant fitness values for differential selection in males and females, in which multiple stable polymorphic equilibria occur (Kidwell *et al.* 1977). In contrast to these systems, however. there can be very sensitive dependence on initial conditions as to which of the attractors is approached in the systems with the density dependence considered here. That is, the basins of attraction can have fractal boundaries. This can be important for invasion scenarios. An example is shown in Fig. 6. In this case, numerical simulations indicate that the system has two polymorphic attractors, a locally stable equilibrium and a locally stable 3-cycle. In particular, a monomorphic population is susceptible to invasion by the other allele, and starting out with only one type of homozygotes, say *BB*, one would like to know the final state of the system after introducing a small amount of heterozygotes in the population. In fact, whether it is the equilibrium point or the 3-cycle depends in a very sensitive way on the exact initial amount of heterozygotes. In Fig. 6, a value of 1 indicates that for the given initial density of heterozygotes the final state is the equilibrium point, while a value of 0 indicates that the final state is the

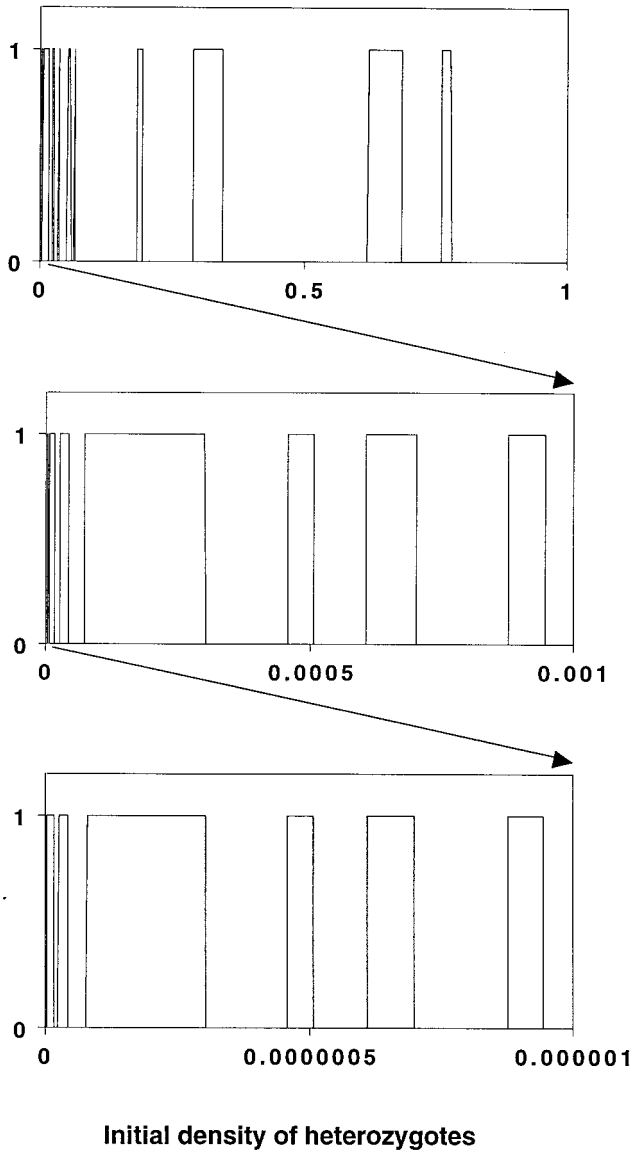

FIG. 6. Sensitive dependence of the final state on the initial density of heterozygotes. For the given parameters system (15) has two polymorphic attractors, a stable equilibrium and a 3-cycle. Starting from a monomorphic *BB*-population and introducing a small amount of *AB*-heterozygotes, the system will move to one of these attractors. In each panel 1000 equally spaced values of initial heterozygote density were classified as leading to the stable equilibrium (1) or to the 3-cycle (0). The test intervals were [0, 1] in the upper panel, [0, 0.001] in the middle panel and [0, 0.000001] in the lower panel. The monomorphic *BB*-population into which the heterozygotes were introduced was the same for each invasion trial. Panels b and c indicate the self-similar nature of the set of initial heterozygote frequencies that lead to either of the two attractors. The parameter values in (15) were $a_1 = 0.1$, $a_2 = 0.06$, and $a_3 = 0.01$ in males, and $a_1 = 0.01$, $a_2 = 0.06$, and $a_3 = 0.1$ in females; growth rate in females $\lambda = 14$ (hence effective growth rate $= 7$); $b = 4.1$ in females and $b = 5$ in males.

3-cycle. In each of the three panels, 1000 equally spaced values of initial heterozygote density were tested. The width of the test interval decreased from 1 to 1/1000 to 1/1,000,000. Initial values leading to the stable equilibrium are densely interspersed with initial values leading to the 3-cycle, and the pattern of 0's and 1's seems to repeat itself on ever finer scales, indicating a fractal structure of the set of initial heterozygote frequencies that lead to either the steady state or the 3-cycle. This implies that the final state of the system depends on the mutation rate and on chance events determining the amount of *A*-mutants occurring at any one point in time.

4. MAINTENANCE OF POLYMORPHISM

In the last section we assumed parameter values that induced a polymorphism in the scaling parameter in system (15) and then studied the effect of this genetic variability on population dynamics. However, the maintenance of polymorphisms is in itself an interesting problem, and we now briefly discuss the various ways in which polymorphisms can be maintained. So far, we have discussed two obvious ways to keep both alleles in the population: with heterozygote advantage in the simple system (5), or with opposite directional selection in males and females in system (15). In the latter system, polymorphism can also be maintained with overdominance in one or both sexes (Kidwell *et al.* 1977, Hartl and Clark 1989). However, none of these conditions is necessary for the maintenance of polymorphisms if constraints between the demographic parameters λ, b, and a in the basic fitness function (1) are included.

To incorporate tradeoffs in the models, one has to know the selection pressures on each parameter when the other two parameters are kept constant. It is clear that, independent of the density of the population, higher intrinsic growth rates λ will present an evolutionary advantage, i.e., that $\partial f/\partial\lambda(N) > 0$ for all N. Similarly, $\partial f/\partial a(N) < 0$ for all N, i.e., a lower a will always be selected for. The situation is more complicated for the competition parameter b, and in fact an analytical solution to the problem of the selection pressure on b is in general lacking. However, numerical results show that small b's are selectively advantageous (Doebeli and Koella 1995). Given the separate selection pressures on each parameter, the selection pressure on the combined sets of parameters (λ, b, a), i.e., on the phenotypes in the population, depends on the covariance between these parameters, i.e., on the constraining relations between them. With tradeoffs, it is possible that two phenotypes

can coexist. For example, if one phenotype has a high growth rate λ, but also a high competition parameter b, it might not be able to outcompete another phenotype with a low growth rate and a low competition parameter, and vice versa. Or a phenotype with a high a and a high λ might coexist with a phenotype with a low a and a low λ, etc. In this way, it is possible to maintain polymorphisms in the genetic model (5) even with complete dominance, i.e., if the heterozygote is phenotypically identical to one of the homozygotes. This can be achieved even if the two phenotypes present in the system do not have the same equilibrium density, i.e., the same carrying capacity. This is worth mentioning because it is often thought that the carrying capacity is the main evolutionary determinant in the type of competition models considered here. This belief is partly based on results for the Ricker fitness function

$$f(N) = \lambda \cdot \exp(-aN), \tag{16}$$

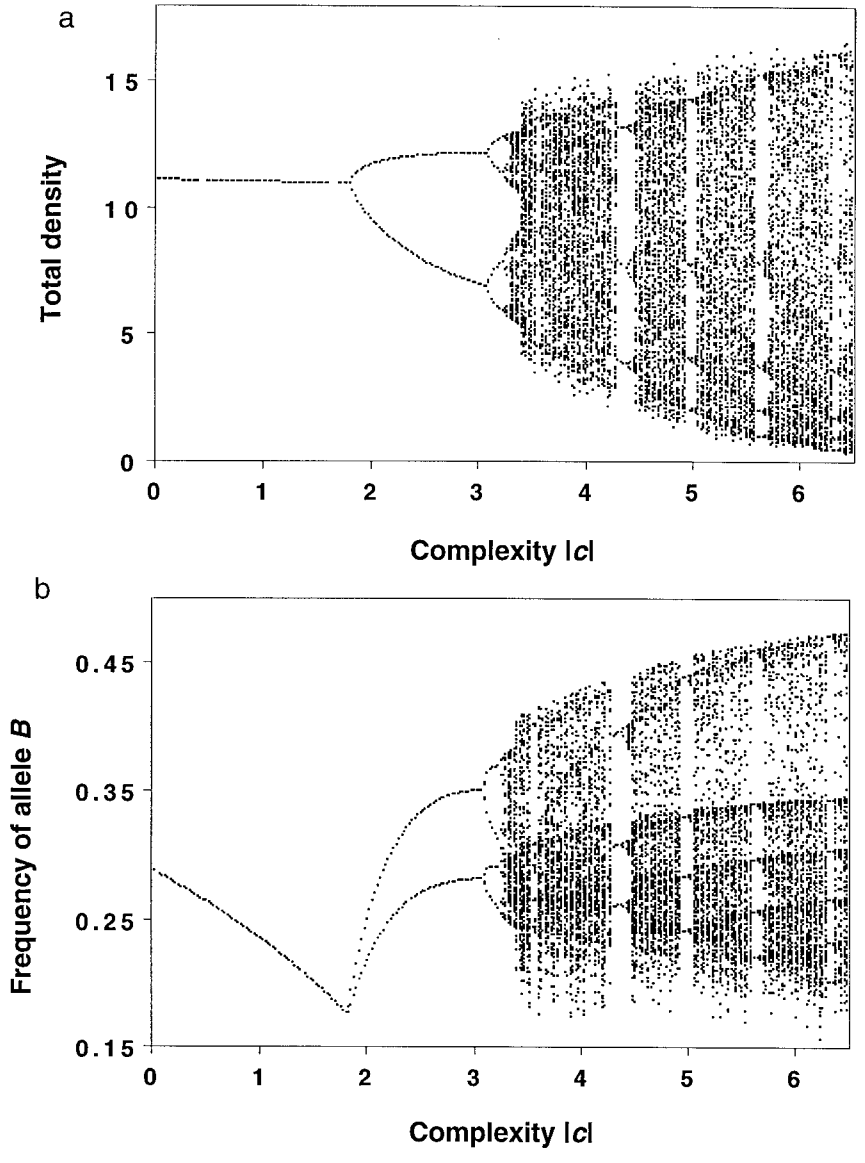

FIG. 7. Bifurcation diagram for model (15) with one inviable homozygote (*BB*). The same procedure as for Fig. 4 was used. The (nominal) growth rates in the females were $\lambda_1 = \lambda_2 = 5$ and $\lambda_3 = 0$ (hence *BB* had no offspring). The *BB* homozygote was favoured in males by setting the competition parameters in males to $b_1 = b_2 = 13$ and $b_3 = 1$. The competition parameter in the females was the same for all genotypes and was calculated from Eq. (4) for each value of the bifurcation parameter c. The sensitivity parameter was $a = 0.1$ for all genotypes in males and females. In (a) is shown the dynamics of the total population size as a function of $|c|$, while in (b) is shown the dynamics of the frequency of allele B in the females.

for which it is indeed true that the only evolutionary criterion is the carrying capacity $N^* = \ln \lambda / a$ (Gatto 1993). However, as far as difference equations are concerned, the Ricker equation is a special case, and more general equations such as the one used here, Eq. (2), lead to different and more general evolutionary results.

In system (15) with different selection in males and females, tradeoffs can be translated into selection differentials in different parameters in males and females. For example, female genotypes could be ordered with respect to the growth rate λ in one direction, while the male genotypes are ordered with respect to the competition parameter b or the scaling parameter a in the other direction. In this way, it is very easy to maintain polymorphisms in system (15).

In fact, numerical investigations indicate that in system (15) polymorphisms occur over a much wider range of parameters than in system (5). While polymorphisms through constraints require parameters that are relatively finely balanced against each other in system (5), very large selection differentials in one sex can be offset by very small selection differentials in the other sex in system (15). For example, with $\lambda_1 = \lambda_2 = 2$ and $\lambda_3 = 200$ in females, and with $b_1 = b_2 = 3$ and $b_3 = 4$ in males (large advantage for *BB* in females and small disadvantage in males), polymorphism is maintained over a wide range of the other parameters in the system. Thus, the decoupling of the selection pressures on different parameters in males and females is favourable for the maintenance of polymorphism. Again, similar observations have been made in the simpler case with constant fitness values in males and females (Kidwell *et al.* 1977, Hartl and Clark 1989).

One particular case in which a polymorphism can be maintained in system (15) but not in system (5) seems worth mentioning. It occurs when one of the phenotypes is not viable when alone, i.e., when the females of that phenotype have an intrinsic growth rate λ that is smaller than 1. Numerical simulations suggest that it is impossible to keep such a phenotype in a population described by system (5). However, with differential selection in males and females, an inviable phenotype can be maintained if the males of that phenotype have a competitive advantage, i.e., a low b. An extreme example of this is shown in Fig. 7, in which the growth rate of females of genotype *BB* was set to 0. However, the *B*-allele is kept in the population through its selective advantage in males. In addition—and going back to the theme of the last section about the effects of genetic variability on population dynamics—the maintenance of the inviable phenotype has an effect on the dynamics of the system. The inviable phenotype acts as a "genetic sink," which simplifies the ecological dynamics. Source-sink structures in heterogenous environments can have a stabilizing effect on the dynamics of metapopulations (Doebeli 1995a), and a similar mechanism seems to be at work here: the sink genotype absorbs the overproduction of the other genotypes and thereby dampens the fluctuations in the system.

4. CONCLUSIONS

In most 1-dimensional discrete time ecological models, there is a parameter that measures the sensitivity to population density but does not affect the dynamic complexity of the model (Bellows 1981). More precisely, this parameter does not influence the slope of the function relating the population densities in successive generations at the equilibrium population size. However, here we argue that genetic variability in this parameter can have a very strong effect on the complexity of population dynamics.

Starting out from the Maynard Smith and Slatkin (1973) model, Eq. (1), we show this by combining this ecological model with two different population genetic models involving one diploid locus with two alleles. In the classical genetic model of a randomly mating population in Hardy–Weinberg equilibrium, variability in the sensitivity to density can be maintained by overdominance. In this case, the dynamics of the genetically variable population are typically simpler than those of the corresponding monomorphic population (Fig. 3). This basic observation also holds for the second genetic model, in which genetic variability can be maintained by opposing selective forces in males and females. Such populations are in general not in Hardy–Weinberg equilibrium, and they exhibit a number of interesting dynamic behaviours. For example, models with different selection in males and females do not necessarily follow the period-doubling route to chaos (Fig. 4), and they can have multiple attractors with complicated basins of attraction. Multiple attractors were also reported by Asmussen (1979) for the simple genetic model (5), but not in the overdominant case that we have considered here. The existence of multiple attractors with complicated geometries of the corresponding basins of attraction can have consequences for the outcome of invasion by mutants, as the final state of the system after invasion can depend very sensitively on the mutation rate and on the distribution in which mutational steps occur (Fig. 6). In addition to usually having a simplifying effect on population dynamics, variability maintained by different selection in males and females can also destabilize a

stable equilibrium in the basic model (2) (Fig. 4c, Fig. 5). It is interesting to note that different selection in males and females can maintain genetic variability even if one of the homozygotes is inviable (Fig. 7). The resulting genetic source-sink structure again tends to have a simplifying effect on population dynamics (Fig. 7).

Even though we have used Eq. (1) for all our results, similar effects as the ones reported here can be observed for other models such as Hassell's (1975) equation

$$N_{t+1} = \frac{\lambda}{(1+aN_t)^b} \cdot N_t \qquad (17)$$

or Bellow's (1981) model

$$N_{t+1} = \frac{\lambda}{\exp[(aN_t)^b]} \cdot N_t. \qquad (18)$$

In contrast to the Maynard Smith and Slatkin (1973) model (2), analytical results cannot be obtained for these models, essentially because the equations for the equilibrium density corresponding to Eq. (10) have no analytical solution. However, numerical simulations showed that if the ecological models (17) and (18) are combined with the two genetic models discussed, then variability in the scaling parameter a has very similar effects to those reported in Sections 2 and 3. In particular, variability in a simplifies the dynamics if the growth rate λ is not too large. The situation is slightly different for the Ricker model, Eq. (16). In this model the growth rate λ alone determines the dynamic complexity, and since the simplifying effect of genetic variability in a disappears for high growth rates, it cannot be seen above a certain complexity in the Ricker model. This reiterates previous observations (e.g., Doebeli 1995a) that, mathematically speaking, the Ricker equation is a somewhat rigid and rather special 1-dimensional model. Nevertheless, Asmussen (1979) has made the observation that overdominance in the carrying capacity can simplify the dynamics in the Ricker model. As the sensitivity parameter in our models directly influences the carrying capacities of the genotypes, our work can be seen as an elaboration of this particular observation in Asmussen (1979).

The genetic model for one locus with two alleles in Hardy–Weinberg equilibrium is of course a classic, and the model with differential selection in males and females and constant fitness values has also received quite a lot of attention in the population genetics literature (e.g., Kidwell *et al.* 1977, Feldman, Christiansen and Liberman 1983, Karlin and Lessard 1986, Selgrade and Ziehe 1987, Hartl and Clark 1989, Szucs 1991a,b). In ecology, 1-dimensional difference equations have been the basis of numerous population dynamic investigations, particularly since May (1974, 1976) used these equations to introduce the paradigm of chaos into ecology. However, the combination of these modelling approaches—simple population genetic models on the one hand and simple ecological models with potentially complicated dynamics on the other hand—is much less common, and since Asmussen's (1979) pioneering work, not much has come forth in this direction. Some exceptions are Doebeli and Koella (1994), Ruxton (1995) and Doebeli (1995b, 1996, 1997). In Doebeli (1996), the basic model (2) was extended by multilocus genetics of a quantitative character that determines ecological interactions. Different phenotypes had different character values and occurred in different frequencies. All phenotpyes had the same basic fitness function (2), but due to frequency dependent interactions (competition is more intense between similar phenotypes than between phenotypes with very different characters), the effective density that was plugged into the fitness function in a given generation was different for each phenotype. Since only the product aN enters the fitness function (2), this is very similar to assuming that all phenotypes experience the same density but differ in their sensitivity a. Indeed, one of the conclusions of Doebeli (1996) was that genetic variability in the quantitative character tends to simplify population dynamics. This conforms with the results presented here and supports our belief that these results are fairly robust.

Mathematically, the reason for the simplifying effect observed here is that the next generation map showing the total population size at time $t+1$ as a function of the total population size at time t can have two maxima in the genetically variable population as opposed to the usual one-humped maps for monomorphic populations (Fig. 2). The simplifying effect occurs for parameter values for which the equilibrium population size lies in the shallow region between the two maxima of the next generation map. In general, if one is thinking about density-dependent fitness, but not about polymorphic populations, then one naturally expects a single maximum in the next generation map, to the right of which conditions get only worse due to increasing density. It is worth remembering that this picture may be wrong for polymorphic populations, in which different types have different density dependent fitness functions. In such populations multiply peaked next generation maps can occur, which in turn can have strong consequences for the dynamics of the populations.

The fact that it is really variability in the sensitivity parameter a which accounts for the phenomena reported can be nicely seen by considering sources of variability

that are not genetic, but imposed by the environment. For example, consider a population that is subject to parasitism. Assume that in each generation, a constant fraction of the population is parasitized, and that the parasitized individuals have a higher sensitivity to population density, but that the other demographic parameters determining the density dependent fitness, Eq. (1), are unaffected. Further assume that reproduction is asexual, and that the chance of being parasitized does not depend on whether the parent was parasitized or not. Then, if the chance of being parasitized in each generation is 0.5, numerical experiments reveal very similar effects as in the simple genetic model with Hardy–Weinberg equilibrium. (In fact, the analytical calculations for the complexity of the model just described are exactly the same as for the genetic model, cf. Eq. (8).) Thus, environmental variability in the scaling parameter can do the same job as genetic variability in this parameter. A similar analogy between environmental heterogeneity and genetic variability also holds with regard to the source-sink structures described at the end of Section 4 (Fig. 7): spatial source-sink structures (Doebeli 1995a) as well as genetic source-sink structures (Fig. 7) can have a stabilizing effect on population dynamics.

In the "parasite" model above the maintenance of variability is imposed by assuming that a certain proportion of the population is parasitized in each generation. By varying this proportion, one can check numerically that the simplifying effect of the environmentally imposed variability is greatest if the chance of being parasitized is exactly 0.5. This suggests that the effect in the genetic system with overdominance is largest if both homozygotes are equal, and that the effect becomes smaller with increasing asymmetry between the two homozygotes.

Besides asymmetry between the homozygotes, constraining relations between the demographic parameters in the model can also blur the effect of variability in the scaling parameter a. With constraints polymorphisms can be maintained in both genetic models under many conditions, e.g., even with complete dominance, i.e., if one of the homozygotes is equal to the heterozygote. However, numerical simulations suggest that with constraints, the dynamic properties of the system are intermediate between those of the different types present in the population. This additivity tends to mask the simplifying effect of pure variability in the scaling parameter.

In general, it is fairly easy to maintain polymorphisms in the systems considered here, either by demographic constraints balancing different types against each other, or by mechanisms such as overdominance or opposing selection in males and females. Typically, polymorphisms are more likely in the genetic system with different selection in males and females than in the system with Hardy–Weinberg equilibrium. This reiterates earlier findings about these models using constant fitness values (Kidwell *et al.* 1977, Hartl and Clark 1989, p. 177). Our results show that combining genetic models with density dependent fitness functions can lead to a number of new dynamic phenomena. This is particularly true for the genetic model with different selection in males and females, and we are confident that similar models will yield further interesting results in the future. For example, one promising approach is to consider fertility selection models, in which fitnesses are assigned to mating pairs rather than to individuals. In contrast to the genetic models considered here, such models can have non-equilibrium dynamics even with constant fitness values (Hadeler and Libermann 1975, Pollak 1978, Doebeli and De Jong 1998). Combining these models with ecological dynamics should prove to be a fruitful extension.

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REFERENCES

Asmussen, M. A. 1979. Regular and chaotic cycling in models of ecological genetics, *Theor. Popul. Biol.* **16**, 172–190.

Bellows, T. S., Jr. 1981. The descriptive properties of some models for density dependence, *J. Anim. Ecol.* **50**, 139–156.

Doebeli, M. 1995a. Dispersal and dynamics, *Theor. Popul. Biol.* **47**, 82–106.

Doebeli, M. 1995b. Phenotypic variability, sexual reproduction, and evolutionary population dynamics, *J. Evol. Biol.* **8**, 173–194.

Doebeli, M. 1996. Quantitative genetics and population dynamics, *Evolution* **50**, 532–546.

Doebeli, M. 1997. Genetic variation and the persistence of predator–prey interactions in the Nicholson–Bailey model, *J. Theor. Biol.* **188**, 109–120.

Doebeli, M., and Koella, J. C. 1994. Sex and population dynamics, *Proc. Roy. Soc. London Ser. B* **257**, 17–23.

Doebeli, M., and Koella, J. C. 1995. Evolution of simple population dynamics, *Proc. Roy. Soc. London Ser. B* **260**, 119–125.

Doebeli, M., and De Jong, G. 1998. A simple genetic model with non-equilibrium dynamics, *J. Math. Biol.* **36**, 550–556.

Feldman, M. W., Christiansen, F. B., and Liberman, U. 1983. On some models of fertility selection, *Genetics* **105**, 1003–1010.

Ferrière, R., and Fox, G. A. 1996. Chaos and evolution, *Trends Ecol. Evol.* **10**, 480–485.

Hadeler, K. P., and Liberman, U. 1975. Selection models with fertility differences, *J. Math. Biol.* **2**, 19–32.

Hartl, D. L., and Clark, A. G. 1989. "Principles of Population Genetics," 2nd ed., Sinauer, Sunderland, MA.

Hassell, M. P. 1975. Density-dependence in single-species models, *J. Anim. Ecol.* **44**, 283–296.

Hassell, M. P., Lawton, J. H., and May, R. M. 1976. Patterns of dynamical behaviour in single-species populations, *J. Anim. Ecol.* **45**, 471–486.

Hastings, A. 1993. Complex interactions between dispersal and dynamics: Lessons from coupled logistic equations, *Ecology* **74**, 1362–1372.

Hastings, A., Hom, C. L., Ellner, S., Turchin, P., and Godfray, H. C. J. 1993. Chaos in ecology: Is mother nature a strange attractor? *Ann. Rev. Ecol. Syst.* **24**, 1–33.

Karlin, S., and Lessard, S. 1986. "Sex Ratio Evolution," Princeton Univ. Press, Princeton, RI.

Kidwell, J. F., Clegg, M. T., Stewart, F. M., and Prout, T. 1977. Regions of stable equilibria for models of differential selection in the two sexes under random mating, *Genetics* **85**, 171–183.

May, R. M. 1974. Biological populations with non-overlapping generations: stable points, stable cycles and chaos, *Science* **186**, 645–647.

May, R. M. 1976. Simple mathematical models with very complicated dynamics, *Nature* **261**, 459–467.

May, R. M., and Oster, G. F. 1976. Bifurcations and dynamic complexity in simple ecological models, *Am. Nat.* **110**, 573–599.

Maynard Smith, J., and Slatkin, M. 1973. The stability of predator–prey systems, *Ecology* **54**, 384–391.

Mueller, L. D., and Ayala, F. J. 1981. Dynamics of single-species population growth: Stability or chaos? *Ecology* **62**, 1148–1154.

Philippi, T. E., Carpenter, M. P., Case, T. J., and Gilpin, M. E. 1987. *Drosophila* population dynamics: Chaos and Extinction, *Ecology* **68**, 154–159.

Pollak, E. 1978. With selection for fecundity the mean fitness does not necessarily increase, *Genetics* **90**, 383–389.

Roughgarden, J. 1979. "Theory of Population Genetics and Evolutionary Ecology: An Introduction," Macmillan, New York.

Ruxton, G. D. 1994. Low levels of immigration between chaotic populations can reduce system extinctions by inducing asynchronous regular cycles, *Proc. Roy. Soc. London Ser. B* **256**, 189–193.

Ruxton, G. D. 1995. Population models with sexual reproduction show a reduced propensity to exhibit chaos, *J. Theor. Biol.* **175**, 595–601.

Schoener, T. 1976. Alternatives to Lotka–Volterra competition: Models of intermediate complexity, *Theor. Popul. Biol.* **10**, 309–333.

Selgrade, J. F., and Ziehe, M. 1987. Convergence to equilibrium in a genetic model with differential viability between the sexes, *J. Math. Biol.* **25**, 477–490.

Smith, R. H., and Mead, R. 1980. The dynamics of discrete-time stochastic models of population growth, *J. Theor. Biol.* **86**, 607–627.

Stone, L. 1993. Period-doubling reversals and chaos in simple ecological models, *Nature* **365**, 617–620.

Szucs, J. M. 1991a. Selection at a diallelic autosomal locus in a dioecious population, *J. Math. Biol.* **29**, 693–713.

Szucs, J. M. 1991b. Selection-mutation at a diallelic autosomal locus in a dioecious population, *J. Math. Biol.* **30**, 1–14.

Thomas, W. R., Pomerantz, M. J., and Gilpin, M. E. 1980. Chaos, asymmetric growth and group selection for dynamic stability, *Ecology* **61**, 1312–1320.

Turchin, P., and Taylor, A. D. 1992. Complex dynamics in ecological time series, *Ecology* **73**, 289–305.