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Phenotypic variation, sexual reproduction and evolutionary population dynamics

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Abstract

I studied the effects of introducing phenotypic variation into a well-known single species model for a population with discrete, non-overlapping generations. The phenotypes differed in their dynamic behaviour. The analysis was made under the assumption that the population was in an evolutionary stable state. Differences in the timing of the competitive impacts of the phenotypes on each other had a strong simplifying effect on the dynamics. This result could also be applied to competition between species. The effect of sexual reproduction on the dynamics of the population was analysed by assuming the simplest genetic model of one locus with two alleles. Sexual reproduction made the system much more stable in the (mathematical) sense that the number of attractors was reduced and their basins of attraction enlarged. In a dominant system sex tended to increase the frequency of the recessive allele, and in an overdominant system it induced gene frequencies of 1/2. Whether the attractors in the dominant system tended to be simpler or more complex than the attractors in the asexual system depended on the phenotype of the recessive homozygote. The overdominant sexual system tended to have simpler dynamics than the corresponding asexual population. A 2-locus model was used to study whether sexuals can invade an asexual population and vice versa. One locus coded for sexual and asexual reproduction, while the other coded for the dynamics. Enhanced stability through sexual reproduction seemed to be the reason why there was a clear asymmetry favouring sex in this evolutionary context.

Introduction

Deterministic ecological models can give rise to very complex dynamical behaviour. Chaos occurs as a rule, even in mathematically very simple models (May,

1974, 1976). For example, consider the following model for a population with discrete, non-overlapping generations (Hassell, 1975):

$$N_{t+1} = \lambda N_t (1 + aN_t)^{-b} . \tag{1}$$

Here N_t is the density of the species at time t, λ is the intrinsic growth rate of the population, and the term $(1 + aN_t)^{-b}$ describes the density dependence. The parameter $b \geq 1$ can be seen as describing the type of competition that causes the density dependence: $b \approx 1$ corresponds to contest competition, while $b \gg 1$ corresponds to scramble competition (Hassell, 1975). The parameter $a > 0$ measures how well the organisms can cope with the environment. It influences the equilibrium density N^*, which is defined by being the non-zero fixed point of eq. (1), i.e., by $\lambda N^*(1 + aN^*)^{-b} = N^*$. Thus

$$N^* = \frac{\lambda^{1/b} - 1}{a} . \tag{2}$$

If we write $N_{t+1} = f(N_t)$, the dynamical properties of the system in the neighborhood of the equilibrium N^* are determined by $|df/dN\,(N^*)|$ (May and Oster, 1976). We have

$$\begin{aligned} \frac{df}{dN}(N^*) &= 1 - b(1 - \lambda^{-1/b}) \\ &= 1 - \tilde{b}, \end{aligned} \tag{3}$$

where $\tilde{b} = b(1 - \lambda^{-1/b}) > 0$. Then, if $\tilde{b} < 2$ the equilibrium N^* is locally stable. If $\tilde{b}$ increases above 2 the system exhibits the familiar bifurcation behaviour, first giving rise to a stable 2-cycle, then to a stable 4-cycle and more generally to a stable 2^n-cycle as $\tilde{b}$ further increases. As $\tilde{b}$ reaches a critical value the system starts to exhibit chaotic behaviour (May and Oster, 1976).

In model (1) all individuals are assumed to behave in exactly the same way. In this paper I study the consequences of relaxing this assumption by introducing phenotypic and genotypic variation into model (1). It is assumed that the phenotypes differ in their dynamic behaviour. Although variability complicates the model, the dynamical properties of the system can be simplified.

In section 2 I describe a general criterion for a mutant phenotype to be able to invade a resident population. This criterion was introduced by Metz et al. (1992). It follows from this condition that the system considered is subject to a form of K-selection. This leads to the notion of evolutionary stability for model (1).

In section 3 I study the dynamical consequences of variability at the evolutionary stable state. I show that a difference in the timing of the competitive impacts of different phenotypes on each other has a strong stabilising effect. May et al. (1981) realized, in the framework of host-parasitoid interactions, that whether density dependence acts before or after parasitism can be crucial for dynamical properties. Section 3 describes another situation where the timing of interactions plays an essential role.

In sections 4 and 5 I study the influence of sexual reproduction on the dynamics of competition between phenotypes, and some evolutionary consequences. I assume the simplest genetical model wtih one locus and two alleles. To compare the sexual with an asexual system with two phenotypes, I also assume complete dominance, so that the three genotypes produce two phenotypes with different dynamics. The general effect of sex is that the dynamics of the system become much more uniform. Whereas in the asexual system many different attractors coexist, so that many different dynamic behaviours are possible depending on initial conditions, sex reduces the number of attractors and enlarges their basins of attraction. In this mathematical sense the sexual system is much more stable. Whether the few remaining asymptotic dynamic behaviours in the sexual population tend to be simpler or more complex than those of the asexuals depends on the dominance behaviour of the alleles.

I next use a 2-locus model to analyse whether sexuals can invade an asexual population and vice versa. In this model one locus codes for sexual and asexual reproduction, while the other codes for the dynamics. In general, no resident population exhibiting a stable equilibrium can be invaded, and there is a clear tendency for the system with the simpler dynamics to be able to invade. Thus, in this model simple dynamics are favoured by individual selection. However, only invasion of sexuals can lead to simpler dynamics for the whole system.

Concerning the evolution of sex, in this model there is a clear asymmetry in invasion ability in favour of sexual populations with overdominance. In this case the sexuals can invade every asexual system that does not exhibit a stable equilibrium, whereas asexuals can never invade an overdominent sexual system. Although the results are derived from a very simple model, they suggest that dynamical considerations might be important when studying the evolution of sex. In the system considered, individuals that reproduce sexually have an evolutionary advantage for purely dynamical reasons. This advantage seems to be that sexual systems are more stable in the sense that they have fewer attractors, each with a large set of initial conditions that are eventually drawn to it.

Although a substantial amount of research has been devoted to the study of the evolution of sex (e.g. Williams, 1975; Maynard Smith, 1978; Bell, 1982; Stearns, 1987; Michod and Levin, 1988), very rarely have there been attempts to analyse theoretically the consequences of sexual reproduction on population dynamics. Some studies have considered sexual reproduction in host-parasite models (e.g. May and Anderson, 1983), but how this might affect population dynamics as compared to asexual systems was mentioned only marginally and not considered to be interesting in its own right. The only studies I am aware of that explicitly address the issue are Koella (1988) and Doebeli and Koella (1994). These papers suggest that sexual reproduction can simplify population dynamics, which implies that sexual populations exhibit fewer fluctuations and are therefore less prone to extinction due to chance events. This fits into one line of argument suggesting a general scarcity of complex dynamics in natural systems (Thomas et al., 1980; Berryman and Millstein, 1989), and might be one of the evolutionary advantages of sex. However, the argument is based on group selection. After setting the stage by

introducing phenotypic variability at an evolutionarily stable state, this paper explores the consequences of variability on the dynamics of a difference equation model. It examines the effects of sexual reproduction, and it addresses the question of whether these effects can confer an evolutionary advantage for sex based on individual selection.

2. Evolutionary stability

Following Metz et al. (1992), I write model (1) in the form

$$N_{t+1} = w(N_t) \cdot N_t,$$

where $w(N_t) = \lambda(1 + aN_t)^{-b}$ is the fitness function. This function calculates the reproductive output per individual for a given density. In an evolutionary context N_t is interpreted as a component of the environment experienced by the population at time t, and the parameters λ, a, b are properties of a particular phenotype (see Metz et al., 1992, Box 2). Consider a mutant phenotype N_m with corresponding parameters λ_m, a_m, b_m. If the mutant is rare, the relevant component of the environment is the resident density N_t, hence the mutant initially obeys the equation

$$N_{m,t+1} = w_m(N_t) \cdot N_{m,t},$$

where $w_m(N_t) = \lambda_m(1 + a_m N_t)^{-b_m}$. I assume here that the competitive impact of the two phenotypes on each other and on themselves is the same. This is reasonable since they belong to the same species. The situation is different if the model describes the interaction of separate species. I will return to this question at the end of section 3. Invasion is now possible if the geometric mean fitness of the mutant is >1, which is the same as requiring that the Lyapunov exponent

$$s_m := \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log w_m(N_t) \tag{4}$$

is positive (Metz et al., 1992). Suppose for the moment that the resident phenotype has stable equilibrium dynamics. Then, assuming that the resident has already reached this equilibrium, we can replace N_t by the equilibrium density N^* in (4). Equation (4) now becomes

$$\begin{aligned} s_m &= \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log w_m(N^*) \\ &= \lim_{T\to\infty} \frac{1}{T} \cdot T \cdot \log w_m(N^*) \\ &= \log w_m(N^*) \\ &= \log \lambda_m(1 + a_m N^*)^{-b_m}. \end{aligned}$$

Thus the condition for initial increase in this case is

$$\lambda_m(1 + a_m N^*)^{-b_m} > 1 ,$$

which is easily seen to be equivalent to

$$N_m^* > N^* ,$$

i.e., to the condition that the equilibrium density of the mutant be larger than that of the resident.

While I have assumed that the resident equilibrium N^* is stable, there is no condition for stability imposed on the mutant equilibrium N_m^*. Therefore, if invasion is successful, interesting dynamical phenomena can occur. For example, I showed in Doebeli (1994) that a successful invasion of a mutant with complex dynamics into a resident population with stable equilibrium dynamics can lead to intermittent chaos. In this form of chaotic motion, the total population density stays close to the equilibrium density of the resident for a long time, during which the invading population gradually builds up and the resident's density correspondingly decreases. Then, the complex dynamics of the invader causes a short series of erratic outbursts and subsequent crashes, after which its density returns to very low values, and the total density is again stable for a long time.

If the resident phenotype does not have stable equilibrium dynamics, the invasion criterion can in general not be stated as neatly as before. However, note that the parameter a does not influence the dynamical properties of model (1), i.e., it does not appear in eq. (3). If one is only interested in dynamical properties of the system, one can therefore assume that this parameter is the same for the resident and the mutant. This leads to a simplification of the criterion (4), as was shown by Metz. et al. (1992). To derive the simplification, note that the Lyapunov exponent of the resident must be 0,

$$s = \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log[w(N_t)] = 0 ,$$

because the resident phenotype is not increasing on average and persists through time. Hence

$$0 = \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log\left[\frac{\lambda}{1 + aN_t)^b}\right]$$

$$= \log\lambda - b \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log[1 + aN_t] ,$$

and thus

$$\lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log[1 + aN_t] = \frac{\log\lambda}{b} .$$

Since we assume $a_m = a$, eq. (4) now becomes

$$s_m = \lim_{T\to\infty} \frac{1}{T} \sum_{t=0}^{T-1} \log\left[\frac{\lambda_m}{(1+aN_t)^{b_m}}\right]$$
$$= \log \lambda_m - b_m \lim_{T\to\infty} \sum_{t=0}^{T-1} \log[1+aN_t]$$
$$= \log \lambda_m - b_m \frac{\log \lambda}{b}.$$

Therefore, the condition $s_m > 0$ for invasion translates to

$$\frac{\log \lambda_m}{b_m} > \frac{\log \lambda}{b}. \tag{5}$$

Note that, since $a_m = a$, taking exponentials conditon (5) is equivalent to

$$\frac{\lambda_m^{1/b_m} - 1}{a_m} > \frac{\lambda^{1/b} - 1}{a}.$$

Thus invasion is again possible if the mutant has a larger equilibrium density than the resident, but now it is unnecessary to assume that the resident has a stable equilibrium. This means that the system considered is subject to a form of K-selection. Higher equilibrium densities confer a competitive advantage, regardless of whether these equilibria are stable or not. K-selection is a consequence of the simple form of the model and is typical for 1-dimensional difference equations. For example, it can be shown along the above line that selection on the parameters of the well-known Ricker equation $N_{t+1} = \lambda N_t \exp(-aN_t)$ always maximizes the equilibrium density $\ln \lambda/a$ (Gatto, 1993). More complicated models, including for instance stochastic effects, induce more complex selection pressures, under which a higher K-value is not always favoured, see e.g. Heckel and Roughgarden (1980), Turelli and Petry (1980).

It must again be emphasised that invasion of a mutant does not necessarily lead to the extinction of the resident. Thus coexistence of phenotypes with different equilibrium densities (i.e., different K-values) is possible. However, if the differences between the equilibrium densities of the phenotypes are too large, coexistence is impossible. More precisely, suppose that one is given two phenotypes N_1 and N_2, where we can assume that $N_1^* \geq N_2^*$. Then there is a number $\epsilon \geq 0$ such that if $N_2^* < N_1^* - \epsilon$, then the phenotype N_2 cannot coexist with N_1 and goes extinct if it competes with N_1. For example, if N_1 has a stable equilibrium then $\epsilon = 0$: every phenotype with a lower equilibrium, stable or not, is outcompeted by N_1. In general, ϵ is not 0 but still very small, typically at most a few percent of the equilibrium density N_1^*, say $\epsilon/N_1^* \sim 0.02$. This can easily be seen via numerical simulations. To do the simulations, one assumes that there are two phenotypes, one of them with a given equilibrium density N_1^*. For various values of eq. (3) for the given equilibrium, i.e., for various dynamical behaviours of the corresponding phenotype, one then checks for which equilibrium densities of the second phenotype coexistence is possible, again testing a whole range of dynamical behaviours for this

phenotype. An analogous procedure can be performed with more than two phenotypes, yielding the same qualitative result: coexistence is only possible if the equilibrium densities of the phenotypes are very close to each other.

In practice, evolution tends to maximise the equilibrium densities of the phenotypes according to biological constraints. The endpoint of evolution in a given system depends on the tradeoffs between the parameters λ, a and b. All phenotypes present in the population in an evolutionary stable state will have approximately the same equilibrium densities, because otherwise they could not coexist. However, a given equilibrium density can be achieved under any dynamic regime, as can be seen from eqs. (2) and (3) above. Therefore, the phenotypes present in an evolutionary stable state can have very different dynamical properties. In fact, it is the dynamical behaviour which defines the phenotype. It follows that the dynamical behaviour of the whole population can depend very much on the relative proportions of the phenotypes. If a phenotype with simple dynamics is abundant, its dynamics tend to stabilize the system; if a phenotype with complex dynamics is abundant, the whole population also tends to have complex dynamics. Thus the dynamics of the system depend on the initial conditions, i.e., on the distribution of the different phenotypes in an evolutionary stable state. In other words, multiple attractors may occur in a system with several phenotypes. Although this might seem unnatural for an evolutionarily stable system, it is not at all unusual from a dynamical point of view: 'The coexistence of multiple attractors is always a possibility in nonlinear models, and is likely to be common for density-dependent [matrix] population models' (Caswell, 1989, p. 253). I show in section 4 that the main effect of sexual reproduction is to reduce the number of attractors and thus make the dynamics more uniform.

For the remainder of the paper I make the simplifying assumption that all the phenotypes in the evolutionarily stable state have exactly the same equilibrium densities, instead of assuming coexistence at the evolutionarily stable state, which would only imply approximately the same equilibrium densities of the phenotypes present. Under the assumption of equal equilibria, some results can be made more transparent with analytical arguments. The justification for this assumption is that the qualitative nature of the results does not change if it is relaxed to imply coexistence of the phenotypes. Such phenotypes would still have almost the same equilibrium densities, as was explained above, and qualitatively the same general picture would emerge as when all phenotypes have the same equilibrium densities.

3. Effects of variability on the dynamics

In this section I assume that the model population consists of several phenotypes that reproduce asexually and are evolutionarily stable, i.e., that all have the same equilibrium densities (see section 2).

For simplicity, I assume first that there are two phenotypes N_1 and N_2. The density dependence for each phenotype is assumed to be induced by the total

population density. Then the new model has the following form:

$$N_{1,t+1} = \lambda_1 N_{1,t}(1 + aN_t)^{-b_1} = f_1(N_{1,t}, N_{2,t})$$
$$N_{2,t+1} = \lambda_2 N_{2,t}(1 + aN_t)^{-b_2} = f_2(N_{1,t}, N_{2,t}), \quad (6)$$

where $N_t = N_{1,t} + N_{2,t}$ is the total population density at time t. Since both phenotypes have the same equilibrium density, an equilibrium of system (6) is a state in which the total density N^* is equal to the equilibrium densities of the two phenotypes. Every combination of densities $\bar{N}_1$ and $\bar{N}_2$ of the phenotypes such that $\bar{N}_1 + \bar{N}_2 = N^*$ defines an equilibrium state. Consider the Jacobian of the system at such a point, i.e., the linearized form of (6) given by the matrix of partial derivatives $\partial f_i/\partial N_j(\bar{N}_1, \bar{N}_2)$. Let $\tilde{b}_1$ and $\tilde{b}_2$ be defined as in eq. (3), i.e., $\tilde{b}_1$ and $\tilde{b}_2$ determine the dynamics of the phenotypes. Let $\gamma_1 := (\bar{N}_1/N^*)\tilde{b}_1$ and $\gamma_2 := (\bar{N}_2/N^*)\tilde{b}_2$. By definition, γ_1 and γ_2 are the parameters determining the dynamics of the two phenotypes, weighted according to their relative abundance in the equilibrium. Then the Jacobian at $(\bar{N}_1, \bar{N}_2)$ has the form

$$\begin{pmatrix} 1-\gamma_1 & -\gamma_1 \\ -\gamma_2 & 1-\gamma_2 \end{pmatrix}.$$

The eigenvalues of this matrix are 1 and

$$\zeta := 1 - \gamma_1 - \gamma_2 . \quad (7)$$

The eigenvalue 1 corresponds to the fact that, if $0 \le s \le \bar{N}_1$, an initial change of the equilibrium point $(\bar{N}_1, \bar{N}_2)$ to the new equilibrium $(\bar{N}_1 - s, \bar{N}_2 + s)$ persists through time. The dynamics in the neighborhood of $(\bar{N}_1, \bar{N}_2)$ are therefore determined by $|\zeta| = |1 - \gamma_1 - \gamma_2|$. This means that the dynamics of the whole system, if it is perturbed from an equilibrium, are determined by the dynamical properties of the phenotypes according to their relative abundance at this equilibrium (Fig. 1). Consequently, depending on initial conditions, many different dynamical behaviours may be observed in the same system. In general, if there are r phenotypes, the behaviour around an equilibrium point $(\bar{N}_1, \ldots, \bar{N}_r)$ with $\bar{N}_1 + \cdots + \bar{N}_r = N^*$ is determined analogously. Now there are $r - 1$ eigenvalues of the Jacobian which are equal to 1, corresponding to free movement on the hyperplane in N^r-space given by $N_1 + \cdots + N_r = N^*$. The last eigenvalue ζ is given by $\zeta = 1 - \gamma_1 - \cdots - \gamma_r$, where the $\gamma_i := (\bar{N}_i/N^*)\tilde{b}_i$ are again the parameters $\tilde{b}_i$ weighted according to the position of the equilibrium point on the hyperplane mentioned above. Again, the dynamics of the system around an equilibrium point depend on the relative abundance of the different phenotypes at this equilibrium, and the system typically has many different attractors.

It was pointed out by May et al. (1981) that the timing of density effects and parasitism in certain host-parasitoid systems can have a profound effect on the dynamics of the system. A similar observation was made by Doebeli (1995). By analogy, I now analyse the system considered so far when the timings of the competitive impacts of the phenotypes on each other are asymmetrical. To be more precise, consider again two phenotypes N_1 and N_2. At time t they have densities $N_{1,t}$

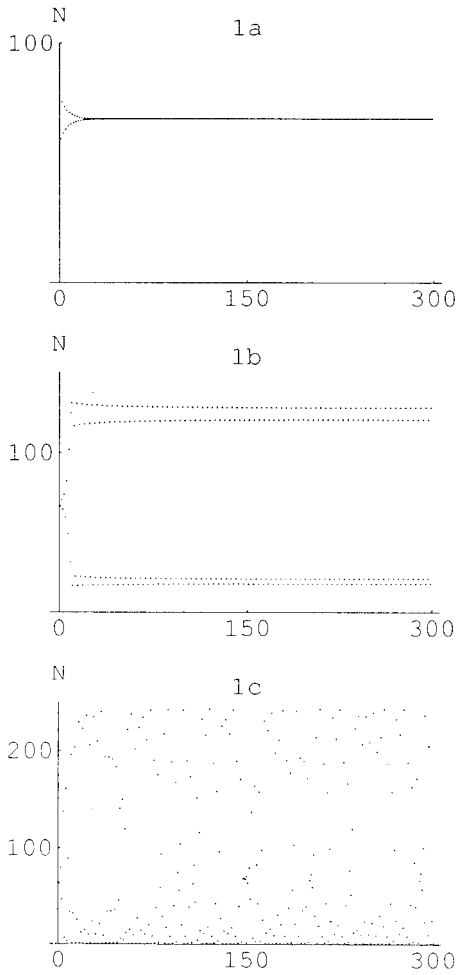

Fig. 1. The behaviour of the asexual system after a perturbation from an equilibrium point $(\bar{N}_1, \bar{N}_2)$ with $\bar{N}_1 + \bar{N}_2 = N^*$ (see text). The parameter values are $a = 0.003$, $b_1 = 10$, $\tilde{b}_1 = 1.7$, $b_2 = 20$ and $\tilde{b}_2 = 3.4$, hence $N^* \sim 68.3$. The phenotype represented by N_1 has simple dynamics, while the phenotype represented by N_2 has complex dynamics. The total density of the population is plotted against time. 1a: $\bar{N}_1 = 0.9\ N^*$, $\bar{N}_2 = 0.1\ N^*$. This equilibrium is locally stable. 1b: $\bar{N}_1 = \bar{N}_2 = 0.5\ N^*$. Locally unstable equilibrium; the modulus of the dominant eigenvalue of the Jacobian is >1, and the system exhibits a locally stable 4-cycle 1c: $\bar{N}_1 = 0.05\ N^*$, $\bar{N}_2 = 0.95\ N^*$. As in 1b, but now the dominant eigenvalue of the Jacobian codes for chaotic motion.

and $N_{2,t}$. In the model used so far it is assumed that the relevant densities to determine the reproductive output during the next time unit from t to $t+1$ are $N_{1,t}$ and $N_{2,t}$ for both phenotypes. Now suppose that phenotype 1 reproduces first, and that its juveniles have the same impact on phenotype 2 as the adults. Then the reproductive output of phenotype 1 is again determined by $N_{1,t}$ and $N_{2,t}$, but the

output of phenotype 2 is now determined by $N_{1,t+1}$ and $N_{2,t}$, since the new generation of phenotype 1 is already present at the time of reproduction of phenotype 2. The model corresponding to this new situation has the following form:

$$N_{1,t+1} = \lambda_1 N_{1,t}\{1 + a(N_{1,t} + N_{2,t})\}^{-b_1}$$
$$N_{2,t+1} = \lambda_2 N_{2,t}\{1 + a(N_{1,t+1} + N_{2,t})\}^{-b_2}$$
$$= \lambda_2 N_{2,t}\{1 + a(\lambda_1 N_{1,t}[1 + a(N_{1,t} + N_{2,t})]^{-b_1} + N_{2,t})\}^{-b_2}.$$

This system has the same equilibrium points as the previous: they are the points $(\bar{N}_1, \bar{N}_2)$ with $\bar{N}_1 + \bar{N}_2 = N^*$. Again, one of the eigenvalues of the Jacobian at such a point is 1. The second is

$$\zeta = 1 - \gamma_1 - \gamma_2 + \gamma_1\gamma_2,$$

where the $\gamma_i = (\bar{N}_i/N^*)\tilde{b}_i$ are defined as above. Since γ_1 and γ_2 are both ≥ 0, it is immediately clear that such a system is much more stable than the previous one: the range of parameters $\tilde{b}_1$, $\tilde{b}_2$ and of equilibrium points $(\bar{N}_1, \bar{N}_2)$ which results in local stability is much larger. In particular, in this new setting both phenotypes can have chaotic dynamics when alone, yet every equilibrium point which is sufficiently far from the extreme points $(N^*, 0)$ and $(0, N^*)$ is locally stable. The same general conclusion holds if more than two phenotypes are present. In fact, the more phenotypes there are, the more chaotic the single types are allowed to be for local stability to still hold over most of the equilibrium plane given by $N_1 + \cdots + N_r = N^*$ (when r types are present). Again, I assume that the times of reproduction are mutually different for all phenotypes present. After possible reordering, this leads to the following system:

$$N_{1,t+1} = \lambda_1 N_{1,t}\{1 + a(N_{1,t} + \cdots + N_{r,t})\}^{-b_1}$$
$$\vdots$$
$$N_{i,t+1} = \lambda_i N_{i,t}\{1 + a(N_{1,t+1} + \cdots + N_{i-1,t+1} + N_{i,t} + \cdots + N_{r,t})\}^{-b_i}$$
$$\vdots$$
$$N_{r,t+1} = \lambda_r N_{r,t}\{1 + a(N_{1,t+1} + \cdots + N_{r-1,t+1} + N_{r,t})\}^{-b_r}.$$

Again there are $r - 1$ eigenvalues 1 for the Jacobian at an equilibrium point. The expression for the last eigenvalue ζ is as follows: Let $\gamma_i = (\bar{N}_i/N^*)\tilde{b}_i$, $i = 1, \ldots, r$, be as above, and let σ_l, $l = 1, \ldots, r$, denote the lth elementary symmetric function in $\gamma_1, \ldots, \gamma_r$, i.e.:

$$\sigma_1(\gamma_1, \ldots, \gamma_r) = \gamma_1 + \cdots + \gamma_r$$
$$\vdots$$
$$\sigma_j(\gamma_1, \ldots, \gamma_r) = \sum_{1 \leq i_1 < i_2 < \cdots < i_j \leq r} \gamma_{i_1} \cdot \gamma_{i_2} \cdots \gamma_{i_j}$$
$$\vdots$$
$$\sigma_r(\gamma_1, \ldots, \gamma_r) = \gamma_1 \cdot \gamma_2 \cdots \gamma_r.$$

Then one can prove:

$$\zeta = 1 - \sum_{l=1}^{r} (-1)^{l-1} \sigma_l(\gamma_1, \ldots, \gamma_r) .$$

For example, if $r = 3$, then $\zeta = 1 - \gamma_1 - \gamma_2 - \gamma_3 + \gamma_1\gamma_2 + \gamma_1\gamma_3 + \gamma_2\gamma_3 - \gamma_1\gamma_2\gamma_3$. In addition to the fact that the stabilising effect described above is greater when more phenotypes are present, numerical simulations show that the effect is also present when the ordering of the different times of reproduction changes randomly every season. I have also verified numerically that if the assumption of different impact times holds only for a subset (of magnitude ≥ 2) of the phenotypes present, then the effect is still present, although weaker, as one would expect.

The model has so far assumed asexual reproduction, and could thus also serve to describe competition between different species. However, in this situation it is less likely that the impact of a competitor would be equal to the density dependence caused by members of the same species. To be more realistic, one should introduce competition coefficients α_i so that the difference equation for species j in the traditional model now becomes

$$N_{j,t+1} = \lambda_j N_{j,t} \left\{ 1 + a\left(N_{j,t} + \sum_{i \neq j} \alpha_i N_{i,t} \right) \right\}^{-b_j} .$$

Hassell and Comins (1976) have studied such models. They derived the conditions under which coexistence is possible, and further described when such coexistence is stable. One can introduce the effect of different impact times as above into this model. Along the same lines as before, it is possible to show analytically and numerically that this is a strong stabilising force in this setting as well, even enabling coexistence under conditions when it was not possible in the traditional model.

4. Effects of sexual reproduction on the dynamics

I now describe the effects of introducing sexual reproduction in model (6). I assume the simplest case of one locus with two alleles A and a. Let $w_{AA}(N)$, $w_{Aa}(N)$ and $w_{aa}(N)$ be the fitness functions of the genotypes. By assumption, these functions have the general form $\lambda(1 + aN)^{-b}$, where N is the total population density. Let p_t be the frequency of allele A at time t. Assuming random mating, to describe the new system it is enough to know the frequency of allele A, p_t, and the total population density N_t, at each time t (Roughgarden, 1979, chap. 3). The densities of the genotypes AA, Aa and aa are then given by $p_t^2 N_t$, $2p_t(1-p_t)N_t$ and $(1-p_t)^2 N_t$ respectively. Let

$$\bar{w} = p_t^2 w_{AA} + 2p_t(1-p_t)w_{Aa} + (1-p_t)^2 w_{aa}$$

be the mean fitness. (Here, as in the following section, I have suppressed the dependency of the fitness functions on the total density for notational convenience.) Then the two equations describing the system are the following (Roughgarden,

1979, chap. 3):

$$p_{t+1} = \frac{p_t(p_t w_{AA} + (1-p_t)w_{Aa})}{\bar{w}}$$

$$N_{t+1} = N_t \bar{w} . \tag{8}$$

This is again a 2-dimensional system. If N^* is the equilibrium density, then for every $p^* \in [0, 1]$, (p^*, N^*) is an equilibrium point of system (8). To determine the dynamics around such a point, one must calculate the eigenvalues of the Jacobian at this point. One of these will be 1 again, corresponding to the fact that p^* can be replaced by any other value in the interval [0, 1], yet still yield an equilibrium point. To calculate the other eigenvalue ζ is straightforward: If $\tilde{b}_{AA}$, $\tilde{b}_{Aa}$ and $\tilde{b}_{aa}$ are the parameters determining the dynamics of each phenotype (cf. eq. (3)), then

$$\zeta = 1 - p^{*2}\tilde{b}_{AA} - 2p^*(1-p^*)\tilde{b}_{Aa} - (1-p^*)^2\tilde{b}_{aa} . \tag{9}$$

In essence, this is the same formula as (7): again the parameters determining the fitnesses of the genotypes appear weighted according to their relative abundance in the total population. For example, if the genotypes produce only two phenotypes, say $w_{Aa} \equiv w_{aa}$, then (9) reduces to $\zeta = 1 - p^{*2}\tilde{b}_{AA} - (1-p^{*2})\tilde{b}_{aa}$, which is just eq. (7) for the two phenotypes AA and $Aa = aa$. So why should there be any difference in stability?

The results described below were obtained from numerical simulations. For the simulations, I assumed, as always, that all genotypes had the same equilibrium density, and that they differed in their dynamical behaviour. The sexual system (8) was primarily compared to an asexual system with two phenotypes, that is, I assumed that the three genotypes in the sexual system produced only two phenotypes. For a given dynamical behaviour of one of the phenotypes, I then examined the dynamics of system (8) for many different dynamical behaviours of the second phenotype. Thus, in the 2-dimensional parameter space whose axes are given by the moduli of eq. (3) for the two phenotypes, I examined a lattice of points ranging in each direction from values coding for stable equilibria to values coding for chaos. Since the three genotypes produced two phenotypes, two genetic scenarios had to be considered: dominance, where the heterozygote produces the same phenotype as one of the homozygotes, and overdominance, where the two homozygotes produce the same phenotypes. I will turn briefly to the case where the sexual system has three phenotypes at the end of this section.

Dominance

In this case the main effect of sexual reproduction is to increase the frequency of the recessive allele. If the sexual system is perturbed away from an equilibrium point in which the density of the recessive homozygote is low, it increases this density until it finally behaves as if it would have been perturbed from an equilibrium with a high frequency of the recessive allele. Thus, sexual reproduction destroys the stability of most of the attractors in the asexual system: only a few

asymptotic behaviours are possible, and the remaining attractors have much larger basins of attraction. Whether these attractors are simpler or more complex than the ones typically found in the asexual system depends on the dynamical properties of the recessive homozygote: Since the system eventually moves on an attractor around an equilibrium with a high density of this phenotype, its dynamics determine the dynamics of the whole sytem. The phenomenon is displayed in Fig. 2.

Overdominance

With this assumption the sexual system has a very strong tendency to equalise gene frequencies at 1/2: except for extreme equilibria with either a very low or a very high frequency p^*, when perturbed away from them the system finally behaves as if it had been perturbed from the midpoint equilibrium with $p^* = 1/2$ (Fig. 2). Again, sexual reproduction greatly reduces the number of attractors. Typically, both simpler and more complex attractors can be found in the asexual system, so that whether the sexual system has simpler dynamics than the asexual system depends on the initial conditions in the latter. Note, however, that if both systems are perturbed away from the midpoint equilibrium in which both phenotypes are equally abundant (i.e. $\bar{N}_1 = \bar{N}_2 = N^*/2$ respectively $p^* = 1/2$), they behave differently: the sexual system then typically has simpler dynamics (see Figs. 1 and 2). In this case sexual reproduction leads to simpler dynamics.

In conclusion, the sexual differs from the asexual system in that it can move along the line of equilibrium points in the 2-dimensional system considered. Sexual reproduction induces shifts in the set of equilibrium points, thereby reducing the number of attractors and hence possible asymptotic behaviours. A shift can occur from an unstable to a stable equilibrium. When perturbed away from the unstable equilibrium, the sexual system approaches the stable equilibrium, whereas the asexual system remains fluctuating (Fig. 2). On the other hand, shifts away from stable equilibria never occur (by definition of stability). However, shifts from unstable to even more unstable equilibria (i.e. equilibria with larger dominant eigenvalue of the Jacobian matrix) are possible.

The results described hold for sexual systems with two phenotypes. They did not change qualitatively if this assumption was relaxed by introducing a difference between the genotypes previously assumed to be equal, as long as this difference was small compared to the original difference between the phenotypes. The next step would be to do the full numerical analysis for sexual systems with three phenotypes. They should be compared to asexual systems with three phenotypes, which would then have a plane of equilibrium points instead of a line. Although I have not done the analysis as rigorously as for the case with two phenotypes, the basic difference between the sexual and asexual systems again seems to be that the sexual systems move along the plane of equilibria, whereas the asexual systems always behave according to the equilibrium point from which they were perturbed away. For example, in an additive system, where the heterozygote has a dynamic complexity that is intermediate between the two homozygotes, one would expect, on the basis of the results for two phenotypes, that the sexual system would always tend to gene frequencies of 1/2, regardless of the equilibrium from which it was

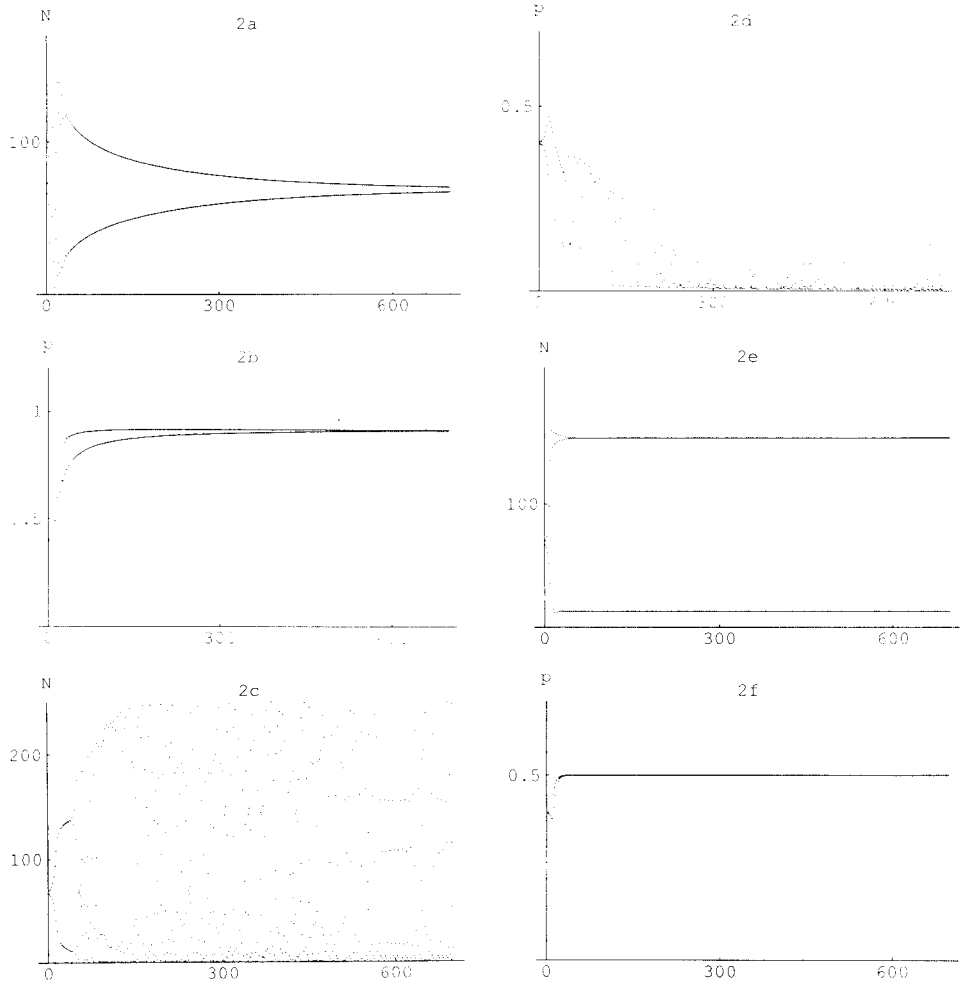

Fig. 2. The behaviour of the sexual model is shown for the same parameter values as in Fig. 1. In 2a–2d the allele A is dominant. In 2a the phenotype produced by the genotype AA has simple dynamics (corresponding to phenotype N_1 for Fig. 1). Genotypes Aa and aa produce the same phenotype, which has complex dynamics (corresponding to phenotype N_2 for Fig. 1). When perturbed from the equilibrium point ($p^* = 0.4$, N^*) (which corresponds to the unstable equilibrium (0.16 N^*, 0.84 N^*) of the asexual system), the sexual system evolves to a stable equilibrium by increasing the frequency p of allele A. This increase is shown in 2b. In 2c the phenotype produced by AA has complex dynamics (the same as phenotype N_2 for Fig. 1), while the other phenotype, produced by genotypes Aa and aa, has simple dynamics (the same as phenotype N_1 for Fig. 1). When perturbed from the same equilibrium point as before the sexual system reacts by decreasing the frequency p, which is shown in 2d. This makes the dynamics of the system more complex, and the total density N starts to exhibit chaotic motion, see 2c. In 2e and 2f the behaviour of the sexual system is shown when the phenotype with the complex dynamics is produced by the heterozygote, while the homozygotes have simple dynamics. The frequency p develops to a value of 0.5, and the total density N behaves according to the dominant eigenvalue of the Jacobian at the equilibrium (0.5, N^*). In this case it moves on a 2-cycle. A similar picture can be seen if the

perturbed away. Indeed, this is what happens. Thus, for systems with three phenotypes the same phenomenon can be observed: sexual reproduction reduces the number of attractors, and thereby stabilizes the sytem in the sense of making its dynamic behaviour much more uniform than that of asexual systems.

5. Evolution of sex through population dynamics?

To compare the effects of sexual and asexual reproduction on the dynamics in an evolutionary context, I constructed a 2-locus model for a population whose genotypic fitnesses are described by difference equations as in the previous sections. In this model one locus determines whether to reproduce sexually or asexually, while the other locus determines the fitness function. Alleles at the fitness locus are denoted by A and a as before. Alleles at the other locus are denoted by B and b. I assume that B is dominant for sexual reproduction, hence that genotypes BB and Bb reproduce sexually, while bb reproduces asexually. The construction of the model follows Roughgarden (1979), chap. 8. As in his models, the census is made in the gametic phase, and I therefore assume that asexuals also produce 2 gametes each, which are then fused to form the zygote. The gamete types are AB, aB, Ab and ab. Let $N_{s,t}$ denote the density at time t of the sexual portion of the population (i.e., of individuals not homozygous for b), and let $N_{a,t} = N_t - N_{s,t}$ denote the density of the asexuals, where N_t is the total density as usual. The gamete frequencies in the sexuals at time t are $x_{1,t}$, $x_{2,t}$, $x^s_{3,t}$ and $x^s_{4,t}$ for AB, aB, Ab and Ab respectively, where the superscript s indicates that those are frequencies of gametes carrying the b allele in the sexual part of the population, i.e., coming from Bb-parents. Let x^a_3 denote the frequency of Ab-gametes in the asexual gamete pool coming from parents which are AA-homozygotes at the fitness locus. Let x^a_{34} denote the frequency of Ab-gametes coming from asexual parents which are heterozygous at the fitness locus. Then x^a_{34} is also the frequency of ab-gametes in the asexual gamete pool coming from fitness heterozygotes. Finally, let x^a_4 denote the frequency of ab-gametes in the asexuals coming from asexual parents which are aa-homozygotes. The following table summarizes these definitions:

Gamete type:	Frequency in sexual pool:	Frequency in asexual pool:
AB	x_1	0
aB	x_2	0
Ab	x^s_3	$x^a_3 + x^a_{34}$
ab	x^s_4	$x^a_4 + x^a_{34}$

As in section 4 let w_{AA}, w_{Aa} and w_{aa} denote the density dependent fitness functions of the different genotypes at the fitness locus. Again, these fitness

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heterozygote produces the simple and the homozygotes the complex dynamics. Note that the behaviour of the asexual system, when perturbed from the midpoint $((N^*/2), (N^*/2))$ (which corresponds to the equilibrium $(0.5, N^*)$ of the sexual system), is more complex than that of the sexuals, see Fig. 1b.

functions all have the general form $\lambda(1+aN_t)^{-b}$, where N_t is the total density at time t. The following formulas for the densities of the sexual and the asexual portions of the population density are now straightforward, where for notational convenience I have suppressed time dependence of the gamete frequencies:

$$\begin{aligned} N_{s,t+1} &= \{w_{AA}(x_1^2 + 2x_1x_3^s) + 2w_{Aa}(x_1x_2 + x_2x_3^s + x_1x_4^s) \\ &\quad + w_{aa}(x_2^2 + 2x_2x_4^s)\}N_{s,t} \\ N_{a,t+1} &= \{w_{AA}(x_3^s)^2 + w_{aa}(x_4^s)^2 + w_{Aa}2x_3^sx_4^s\}N_{s,t} \\ &\quad + \{w_{AA}x_3^a + w_{Aa}2x_{34}^a + w_{aa}x_4^a\}N_{a,t}\,. \end{aligned}$$

The new gamete frequencies can be easily obtained; the calculations are basically the same as in Roughgarden (1979), chap. 8. I therefore omit the details. In the following formulas I again suppress time dependence in the fitness functions and the frequencies occurring on the right hand side. The parameter r appearing in the formulas is the recombination factor and lies in the interval $[0, \frac{1}{2}]$.

$$x_{1,t+1} = \frac{N_{s,t}}{N_{s,t+1}}\{w_{AA}(x_1^2 + x_1x_3^s) + w_{Aa}(x_1x_2 + x_1x_4^s(1-r) + x_2x_3^sr)\}$$

$$x_{2,t+1} = \frac{N_{s,t}}{N_{s,t+1}}\{w_{Aa}(x_1x_2 + x_1x_4^sr + x_2x_3^s(1-r)) + w_{aa}(x_2^2 + x_2x_4^s)\}$$

$$x_{3,t+1}^s = \frac{N_{s,t}}{N_{s,t+1}}\{w_{AA}x_1x_3^s + w_{Aa}(x_1x_4^sr + x_2x_3^s(1-r))\}$$

$$x_{4,t+1}^s = \frac{N_{s,t}}{N_{s,t+1}}\{w_{aa}x_2x_4^s + w_{Aa}(x_1x_4^s(1-r) + x_2x_3^sr)\}$$

$$x_{3,t+1}^a = w_{AA}\left\{(x_3^s)^2\frac{N_{s,t}}{N_{a,t+1}} + x_3^a\frac{N_{a,t}}{N_{a,t+1}}\right\}$$

$$x_{4,t+1}^a = w_{aa}\left\{(x_4^s)^2\frac{N_{s,t}}{N_{a,t+1}} + x_4^a\frac{N_{a,t}}{N_{a,t+1}}\right\}$$

$$x_{34,t+1}^a = w_{Aa}\left\{x_3^sx_4^s\frac{N_{s,t}}{N_{a,t+1}} + x_{34}^a\frac{N_{a,t}}{N_{a,t+1}}\right\}.$$

For these formulas to hold it is of course necessary that the densities N_s and N_a are non-zero when they appear in the denominator.

I examined this sytem via numerical simulation. At the locus coding for the dynamics, I examined a similar range of parameters as in the previous section for systems with two phenotypes. For each set of parameters, I assumed a resident population that was homozygous at the sex locus, being either sexual (*BB*) or asexual (*bb*). After several hundred generations, I introduced a small amount of individuals carrying the mutant allele (*b* or *B*). Invasion was possible by definition if the mutant reached densities which were larger than the resident's density in some generations (recall that the system may fluctuate). I thus investigated whether under

the given circumstances asexuality can invade a sexual population and vice versa. For the results below the value of the recombination factor r was of no importance. Also, the results did not change qualitatively if the model was altered such that the gene b coding for sex was recessive.

The following general picture emerged. If the sexual system has dominance, the abilities for invasion are, on average, more or less symmetric. First of all, invasion is never possible if the resident exhibits stable equilibrium dynamics, regardless of whether it is sexual or asexual. Since a dominant sexual system always develops a high frequency of the recessive allele, such a system should be compared to an asexual system that is perturbed away from an equilibrium with a high density of the phenotype corresponding to the recessive homozygote. If this is done, invasion ability is symmetric. For a given set of parameters, either both or no resident system can be invaded. If the restriction above is dropped and the sexual system was compared with asexual systems perturbed away from other equilibria, invasion was typically possible for the population with the simpler dynamics. Suppose, for example, that the recessive homozygote has stable equilibrium dynamics. Then so does the sexual population (Fig. 2a), and hence it is never invaded. On the other hand, this sexual population can invade every asexual system that has been perturbed away from an unstable equilibrium (i.e., from an equilibrium in which the phenotype of the dominant homozygote, which is assumed to have complex dynamics, has a high density). This is shown in Fig. 3. If the recessive homozygote in the sexual population has complex dynamics, the opposite can be observed (Fig. 3c). Now it is possible that the asexuals have simpler dynamics and can therefore invade. Note, however, that invasion of asexuals was only observed when the resident sexual population was highly chaotic (Fig. 3c), and that invasion never resulted in simple dynamics for the whole system. If invasion of asexuals is possible, it always leads to coexistence, and in such systems the sexuals always dominate the dynamics. This is due to the fact the they have much more stable attractors.

Quite a different picture emerged with overdominance in the sexual system. A stable resident could still never be invaded, but now the sexuals were able to invade every unstable asexual population (Fig. 4). Moreover, the asexuals could never invade an asexual population. Thus, with overdominance, the sexual system seems to be much more stable in an evolutionary sense. This seems to happen for purely dynamical reasons. Sexual reproduction changes the dynamics of the system. The change consists in enhancing the stability of attractors, which confers an evolutionary advantage.

6. Discussion

This paper is about the effects of phenotypic variability on the dynamics of a population. I examined how these effects differ in sexual and asexual systems, and I compared sexual and asexual reproduction in the framework of evolutionary population dynamics by studying invasion scenarios. The basic model is a difference equation introduced by Hassell (1975). Variability is incorporated by assuming that

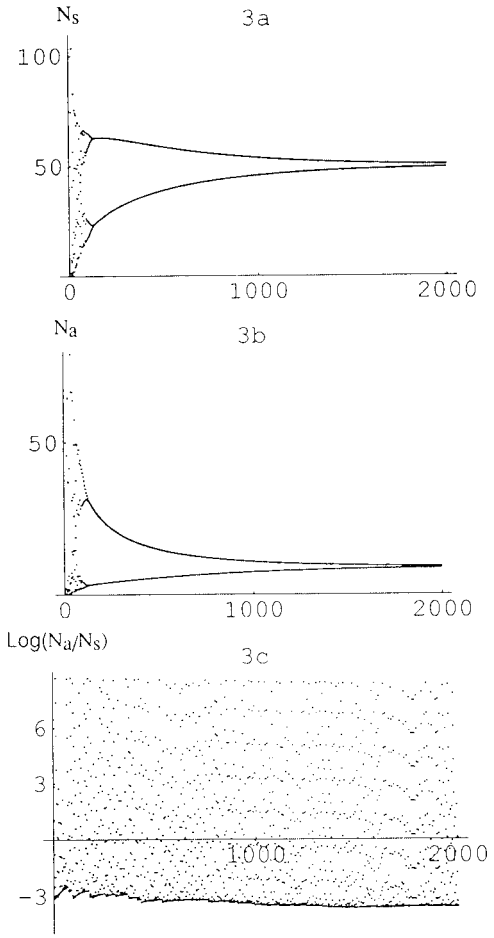

Fig. 3. In 3a the behaviour of the sexual fraction of the population is shown after a small amount of sexually reproducing individuals has been introduced into an asexual population that has been present for a few hundred generations. The asexuals alone exhibit chaotic motion corresponding to a perturbation from an equilibrium $(\bar{N}_1, \bar{N}_2)$ with $\bar{N}_2 = 0.9\,N^*$. Here genotype AA produces the phenotype N_1 of the asexual system with simple dynamics, while the other genotypes Aa and aa produce phenotype N_2 with complex dynamics. The labels N_s and N_a indicate the sexual and asexual parts of the population. The parameter values (see text for further explanation) are $a = 0.003$, $b_{AA} = 10$, $\tilde{b}_{AA} = 1.5$, $b_{Aa} = 40$ and $\tilde{b}_{Aa} = \tilde{b}_{aa} = 6$, hence $N^* \sim 58.8$. The simultaneous behaviour of the asexual part of the population is shown in Fig. 3b. In this situation the sexuals can invade, in the sense that they become more abundant than the asexuals. The sexuals cannot eliminate the asexuals, but the invasion transforms chaotic motion into stable equilibrium dynamics. (For a case where sexual invasion is followed by the extinction of the asexuals see Fig. 4b.) With the given parameter values the sexuals exhibit stable equilibrium dynamics when alone. Therefore the opposite invasion of the asexuals is not possible. In 3c a situation is depicted in which the asexuals can invade a sexual population. Since the system is highly chaotic with large fluctuations, the logarithm of the quotient N_a/N_s of the density of the asexuals divided by the density of the sexuals is shown. At times when this logarithm is positive, the asexuals are more abundant than the sexuals. Here the phenotype produced by the homozygote AA has complex dynamics, while the other

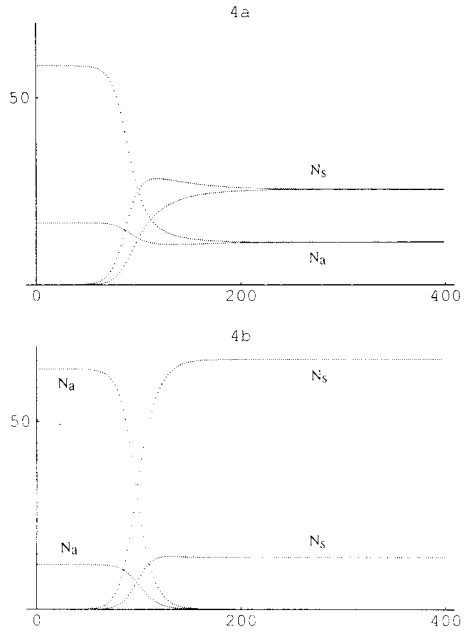

Fig. 4. In this figure two situations are shown in which a sexual population with overdominance can invade an asexual population. In both cases the heterozygote produces the simpler dynamics, but the situation is similar in the opposite case. 4a is similar to 3a: After introducing a small amount of sexually reproducing individuals into an asexual population exhibiting 2-cyclic behaviour, the system evolves to stable equilibrium dynamics such that the sexuals are more abundant than the asexuals. The labels N_s and N_a indicate the sexual and asexual portions of the population. The parameter values for this figure are $a = 0.003$, $b_{AA} = \bar{b}_{aa} = 30$, $\bar{b}_{AA} = \bar{b}_{aa} = 3$, $b_{Aa} = 11$ and $\bar{b}_{Aa} = 1.1$, hence $N^* \sim 11.5$. In this situation the equilibrium ($p^* = 0.5$, N^*) of the sexual system is locally stable. The asexuals were alone for several hundred generations after a perturbation from an equilibrium with a fraction 0.8 of the population having complex dynamics. (This equilibrium is unstable; recall that a resident with stable equilibrium dynamics could never be invaded.) In 4b the same parameter values hold, except that the heterozygote dynamics are made a bit more complex to render the equilibrium (0.5, N^*) of the sexual system unstable. Here $b_{Aa} = 15$, hence $\bar{b}_{Aa} = 1.5$. Now the invasion of the sexuals into an asexual population (again exhibiting 2-cycles) leads to the extinction of the latter. The remaining sexual system exhibits a 2-cycle, corresponding to the dominant eigenvalue of the Jacobian at the (unstable) equilibrium (0.5, N^*). The situation here is typical: whenever the equilibrium ($p^* = 0.5$, N^*) of the sexual system is unstable, sexual invasion is always followed by the extinction of the asexuals.

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genotypes produce simpler dynamics. The parameter values for this figure are $a = 0.003$, $b_{AA} = 31$, $\bar{b}_{AA} = 5.58$, $b_{Aa} = b_{aa} = 10$ and $\bar{b}_{Aa} = \bar{b}_{aa} = 1.8$, hence $N^* \sim 73.2$. A small amount of asexuals is introduced, of which a fraction 0.9 have simple dynamics. Sexual invasion into a corresponding resident population of asexuals is not possible.

different phenotypes (or genotypes) have different dynamical behaviours, ranging from stable equilibria to chaos. If reproduction is asexual, the system typically has many different attractors, and consequently many different dynamic behaviours are possible. Which of these is displayed depends on the point in the set of equilibria from which the system is perturbed away.

The main consequence of sexual reproduction is to drastically reduce the number of attractors, hence the number of possible asymptotic behaviours. Sex is a strong stabilising force in the sense that the basins of attraction become much larger with sexual reproduction, so that the dependence of the type of dynamics on initial conditions is greatly reduced. This happens because sex induces shifts of the system along the set of equilibrium points. With dominance, this shift consists of increasing the frequency of the recessive allele. Whether the sexual system tends to have simpler or more complex dynamics than the asexual depends on the phenotype of the recessive homozygote. With overdominance, the sexual system develops gene frequencies of 1/2, and hence behaves as if it were perturbed away from the mid-point in the line of equilibria. In this state it exhibits simpler dynamics than the corresponding asexual system (Figs. 1 and 2).

The strong stabilising effect of reducing the number of attractors and enlarging their basins of attraction can be thought of as being due to the mixing of interactions brought about by sexual reproduction. It is generally believed that mixing and asynchrony of interactions have a stabilising effect (May, 1985; Gilpin and Hanski, 1991; Hastings, 1993; Doebeli and Koella, 1994; Doebeli, 1995), but usually stabilisation is understood not in the mathematical sense above, but in the intuitive sense of inducing simple dynamics. Such a stabilising effect was also observed in the system with asexual reproduction considered in the present study. When an asynchrony of interactions is introduced by assuming a difference in the timing of the competitive impact of the phenotypes on each other, the system tends to have much simpler dynamics. The new system can have a stable equilibrium even if the phenotypes exhibit chaotic dynamics when alone. This mechanism also works for different species competing with each other, and it is enhanced when the number of species interacting increases. It would be interesting to see to what extent such an asynchrony is present in real systems.

Enhancing stability through sexual reproduction seems to confer an evolutionary advantage based on individual selection, at least in the model considered in this paper. To study the different dynamical behaviour of sexual and asexual systems in an evolutionary context, I used a 2-locus model to analyse invasion scenarios. In general, invasion was never possible when the resident exhibited a stable equilibrium, irrespective of the mode of reproduction. This suggests that systems with simple dynamics are evolutionarily more stable than systems exhibiting fluctuations, and relates to the debate about how often complex dynamics occur in real systems (Berryman and Millstein, 1989). That evolution tends to favour simple dynamics was further supported by the fact that when dominant sexual systems were compared with asexual populations, there was a clear tendency for the system with the simpler dynamics to be able to invade. Simple dynamics of sexuals enabled them to invade a chaotic asexual population and induce a stable equilibrium for the whole population (Fig. 3). Invasion of asexuals into a sexual population was much

less frequent, and never lead to simple dynamics (Fig. 3c), despite the fact that it was simpler dynamics that enabled invasion. The reason was that invasion of asexuals always lead to coexistence, and in this state the sexuals always dominated dynamically. This seems to be due to the greater stability of the attractors in the sexual system. Overdominant sexual populations could invade every asexual population not exhibiting a stable equilibrium, but the reverse never occurred (Fig. 4). Again it seems that this evolutionary advantage of sexuals is caused by the fact that the asymptotic dynamical behaviours in the sexual system have much larger basins of attraction. Thus, in this model sex was favoured by evolution for purely dynamical reasons: the way it changes the dynamics implies an evolutionary advantage.

Most of the literature on the evolution of sex is concerned with recombination (e.g. Bell, 1982; Stearns, 1987; Michod and Levin, 1988). In contrast, the effect of sexual reproduction in the present model is segregation at one locus, not recombination between loci. That segregation can confer an evolutionary advantage has been observed by Kirkpatrick and Jenkins (1989), whose results have been partly confirmed and partly refuted by Wiener et al. (1992). In their models, segregation leads to a faster rate of accumulating advantageous mutant alleles under constant selection pressures. This is very different from the present situation. Here segregation has an effect because of variable selection pressures, which are given by the population densities that are changing from generation to generation. That sex might be favoured when selection pressures are variable is one of the classic theories for the evolution of sex (e.g. Williams, 1975; Maynard Smith, 1978), but it is usually assumed that the variability is due to the environment and not to processes inherent in the population. Nevertheless, that segregation influences the dynamics of frequency and density dependent systems has been noted before (Hamilton, 1980; May and Anderson, 1983; Doebeli and Koella, 1994). Generally, it reduces the variance in the fitness of the population, which can imply a competitive advantage over asexuals. It has also been noted by Doebeli and Koella (1994) that segregation can lead to simpler population dynamics. Two things are new here. First, segregation can not only lead to simpler dynamics, but it can also stabilise the system in the mathematical sense of reducing the number of attractors, hence the number of possible dynamic behaviours. Second, this stabilising effect can imply an evolutionary advantage over asexuals based on individual selection. In contrast to the traditional way of using competition models, this was shown here by considering invasion scenarios in a population genetic framework.

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