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Sex and population dynamics

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SUMMARY

Chaos has been found in many mathematical models of population dynamics. This suggests that it should be a common feature of natural populations, quite in contrast to the scarcity of systems where it has been demonstrated. We suggest in this paper that the reason for this contrast may lie in the reproductive system. Whereas ecological modelling traditionally deals with asexual organisms, we introduce sexual reproduction (and thus explicit population genetics) into the population dynamic models. As specific examples, we describe the effect of sexual reproduction on two models (a host–parasite model and a predator–prey model) which exhibit chaotic behaviour for many sets of parameters. The results show that sexual reproduction generally reduces the complexity of the system, leading to a stable equilibrium or other forms of simple dynamics, or at least reducing the fluctuations of the system. Thus the chaotic behaviour predicted by many population dynamic models may be restricted to systems with asexual reproduction.

1. INTRODUCTION

The realization that ecological models can have very complicated dynamics (May 1974, 1976) has attracted much interest among population biologists. Whereas early studies focused on single species with discrete generations (May 1974), it has later been shown that complex dynamics are a likely outcome in systems with continuous time if three or more species interact (Gilpin 1979; Takeuchi & Adachi 1983; Hastings & Powell 1991). The extent, however, to which models exhibiting chaos reflect the dynamics of natural systems remains controversial. Hassell *et al.* (1976) estimated the parameters of a one-dimensional difference equation for 24 insect populations and found that in all but two cases these parameters corresponded to a stable equilibrium. This study led to the belief that chaos is scarce in natural populations. Other studies also failed to reveal chaos: Thomas *et al.* (1980) concluded that *Drosophila* systems in the laboratory do not exhibit chaos; Godfray & Blyth (1990) showed that the long-term data of plankton in the North Sea and Atlantic have annual, four-yearly or four-monthly cycles, but fail to demonstrate any chaotic patterns. However, Schaffer and his colleagues (Schaffer 1984; Schaffer & Kot 1986) argued that chaos is much more common than ecologists had earlier believed. In particular, several simple ecological and epidemiological systems with seasonality in contact rates have chaotic dynamics (Schaffer & Kot 1985). Measles in many cities (Sugihara & May 1990) or monocultures of the grass *Agrostis scabra* (Tilman *et al.* 1991) could be particular examples of this. In a recent article, Hanski *et al.* (1993) reported chaotic dynamics for microtine rodents in Arctic regions. Hastings *et al.* (1993) also promote the view that chaos might be the rule rather than the exception. Yet the question of how often chaos occurs in nature is far from being settled, and there is still a tendency to believe that it does not occur frequently (Berryman & Millstein 1989). But what could be the cause for a prevalence of simple dynamics? One reason may lie in the fact that blurring the details of interactions leading to chaos can lead to more simple dynamics. Blurring is usually modelled as demographic and environmental stochasticity or as spatial heterogeneity (see, for example, Hassell *et al.* 1991; Hastings 1993). Sexual reproduction, usually not considered when studying population dynamics, might also be expected to have such blurring effects, because it can dampen fluctuations in allele frequencies. Hamilton (1980, 1982; see also Hamilton *et al.* 1990) studied the effect of sexual reproduction on the dynamics of gene frequencies in host–parasite models. He showed that the way sex changes these dynamics can imply an evolutionary advantage. However, sex did not generally lead to simpler gene frequency dynamics. May & Anderson (1983) extended these studies by assuming density-dependent population dynamics. They examined competition between sexual and asexual subpopulations, and they showed that density dependence destroys the favourable effect of sex on the dynamics of gene frequencies. However, they did not compare the population dynamics of sexual and asexual systems. Koella (1988) also analysed competition between sexual and asexual populations. In this model, which is an extension of the Lotka–Volterra competition equations with the quantitative genetic equations developed by Lande (1976), the sexuals have a stable equilibrium when alone, whereas the asexuals exhibit chaos. This leads to a lower variance in fitness for the sexuals when they compete with the asexuals, and as a consequence the sexuals

win. The advantage of sex is due to its simplifying effect on the dynamics. In another study, M. Doebeli (unpublished results) extended a model by Hassell (1975) with the equations describing the population genetics of a system with one locus and two alleles. Here the general effect of sexual reproduction is to increase the stability of the system in the mathematical sense of enlarging basins of attraction. In addition, under certain conditions the attractors themselves are simpler with sexual reproduction. In the present study we will argue that such a simplifying effect can be expected under rather general conditions. We show the effect of sex in two models from different areas of population biology. The first model describes the regulation of populations by microparasites, and the second describes competition among genotypes with a common predator. For each model, we will first give an account of the dynamics of asexual genotypes and the conditions that lead to chaos. Then we will show how the dynamics are changed if sexual reproduction is introduced.

2. SEXUAL REPRODUCTION IN A HOST–MICROPARASITE MODEL

May & Anderson (1983) described the population dynamics of a host population with discrete, non-overlapping generations whose density is regulated by a pathogen. In their model, the pathogen spreads throughout each generation of the host before reproductive age is reached, and a fraction I of the host population is infected and killed. If the reproductive rate of the hosts in the absence of disease is $\lambda > 1$, and if N and N' denote host densities in successive generations, this system is governed by the equation

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

The fraction I of infected hosts depends on the density N and is given by the equation

$$1-I = \exp[-I(N/N_T)] \tag{2}$$

(May & Anderson 1983, Appendix). Here N_T is the threshold density of the host below which the pathogen cannot establish itself in the population. An extensive analysis of this system was done by May (1985). He showed that it exhibits chaos for all growth rates, λ. To study competition between sexual and asexual populations, May & Anderson (1983) introduced population genetics in this model by assuming that the diploid hosts mate randomly and that their dynamics are governed by one locus with two alleles, A and a. Because they were only interested in competition between sexuals and asexuals, they did not compare the dynamics of sexual and asexual systems separately. In fact, if they had, they would not have noted any difference, as they assumed complete dominance in the sexual system, i.e. they assumed that the three genotypes produce two phenotypes: it can be shown along the lines of the analysis in May (1985) that the sexual system with two (identical) phenotypes also exhibits chaos for all growth rates λ. However, this changes rather drastically when the three genotypes produce three phenotypes, as we will now show. We assume that each of the three genotypes is susceptible to a unique pathogen, i.e. the growth rate of each genotype is governed by equations (1) and (2) independently of the density of the other genotypes. Thus, for each genotype ij, the fitness function is

$$w_{ij} = \lambda_{ij}(1-I_{ij}),$$

and the infection rate I_{ij} is determined by

$$1-I_{ij} = \exp[-I_{ij}(p_{ij}N/N_{T_{ij}})],$$

where $N_{T_{ij}}$ is the threshold density of genotype ij, p_{ij} is its frequency, and N is the total host density. The equations for the dynamics of the frequency p of allele A and the total density N are

$$\left.\begin{aligned} p' &= p[pw_{AA}+(1-p)w_{Aa}]/\bar{w}, \\ N' &= \bar{w}N, \end{aligned}\right\} \tag{3}$$

where

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

is the mean fitness. For our results we compared this sexual population with a population consisting of three phenotypes that reproduce asexually. We assumed that in both populations all phenotypes have the same growth rate and the same threshold density. Figure 1 shows the dynamic behaviour of the sexual and the asexual systems in dependence on the growth rate, λ.

The asexual system consists of three uncoupled subpopulations and therefore still exhibits chaos for all λ. The genetic coupling in the sexual system has two consequences: it leads to a stable 2-cycle for low values of λ ($\lambda \lesssim 1.6$), and to smaller fluctuations in density for all values of λ. Whereas the latter result is simply obtained by observation (figure 1), the former can be proved analytically. One can also show, by using May's (1985) results, that the sexual system never has a stable equilibrium. However, for small values of λ, the fluctuation in the stable 2-cycle is very small, so that in practice one could not distinguish the 2-cycle from a stable equilibrium. Just after the 2-cycle becomes unstable ($\lambda \approx 1.6$), the fluctuations are again very small and the system stays close to an equilibrium. If the sexual system is on a stable 2-cycle, one of the homozygotes is immune against infection because its density is always below the threshold, the heterozygote is always above the threshold and the other homozygote alternates between densities below and above the threshold. Thus the stable 2-cycle is due to sexual reproduction immunizing one of the phenotypes by keeping its density low.

We have seen that the transition from two to three phenotypes changes the influence of sexual reproduction on the dynamics. That variability enhances the stabilizing effect of sex becomes clear when one considers a system with three alleles. Then the host population has six genotypes, and the dynamics of the gene frequencies and of the total density are again described by standard population genetic equations (see, for example, Crow & Kimura 1970, chapter 5). For the sake of brevity we omit the details, but we emphasize that, in the resulting system, the stabilizing effect of sex is much greater (figure 1*c*). In particular, the sexual population now has a stable equilibrium for growth rates $\lambda \lesssim 2$. These simple dynamics result

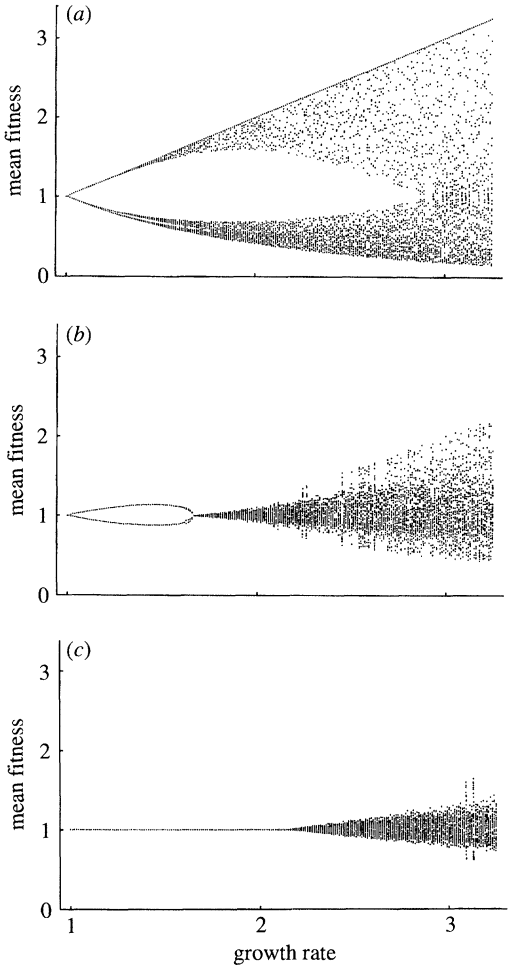

Figure 1. Time distribution of the mean fitness in the host–pathogen system for a range of values of the basic growth rate λ. The mean fitness of the host populations is defined as N'/N, where N and N' denote total densities in successive generations. (*a*) The asexual system with three phenotypes is chaotic for all growth rates λ. For low values of λ the system stays close to a 2-cycle. (*b*) In the sexual system with one locus and two alleles, hence with three genotypes, which produce three phenotypes, low values of λ lead to a stable 2-cycle, which gives way to chaos for $\lambda \gtrsim 1.6$. For all values of λ the fluctuations in the fitness are considerably smaller than in the asexual system. (*c*) In the sexual system with one locus and three alleles, hence with six genotypes, which produce six phenotypes, low values of λ lead to a stable equilibrium, which gives way to chaos for $\lambda \gtrsim 2$. For all values of λ the fluctuations in the fitness are considerably smaller than in the sexual system with three phenotypes or in the asexual system. The results were obtained with numerical simulation of equations (1)–(3), respectively their analogues for the system with three alleles. All threshold densities are set equal to 1. The value of the threshold density has only a scaling effect and does not influence the type of dynamics displayed by the system.

again because sexual reproduction immunizes some phenotypes by keeping their density below the infection threshold: at the equilibrium all homozygotes are immune. For growth rates around 2 the system starts to fluctuate erratically, although first with very small amplitudes. The size of the fluctuations gradually increases, but it is always smaller than in the corresponding sexual population with three phenotypes or in the asexual population.

3. SEXUAL REPRODUCTION IN A PREDATOR–PREY MODEL

The second model describes competition among two species with a common predator. It is again set in discrete time and based on the Ricker equation (Ricker 1954) for a population with non-overlapping generations. This equation is first extended to describe competition between species, and then the predator is included. The model was originally introduced by Comins & Hassell (1976) to study stabilizing effects of predation on a system of competing prey species. Their equations were:

$$\left.\begin{aligned} N_1' &= \lambda_1 N_1 \exp\left[-q_1(N_1+\alpha_1 N_2)-\beta_1 M\right],\\ N_2' &= \lambda_2 N_2 \exp\left[-q_2(N_2+\alpha_2 N_1)-\beta_2 M\right],\\ M' &= N_1[1-\exp(-\beta_1 M)]\\ &\quad + N_2[1-\exp(-\beta_2 M)]. \end{aligned}\right\} \tag{4}$$

Here N_1 and N_2 are the densities of the prey competitors, M is the density of the predator, and the prime indicates successive generations. The parameters λ_i are the basic growth rates of the prey species, the α_i describe the competitive impacts of the species on each other, and the q_i measure the ability to cope with the environment in the absence of the predator. The predator finds, kills and converts prey N_i into offspring at a rate $1-\exp(-\beta_i M)$, $i = 1, 2$. The parameters β_1 and β_2 are the searching efficiencies of the predator. Hassell & Comins (1976) showed that the system of competitors without predator tends to have complex dynamics for high competitive impacts, $\alpha_1\alpha_2 > 1$, and for high growth rates λ_i. In Comins & Hassell (1976) they showed further that a predator can dampen fluctuations in prey densities and lead to simpler dynamics. Typically, in the purely competitive system, where $M = 0$, one of the prey species goes extinct. Predation can then induce a stable three-species equilibrium if predation on the superior competitor is sufficiently intense. We will now show that this stabilizing effect is much stronger if the prey reproduces sexually. To do so, we again assume the simplest genetic model of one locus with two alleles A and a. The fitness function for the genotype AA is

$$w_{AA} = \lambda_{AA}\exp\left[-q_{AA}(N_{AA}+\alpha_{AA}^{Aa}N_{Aa}+\alpha_{AA}^{aa}N_{aa}) - \beta_{AA}M\right],$$

and similarly for the other two genotypes. Here α_{AA}^{Aa} and α_{AA}^{aa} measure the competitive impact of genotypes Aa and aa on AA. The sexual system is now determined by the three equations

$$\left.\begin{aligned} p' &= p[pw_{AA}+(1-p)w_{Aa}]/\bar{w},\\ N' &= \bar{w}N,\\ M' &= p^2N[1-\exp(-\beta_{AA}M)]\\ &\quad + 2p(1-p)N[1-\exp(-\beta_{Aa}M)]\\ &\quad + (1-p)^2N[1-\exp(-\beta_{aa}M)], \end{aligned}\right\} \tag{5}$$

where p is the frequency of allele A, N is the total prey density, and

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

is the mean fitness. To compare equations (5), which

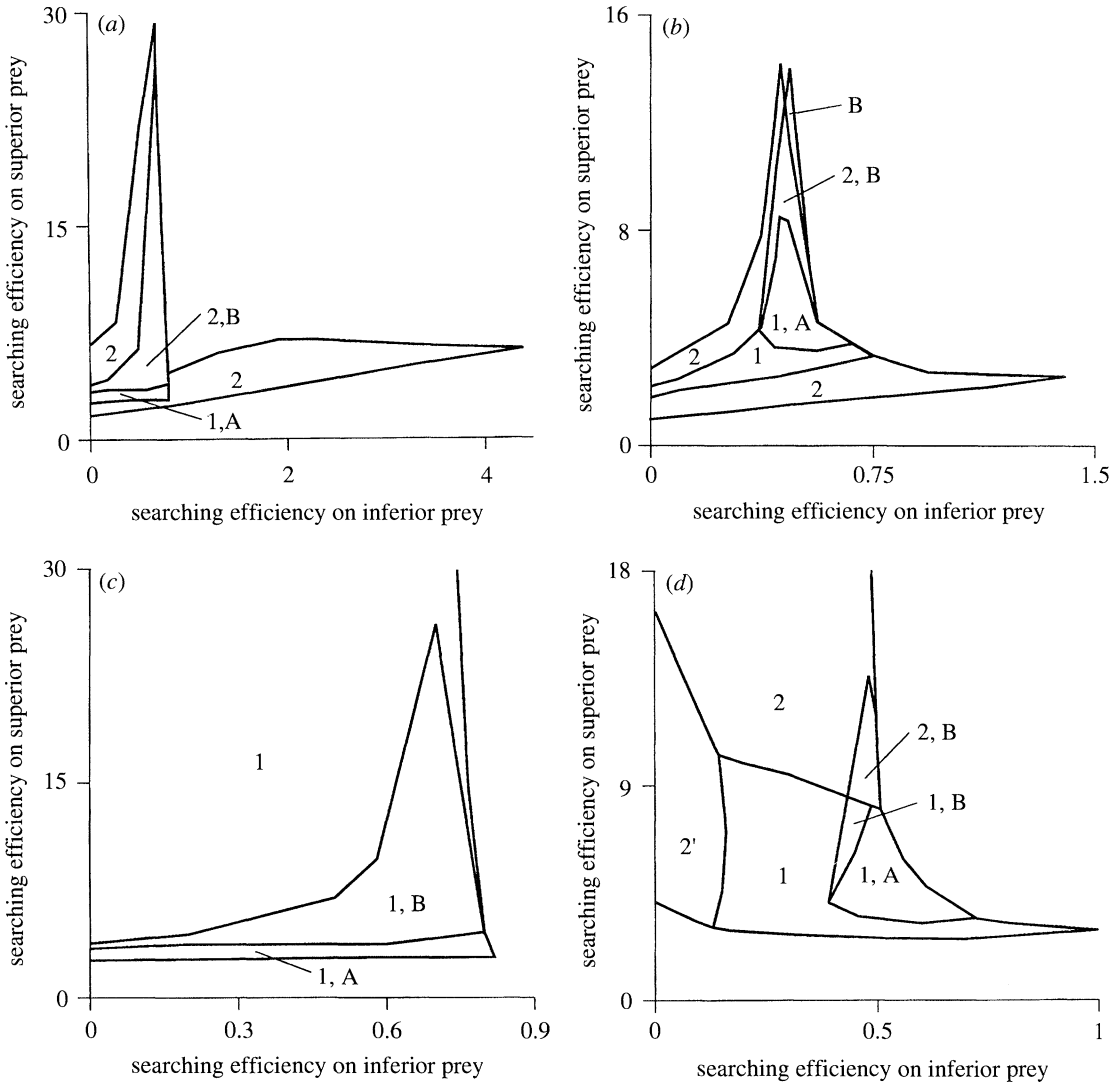

Figure 2. Regions in parameter space are shown for which coexistence in the predator–prey model is possible. The axes in the parameter space are the predation efficiencies on the inferior (x-axis) and on the superior (y-axis) prey phenotype (see text). The areas are labelled as follows: 1 refers to coexistence with a stable equilibrium in the sexual system; 2 refers to coexistence with cyclic or chaotic motion in the sexual system; A and B correspond to stable coexistence and coexistence with cyclic or chaotic motion in the asexual system. In (*a*) and (*b*) the asexual system is compared with the sexual system in which the dominant allele produces the superior competitor. This is done for two sets of parameters. In both figures the combined regions labelled either 1 or 2, corresponding to coexistence in the sexual system, are considerably larger than the regions labelled A or B, corresponding to coexistence in the asexual system. In (*a*) the region of stable coexistence is the same for both systems (area 1 A), but in (*b*) the region labelled 1 is larger than that labelled A, hence stable coexistence is more likely in the sexual system. Note the different scales used in (*a*) and (*b*). In (*c*) and (*d*) the asexual system is compared with the sexual system in which the dominant allele produces the inferior competitor. The same two sets of parameters were used as in (*a*) and (*b*). Again, in both cases the regions of parameters allowing coexistence are much larger in the sexual system. In (*c*), coexistence implies stable coexistence in the sexual system (hence no area labelled 2 occurs), and in both figures the regions for stable coexistence in the sexual system are larger. In (*d*), regions labelled 2 correspond to 2-cyclic motion, and the label 2′ indicates more complex dynamics. Note again the different scales in (*c*) and (*d*). The two sets of parameters used are (the subscript inf refers to the inferior, the subscript sup to the superior phenotype), for (*a*) and (*c*); $\lambda_{inf} = e^{1.3}$, $\lambda_{sup} = e^{1.7}$, $\alpha_{inf} = 2$, $\alpha_{sup} = 1.5$, $q_{inf} = 1$, $q_{sup} = 0.8$; and for (*b*) and (*d*); $\lambda_{inf} = e^{2.2}$, $\lambda_{sup} = e^{3.3}$, $\alpha_{inf} = 1.2$, $\alpha_{sup} = 0.7$, $q_{inf} = 1$, $q_{sup} = 0.8$. All results are approximations obtained by numerical simulations of equations (4) and (5).

describe three genotypes, with equations (4), we assume that the three genotypes produce two phenotypes. Then the sexual system is determined by pairs of parameter values corresponding to the two phenotypes and by the dominance behaviour of the alleles. In a system in which the heterozygote produces one phenotype and the two homozygotes produce the other (overdominance), sufficiently large predation rates on the homozygotes stabilize the system: whereas the fitness function of the homozygotes tends to 0 for high predation rates, the heterozygotes produce new homozygotes through random mating in each generation. This stabilizes the system at a constant density and a gene frequency of $\frac{1}{2}$, guaranteeing polymorphism. Obviously, such a mechanism cannot work in the asexual system.

More interesting is the dominant system, where the heterozygote produces the same phenotype as one of

the homozygotes. To compare the sexual with the asexual system, we chose two sets of parameters for the prey phenotypes which imply competitive exclusion in both systems in the absence of the predator. In the first set of parameters, exclusion is caused by high competitive impacts α_i, in the second by high growth rates λ_i. Figure 2 shows the combination of searching efficiencies β_i for which predation induced coexistence. We considered two kinds of coexistence: stable equilibrium, and cyclic or chaotic motion. For both sets of parameters, we considered a sexual system in which the dominant allele produces the superior competitor (figure 2*a*, *b*) and one in which it produces the inferior competitor (figure 2*c*, *d*). In general, the parameter regions allowing coexistence are much larger in the sexual than in the asexual system. When the dominant allele produces the superior competitor (figure 2*a*, *b*), coexistence with complex dynamics is much more likely for the sexual than for the asexual system, whereas the region allowing stable coexistence is larger for sexuals in figure 2*b*, but almost the same for asexuals and sexuals in figure 2*a*. When the dominant allele produces the inferior competitor (figure 2*c*, *d*), the regions for both types of coexistence are larger for the sexual than for the asexual system. When competition is intense (figure 2*c*), coexistence implies stability for the sexuals, but not for the asexuals. Although this is not the case when growth rates are high (figure 2*d*), the dynamics of the sexual system, in form of a 2-cycle, are simpler than that of the asexual system for a wide range of parameters.

It is clear from these results that sexual reproduction can fundamentally alter the dynamics of the predator–prey system. It can be a strong stabilizing force as well as an agent maintaining coexistence while the system exhibits complex dynamics. Of course, the two sets of parameters are just two cases in an infinite variety, but numerous numerical simulations showed that these cases are typical. The conclusions hold in general: the sexual system tends to be more polymorphic and to have simpler dynamics than the asexual system.

4. DISCUSSION

In this note we have shown that sexual reproduction can stabilize population dynamics in two ways: (i) sex can increase the parameter space that leads to the coexistence of different genotypes; and (ii) sex generally decreases fluctuations in the density of individual genotypes and of the population as a whole. We have used two models to illustrate this phenomenon. The first model describes a host population regulated by a pathogen. It was introduced by May & Anderson (1983) and analysed by May (1985). May & Anderson (1983) have already studied the influence of sex in this model, but only in the context of competition with an asexual population. Moreover, they only considered sexual systems with two host phenotypes which exhibit the same population dynamic behaviour as asexual systems. We have shown that this changes quite radically when there are three or more genotypes. Whereas the asexual system still exhibits chaos for all host growth rates, low growth rates lead to a stable 2-cycle in a sexual population with three genotypes and to a stable equilibrium in a sexual population with six genotypes (figure 1). These results also suggest that the stabilizing effect of sex is enhanced when there is greater genetic and phenotypic variability. The second model was introduced by Comins & Hassell (1976) and describes the interaction of a predator with two prey phenotypes. Introducing sexual reproduction in the prey enlarges the regions in parameter space for which the system has a stable equilibrium with the predator and both prey present. Moreover, the regions for which more complicated coexistence is possible, ranging from cycles to chaotic motion, is also greatly enlarged (figure 2). The intuitive explanation for the stabilizing effect of sex is that sexual reproduction evens out the fluctuations of the phenotypes. This cannot only lead to a stable equilibrium or otherwise regular motion when the asexual system exhibits chaos, but it also decreases the fluctuations when both the sexual and the asexual system exhibit coexistence with complex dynamics. This is illustrated in figure 3, which shows time series of the sexual and asexual systems in the predator–prey model. For the chosen parameter values, both systems exhibit chaotic motion, despite the apparent patterns. It is obvious, however, that the fluctuations of the sexual system are much smaller. A similar observation holds for the host–pathogen system (figure 1).

Following Thomas *et al.* (1980) and Berryman & Millstein (1989), this observation could be used to explain an evolutionary advantage of sex with group selection arguments: systems with smaller fluctuations are less threatened by extinction due to chance events. However, because a smaller variance in fitness implies a higher geometric mean fitness (Gillespie 1977), decreased fluctuations can in principle lead to an evolutionary advantage of sexual reproduction based on individual selection. This fits into the line of arguments used by Hamilton (1980, 1982) to show the advantage of sex in host–parasite coevolution. As an example, consider again the host–parasite model (equation (1)). May & Anderson (1983) showed that there is no advantage of sex in this model when there are two phenotypes. This is because sex does not change the dynamics as compared to an asexual system with two phenotypes. However, with three or more phenotypes the dynamics of the sexual and asexual populations differ as described above, and it can be shown that this implies an advantage of sex under certain conditions. For example, with the same parameter values as were used for figures 8 and 9 in May & Anderson (1983) to show that asexuals outcompete a sexual population with two phenotypes, the sexual population with three phenotypes outcompetes the corresponding asexual population. A similar remark holds for the sexual population with six phenotypes. Most often, an evolutionary advantage of sex is assumed to be due to recombination. In contrast, in our situation the advantage of sex is due to segregation, as we consider only one locus. That segregation without recombination can be sufficient for sex to be advantageous has been noted by Kirkpatrick

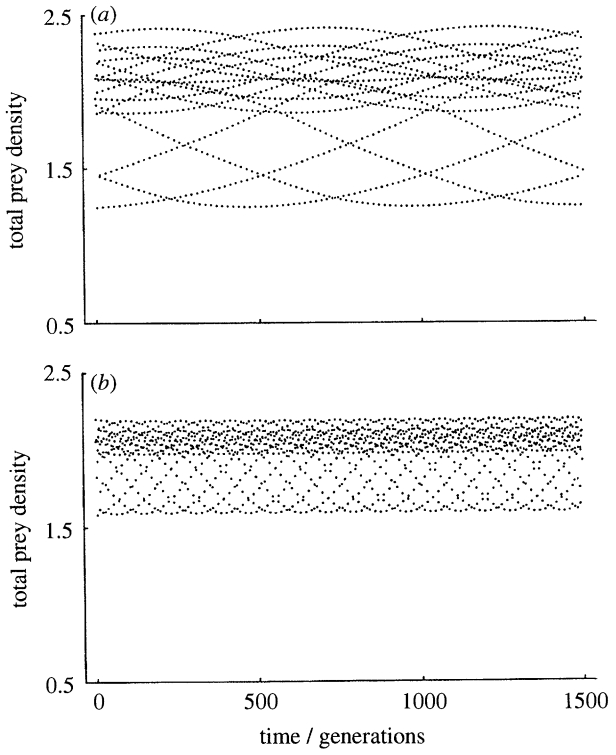

Figure 3. Time series for (*a*) the asexual and (*b*) the sexual predator–prey systems corresponding to figure 2*b* with parameters $\beta_{inf} = 0.4$ and $\beta_{sup} = 4.65$. The total density of the prey population is shown in successive generations. Despite the apparent patterns, both systems exhibit chaotic motion, as can be seen by computing dominant Liapunov exponents. (Liapunov exponents can be used to detect chaos (Schuster 1984). If they are positive, the system exhibits sensitive dependence on initial conditions, a hallmark of chaos.) It is clear that the size of the density fluctuations is smaller in the sexual system.

& Jenkins (1989) in a different context. We remark, however, that results similar to the ones reported here can be obtained with a haploid 2-locus model, where the advantage of sex is due to recombination.

In conclusion, we have shown that the intuitive idea of sexual reproduction decreasing fluctuations and preventing phenotypes from going extinct is correct in two models from different areas of population biology. Together with other work (Koella 1988; M. Doebeli, unpublished results), the study suggests that the stabilizing effect of sex on population dynamics could be a general phenomenon. In view of this idea, it is remarkable that the one clearest example of chaotic dynamics in nature, the dynamics of measles (Sugihara & May 1990), occurs in an asexual system.

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