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Stabilizing factors interact in promoting host–parasite coexistence

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Abstract

Understanding the mechanisms that promote coexistence among species is a fundamental problem in evolutionary ecology. Such mechanisms include environmental noise, spatial population structure, density dependence, and genetic variation. In natural populations such factors may exert combined effects on coexistence. Thus, to disentangle the contribution of several factors to coexistence, their effects have to be considered simultaneously. Here we investigate the effects of Ricker-type density dependence, genetic variation, and the frequency of sex on host–parasite coexistence, using Nicholson–Bailey models with and without host density dependence. Interestingly, a low frequency of sex (and the genetic variation induced by sex) is the most important factor in explaining the stability of the host–parasite interaction. However, the carrying capacity K and the frequency of sex interact in affecting coexistence. If K is low (strong density regulation), coexistence is easily attained in the density-dependent model, independently of the frequency of sex. In contrast, for high values of K (weak density regulation), low frequencies of sex considerably improve coexistence. Thus, our results suggest that coexistence among species may strongly depend on interactions among several stabilizing factors. These results seem to be robust since they remain qualitatively unchanged if one assumes (1) Beverton–Holt-type or genotype-specific rather than Ricker-type density dependence in the host, or (2) different genotype-specific susceptibilities of hosts to their parasites, or if one adds (3) moderate levels of environmental stochasticity.

Keywords: Coexistence; Host–parasite dynamics; Nicholson–Bailey model; Stability

1. Introduction

The conditions promoting coexistence among species are a central issue in evolutionary ecology (e.g. Anderson et al., 1992; Jansen and Sigmund, 1998; Abrams, 2001; Vandermeer et al., 2002). For instance, early microcosm experiments demonstrated that a predator–prey system consisting of predatory ciliates and bacteriophagous *Paramecium* exhibited diverging oscillations, leading to the extinction of the predator or the whole system (Gause, 1934). Similarly, simple predator–prey and host–parasite models showed unstable dynamics causing extinction (e.g. Nicholson and Bailey, 1935). These findings prompted a search for mechanisms that prevent or promote coexistence. For instance, theory has shown that spatio-temporal environmental heterogeneity, density dependence, interference among parasitoids, spatial aggregation of predators in areas of high prey density, dispersal, and genetic variation may promote coexistence (e.g. Hassell and May, 1973, 1974; Beddington et al., 1975, 1978; Hassell, 1978; Chesson and Murdoch, 1986; Hassell et al., 1991; Doebeli and Koella, 1994; Doebeli, 1995, 1997; Weisser et al., 1997). Some of these findings have received empirical support. For instance, a field study by Walde et al. (1992) on a herbivorous mite and its predator shows that spatial structure promotes persistence, and work by Holyoak and Lawler (1996) demonstrates that asynchronous metapopulation dynamics can lead to coexistence in a protist predator–prey system.

Although many factors promoting coexistence have been investigated, it is not clear which of them actually operate in natural populations. For example, long-term studies by Murdoch and collaborators on the coexistence between the red scale (*Aonidiella aurantii*) and its

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parasitoid *Aphytis melinus* have failed to demonstrate the effect of any tested single mechanism (reviewed in Murdoch, 1994). One potential reason for the difficulty in detecting the action of such single factors is that several of them may interact, exerting combined effects on the persistence of species interactions. Thus, to disentangle the contributions of several factors to coexistence, their effects must be considered simultaneously (e.g. Hilborn and Stearns, 1982). However, this has only rarely been attempted.

The Nicholson–Bailey model (Nicholson and Bailey, 1935) offers an ideal model system to address the question of whether and how multiple mechanisms affect coexistence. It is one of the simplest two-species models to study discrete-time predator–prey, host–parasite, or host–parasitoid dynamics (e.g. Hassell, 1978). Furthermore, since coexistence is impossible in the classical model, the effects of various factors on coexistence have been particularly well investigated in model extensions of the classical version (e.g. Beddington et al., 1975, 1978; Hassell and May, 1974; Doebeli, 1997; Flatt et al., 2001). First, as shown by Beddington et al. (1975), including host density dependence can lead to coexistence in the Nicholson–Bailey model. Second, by incorporating quantitative genetic variation, coexistence becomes possible in the classical and is improved in the density-dependent Nicholson–Bailey model (Doebeli, 1997). Third, sexual reproduction dynamically stabilizes a genetic and density-dependent version of the Nicholson–Bailey model, the effects critically depending on the frequency of sexual reproduction (Flatt et al., 2001). However, if considered simultaneously, do these factors interact in promoting coexistence and, if so, how?

Here we extend the theory on these single factors by examining their effects on coexistence simultaneously. First, we consider two ways of taking host density dependence into account: (1) the classical model without host density dependence (Nicholson and Bailey, 1935), and (2) the model version with host density dependence (Beddington et al., 1975). Second, to investigate how genetics influence coexistence, we introduce genetic variation by assuming that the susceptibility of hosts to parasites is genetically determined. Third, we investigate how different frequencies of sex (cf. Flatt et al., 2001) affect coexistence in these models. We show that a low frequency of sex (and the genetic variation at one diallelic locus induced by sex) generally induces or improves coexistence. However, the magnitude of this effect critically depends on the strength of host density dependence. Thus, our results highlight the importance of simultaneously considering multiple factors promoting coexistence. This is particularly important because the mechanistic details of interactions among such factors may be complex and difficult to predict from their effects considered separately.

2. Models of the Nicholson–Bailey type

2.1. Classical Nicholson–Bailey model

We start by describing the classical model of Nicholson and Bailey (1935). This deterministic model is set in discrete time and can be applied quite generally to organisms with non-overlapping generations. Let H_t and P_t be the host and parasite densities at time t. Then, the model has the form

$$H_{t+1} = H_t \lambda \exp(-aP_t),$$

$$P_{t+1} = cH_t(1 - \exp(-aP_t)). \tag{1}$$

Here λ is the host population growth rate, and, in absence of the parasite ($P_t = 0$), the host population grows exponentially at rate λ. Thus, there is no host density dependence. The parameter a is the parasite's searching efficiency, and the term $\exp(-aP_t)$ is the probability that a host individual escapes parasitism. This exponential term corresponds to the zeroth term of a Poisson distribution, describing the number of encounters of a host facing a population of P_t parasites with searching efficiency a. In contrast, a particular host individual is parasitized and converted with probability $[1-\exp(-aP_t)]$, the probability of *not* escaping parasitism, into c parasite individuals. Thus, c is the conversion rate of attacked hosts into parasites, i.e. a measure of the parasite's fecundity in terms of the average number of parasites emerging from a successfully attacked host. The model has a (non-zero) unstable equilibrium at

$$H^* = \frac{\lambda \ln \lambda}{(\lambda - 1)a} \quad \text{and} \quad P^* = \frac{\ln \lambda}{a}. \tag{2}$$

(The other fixed point is $H^{**} = P^{**} = 0$.) Because of the instability of the fixed point H^*, P^*, the size of the host–parasite oscillations increases rapidly so that the parasite goes extinct after a few generations. The host either also goes extinct (since $H^{**} = P^{**} = 0$ is a stable fixed point) or increases without bounds. Thus, there is no coexistence.

2.2. The host density-dependent Nicholson–Bailey model

The rather unrealistic classical model can be modified by incorporating Ricker-type density dependence of the host population (Beddington et al., 1975):

$$H_{t+1} = H_t \exp(\ln \lambda (1 - H_t/K)) \exp(-aP_t)$$

which can be rewritten as

$$H_{t+1} = H_t \lambda \exp(-H_t \ln \lambda / K) \exp(-aP_t)$$

and

$$P_{t+1} = cH_t(1 - \exp(-aP_t)), \tag{3}$$

where K represents the host's carrying capacity. The dynamics of this system can range from unstable and

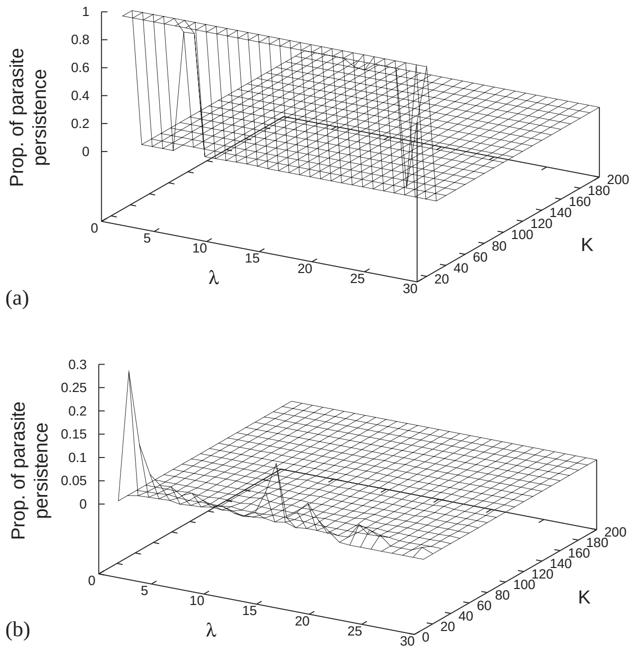

Fig. 1. (a) The monomorphic Nicholson–Bailey model with host density dependence. As compared to the monomorphic density-independent model, host–parasite coexistence becomes possible due to density dependence, even in the absence of genetic variation. (b) The polymorphic Nicholson–Bailey model with host density dependence, but without sex. Cases a and b are qualitatively similar for low values of K. Panels a and b show the proportion of cases in which the parasite coexists with its host as a function of λ and K. Since the occurrence of coexistence depends on the initial conditions, the plots show average densities, averaged over 1000 randomly chosen initial host and parasite densities. Host genotype densities were chosen randomly from the interval [0,10]; parasite genotype densities were chosen from the interval [0,1]. The other parameters were $c=1$, $a=0.45$.

stable equilibria to limit cycles and chaos (Beddington et al., 1975). The dynamics are mainly determined by λ, a, and the quantity $q=H^*/K$, where H^* is the host density at equilibrium (Beddington et al., 1975). The host dynamics can then undergo a series of period-doubling bifurcations as λ increases, and the dynamics can be chaotic if λ is large enough (Beddington et al., 1975). Here we are interested in the parameter range for which coexistence is possible, which is illustrated in Fig. 1a.

2.3. Polymorphic Nicholson–Bailey models

To study how genetic variation and different frequencies of sex affect coexistence, we extend systems (1) and (3) by including simple Mendelian population genetics. Let the genetics of host and parasite be governed by one diallelic locus. This simplifying assumption rests on the observation that susceptibility and resistance are often determined by single loci (e.g. Richards, 1970). Furthermore, work by Doebeli and Koella (1994) and Flatt et al. (2001) shows that genetic variation at a single locus is sufficient for observing stabilizing effects on the dynamics of single- and multiple-species models. Thus, for simplicity, we consider a single- rather than a multi-locus model (cf. Doebeli (1997) for a multilocus model). We further assume, using a diploid matching-alleles model, that the three host genotypes produce three different phenotypes, each of them being susceptible to a specialized parasite. The host genotypes AA, Aa and aa are susceptible to parasite genotypes BB, Bb, and bb, respectively. Thus, in all other host–parasite genotype combinations the parasite is assumed to be ineffective. This matching-alleles model is based on the assumption that different parasite genotypes will be specifically adapted to particular host genotypes, i.e. no parasite genotype can be optimally adapted to two or more host genotypes (e.g. Parker (1994), Frank (1996), see Carius et al. (2001) for empirical evidence supporting this assumption). Other models of host–parasite resistance and susceptibility (e.g. gene-for-gene interaction) may change the effects of genetics and sexual reproduction on the dynamics, but this is not the focus of our attention here. Furthermore, we assume in all three genetic models that different host and parasite genotypes only differ in their compatibility to each other, but not in their demographic parameters. Finally, we assume that only females contribute to population growth, independently of the relative abundance of males (Charlesworth, 1994).

Let $H_{AA,t}$, $H_{Aa,t}$, $H_{aa,t}$, and $P_{BB,t}$, $P_{Bb,t}$, $P_{bb,t}$, be the densities of host and parasite genotypes at time t. At the beginning of each generation, three pairs of recursion equations determine the host–parasite interaction. Each pair consists of the equations for the population dynamics of the parasite genotype IJ ($I=B$, b; $J=B$, b) and its respective susceptible host genotype ij ($i=A,a; j=A,a$), where (A,a) designate the two alleles at the A locus of the host and (B,b) the alleles at the B locus of the parasite. With this definition, we specify the polymorphic model versions:

$$H'_{ij} = H_{ij}\lambda \exp(-aP_{IJ}),$$

$$P'_{IJ} = cH_{ij}(1-\exp(-aP_{IJ})), \tag{4}$$

defines the set of three pairs of equations for the model without density dependence, and

$$H'_{ij} = H_{ij}\lambda \exp(-H \ln \lambda/K)\exp(-aP_{IJ}),$$

$$P'_{IJ} = cH_{ij}(1-\exp(-aP_{IJ})), \tag{5}$$

defines the set of three pairs of equations for the model with density dependence, where $H = H_{AA}+H_{Aa}+H_{aa}$ is the total host density before selection. Note that we have omitted the subscript t in Eqs. (4) and (5), where the superscript $'$ denotes densities after selection. After selection, reproduction occurs in both host and parasite. Allele frequencies change due to selection only, i.e. we do not allow for mutation.

Most previous models investigating the effects of population genetics on population dynamics have assumed obligate sexuality (but see Flatt et al., 2001). However, asexuality and sexuality are only two end-points of a continuum of varying degrees of sexual reproduction. Considering the effects of the full range of different frequencies of sexual reproduction, from asexuality to sexuality, is important because many species, including many hosts and parasites, may not be either strictly asexual or sexual (e.g. Hebert, 1987; Maynard Smith et al., 1993; Hurst and Peck, 1996; Birky, 1999; Seger, 1999). To investigate the effects of the frequency of sex on coexistence we introduce the control parameter ϑ, determining the amount of sexual reproduction (Flatt et al., 2001). The parameter ϑ is defined as the frequency of sex, its values ranging between pure asexuality ($\vartheta = 0$), partial sexuality ($0 < \vartheta < 1$), and pure sexuality ($\vartheta = 1$). If $\vartheta > 0$, sex is assumed to occur through random mating. Thus, hosts and parasites may be asexual, periodically sexual, or fully sexual. The parameter ϑ_h and ϑ_p determine the frequency of sex in host and parasite, respectively. With this parameterization understood the model becomes completely defined and we can consider the dynamics of the change of allele frequencies in host and parasite for systems (4) and (5). Note that the following equations are identical for both polymorphic models considered. In the host, the frequency of allele A after selection, p', is given by

$$p' = \frac{H'_{AA} + (H'_{Aa}/2)}{H'}, \tag{6}$$

where H'_{AA} and H'_{Aa} are given by Eq. (4) or (5) and where H' is the total host density after selection. Under Mendelian segregation of alleles, the new densities of the host genotypes AA, Aa and aa at time $t+1$ after random mating are given by

$$H_{AA,t+1} = (1 - \vartheta_h)H'_{AA} + \vartheta_h p'^2 H',$$

$$H_{Aa,t+1} = (1 - \vartheta_h)H'_{Aa} + \vartheta_h 2p'(1 - p')H',$$

$$H_{aa,t+1} = (1 - \vartheta_h)H'_{aa} + \vartheta_h (1 - p')^2 H'. \tag{7}$$

The total density at time $t+1$ is $H_{t+1} = H' = H_{AA,t+1} + H_{Aa,t+1} + H_{aa,t+1}$. Similarly for the parasite, let

$$q' = \frac{P'_{BB} + (P'_{Bb}/2)}{P'} \tag{8}$$

be the frequency of allele B after selection and P' the total density of the parasite. Then,

$$P_{BB,t+1} = (1 - \vartheta_p)P'_{BB} + \vartheta_p q'^2 P',$$

$$P_{Bb,t+1} = (1 - \vartheta_p)P'_{Bb} + \vartheta_p 2q'(1 - q')P',$$

$$P_{Bb,t+1} = (1 - \vartheta_p)P'_{Bb} + \vartheta_p (1 - q')^2 P' \tag{9}$$

give the densities of the parasite genotypes at time $t+1$. The total parasite population density at time $t+1$ is $P_{t+1} = P' = P_{BB,t+1} + P_{Bb,t+1} + P_{bb,t+1}$.

3. Numerical methods

We studied the dynamics of these systems numerically by examining a wide range of parameter combinations. The existence and stability of fixed points in the monomorphic models depend on λ and, in the case of density dependence, on the product acK (e.g. Brown and Rothery, 1993). Thus, there are only two independent parameters, λ and acK, in the system. Therefore it is sufficient to investigate the dynamics as a function of λ and, in the case of density dependence, as a function of λ and K for fixed values of ac (or, if K is fixed, as a function of λ, and ac). We let a given system run for 1100 generations to remove transient effects and recorded the host and parasite population densities for the next 100 generations. Exploratory simulations showed that increasing the number of generations above 1100 does not qualitatively change our results. In all simulations initial allele frequencies for host and parasite were randomized to avoid starting the system at an unstable fixed point (e.g. $p = 0.5$) and to find the different stable attractors with their basins of attraction. We considered host and parasite to coexist if their densities were larger than a defined small positive value after some time (cf. Hofbauer and Sigmund, 1998); the populations were assumed to be extinct if their densities fell below 10^{-14}. The percentage of cases where densities are greater than the extinction threshold is a rough measure for coexistence under deterministic conditions (i.e. demographic stochasticity is assumed to be unimportant). Exploratory simulations showed that increasing the threshold (e.g. to 10^{-5}) does not qualitatively change the results. Finally, we checked the robustness and generality of our results by assuming a generalized Beverton–Holt-type and a genotype-specific Ricker-type density dependence for the host, by changing the genotype-specific susceptibilities of hosts to their parasites, and by adding environmental stochasticity.

4. Results

Our extensive simulations show that (4.1) whether and how genetic variation improves coexistence depends on the frequency of sex, (4.2) coexistence is affected by an interaction between the strength of density dependence and the frequency of sex, and (4.3) these results appear to be general and robust.

4.1. Frequency of sex

In both models, the coexistence-improving effects depend considerably on the frequency of sex, determining the degree of genetic coupling among genotypes. Fig. 2 illustrates the effects of different frequencies of sex in host and parasite on coexistence in the polymorphic density-independent and the polymorphic host density-dependent Nicholson–Bailey model. Generally, small frequencies of sex in host and parasite improve coexistence most. Yet, in the absence of sex, coexistence is typically not possible (also see Fig. 1b). Note that for $\vartheta_h > 0.2$ (not shown), the results do qualitatively not change, suggesting that the effects of changing ϑ_h and ϑ_p are not symmetrical.

For the case of the density-independent model, an analysis of the distributions of the times to extinction (simulations not shown; using $\lambda = 10$, $a = 0.45$, $c = 1$, and H_{ij}, P_{IJ} picked from [0, 10] and [0, 1], respectively) shows that there is no coexistence if host and parasite are asexual ($\vartheta_p = \vartheta_h = 0$; also see Fig. 2a). This is because, if $\vartheta_p = \vartheta_h = 0$, there is no genetic coupling among the three host–parasite genotype pairs. Similarly, there is no density-dependent coupling among the host–parasite oscillators. Thus, each pair of oscillators exhibits the dynamics of the unstable monomorphic Nicholson–Bailey model. Typically, coexistence is largest (up to 2%, depending on parameter settings) if the frequencies of sex in host and parasite are low, but much smaller for higher frequencies of sex (see Fig. 2a).

For the density-dependent model (cf. Figs. 1b, 2b, and 3), if host and parasite are asexual ($\vartheta_p = \vartheta_h = 0$; see Fig. 1b), there is no random mating among genotypes, and the system is similar to the monomorphic case, which, depending on parameter settings, allows coexistence due to density dependence (cf. Figs. 1b and 1a). The system shown in Fig. 1b is similar to the monomorphic case (Fig. 1a) in the sense that there is no genetic coupling among genotypes (i.e., the system does not include sex and segregation). Yet, in contrast to the monomorphic case, even in the absence of genetic coupling (i.e., $\vartheta_p = \vartheta_h = 0$), there are three host–parasite oscillators that are linked due to density dependence (Fig. 1b). Consequently, depending on parameter settings, the dynamics may be different from the purely monomorphic case consisting of only a single host–parasite oscillator.

However, if the host reproduces sexually at a low frequency and the parasite is asexual ($\vartheta_h = 0.01, \vartheta_p = 0$), the parameter space for coexistence increases significantly (Fig. 3a) as compared to the monomorphic and the asexual case. However, the results change drastically for the reversed situation ($\vartheta_h = 0, \vartheta_p = 0.01$): the parasite coexists with the host only under restrictive conditions (Fig. 3b), showing that the effects of sexual reproduction in host and parasite on coexistence are asymmetric. Most interestingly, if host and parasite reproduce sexually at a very low frequency ($\vartheta_p = \vartheta_h = 0.01$), the parasite persists for a very large range of K and λ values, with the percentage of cases with persistence ranging between 80% and 100% for a wide range of K values (Fig. 3c). Thus, low frequencies of sex in host and parasite allow the polymorphic system to persist for a much broader range of parameters than the monomorphic case. Increasing the frequency of sex from 0 to 1 in the host ($\vartheta_h = 1, \vartheta_p = 0.01$; Fig. 3d) or the parasite ($\vartheta_p = 1, \vartheta_h = 0.01$; 3e) decreases the parameter space allowing coexistence as compared to the previous case, but coexistence is still possible for a much larger range of parameter space than in the monomorphic model. A qualitatively similar result holds if both species are fully sexual (Fig. 3f). Coexistence is improved as compared to the monomorphic case, but not as strongly as in the case in which both host and parasite have a low frequency of sex.

4.2. Density dependence and frequency of sex

As is well known, low K values imply strong density dependence, and strong-density dependence promotes

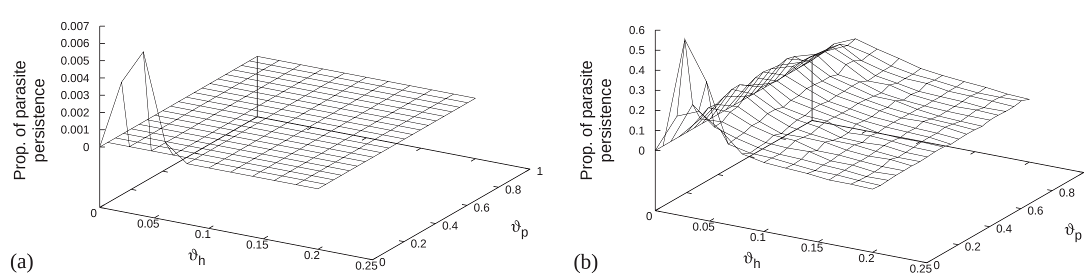

Fig. 2. The effects of varying frequencies of sex in host and parasite on coexistence in both polymorphic Nicholson–Bailey models. The figures show the proportions of cases in which host–parasite coexistence is possible as a function of ϑ_h and ϑ_p. (a) polymorphic density-independent model. (b) Polymorphic host density-dependent model, showing the case for which there is no coexistence in the absence of sex (also see Fig. 1b). Generally, the figures show that low frequencies of sex in host and parasite improve coexistence most. Also note that ϑ_h is shown here only for the interval [0,0.2], because for $\vartheta_h > 0.2$ the results remain qualitatively unchanged, showing that the effects of changing ϑ_h and ϑ_p are not symmetrical. The plots show average densities, averaged over 1000 randomly chosen initial host and parasite densities. Host genotype densities were chosen randomly from the interval [0,10], parasite genotype densities from the interval [0,1]. The other parameters used were $K = 150$, $\lambda = 20$, $a = 0.45$, and $c = 1$.

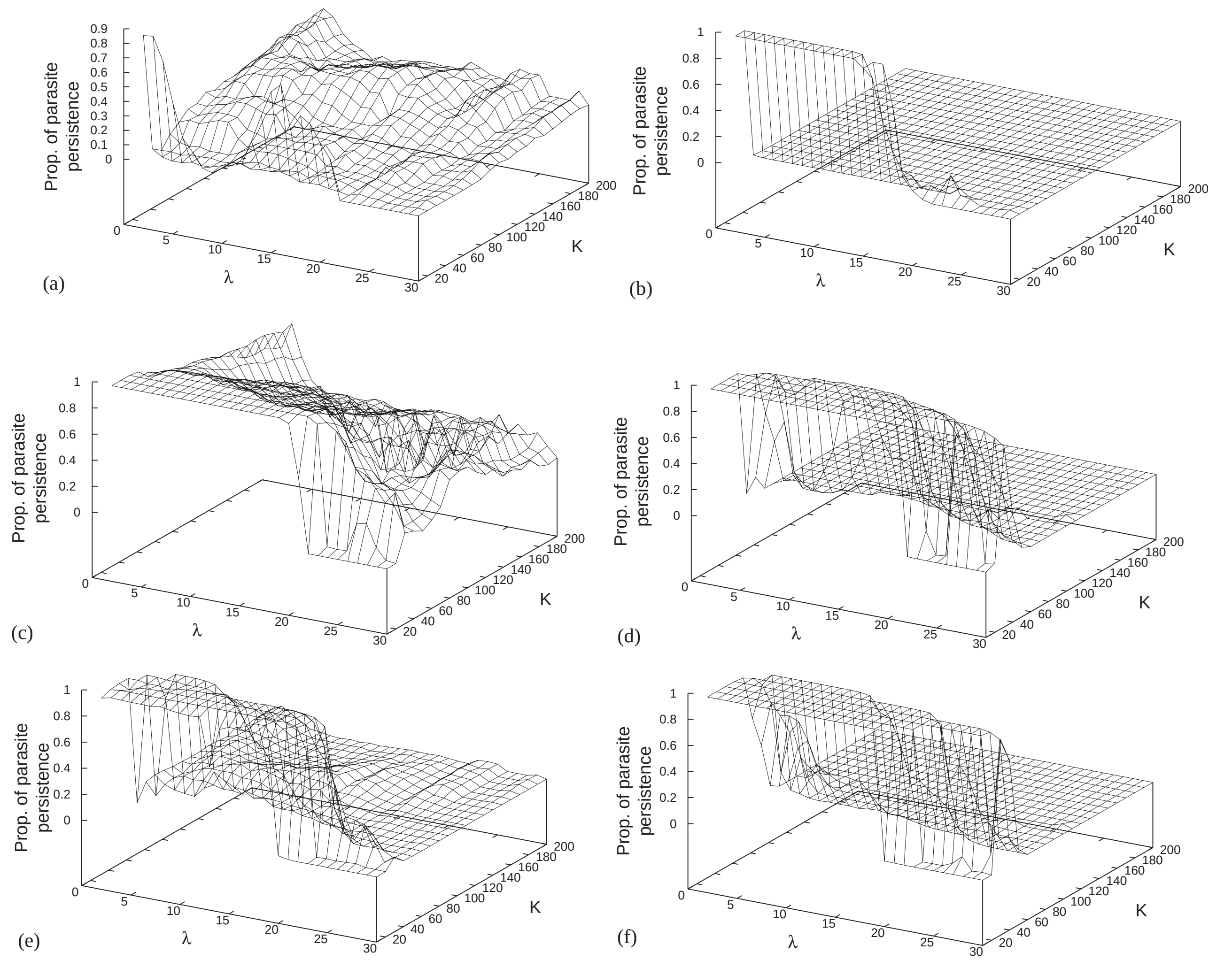

Fig. 3. Effects of different frequencies of sex on the persistence of the parasite in the polymorphic density-dependent Nicholson–Bailey model. The graphs show, for different combinations of ϑ_h and ϑ_p, the average proportion of cases in which the parasite can persist as a function of λ and K. The plots show average densities, averaged over 1000 randomly chosen initial host and parasite densities. Host genotype densities were chosen randomly from the interval [0,10], parasite genotype densities from the interval [0,1]. The other parameters were $c = 1$, $a = 0.45$. Although changes are continuous as a function of ϑ_h and ϑ_p we show only the qualitatively interesting cases: no sex, facultative sex with a low frequency, and obligate sex. (a) Host with a low level of sex, asexual parasite ($\vartheta_h = 0.01$, $\vartheta_p = 0$). (b) Parasite with a low level of sex, asexual host ($\vartheta_p = 0.01$, $\vartheta_h = 0$). (c) Both host and parasite reproduce sexually at a low frequency ($\vartheta_h = 0.01$, $\vartheta_p = 0.01$). (d) Fully sexual host, parasite with a low level of sex ($\vartheta_h = 1$, $\vartheta_p = 0.01$). (e) Fully sexual parasite, host with a low level of sex ($\vartheta_p = 1$, $\vartheta_h = 0.01$). (f) Fully sexual host and parasite ($\vartheta_h = 1$, $\vartheta_p = 1$). The results suggest that low frequencies of sex in both host and parasite improve coexistence most. At higher frequencies of sex, coexistence can decrease again, suggesting that a too strong genetic coupling may be 'destabilizing'.

stability in the Nicholson–Bailey model (e.g., Beddington et al., 1975; Doebeli, 1997). Interestingly, the density-dependent model shows a so-called 'paradox of enrichment': for low K values, the full system undergoes oscillations of increasing magnitude, and if K is too high the system goes extinct (e.g., Doebeli, 1997, and references therein).

Although low values of K typically promote stability, our results indicate that different degrees of density dependence and genetic coupling interact in affecting coexistence. If K values are low (i.e., strong density regulation) coexistence occurs for a wide range of parameter settings in the density-dependent model, independently of the frequency of sex (cf. Fig. 3). Here the overall coupling due density dependence appears to be sufficiently strong to improve coexistence as compared to the density-independent case. The genetic coupling due to sexual reproduction, however, has a minor effect on coexistence. In contrast, if K values are high (i.e., weak density regulation), low frequencies of sex are sufficient and necessary to improve coexistence as compared to the monomorphic and the asexual case (Fig. 3).

4.3. *Generality and robustness of results*

The above results seem to be rather general and robust since assuming (1) a generalized Beverton–Holt-type and a genotype-specific Ricker-type density dependence for the host, (2) different genotype-specific susceptibilities of the hosts to their parasites, and (3) environmental stochasticity does not qualitatively change our results.

First, we examined a model version in which the host population is regulated by a generalized Beverton–Holt-type (Hassell et al., 1976) rather than a Ricker-type of density dependence. This generalized Beverton–Holt model (sometimes also called the Hassell model) results in density dependence having an inverse sigmoidal form and is defined by

$$x_{t+1} = \frac{\lambda x_t}{[(1+x_t)/K]^{\beta}}, \tag{10}$$

where λ is the population growth rate and β is a constant defining the density-dependent feedback term. This model is dynamically similar to the Ricker model: it leads to chaotic behavior through period doubling as the population growth rate increases (Hassell et al., 1976). When incorporating this generalized Beverton–Holt type of density dependence into the Nicholson–Bailey model the host dynamics are described by

$$H_t = \frac{H_t \lambda \exp(-aP_t)}{[(1+H_t)/K]^{\beta}}. \tag{11}$$

Various simulations (assuming $\beta = 5$; results not shown) showed that our results remain qualitatively unchanged for this type of density dependence, suggesting that our findings do not depend on the type of density dependence assumed. Additionally, to further examine this, we investigated a model with host genotype-specific density regulation, i.e. we replaced the first equation in Eq. (5) by

$$H'_{ij} = H_{ij} \lambda \exp(-H_{ij} \ln \lambda/K) \exp(-aP_{IJ}). \tag{12}$$

Again, the results remain qualitatively similar in this model, except that coexistence is typically largest if the host is fully sexual and the parasite is only occasionally sexual ($\vartheta_h = 1, \vartheta_p = 0.01$). Why is this the case? Since this form of genotype-specific density dependence acts only within genotypes, there is no direct coupling via density dependence among host genotypes.

Consequently, the total density dependence in this system is only moderate, and stabilization may require a stronger genetic coupling.

Second, since we have assumed that homozygote host genotypes are susceptible to a specific homozygote parasite genotype and that the heterozygote host genotype interacts with the heterozygote parasite genotype (matching-alleles model), we checked how relaxing this assumption affects our results. Thus, we assumed that a homozygote host genotype interacts with a heterozygote parasite genotype (e.g. *AA* with *Bb*) and vice versa (e.g. *Aa* with *bb*). Our simulations showed that this modification does not cause any qualitative change of our results.

Finally, to check whether adding environmental stochasticity would alter our conclusions we incorporated noise into our models by multiplying the right-hand side of the recursion equations for host and parasite with the term $\exp(\sigma\varepsilon)$, where σ is the variance and ε is a normally distributed random variable with variance 1 and mean 0 (Dennis and Taper, 1994; Dennis et al., 2001). We made the following observations (results not shown). Adding environmental noise evens out small fluctuations in population densities. However, for small and moderately high amounts of noise ($\sigma_P, \sigma_H \leqslant 0.1$), our results remain qualitatively the same as compared to the models without stochasticity. In contrast, for high levels of noise, dynamical instability (high λ values) causes the extinction of the parasite. Finally, if noise is strong for both host and parasite ($\sigma_H = \sigma_P = 1$), host–parasite coexistence is not possible. Thus, adding environmental stochasticity does typically not alter our findings, given that the level of noise is not too high.

5. Discussion

5.1. *Summary of results*

Here we have investigated the effects of genetic variation, frequency of sex, and host density dependence on host–parasite coexistence in Nicholson–Bailey host–parasite models with and without host density dependence. First, we have shown that genetic variation at one diallelic locus induces coexistence in the model without density dependence and improves it in the host density-dependent model. Second, we have found that, for most parameter settings, a low frequency of sex induces or improves coexistence more than other frequencies. Third, we have demonstrated that coexistence critically depends on interactions among genetic variation, the frequency of sex, and host density dependence. While the first two results have been observed previously (Doebeli and Koella, 1994; Doebeli, 1997; Flatt et al., 2001), the third result is novel.

5.2. *Interaction effects on coexistence*

Most previous models studying the mechanisms stabilizing population dynamics have investigated the effects of single factors by including them on a one-by-one basis; the combined effects of several factors are often predictable from the effects of each factor considered in isolation (e.g. Hassell, 1978). However, this may not always be the case. The models presented here provide examples for interactions among stabilizing factors affecting coexistence that cannot easily be predicted from their single effects.

Interestingly, different degrees of density dependence and of genetic coupling interact in affecting coexistence in our models. If density regulation is strong, coexistence is possible for a wide range of parameter settings, independently of the frequency of sex. We hypothesize that the overall coupling due density dependence is sufficiently strong to improve coexistence as compared to the density independent case (cf. Fig. 1 with Fig. 3). The genetic coupling due to sex, however, apparently plays a minor role in affecting coexistence. In contrast, if density regulation is weak, low frequencies of sex are sufficient and necessary to improve coexistence as compared to the monomorphic and asexual case. Although to our knowledge this is the first formal report of such an interaction, it does not seem very surprising that coexistence-affecting factors interact with each other.

Interestingly, however, our results indicate that genetic coupling and density dependence trade off in affecting coexistence. In some cases increasing the frequency of sex from low to higher values does either not affect coexistence or even decreases it (e.g. cf. Fig. 3a with f). Interestingly, this suggests that there may be an optimal frequency of sex that enlarges the parameter space for coexistence most, although it is at present mechanistically unclear why this should be the case. Thus, the overall coupling due to genetics and density dependence seems to be sufficiently strong for already a small frequency of sex to improve coexistence. Higher frequencies of sex, in contrast, may not improve coexistence further or may even be 'destabilizing'. This suggests that, if several factors affect coexistence, interactions among these factors may be rather complex and difficult to predict from their effects considered in isolation.

5.3. Conclusions

In summary, we have confirmed previous results showing that genetic variation positively affects coexistence. However, in contrast to previous work, our simulations show that the coexistence-promoting effects of genetic variation critically depend on both the frequency of sex and the strength of the density-dependent feedback. Although the existence of such interaction effects is not surprising, our simulations revealed some unexpected findings: genetic coupling and density dependence trade off in affecting coexistence and there is an 'optimal' low frequency of sex which improves coexistence most. Thus, our results suggest that extrapolating the combined effects of multiple factors on coexistence from their effects considered singly (e.g. Hassell, 1978) may not be justified.

Why is a weak coupling due to sex most stabilizing for the host–parasite interaction? Interestingly, recent work on metapopulation and community ecology (see also Scheuring, 2001 for a discussion) shows that weak coupling forces may promote coexistence and dynamical stability more than strong coupling. For instance, Gyllenberg et al. (1993) have analysed a metapopulation model of the Ricker-type and found that, if two populations have high growth rates and little dispersal, the dynamics remain complex. However, with a somewhat higher dispersal rate, population fluctuations have decreased amplitudes and the metapopulation dynamics become stable. Yet, if dispersal rate is even higher, the subpopulations are fluctuating synchronously, but show chaotic dynamics. Similarly, McCann et al. (1998) have found that weak to intermediate strength links promote stability and community persistence in food webs. Clearly, the effects of weak coupling forces on the stability of metapopulations, communities and ecosystems deserve further study.

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