Evolutionary Ecology Research, 2011, **13**: 557–569

Parasites can simplify host-population dynamics and reduce extinction risk

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ABSTRACT

Question: Can parasites stabilize host-population dynamics and thereby reduce the risk of host extinction?

Model: We analysed a discrete-time model of logistic growth in an annual, asexual host population. Infection was assumed to reduce host fecundity, but not host survival. Using simulations, we determined whether parasites stabilized or destabilized host-population dynamics. We calculated the coefficient of variation and the effective population size (N_e) as measures of host extinction risk.

Ranges of key variables: Per capita birth rates in the host population varied from just above replacement (1.1) to 50. Equilibrium infection prevalence ranged from 0.25% to 99.75%. Infection caused a two- or ten-fold disadvantage in fitness parameters, with the fitness cost being density-independent, density-dependent, or both.

Conclusions: Parasites stabilized host-population dynamics, thereby reducing the coefficient of variation and increasing host N_e, for a broad range of host birth rates. Thus parasites could, in theory, reduce host extinction probabilities in otherwise unstable populations with high intrinsic birth rates.

Keywords: chaos, density dependence, effective population size, extinction, parasite, population regulation.

INTRODUCTION

The logistic model of population growth attains a stable equilibrium point that transitions to cycles and chaotic dynamics as the birth rate increases (for reviews, see May, 1974; May and Oster, 1976; Cushing *et al.*, 2003). Consistent with this model, laboratory flour beetle populations make transitions from a stable equilibrium point to cycles and chaotic dynamics at the points predicted by a demographic model (Costantino *et al.*, 1997). Natural and laboratory populations also exhibit chaotic dynamics, but stable populations comprise the majority (Ellner and Turchin, 1995). Because the classic logistic models predict chaotic dynamics for a large range of

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biologically reasonable birth rates [e.g. the Ricker model (Coulson and Godfray, 2007)], it is not clear why stable populations are more common than chaotic ones.

More ecologically complex models have shown that variation can stabilize population dynamics (Bjørnstad and Hansen, 1994; Doebeli, 1995; Ruxton, 1995; Doebeli and de Jong, 1999), which may explain the discrepancy between the logistic model and actual populations. In particular, Doebeli and de Jong (1999) showed that genetic variation for sensitivity to population density could help stabilize population dynamics. They also suggested that the variation need not be genetically based, but could be caused by environmental factors, such as parasites. A subsequent model (Lively, 2006) confirmed that parasites can stabilize host populations when infection causes increased sensitivity to density, such that infected individuals produce fewer offspring than uninfected individuals. The study, however, was restricted to a few illustrative cases.

Here, we explore a large parameter space to examine, in general, how parasites alter host-population dynamics. We used simulations of a logistic model to determine when parasites stabilized host dynamics, or at least damped the oscillations in host-population size. We estimated risk of extinction in the host population using the coefficient of variation for population size over time; we also estimated the potential for loss of genetic variation by calculating the effective population size (N_e) (for a review, see Charlesworth, 2009).

Large fluctuations in population size increase the risk of extinction. If host-population dynamics are so unstable that the population crashes close to zero, only a small perturbation is needed to drive the population extinct. We estimated the size of fluctuations using the coefficient of variation, since past work has shown a link between the coefficient of variation and population-level extinction. An analysis of ornithological field studies showed a positive correlation between the risk of extinction and the coefficient of variation (Pimm *et al.*, 1988; but see Schoener and Spiller, 1992). Furthermore, previous models have defined the time to extinction in terms of the coefficient of variation (Leigh, 1981). A subsequent analysis of turnover studies supported the prediction that a population's lifetime declines with increasing coefficient of variation (Diamond, 1984).

We simulated the model over a large parameter space to determine when parasites are expected to stabilize (or partially stabilize) host-population dynamics. We found that over much of the parameter space, when virulent parasites were introduced to unstable host populations, dynamics were simplified, oscillations in host density were damped, and the effective population size was increased.

MODEL

We used a discrete-time model of logistic population growth in the presence of parasites, as described previously (Lively, 2006); a flowchart of the model framework is shown in Fig. 1. Hosts were assumed to be annuals, and all hosts were susceptible to infection. The only variation in host fitness within a generation was caused by infection. We began by calculating the probability of infection at time t (P_t). Parasites contacted hosts according to a Poisson distribution, with hosts becoming infected if they were exposed to one or more parasite propagules,

$$P_t = 1 - e^{(-\beta I_{t-1}/N_t)}, \tag{1}$$

where β is the number of spores produced per infection that contact a host in the next generation, I_{t-1} is the number of infected hosts in the previous generation, and N_t is the

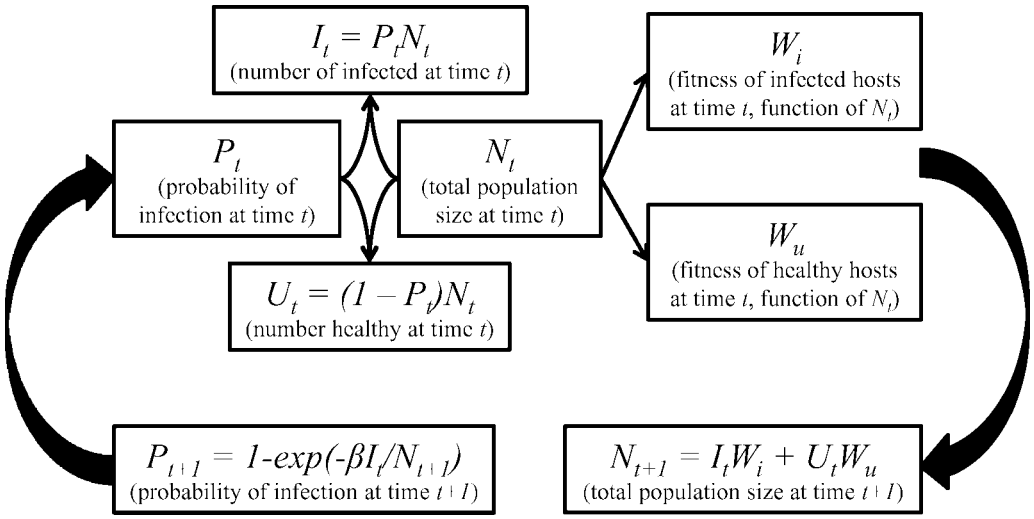

Fig. 1. Model structure. Given the number of infected (I_t) and uninfected hosts (U_t) in generation t, the total number of hosts (N_t) and the fitness for infected and uninfected hosts (W_i and W_u, respectively) can be calculated. The number of hosts ($N_t + 1$) and the probability of infection ($P_t + 1$) in generation $t + 1$ can then be calculated. Finally, the number of infected ($I_t + 1$) and uninfected hosts ($U_t + 1$) in generation $t + 1$ can be determined.

number of hosts in the current generation. Thus, $\beta I_{t-1}/N_t$ can be thought of as the mean number of propagules contacting each host in the current generation. We calculated the number of infected ($I_t = N_t P_t$) and uninfected ($U_t = N_t[1 - P_t]$) hosts.

I_t and U_t can be used to obtain the host-population size for the next generation,

$$N_{t+1} = I_t W_i + U_t W_u, \tag{2}$$

where W_u and W_i are the fitness (i.e. the per capita birth rates) of uninfected and infected hosts, respectively. Following Maynard Smith and Slatkin (1973), we assumed that fitness asymptotically decreased to zero as host density increased, with

$$W_u = \frac{b_u}{1 + (a_u N_t)^x}, \tag{3}$$

$$W_i = \frac{b_i}{1 + (a_i N_t)^x}. \tag{4}$$

The variable b_u denotes the intrinsic (maximum) birth rate of uninfected hosts, while b_i is the intrinsic birth rate of infected hosts. Similarly, a_u denotes the birth-rate sensitivity of uninfected hosts (here we set $a_u = 0.001$), while a_i is the birth-rate sensitivity of infected hosts. Infected hosts may make fewer offspring than their healthy counterparts: $b_i \leq b_u$. Likewise, infected hosts may have a reduced ability to compete with other hosts for resources, making them more sensitive to density than healthy hosts ($a_i \geq a_u$). As described previously (Doebeli and de Jong, 1999), the dynamics of the homogenous system ($b_i = b_u$, $a_i = a_u$) are stable when

$$\left| 1 - x\frac{b-1}{b} \right| < 1. \tag{5}$$

We set the exponent $x = 3$, so that the host population transitions to cycles when the intrinsic birth rate exceeds 3, which is similar to the Ricker model for an annual population (Coulson and Godfray, 2007). Since x is fixed and the density-dependence parameter a is absent from equation (5), stability of the homogenous system is determined by the intrinsic birth rate only.

Stability and persistence: coefficient of variation and effective population size

For each run through the simulation, we set the starting population size to 90% of the carrying capacity of an uninfected population, where

$$K_{\text{uninfected}} = \frac{(b_u - 1)^{1/3}}{a_u}. \tag{6}$$

We then iterated the simulation for 500 generations for each combination of parameters. A single infected host was introduced into the population at generation 101. Simulation results were obtained for a range of β values and intrinsic birth rates for uninfected hosts. At equilibrium, the number of infected hosts, I^*, is constant and equal to P^*N^*. Therefore, equation (1) reduces to:

$$P^* = 1 - e^{-\beta I^*/N^*} = 1 - e^{-\beta P^*}. \tag{7}$$

The equilibrium infection probability, P^*, depends on β but not on N^*. Together with host fitness parameters, parasite fecundity (β) helps to determine the equilibrium host population size (N^*), a quantity that can be solved for numerically. In addition to the trivial solution when $\beta = 0$, there are non-zero equilibrium infection probabilities whenever $\beta > 0$. For each simulation, we set the equilibrium infection frequency, P^*, to a desired value and solved equation (7) for β. This β value was then used in simulations for a range of host birth rates. Larger β values yield P^* values that asymptotically approach 1, but for a given β the system may exhibit a range of dynamics depending on the per capita birth rates of infected and healthy hosts, which are in turn dependent on the host population size.

Host dynamics were simulated for four different cases: (1) avirulent parasites ($a_i = a_u = 0.001$, $b_i = b_u$); (2) virulent parasites with density-independent effects ($a_i = a_u = 0.001$, $b_i < b_u$); (3) virulent parasites with density-dependent effects ($a_i > a_u$, $b_i = b_u$); (4) highly virulent parasites with both density-dependent and -independent effects ($a_i > a_u$, $b_i < b_u$). Virulent parasites imposed either a two- or ten-fold change in the fitness parameters, a and b.

We used the coefficient of variation (CV, the standard deviation of host-population size divided by the mean population size) over the last 100 generations of each simulation to assess population stability and the risk of extinction. The effective population size [N_e, the harmonic mean of population size over time (Hartl and Clark, 1989)] was calculated for the entire population over the last 100 generations of each simulation run, with N_e set to zero if the population went extinct. We calculated how much host N_e was changed by comparing the values obtained from simulations with avirulent parasites with the results for virulent parasites [i.e. case (1) above was compared with cases (2–4)].

To visualize the dynamics underlying the CV and N_e results, we generated bifurcation plots showing the population size for the last 100 generations of each simulation for

different values of b_u. We examined host dynamics in the presence of avirulent parasites and parasites imposing density-independent versus density-dependent fecundity costs. We similarly created bifurcation plots for virulent parasites as parasite fecundity β (and the proportion of hosts infected at equilibrium, P^*) increased. All simulations were run in Mathematica© (version 8.0.0.0).

RESULTS

Avirulent parasites: homogenous host population

In the basic model without costs of infection, the coefficient of variation (CV) for population size increased rapidly as the intrinsic birth rate increased to greater than 3 (Fig. 2A), as follows from equation (5). However, despite the large CV at high birth rates, none of the simulated host populations went extinct during the course of the simulations. Since the birth rate was always greater than replacement, and per capita growth was not reduced by infection, hosts were always able to replace themselves. Nonetheless, a previous synthesis of turnover studies found that populations prone to extinction usually had a CV > 0.4 (Diamond, 1984); and here we found that the CV exceeded 0.4 as the intrinsic birth rate approached 4. Thus, homogenous host populations experienced sizeable oscillations in density as the intrinsic birth rate increased past 4. Populations experiencing fluctuations equal to or greater than the mean (CV > 1, red areas in Fig. 2A) should be at extremely high risk of extinction. The effective population size (N_e) increased until the intrinsic birth rate (b_u) was approximately 3 and then declined (Fig. 2B). The effective population size varied between 447.97 and 1254.71. For a frame of reference, populations with $N_e > 1000$ are expected to be evolutionarily viable (Franklin, 1980; Frankham and Franklin, 1998; Lynch and Lande, 1998), while 500 is the minimum size suggested to be evolutionarily viable (Franklin, 1980). While only host populations with very high birth rates had N_e below the minimum viable size of 500, populations with a large range of birth rates had $N_e < 1000$.

Virulent parasites: coefficient of variation

For parasites that reduced the intrinsic birth rate ($b_i < b_u$), the host populations went extinct, meaning that host numbers reached zero before the 500th generation, when the intrinsic birth rate of healthy hosts was low, and the equilibrium parasite prevalence was high (Fig. 3A, B, E, and F). Under these conditions, individuals in the host population were unable to replace themselves. In contrast, parasites imposing only density-dependent fitness costs did not drive their hosts extinct at any of the birth rates simulated (Fig. 3C and D). For a two-fold change in fitness parameter b, parasites maintained stability through a birth rate of 8, whereas for a two-fold increase in a, stable dynamics were observed through birth rates of 27 (Fig. 3A and C). When parasites imposed either a two-fold change in both fitness parameters, or a ten-fold change in a or b, stable populations were maintained for birth rates through 50 (Fig. 3B, D, E, and F), an order of magnitude higher than the birth rates at which the classic logistic model transitions to cycles and chaos. For all costs of infection, the stable region was extended most prominently when the equilibrium infection prevalence exceeded 50%. Parasites were most prone to destabilizing host dynamics when fitness costs were high and density-dependent (Fig. 3D and F).

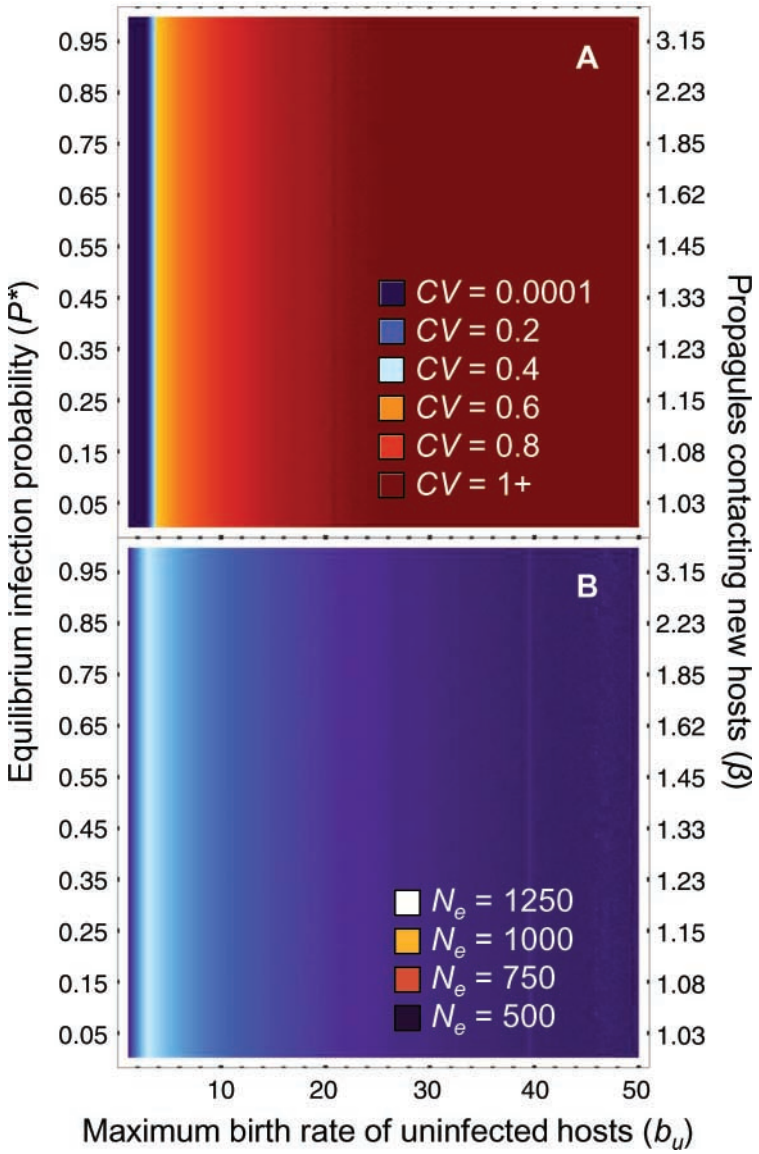

Fig. 2. Dynamics of a homogenous host population ($b_i = b_u$, $a_i = a_u = 0.001$). The coefficient of variation of population size (CV, Panel A) is plotted with cool colours for CV ≤ 0.4 and warm colours for larger CVs. Effective population size (N_e, Panel B) is shown with large values in white and orange, and smaller values in purple and black. Dynamics do not change with equilibrium infection prevalence because parasitic infection imposes no fitness cost (cf. Figs. 3 and 4).

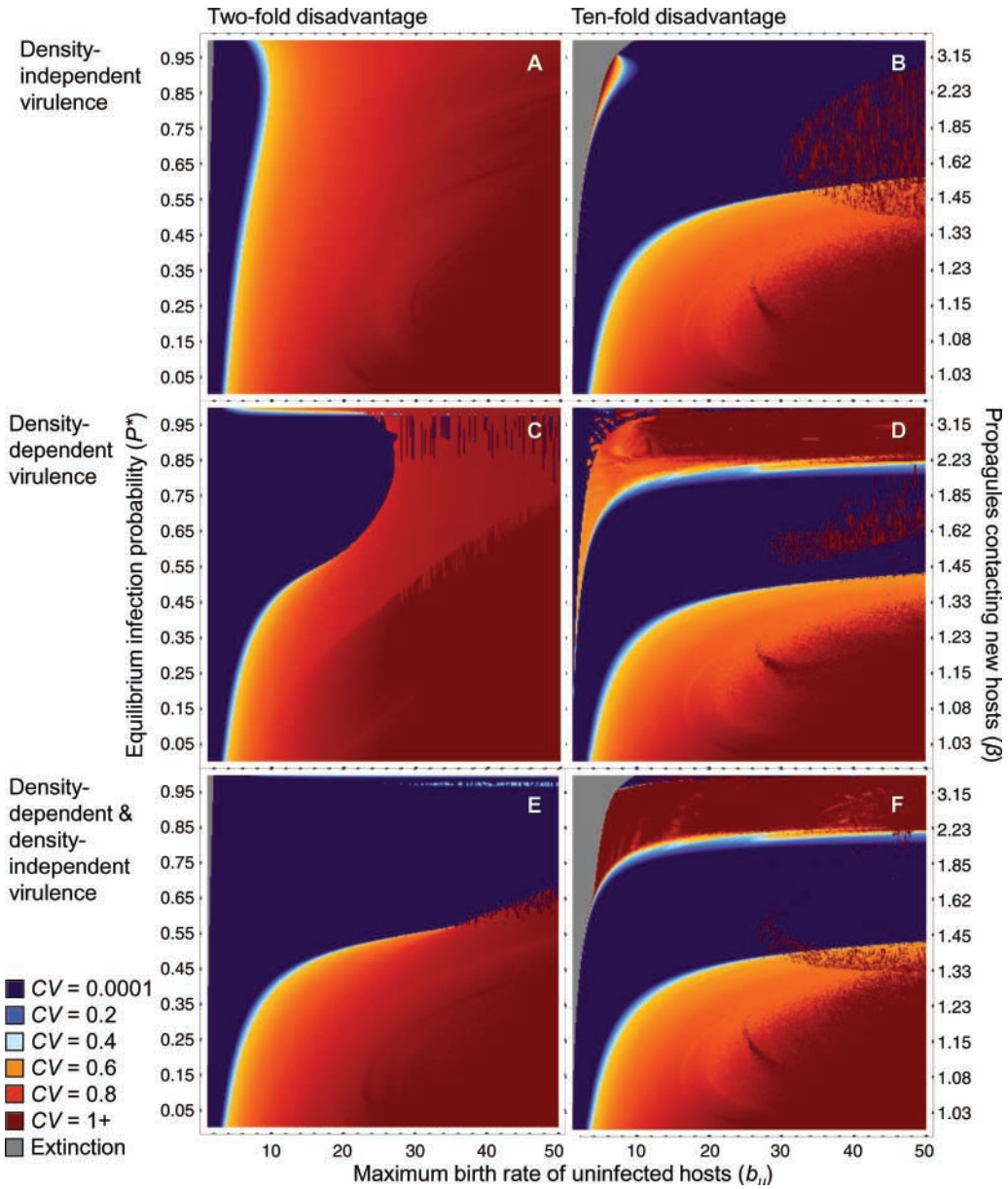

Fig. 3. Coefficient of variation of host population size (CV). Stable population dynamics are indicated by small CVs (CV ≤ 0.0001). CV ≤ 0.4 (cool colours) denotes a population with small oscillations relative to the mean size, while CVs > 0.4 (warm colours) suggest larger fluctuations relative to the mean. Grey areas indicate where the host population reached zero prior to the 500th generation. Infected hosts experience a two- or ten-fold decrease in the intrinsic birth rate ($b_i < b_u$, $a_i = a_u$; Panels A and B, respectively), a two- or ten-fold increase in the sensitivity to density ($b_i = b_u$, $a_i > a_u$; Panels C and D, respectively), or a two- or ten-fold disadvantage in both fitness parameters (Panels E and F, respectively).

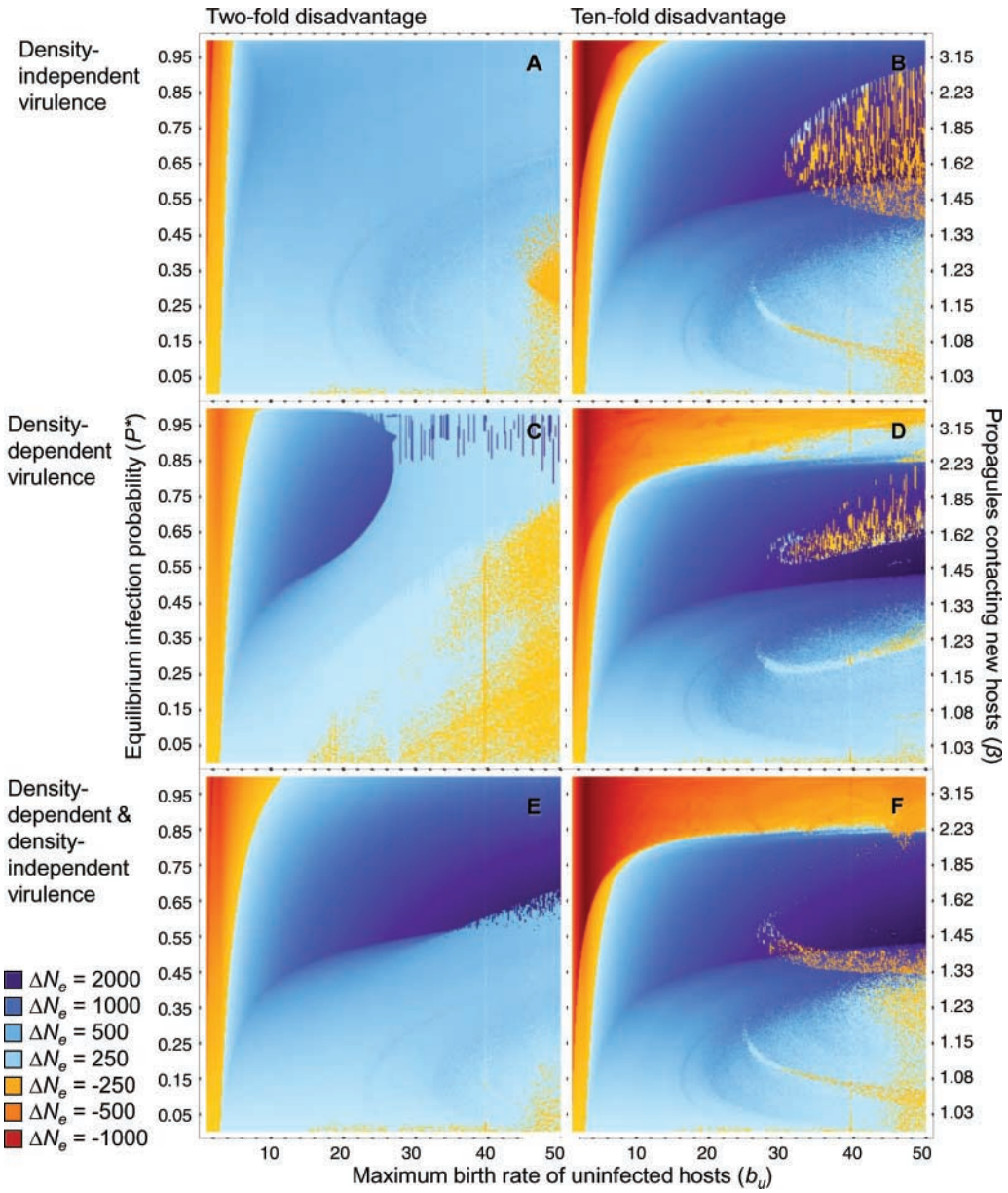

Fig. 4. Virulent parasites substantially increase host effective population size (N_e) for a broad range of the parameter space. Cool (warm) colours indicate where parasites increase (decrease) host N_e, with deeper blues (reds) denoting a greater change compared with N_e for a homogenous host population ($b_i = b_u$, $a_i = a_u$; as shown in Fig. 2). Parameter values as for Fig. 3.

Virulent parasites: effective population size

Virulent parasites increased the effective host-population size for a large range of intrinsic birth rates (b) and parasite fecundities (β) (blue regions in Fig. 4). As β increased, the effective population size decreased in populations with low birth rates (orange and red regions in upper left portion of all panels in Fig. 4). Parasites increased effective population size most when the equilibrium infection prevalence was 50% or more. When parasites were extremely virulent (imposing a ten-fold change in fitness parameters), parasites increased N_e for a narrower range of β values (Fig. 4B, D, and F).

Host dynamics: bifurcation plots

With avirulent parasites, host populations maintained a stable equilibrium size until the intrinsic birth rate exceeded 3 (Fig. 5A). Parasites inducing variation in the per capita birth rate – either through density-dependent or density-independent effects – greatly simplified host dynamics (Fig. 5B, C, and D). With a ten-fold change in b (density-independent fitness parameter), parasites maintained a stable carrying capacity up to intrinsic birth rates of 16 (Fig. 5B), and up to intrinsic birth rates of 30 for a ten-fold change in a (density-dependent fitness parameter; Fig. 5C). As the birth rate increased beyond these threshold values, the host population showed cycling and chaos, but with substantially smaller fluctuations than the homogenous host population. With density-dependent costs, the dynamics switched between large and small fluctuations with small changes in birth rates (Fig. 5C, b_u near 46; Fig. 5D, b_u between 28 and 35).

As average parasite fecundity (β) increases, a greater proportion of hosts are infected at equilibrium. When the healthy intrinsic birth rate, b_u, was held at 4, dynamics were stabilized when an intermediate proportion of hosts were infected (Fig. 6). Large oscillations in population size occurred only when the vast majority of hosts were infected or uninfected – or, in other words, when there was little phenotypic variation in the host population. When parasites imposed density-independent costs, populations approached a stable point at 15% infected at equilibrium and returned to oscillations when more than 70% of hosts were infected at equilibrium (Fig. 6A). Oscillations were amplified with increasing parasite fecundity, until infection exceeded 76% and the population approached extinction. Shifts in dynamics were similar with density-dependent virulence, except that large oscillations were observed when 65–90% of hosts were infected (Fig. 6B). When greater than 90% of hosts were infected, populations again approached a stable equilibrium point. When parasites imposed density-dependent and density-independent fitness costs (Fig. 6C), the results were qualitatively similar to the case when virulence was independent of host density.

DISCUSSION

We examined the effects of parasites on host-population persistence using simulations to distinguish between different amplitudes of oscillatory dynamics. We then linked oscillations in host numbers to extinction risk and evolutionary viability, as estimated by the CV and N_e, respectively. Previous work has shown that both laboratory and field populations span the boundary between stability and chaos, suggesting a continuous rather than sudden shift in dynamics (Ellner and Turchin, 1995). These subtle shifts may cause correspondingly small changes in populations' viability and risk of extinction. Our

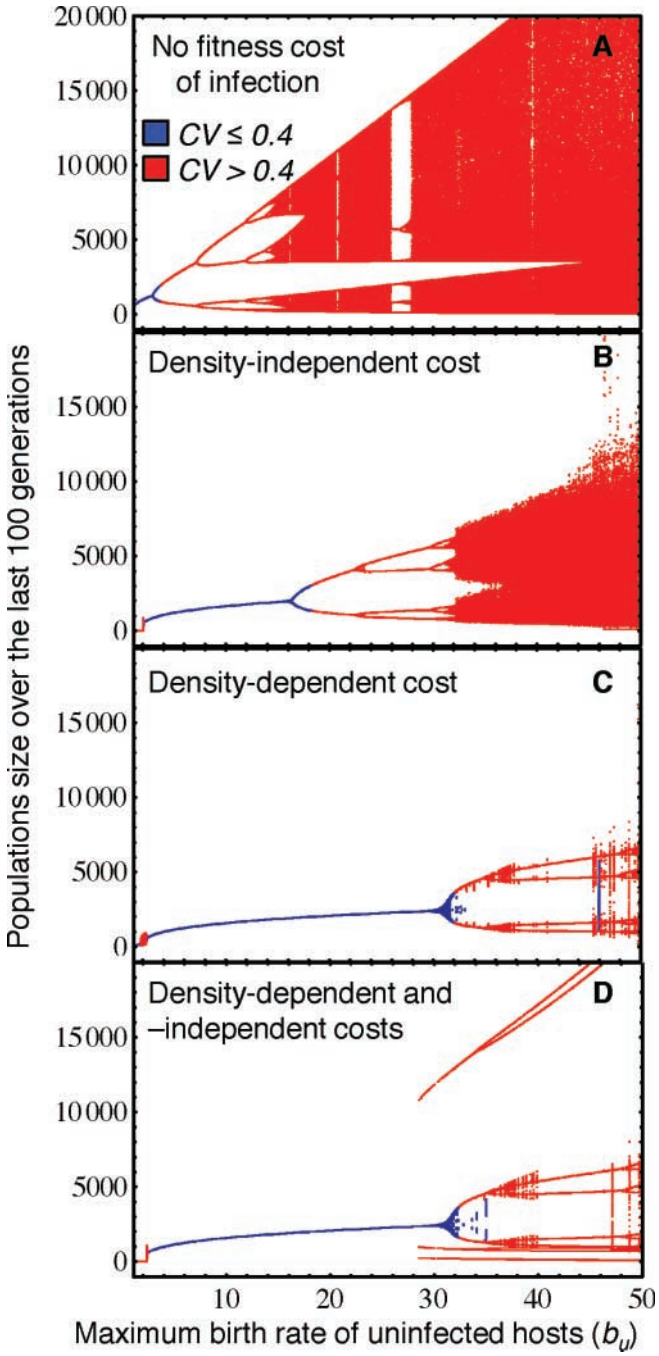

Fig. 5. Host dynamics when equilibrium infection prevalence is 50% ($\beta = 1.39$). Population size is shown for the last 100 generations of the simulation for each intrinsic birth rate (b_u) when parasitic infection imposes no fitness cost ($b_i = b_u$, $a_i = a_u$; Panel A), a cost independent of host density ($b_i = 0.1b_u$, $a_i = a_u$; Panel B), a cost dependent on host density ($b_i = b_u$, $a_i = 10a_u$; Panel C), or density-dependent and density-independent costs ($b_i = 0.1b_u$, $a_i = 10a_u$; Panel D). Black indicates that the CV of host population size is less than 0.4, while grey denotes larger CVs or a mean population size of zero.

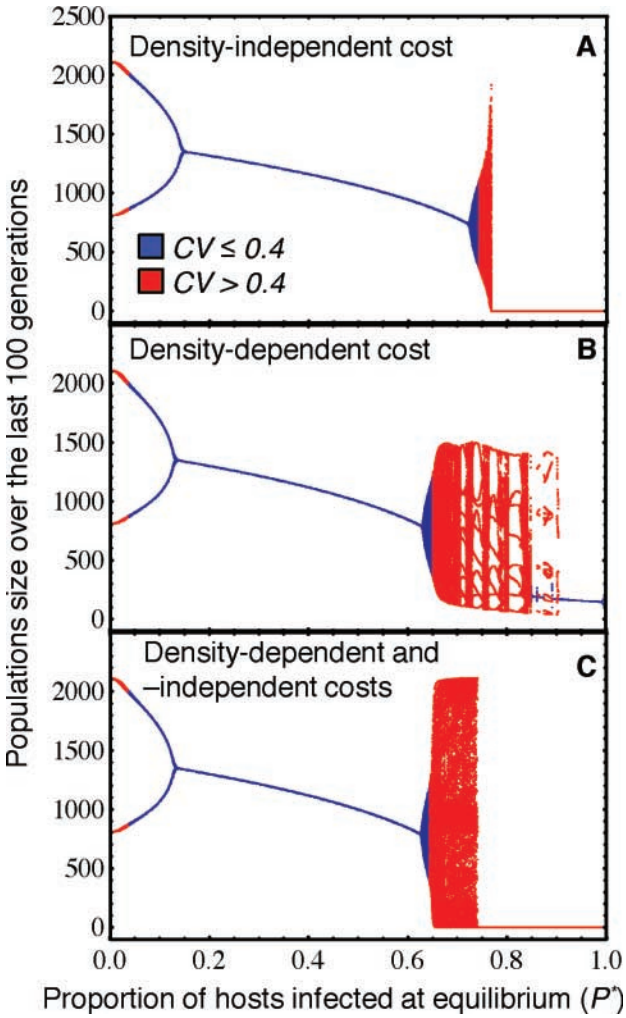

Fig. 6. Host dynamics are shown for the last 100 generations of each simulation for a range of partially infected populations (uninfected intrinsic birth rate, $b_u = 4$). Parasitic infection imposes a cost independent of host density ($b_i = 0.1b_u$, $a_i = a_u$; Panel A), a cost dependent on host density ($b_i = b_u$, $a_i = 10a_u$; Panel B), or density-dependent and density-independent costs ($b_i = 0.1b_u$, $a_i = 10a_u$; Panel C). Black indicates that the CV of host population size is less than 0.4, while grey denotes larger CVs or a mean population size of zero.

simulations suggest that, under a broad range of conditions, parasites promote persistence of their hosts by reducing fluctuations in population size. These findings imply an additional route by which parasites could maintain host diversity. While previous theory holds that parasites may maintain genetic variation through negative frequency-dependent selection (e.g. Haldane, 1949; Hamilton, 1980), our simulations suggest that parasites might also preserve within-population diversity by stabilizing host dynamics.

Previous models have also shown that both phenotypic (Bjørnstad and Hansen, 1994; Doebeli, 1995) and genetic (Ruxton, 1995; Doebeli and de Jong, 1999) variation can stabilize population dynamics.

We found that the phenotypic variation induced by infection is analogous to genetic variation in its ability to stabilize population dynamics, as predicted by Doebeli and de Jong (1999). The stabilizing effect of density-dependent virulence was greatest when hosts had relatively high intrinsic birth rates and when the equilibrium infection prevalence was intermediate (Fig. 3C and D), while density-independent virulence stabilized populations for both intermediate and high prevalence (Fig. 3A and B). At least with respect to density-dependent virulence (where $b_i = b_u$), the stabilizing effect of parasites was independent of any infection-mediated reduction in the intrinsic birth rate. In addition, similar to Doebeli and de Jong (1999), we found that infection can also destabilize dynamics, particularly when infection prevalence is high and virulence is density-dependent (Fig. 3D and F).

Given the abundance of parasitic species, and their importance to many ecological interactions (reviewed in Lafferty *et al.*, 2008), parasites have the potential to simplify population dynamics across a diverse range of host species. Our results suggest that parasites imposing an intermediate degree of virulence can damp oscillations in host-population size, and thus increase the persistence and evolutionary viability of host populations. Parasites may extend host persistence independent of any infection-induced mortality, with the potential to undermine biological control strategies or, equally, to enhance conservation efforts.

ACKNOWLEDGEMENTS

We thank Spencer Hall, Farrah Bashey-Visser, Ottar Bjørnstad, Jennie Lavine, and Lucie Salvaudon for helpful discussion. This study was supported by funding from the Indiana University Science, Technology, and Research Scholars Program, and from the US National Science Foundation (DEB-0640639).

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