The Evolution of Host-Parasite Range

A. Best,[1,*] A. White,[2] É. Kisdi,[3] J. Antonovics,[4] M. A. Brockhurst,[5] and M. Boots[1]

1. Department of Animal and Plant Sciences, University of Sheffield, Sheffield S10 2TN, England, United Kingdom; 2. Department of Mathematics and the Maxwell Institute for Mathematical Sciences, Heriot-Watt University, Edinburgh EH14 4AS, Scotland, United Kingdom; 3. Department of Mathematics and Statistics, University of Helsinki, FIN-00014 Helsinki, Finland; 4. Department of Biology, University of Virginia, Charlottesville, Virginia 22904; 5. School of Biological Sciences, University of Liverpool, Liverpool L69 7ZB, England, United Kingdom

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ABSTRACT: Understanding the coevolution of hosts and parasites is one of the key challenges for evolutionary biology. In particular, it is important to understand the processes that generate and maintain variation. Here, we examine a coevolutionary model of hosts and parasites where infection does not depend on absolute rates of transmission and defense but is approximately all-or-nothing, depending on the relative levels of defense and infectivity of the host and the parasite. We show that considerable diversity can be generated and maintained because of epidemiological feedbacks, with strains differing in the range of host and parasite types they can respectively infect or resist. Parasites with broad and narrow ranges therefore coexist, as do broadly and narrowly resistant hosts, but this diversity occurs without the assumption of highly specific gene interactions. In contrast to gene-for-gene models, cycling in strain types is found only under a restrictive set of circumstances. The generation of diversity in both hosts and parasites is dependent on the shape of the trade-off relationships but is more likely in long-lived hosts and chronic disease with long infectious periods. Overall, our model shows that significant diversity in infectivity and resistance range can evolve and be maintained from initially monomorphic populations.

Keywords: coevolution, infection range, genetic variation, adaptive dynamics.

Introduction

Given the threat of infectious disease, we need a better understanding of the evolution of infectious organisms and their hosts (Dieckmann et al. 2002; Grenfell et al. 2004; Parrish et al. 2008). However, gaining greater insight into host-parasite coevolution and in particular the factors that generate and maintain diversity is challenging for a number of reasons. First, host-parasite interactions are coevolutionary, with both species continually adapting to each other (e.g., May and Anderson 1983; van Baalen 1998; Dieckmann et al. 2002). Second, resistance in the host and infectivity in the parasite may be determined by only a few alleles (e.g., Bergelson et al. 2001), or they may be more quantitative and determined by many alleles at many loci (e.g., Nuismer et al. 2007). Furthermore, infection may be specific, with particular hosts being infected only by particular parasites, or universal, where each parasite strain can potentially infect all host types but at different rates (May and Anderson 1983; van Baalen 1998; Poland et al. 2009). Finally, ecological feedbacks are intrinsic to host-parasite coevolution. Changes in resistance or infectivity in a population alter the selection pressures because they will have changed parasite prevalence (May and Anderson 1983; van Baalen 1998; Boots and Haraguchi 1999). How ecological feedbacks, genetic systems, and infection processes interact to generate and maintain variation in hosts and parasites remains an open question.

In some natural systems, parasite infectivity and host resistance appear to be quantitative traits (Poland et al. 2009). Particular parasite strains are universally more infective against all host types, while particular hosts are universally more resistant to any parasite (Kover and Schaal 2002; Mealor and Boots 2006). In contrast, in other systems particular parasite strains can infect only particular strains of the hosts. This form of specific infection process has been most commonly observed in plant-pathogen (Flor 1956; Thompson and Burdon 1992) and bacteria-phage systems (Chao et al. 1977; Buckling and Rainey 2002) and is also found in invertebrate systems (Lambrechts et al. 2005; Little et al. 2006). Parasite strains may differ in the range of hosts that they can infect; correspondingly, hosts may be infected by different numbers of parasite strains (Forde et al. 2008). While host range is often studied at the interspecific level, especially in the context of disease emergence (Cleaveland et al. 2001), such variation is also seen at the intraspecific level in gene-for-gene systems. Hosts may simultaneously carry resistance alleles at several different loci, giving them variation in

* Corresponding author; e-mail: a.best@shef.ac.uk.

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how many pathogen strains they are resistant to, and vice versa for infectivity in the pathogen (Thrall and Burdon 2003).

There are two principal theoretical frameworks for modeling the evolutionary dynamics of host-parasite systems. Gene-for-gene (GFG) and matching alleles models consider the explicit genetics of the host-parasite interaction and follow gene frequencies in the hosts and parasites. Parasites with particular alleles can either infect or not infect, depending on the genes carried by the host. These models are coevolutionary, assume that only specific parasites can infect specific hosts and that the basis of infectivity is controlled by only a few genes (but see Sasaki 2000), and do not include ecological feedbacks (although see May and Anderson 1983). In contrast, evolutionary-ecology models using the adaptive-dynamics framework (and those that determine an evolutionary singular strategy [ESS]) make the simpler genetic assumption that changes occur as additive gene effects by small mutational steps. These models examine the invasion of rare mutants into populations where there are explicit ecological/epidemiological dynamics and typically assume that the infection process is universal (or absolute); that is, a particular parasite strain's transmission rate is the same against all host types, and a particular host strain's resistance is the same against all parasite types. Explicit tradeoffs are assumed between traits, typically such that increased transmission rates for the parasite will cause greater host mortality (and therefore shorter infectious periods) and that increased defense in the host is at the cost of a lower reproductive rate. These models usually predict either that one strain dominates or that there is evolutionary branching, where disruptive selection around a fitness minimum causes the emergence of two distinct strains. Typically, either the evolution of the parasite (Levin and Pimental 1981; Bremermann and Pickering 1983) or that of the host (Bowers et al. 1994; Boots and Haraguchi 1999) is modeled, although coevolutionary studies are also emerging (van Baalen 1998; Gandon et al. 2002; Best et al. 2009).

Evolutionary-ecology models thus assume absolute rates of infection and defense, where particular parasite strains achieve greater transmission than other strains against any host type. Contrastingly, genetic models assume that infection is relative, such that the infection success of different parasite strains depends critically on the defense characteristics of the prevalent host type. Here we present a fully coevolutionary host-parasite model using the assumptions of adaptive dynamics, but rather than assuming that transmissibility and defense are absolute, we assume something closer to an all-or-nothing infection process, where the success of infection depends on the relative breadths of host resistance and parasite infectivity. Infection success therefore depends on characteristics of both the parasite and the host, which gives an infection process in some sense comparable to that of gene-for-gene models. We find that considerable genetic variation can arise and be maintained in both host and parasite strains because of the ecological feedbacks caused by this infection process, with the resulting strains differing in the range of host and parasite types that they can successfully infect and resist.

Methods

We use a standard susceptible-infected-susceptible (SIS) framework (Anderson and May 1981), with the ecological dynamics of susceptible hosts (X) and infected hosts (Y) governed by the following equations:

$$\frac{dX}{dt} = aX - qX(X + Y) - bX - \beta XY + \gamma Y,$$

$$\frac{dY}{dt} = \beta XY - (\alpha + b + \gamma)Y. \tag{1}$$

Uninfected hosts reproduce at rate a, which is reduced because of crowding by a factor q, and have natural death rate b. The transmission coefficient of the infection is β, and infected hosts have an additional mortality rate due to infection α. We also include the potential for recovery at a rate γ (for mathematical reasons, the analytic results presented assume $\gamma = 0$ [see the appendix in the online edition of the *American Naturalist*], although analysis suggests that the results are qualitatively robust for $\gamma > 0$).

Rather than assuming that infection is universal, we wish to introduce a function whereby infection is all or nothing: if a parasite strain can successfully infect a host strain, then it achieves a positive transmission rate; if not, then the transmission rate is 0. Ideally, we would use a discontinuous step function, but this would complicate our analysis. Instead, we choose a smooth function that approximates the all-or-nothing framework but still permits analysis (in a sense, ours is an all-or-partial-or-nothing infection process), given by

$$\beta = \beta_0\left[1 - \left(1 + e^{-2(u-v)}\right)^{-1}\right]. \tag{2}$$

The infection function is best understood from figure 1. The parameters u and v denote the respective strengths of host resistance and parasite infectivity (here, strength refers to the breadth of infectivity and resistance rather than an absolute level of transmission), and β_0 is the maximum transmission rate that a parasite with infectivity v can achieve. A host with a higher u is able to prevent infection from stronger parasites (corresponding to resis-

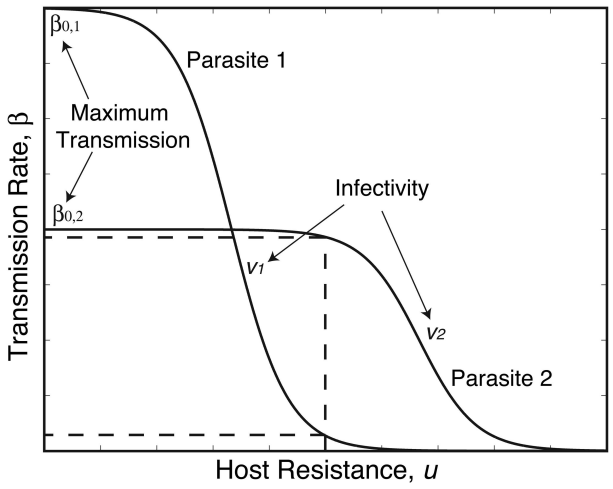

Figure 1: The transmission rate between any host and parasite strain is determined by the respective resistance (u) and infection breadths (v). Here we plot the transmission rates for two parasite types with infectivity v_1 and v_2 ($v_1 < v_2$) and corresponding maximum transmission rates $\beta_{0,1}$ and $\beta_{0,2}$ ($\beta_{0,1} > \beta_{0,2}$) across all host types. To the left-hand end, where u is small, hosts can successfully defend themselves only from parasites with low v (i.e., they have a small resistance range), while toward the right-hand end, u is larger and hosts can defend themselves from parasites with much higher v (a greater resistance range). The dashed lines show how to read off the transmission rate for the interaction between particular host and parasite strains. The figure shows that parasite 1 achieves a high transmission rate against the least resistant hosts but that the transmission rate soon drops toward 0 as the resistance of hosts increases. In contrast, parasite 2 achieves a lower transmission rate against the least resistant hosts (since $\beta_{0,1} > \beta_{0,2}$) but achieves a positive transmission rate against hosts more resistant than those infected by parasite 1.

tance to a broader range of potential parasite types), and similarly a parasite with a higher v is able to infect stronger hosts (similarly, corresponding to an ability to infect a broader range of host types). In particular, when $v \gg u$, the parasite can successfully infect the host ($\beta = \beta_0$), whereas if $v \ll u$, the host successfully resists the parasite ($\beta = 0$), and the transmission rate slopes down between these two extremes (at $v = u$ the transmission rate is $\beta_0/2$). A similar function has been used to consider infection in a host-parasitoid model (Sasaki and Godfray 1999) as well as asymmetric competition in more general ecological models (Kisdi 1999; Adler and Mosquera 2000). Furthermore, this type of all-or-nothing interaction has been used to understand the evolution of complex food webs through recursive evolutionary branching (Ito and Ikegami 2006). We assume a trade-off between β_0 and v, such that a stronger parasite that can infect a broader range of hosts (higher v) cannot infect them as efficiently (lower β_0), while a stronger host that can resist a broader range of parasites (larger u) incurs a cost to its birth rate (lower a). Note that in our model, the cost to the parasite of greater transmission is reduced range, unlike in previous work, where the cost was greater virulence, leading to a reduced infectious period. We can express these trade-offs as $\beta_0(v)$ and $a(u)$, with gradients $\beta_0'(v)$ and $a'(u)$ and curvatures $\beta_0''(v)$ and $a''(u)$, respectively (see the appendix).

We carry out analysis of the system by using adaptive dynamics (Dieckmann and Law 1996; Marrow et al. 1996; Geritz et al. 1998). Accordingly, we assume that rare, small mutations (with strategies $\bar{u}$ and $\bar{v}$) arise in a resident population at equilibrium (i.e., we assume separate ecological and evolutionary timescales). By considering the dynamics of the mutants, we can obtain invasion fitnesses for mutant hosts and mutant parasites, respectively labeled s and r (see the appendix). The coevolutionary dynamics are given by the rate of change (in evolutionary time) of host defenses u and parasite infectivity v:

$$\frac{du}{dt} = \phi_u X^* \left.\frac{\partial s}{\partial \bar{u}}\right|_{\bar{u}=u},$$

$$\frac{dv}{dt} = \phi_v Y^* \left.\frac{\partial r}{\partial \bar{v}}\right|_{\bar{v}=v}. \tag{3}$$

The partial derivatives $\partial s/\partial \bar{u}$ and $\partial r/\partial \bar{v}$ are the selection gradients, X^* and Y^* are the equilibrium densities of susceptible and infected hosts, and ϕ_u and ϕ_v govern the speed of mutation for the host and parasite, respectively, combining the mutation rate and variance (Dieckmann and Law 1996). We assume both ϕ_u and ϕ_v to be constants and divide through both equations by ϕ_v, making the parasite's mutation speed unity and the host's mutation speed $\phi = \phi_u/\phi_v$ (unless otherwise stated, we assume that the mutation speeds are equal, $\phi = 1$). Coevolutionary singularities, (potentially temporary) stopping points of the system, will occur where the selection gradients are simultaneously 0. At such points, the evolutionary behavior of the host or parasite may be a continuously stable strategy (CSS; an evolutionary attractor and a local fitness maximum), a repeller (a point from which the host/parasite strategy always moves away), or an evolutionary branching point (an evolutionary attractor that is a fitness minimum at which the host/parasite undergoes disruptive selection and branches into two distinct strains).

Results

A coevolutionary singular point exists for a wide set of parameter values. For a chosen parameter set (those of fig. 2 but with $\gamma = 0$), the singular point is at ($u^* = 8$, $v^* = 8.5$), where, since $v > u$, the parasite can successfully infect the host. Furthermore, this singular point is co-

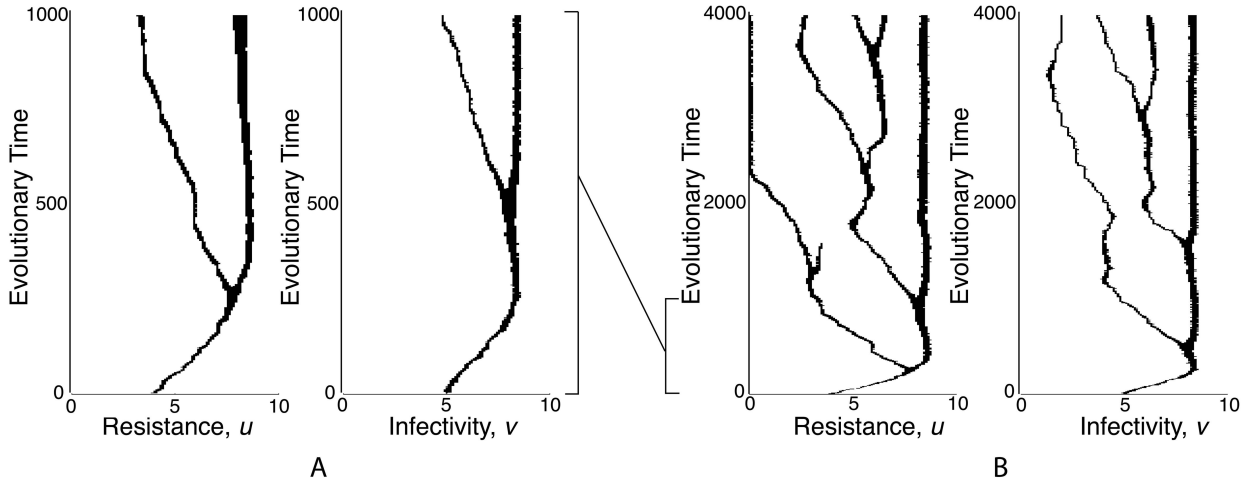

Figure 2: Simulations of the coevolutionary dynamics. *A*, Evolutionary dynamics with first the host and then the parasite branching. The results are for a model that includes recovery, $\gamma > 0$, and display the behavior predicted by the analytic results (where $\gamma = 0$). *B*, Behavior over a longer evolutionary time period. Parameter values: $(u_{\min}, u_{\max}) = (0, 10)$, $(a_{\min}, a_{\max}) = (0.782, 5.454)$, $p_a = 2.615$, $(v_{\min}, v_{\max}) = (0, 10)$, $(\beta_{0,\min}, \beta_{v,\max}) = (0.491, 17.117)$, $p_{\beta_0} = -0.434$, $b = 0.5$, $q = 0.5$, $\alpha = 1.5$, $\gamma = 0.5$, $\phi = 1$.

evolutionarily convergent (attracting) and is evolutionarily stable for the parasite (a local fitness maximum) but evolutionarily unstable for the host (a local fitness minimum; see the appendix for the full mathematical details of the analysis). It is straightforward to show that in the corresponding host-only evolution model, the singularity would be a repeller for the host and resistance would evolve to either the maximum or minimum level but the coevolution of the parasite has forced it to converge. At this cosingularity the host meets the conditions for evolutionary branching (Geritz et al. 1998), and two coexisting host strains will emerge, X_1 and X_2, which have differing densities. Once the host has branched, we have a three-dimensional system (two hosts and one parasite), and the analysis becomes harder. If we assume that the parasite mutates more quickly than the host, then we can show that the parasite can branch when there are two host types present (see the appendix). We may therefore expect the parasite to branch to produce two strains, one that specializes on the susceptible host strain and one that is able to infect both strains.

To confirm the predictions of our analysis and to consider the coevolutionary dynamics over the longer term, we produce numerical simulations of the system (see the appendix for a description of the simulation process). Unlike the case of the analytic results, we here assume that infected hosts are able to recover back to susceptibility, such that our results are applicable to more general host-parasite systems. Furthermore, in these simulations we partially relax the assumption of separate ecological and evolutionary timescales by allowing mutants to arise before the ecological dynamics have fully reached equilibrium. In figure 2*A*, the simulations show the host and parasite evolving toward the singular strategy where the host branches into two strains. The two host strains then diverge, one with lower resistance and one with higher resistance than at the singular strategy. After host branching, the parasite branches into two strains, one specializing on the low-resistance host and the other able to infect both types. In figure 2*B*, we continue the simulations for a further period of time. This shows an array of host and parasite strains evolving. At all times there are either equal numbers of host and parasite strains or one more host than parasite, a pattern determined by the number of ecological feedbacks each species experiences (Dieckmann and Metz 2006). Initially, the host has two feedbacks (the total density and the force of infection) while the parasite has only one (host density), meaning that the host can branch but the parasite cannot. After the host branches, the parasite has two feedbacks (the two separate host densities) but the host still has the same two feedbacks, meaning the parasite can now branch but the host will not, since it has two strains and two feedbacks. When the parasite branches, the number of ecological feedbacks for the host increases to three and the cascade of branching events can continue. This is in contrast to previous studies that employed a universal infection process (Boots and Haraguchi 1999; Best et al. 2009; Svennungsen and Kisdi 2009), in which the initial branching does not increase the number

of feedbacks to the parasite and so only a single branching event occurs.

Trade-off shapes. For a fuller understanding of our model, we apply a geometric form of adaptive dynamics (sometimes called critical-function analysis), which highlights the role of the trade-offs in the evolutionary outcome (de Mazancourt and Dieckmann 2004; Bowers et al. 2005). In particular, we express the fitness and stability conditions in terms of the trade-off shapes at the singularity and partition the evolutionary behavior in terms of these shapes (see the appendix; Kisdi 2006; Best et al. 2009). In figure 3, we show the possible coevolutionary outcomes at the cosingularity ($u^* = 8, v^* = 8.5$) for different trade-off shapes in the host and parasite (in particular the trade-off curvatures, the second derivatives of the trade-offs, $a''(u)$ in the host, plotted on the *X*-axis, and $\beta_0''(v)$ in the parasite, plotted on the *Y*-axis; see the appendix for a detailed explanation). The dark shaded rectangle shows the trade-off shapes for which we would expect the initial evolutionary branching in the host-only evolutionary model. The lighter shaded area shows the additional trade-off shapes for which evolutionary branching is predicted in the fully coevolutionary model. Branching tends to occur when the trade-offs are close to linear (i.e., $a''(u) = 0$, $\beta_0''(v) = 0$). Numerical analysis also suggests that for a wide range of parameter values, whenever the host initially branches, the pattern of multiple branching in both species will follow.

We show this classification diagram for varying life spans ($1/b$) in figure 3*A* and for varying virulence rates (α) in figure 3*B*. In all cases, evolutionary branching in host resistance becomes more likely when we consider the fully coevolutionary model than is predicted from evolution in just the host. Evolutionary branching is predicted for a wider range of trade-off shapes at longer life spans (fig. 3*A*) and at lower virulence rates and so longer infectious periods (fig. 3*B*).

Evolutionary branching is just one potential outcome of the system. A common behavior at the cosingularity is a co-CSS where both the host and parasite remain fixed. This outcome occurs when host resistance incurs moderately to strongly accelerating costs and parasite infectivity incurs accelerating to moderately decelerating costs (in fig. 3, the co-CSS occurs for the dotted region). When either trade-off is strongly decelerating, the cosingularity is a repeller (in fig. 3, the repeller occurs in the plain [unshaded

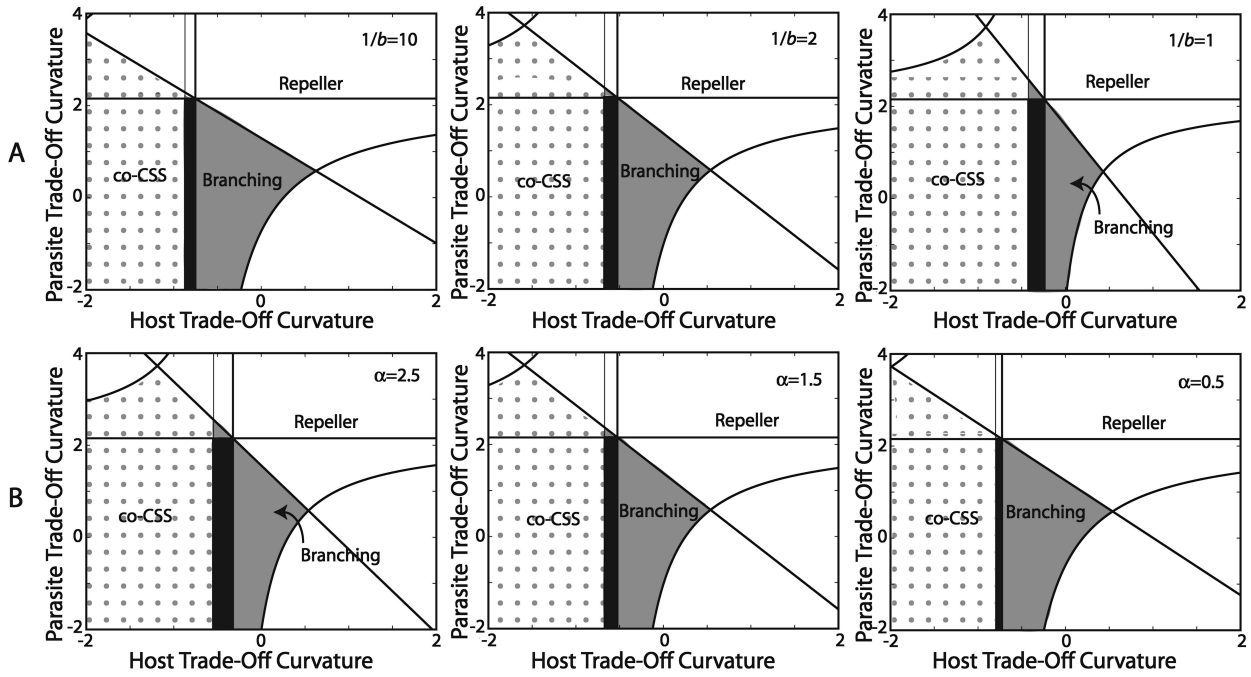

Figure 3: How the coevolutionary outcome at ($u^* = 8, v^* = 8.5$) depends on host and parasite trade-off shapes (i.e., the second derivatives of the trade-offs). *A* shows diagrams for varying life span, *B* those for varying virulence rates. The dark shaded areas are trade-off shapes where branching occurs in the host-only evolution model, and the lighter shaded areas are the additional shapes for which the fully coevolutionary model predicts branching. A co–continuously stable strategy (co-CSS) occurs in the dotted region. For all remaining combinations, the singularity is a repeller or there are cycles. Except where marked, parameter values are as follows: $(u^*, v^*) = (8, 8.5)$, $(a^*, \beta_0^*) = (3, 2)$, $b = 0.5$, $q = 0.5$, $\alpha = 1.5$, $\gamma = 0$, $\phi = 1$.

and undotted] region), and we would either expect both the host and parasite to evolve to maximum or minimum resistance/infectivity or the appearance of evolutionary cycles.

Evolutionary cycles are observed for a small set of trade-off shapes and also by adjusting the relative mutation speeds. In figure 4, we show simulations for a system identical to that in figure 2 (again, recovery is included) but where the host mutates eight times as fast as its parasite ($\phi = 8$). Here the host first increases resistance to escape infection, but the parasite follows by increasing its infectivity. The host then lowers resistance in order to benefit through increased reproduction. The parasite then lowers its infectivity, because it can specialize on the susceptible host and gain greater transmission, and the cycle continues. Evolutionary cycling appears not to be a particularly common outcome of the model, requiring either a limited set of trade-off shapes or the host to mutate much more quickly than its parasite. This contrasts with the predictions of GFG models with costs, which tend to show cycling.

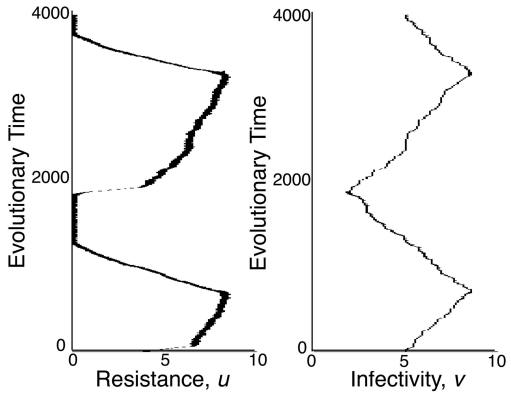

Figure 4: Simulations of the coevolutionary dynamics showing cycling. Parameters are as in figure 2, except the relative mutation speed $\phi = 8$.

Discussion

Our host-parasite model considers the coevolution of resistance and infectivity, where the success of infection depends on the relative levels of parasite infectivity and host resistance in an all-or-nothing manner, in contrast to quantitative, absolute levels of resistance and infectivity. The model allows us to examine how coevolution with ecological feedbacks influences the evolution of the host and parasite and to understand the factors that generate and maintain genetic variation in host-pathogen systems. The analysis shows that (i) a high level of diversity in both hosts and pathogens can arise through repeated evolutionary branching, leading to host strains that vary in the range of parasites that they can successfully resist and parasite strains that vary in the range of hosts that they can successfully infect; (ii) this multiple branching is much more likely in a coevolutionary setting than when the host or parasite evolves alone; (iii) evolutionary branching occurs for a wide range of cost structures; (iv) branching is more likely in hosts with longer life spans and parasites with longer infectious periods; and (v) coevolutionary cycles may occur, but only under a relatively restricted set of conditions.

In genetic models where infection depends on specific alleles in the host and parasite, polymorphisms are widely predicted when costs are incorporated (Frank 1993; Sasaki 2000). Similarly, in adaptive-dynamics models where resistance and infectivity rates are universal, branching can occur, provided that there are costs, because of negative frequency-dependent selection (Boots and Bowers 1999, 2004; Svennungsen and Kisdi 2009). However, these latter models generally predict only one branching event, even when there is coevolution (Best et al. 2009), suggesting a limit on the degree of diversity that can arise when interactions are nonspecific. Here we show that a higher degree of diversity can be obtained, without assuming the tight specificity of genetic models. We have shown that an assumption of all-or-nothing infection, which is similar but not identical to that in GFG models, can generate considerable host-parasite diversity where strains differ in their resistance and infectivity ranges. We therefore emphasize that the nature of infection may be a crucial factor in the generation of diversity.

A key prediction of our model is that we should see host and parasite strains that vary in their respective resistance and infection ranges in populations. Variation in infection and resistance range is particularly well studied in bacteria-phage interactions. For example, a recent study of the *Pseudomonas fluorescens*–phage $\phi 2$ interaction revealed that coevolution leads to rapid within-population diversification into multiple coexisting host and parasite phenotypes that vary in resistance and infectivity range, from specialists to generalists (Poullain et al. 2008). In line with our model, the evolution of increased resistance and infectivity ranges is costly in this system (Brockhurst et al. 2004; Buckling et al. 2006; Poullain et al. 2008). In addition, our model predicts that diversification is likely to occur under coevolution, and this was confirmed in experiments that permitted phage evolution only where no diversification of phage phenotypes was observed (Poullain et al. 2008). Similar diversification of resistance-range and infectivity-range phenotypes has also been observed in coevolving populations of *Escherichia coli* and phage T7, where, again, costs of increased resistance and infectivity range are observed (Forde et al. 2008).

In the classical plant gene-for-gene systems, an increasing number of resistance alleles in an individual results in resistance to a larger number of pathogen strains, and, correspondingly, an increasing number of loci in the pathogen for infectivity (generally termed "virulence" in the plant literature) results in a broader host range. Estimates for levels of polymorphism at these loci in natural populations are difficult to obtain because most studies are confined to agricultural contexts and because it is difficult to obtain precise genetic data without extensive crossing designs. Moreover, detection of host resistance and pathogen infectivity phenotypes depends on the tester strains used. While the emphasis has been on the large number of resistance and virulence phenotypes present in any one system, generally populations are dominated by relatively few phenotypes. Thus, while Bevan et al. (1993) found 27 resistance phenotypes in two populations of groundsel (*Senecio vulgaris*) infected by powdery mildew (*Erisyphe fischeri*), more than 70% of the individuals were of one of two phenotypes, including a strain susceptible to all pathogen tester lines. Similarly, in a study of six populations of wild flax (*Linum marginale*) infected by rust (*Melamspora lini*), Thrall et al. (2001) scored 75 different resistance phenotypes, yet the four most common made up 47% of the individuals, while the five most common of the 44 pathogen lines scored made up 60% of those sampled. There is therefore evidence that while variation is abundant, host and pathogen diversity are limited. It is interesting that in our model we also find that different strains vary in their population sizes.

One difficulty with directly linking our results to field data is that our model assumes that the host and pathogen are asexual and that host and pathogen range are highly heritable. If the host and pathogen were outcrossed, there would be recombination among the genotypes that differed in their ranges. The effect of recombination deserves further rigorous modeling, but intuitively, recombination may have the effect of reducing the heritability of host range and so reducing the rate and extent of divergence among host and pathogen lineages. Despite this, in the wild flax rust system, plains and upland populations of the host and pathogen differ in their breeding systems (Burdon et al. 1999) but do not show significant differences in the number of resistance and infectivity genotypes, and the trends were for more, rather than less, variation in the outcrossed plains populations. The effect of mating system on the evolution of host range is in need of further investigation.

The pattern of diversity generated in our model produces parasites from the very widely infective to the specialist and hosts from those that can be infected by many parasites to those that are susceptible to only a few. Analytically, there is no limit to the level of diversity that could evolve in our model. The simulations suggest that only a few strains of each species can coexist because our infection function is not strictly all-or-nothing but a smooth function that slopes down between maximum and zero transmission. It is the steepness of this slope that determines the potential for coexistence, with a discontinuous step function supporting an infinite continuum of coexisting strains and the smooth function only a few (Adler and Mosquera 2000). Simulations (not shown) taking transmission terms with steeper slopes (by increasing the value of the constant $[-2]$ in the exponential term in eq. [2]) show far greater numbers of strains being generated. In particular, the closer the infection process becomes to being truly all-or-nothing, the greater the variation that will be generated.

In the equivalent version of our model where only the host evolves, the initial evolutionary branching in the host is predicted for only a narrow set of cost structures. However, in our fully coevolutionary model, a much wider set of cost structures (particularly where the relationships are close to linear) results in branching, suggesting that the coevolution of the parasite makes diversity in the host more likely. Furthermore, in models where only the host is evolving there is only this initial branching event, whereas in the coevolutionary model there is often a chain of branching events leading to considerable diversity in both the host and parasite. Evolutionary branching, and the consequent generation of variation in both the host and the parasite, was found to occur for a wider range of trade-off shapes when hosts had longer life spans and when the parasite had lower virulence and therefore a longer infectious period. This therefore suggests that infections in long-lived hosts may produce more diversity in both host and parasite.

Although we have focused on evolutionary branching, this is just one potential outcome of the model. Both the host and the parasite may evolve to a CSS, or they may maximize/minimize their resistance and infectivity. Coevolutionary cycling is also a possible outcome of the model, but a relatively rare one. Genetic models often predict cycling of gene frequencies, particularly in matching-alleles models or where the number of loci is high (Sasaki 2000; Agrawal and Lively 2002). Such complex genetic topologies may encourage cycling, because continually changing combinations of loci will allow hosts to escape infection. In contrast, evolutionary cycling tends to occur less frequently in classical adaptive-dynamics models (although see Dieckmann et al. 1995). The fact that evolutionary cycling in our model is relatively rare, compared to the other possible evolutionary outcomes, suggests that our linear ordering of strains (such that all strains with an equal level of investment are assumed identical) is less

likely to promote evolutionary cycling than highly specific gene-for-gene–type interactions.

There continues to be considerable effort to understand the evolution of hosts and parasites in an attempt to improve disease management, with particular attention paid to understanding the mechanisms that generate and maintain diversity. Theoretical host-parasite models are generally developed either in a genetic context with a specific, tightly linked infection process or in an evolutionary-ecology framework with universal quantitative changes in traits. Here we have developed a model that is built within an evolutionary-ecology framework but approximates the specific-infection process more often seen in genetic models. We have found that ecological feedbacks to the selection pressures can lead to considerable diversity in both species, with strains differing in their resistance and infection ranges. We suggest that the key factors in this diversity are that the interaction is coevolutionary and that infection success is not determined by absolute rates of defense and infectivity but is all-or-nothing, depending on the relative breadths of host resistance and parasite infectivity. In a sense, our model suggests that gene-for-gene–like patterns of specificity can evolve in natural systems. Diversity does not require highly specific, gene-for-gene mutual recognition factors; instead, it requires coevolution in a system where hosts and parasites differ in their respective resistance and infection ranges, with simple, direct trade-offs between increased range and decreased fitness.

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