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Transmission rates and adaptive evolution of pathogens in sympatric heterogeneous plant populations

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Diversification in agricultural cropping patterns is widely practised to delay the build-up of virulent races that can overcome host resistance in pathogen populations. This can lead to balanced polymorphism, but the long-term consequences of this strategy for the evolution of crop pathogen populations are still unclear. The widespread occurrence of sibling species and reproductively isolated sub-species among fungal and oomycete plant pathogens suggests that evolutionary divergence is common.

This paper develops a mathematical model of host–pathogen interactions using a simple framework of two hosts to analyse the influences of sympatric host heterogeneity on the long-term evolutionary behaviour of plant pathogens. Using adaptive dynamics, which assumes that sequential mutations induce small changes in pathogen fitness, we show that evolutionary outcomes strongly depend on the shape of the trade-off curve between pathogen transmission on sympatric hosts. In particular, we determine the conditions under which the evolutionary branching of a monomorphic into a dimorphic population occurs, as well as the conditions that lead to the evolution of specialist (single host range) or generalist (multiple host range) pathogen populations.

Keywords: pathogen evolution; transmission rates; host heterogeneity; trade-offs; adaptive dynamics; sibling species

1. INTRODUCTION

It has long been recognized that monocultures of genetically uniform crops impose strong selection pressures on pathogens to overcome host resistance to infection (Leonard 1977). This has led to an agricultural strategy of diversification, involving spatial and temporal heterogeneity in cropping patterns to delay the build-up of virulence races in pathogen populations that can overcome host resistance. Although this is known to promote balanced polymorphism in pathogens where there is a simple gene-for-gene interaction with the host (Frank 1993), the long-term consequences of diversifying cropping patterns for the evolution of crop pathogen populations are still unclear. We do not know how introducing host diversity into the landscape affects evolutionary divergence towards specialist or generalist pathogens, or even to a switch from pathogenicity on one host to another. The widespread occurrence of sibling species and reproductively isolated sub-species amongst fungal and oomycete plant pathogens (Brasier 1987) suggests that evolutionary divergence is common. Many of these species are listed in Brasier (1987): they include many common and widely distributed, economically important plant pathogens with host-specific *formae speciales*, including powdery mildews, rusts and species of *Fusarium* and *Phytophthora*; they also include pathogens with host-specific anastomosis groups such as the ubiquitous damping-off fungus, *Rhizoctonia solani*. Some of these have frequent sexual phases, notably the powdery mildew fungus *Blumeria graminis* f. sp. *tritici* and *B. graminis* f. sp. *hordei*, which are specialized respectively on wheat and barley. Others, typified by yellow rust *Puccinia striiformis* f. sp. *tritici* and *P. striiformis* f. sp. *hordei*, have no known sexual stage. Because the above pairs are morphologically indistinguishable, it is very likely that they have evolved from a common ancestor. Moreover, it is possible that agricultural practices such as crop diversification could lead to the emergence of new pathogen species, but also to a host range expansion of an existing pathogen. Given that many plant pathogens have relatively short generation times, allowing sometimes more than 20–30 generations in a season, these evolutionary changes may occur in the order of decades rather than centuries. Understanding the mechanisms that govern pathogen evolution is therefore an important practical problem in assessing the sustainability of disease control.

Here, we develop an epidemiological model to study the effect of sympatric host populations, characterized by the agricultural mosaic of fields containing different crop varieties, on the evolution of plant pathogen populations. For simplicity we consider two sympatric host species; however, similar studies could be conducted for the systems with higher degrees of host heterogeneity. The model, in which we consider the fitness of successive mutant strains to invade a succession of resident strains, is based upon adaptive dynamics (Metz *et al.* 1996; Dieckmann 1997; Geritz *et al.* 1998; Doebeli & Dieckmann 2000; Nowak & Sigmund 2004). We use the model

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to show how the trade-off in pathogen transmission between the two hosts affects the outcome of evolutionary change in the pathogen population. The transmission rate encompasses spore production, dispersal and infection that collectively influence secondary infection from infected to susceptible hosts.

Previous theoretical studies on the role of host heterogeneity in plant pathogen diversity have focused on models for gene frequency that ignore population and epidemiological dynamics (Leonard 1997), or on models that focus on competing pathogen strains that differ markedly in virulence (Leonard 1997). These models analyse changes in allele frequency, representing two pathogen strains (virulent and avirulent) in a population of hosts that differ in susceptibility. Studies on the role of host heterogeneity in the diversity of human and animal pathogens have also focused on competition between virulent and avirulent strains that affect transmission (Gupta & Hill 1995), or on contemporary exposure of two hosts to multiple strains that differ in virulence (Regoes *et al.* 2000). These, together with the plant models, show how genetic and epidemiological mechanisms can maintain polymorphisms for pathogen strains when there is differential host susceptibility. Strikingly, Regoes *et al.* (2000) showed competitive exclusion with the strain with the highest basic reproductive number, weighted for the two hosts, outcompeting all other strains. Coexistence occurred only when differences in transmission rates were correlated with differences in virulence (Regoes *et al.* 2000). All of these studies, however, omit the evolutionary time-scale in considering contemporary occurrence of strains. Consequently, the course of evolution cannot be observed over time and the evolving populations can only be described at an evolutionary endpoint.

The adaptive dynamics approach (Metz *et al.* 1996; Dieckmann 1997; Geritz *et al.* 1998; Doebeli & Dieckmann 2000) used here adopts an epidemiological framework that takes account of the density of infected and susceptible hosts and sets it in an evolutionary framework. The epidemiological processes are represented by host–pathogen interactions, whereas the evolutionary process is represented by a sequence of mutations from clonal reproduction that successively challenge the resident pathogen population. This approach assumes that mutations have relatively small effects on the phenotype and occur sufficiently infrequently that the population has reached a steady state before a new mutation occurs. This allows us to incorporate an 'environmental' feedback, whereby the ability of a mutant to invade the resident population depends on the conditions set out by the resident. The fate of the mutant is analysed using pairwise invasibility plots (PIPs) (Dieckmann 2002). If the mutant manages to replace the resident, it becomes the new resident and in turn sets out new conditions for invasibility of new mutants, forming a feedback loop. The evolutionary dynamics are governed by a trade-off relationship, where an increase in the pathogen transmission on host 1 leads to a decrease in the pathogen transmission on host 2. We address the following questions regarding the trade-off in pathogen transmission on two hosts: (i) what are the conditions for the occurrence of evolutionary branching and the divergence of pathogen populations, and (ii) what are the conditions for the evolution of specialist (single-host range) or generalist (two-host range) pathogen populations?

2. THE MODEL

In the modelling procedure we separate the ecological and the evolutionary time-scales and assume that the population dynamics occur on an ecological time-scale that is much faster than the evolutionary time-scale (Roughgarden 1983).

(a) *The host–pathogen dynamics*

We consider a system of two plant hosts and a resident pathogen species, assuming that the pathogen is a microparasite pathogen numbers are therefore not represented explicitly; Anderson & May (1981). We assume that the two hosts are grown on non-overlapping agricultural fields and therefore that there is no between-host competition.

Depending on the site of pathogen activity, the host population can be defined relative to small units such as roots, stems, leaves or other organs (Gilligan *et al.* 1997). Using the whole plant as the unit of measurement is not appropriate in this case because the majority of activities between hosts and pathogens occurs within the plant. Consequently, the disease impact on the agricultural yield is closely linked to the amount of tissue lost because of disease, whereas the number of plants within fields is not usually affected.

Owing to complexities associated with combining ecological and evolutionary processes, multiple infections are not included in this study. This assumption makes the problem more tractable for analysis, but also allows results to be clearly interpreted in terms of the underlying mechanisms.

Therefore, we consider a model with state variables H_i, representing the density of healthy tissue of host i (with $i = 1, 2$), and P, representing the density of infected plant tissue from both hosts. We assume that the rate at which host tissue becomes infective is proportional to the density of infected tissue and that there is no recovery from infection.

Hence, the dynamics of two hosts interacting with the resident pathogen is formulated as follows:

$$\frac{\mathrm{d}H_1}{\mathrm{d}t} = r_1 - \mu H_1 - x_1 P H_1,$$

$$\frac{\mathrm{d}H_2}{\mathrm{d}t} = r_2 - \mu H_2 - f(x_1) P H_2,$$

$$\frac{\mathrm{d}P}{\mathrm{d}t} = x_1 P H_1 + f(x_1) P H_2 - \Gamma P, \tag{2.1}$$

where r_i denotes the planting rate of the host population i with $i = 1, 2$. This term has been chosen to reflect planting in agricultural systems, where the planting is controlled manually and is not a function of the plant density. Note that a similar form can be found in the model of experimental epidemiology in Anderson & May (1979), where the host species is introduced into the system manually at a constant rate, and more recently in Bonhoeffer & Nowak (1994). The parameter μ represents the harvest rate, while the mortality rate of infected hosts, Γ, incorporates both the harvest rate and disease-induced mortality. Because of the nature of system (2.1), if the mortality rate of the infected hosts is sufficiently large the density of the infected plant tissue decreases to zero, which in turn leads

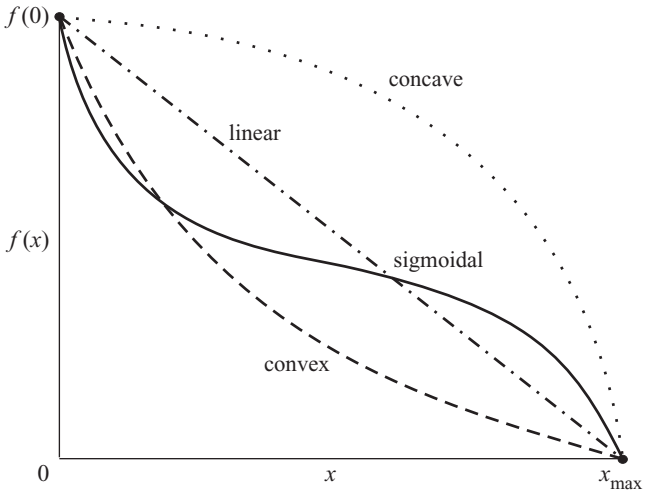

Figure 1. Shapes of the trade-off curves: pathogen transmission on host 1, denoted by x, against pathogen transmission on host 2, denoted by $f(x)$.

to an extinction of the healthy population. Therefore, to study the evolution of pathogens, we assume, throughout the paper, that Γ is sufficiently small for a disease-mediated non-trivial coexistence steady state of system (2.1) to be possible. The parameter x_1 represents the pathogen transmission rate on host 1, while $f(x_1)$ represents the pathogen transmission rate on host 2. These transmission rates encompass spore production, dispersal and infection that collectively influence secondary infection from infected to susceptible hosts. We assume a trade-off relationship described by the function f, as shown in figure 1, where an increase in transmission on host 1 carries a cost in terms of reduced transmission on host 2. Note that all parameter values in system (2.1) are positive and their choice has been motivated by parameter estimates in Van den Bosch *et al.* (1988), Campbell & Madden (1990), Gilligan *et al.* (1997) and Gubbins & Gilligan (1997).

Because there will be a biologically feasible maximum to any transmission rate, we denote, by $x_{\max}$ and $f(0)$, the maximal pathogen transmission rates on hosts 1 and 2, respectively.

(b) *The evolutionary dynamics*

Let x denote an element of $\Omega = (0,\ x_{\max})$, which represents a one-dimensional phenotypic trait space. We choose pathogen transmission on host 1 to be the evolving trait, noting that the evolution of pathogen transmission on host 2 follows from the trade-off relationship (see figure 1). At $x = 0$, the pathogen is completely specialized to host 2 with transmission rate $f(0) \neq 0$, while at $x = x_{\max}$ the pathogen is completely specialized to host 1 with transmission rate $x_{\max} \neq 0$.

We consider the effect of adding a mutant pathogen P_{m}, with phenotypic characteristic $x_2 \in \Omega$, into the resident pathogen population P, with phenotypic characteristic $x_1 \in \Omega$, in the mutation-free system (2.1). We assume that system (2.1) has a locally stable steady state at which the two hosts coexist with the resident pathogen $(H_1^*(x_1),\ H_2^*(x_1),\ P^*)$. The equations for the new (mutated) system are given by:

$$\frac{\mathrm{d}H_1}{\mathrm{d}t} = r_1 - \mu H_1 - x_1 P H_1 - x_2 P_{\mathrm{m}} H_1, \tag{2.2a}$$

$$\frac{\mathrm{d}H_2}{\mathrm{d}t} = r_2 - \mu H_2 - f(x_1) P H_2 - f(x_2) P_{\mathrm{m}} H_2, \tag{2.2b}$$

$$\frac{\mathrm{d}P}{\mathrm{d}t} = x_1 P H_1 + f(x_1) P H_2 - \Gamma P, \tag{2.2c}$$

$$\frac{\mathrm{d}P_{\mathrm{m}}}{\mathrm{d}t} = x_2 P_{\mathrm{m}} H_1 + f(x_2) P_{\mathrm{m}} H_2 - \Gamma P_{\mathrm{m}}. \tag{2.2d}$$

The fitness of the invader is the largest eigenvalue of equations (2.2) at the steady state $(H_1^*(x_1),\ H_2^*(x_1),\ P^*,\ 0)$ (see Rand *et al.* (1994)), and is denoted by $\lambda_{x_1}(x_2)$ which takes the following form:

$$\lambda_{x_1}(x_2) = x_2 H_1^*(x_1) + f(x_2) H_2^*(x_1) - \Gamma. \tag{2.3}$$

For a discussion of the notion of fitness see Metz *et al.* (1996). The invader's success will depend on its fitness in the following way: an invader with phenotypic characteristic x_2 when initially rare will be able to invade the resident population with phenotypic characteristic x_1 if $\lambda_{x_1}(x_2) > 0$. Alternatively, if $\lambda_{x_1}(x_2) < 0$, the invading population will die out.

A phenotypic value for which the local fitness gradient is zero is called an 'evolutionarily singular strategy' (Metz *et al.* 1996). According to Metz *et al.* (1996) and Geritz *et al.* (1998), at a singular strategy several evolutionary outcomes are possible. A singular strategy can: lack convergence stability and therefore act as an evolutionary repeller; be both evolutionarily and convergence stable and therefore be the final outcome of the evolution (also called 'continuously stable strategy'); and, finally, be convergence stable but not evolutionarily stable, in which case it is called a 'branching point'.

These classifications are based on the assumption that, away from a singular strategy, the principle of mutual exclusion holds so that, after a successful invasion, the nearby invading population takes over and replaces the resident population. However, in a small neighbourhood of a singular strategy, the successful invasion by a nearby mutant can, under certain conditions, result in the coexistence of the invader and of the resident type populations (Geritz *et al.* 1998).

3. MODEL OUTCOMES

The outcomes of the evolution of a pathogen population is investigated graphically using PIPs (Metz *et al.* 1996; Dieckmann 1997; Geritz *et al.* 1998). In this paper, the PIP is a graphical representation of the sign of the fitness of an invader population, with the phenotypic characteristic x_2 in a locally stable coexistence between two hosts and the pathogen population, with the resident phenotypic characteristic x_1, for a fixed set of model parameters. The mutant's fitness and its sign are plotted as a function of x_1 and x_2 (figure 2). Identical calculations have been carried out for a wide range of parameter values and trade-off curves, and the results obtained were of the same nature as those described in this section. For ease of interpretation and illustration, we also include numerical simulations of evolutionary trajectories over time (see figure 3).

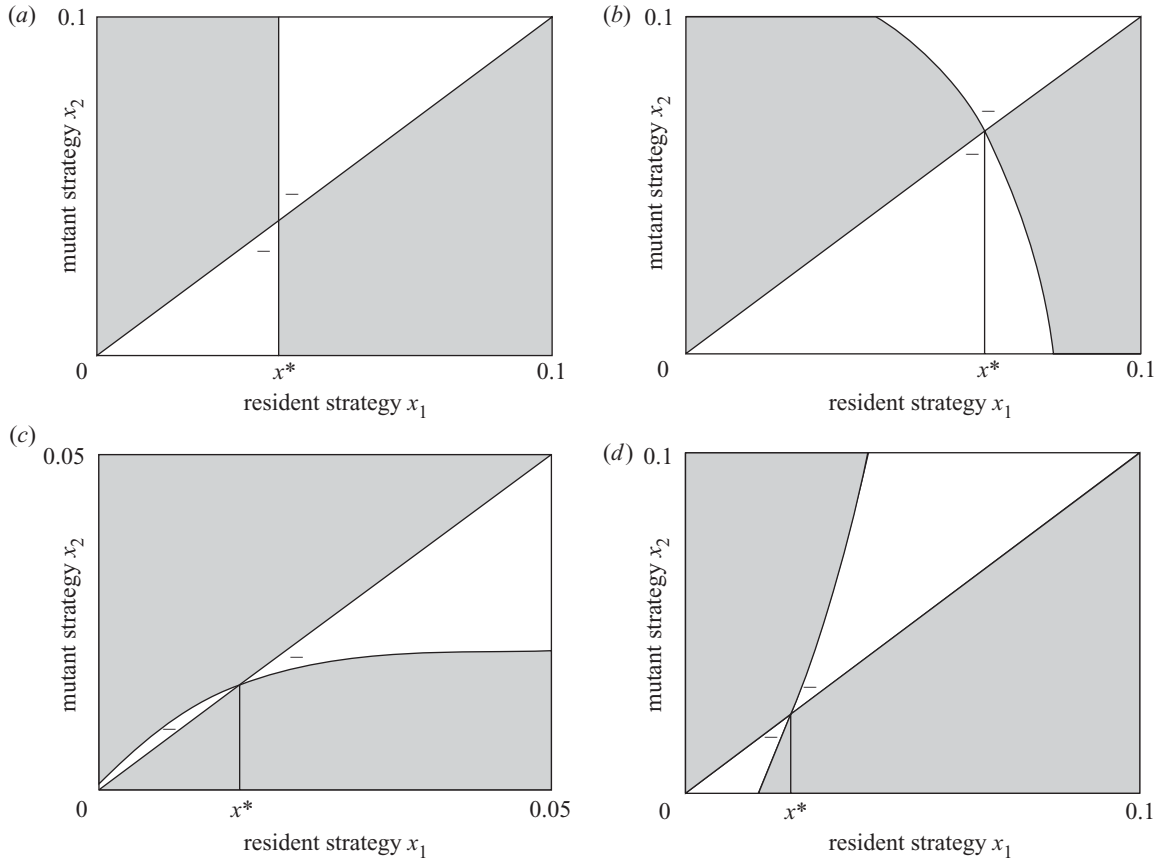

Figure 2. A PIP. The resident and mutant strategies are denoted by x_1 and x_2, respectively. The shaded areas indicate the combinations of x_1 and x_2 for which the fitness of the mutant, $\lambda_{x_1}(x_2)$, is positive. The singular strategy is denoted by x^*. (*a*) Linear trade-off, $f(x) = 0.1 - x$; (*b*) concave trade-off, $f(x) = 0.12(x - 0.1)/(x - 0.12)$; (*c*) steep convex trade-off, $f(x) = -0.000\,04(x - 0.1)/(x + 0.000\,04)$; and (*d*) shallow convex trade-off, $f(x) = -0.04(x - 0.1)/(x + 0.04)$. Model parameters: $r_1 = 1$, $r_2 = 1.5$, $\mu = 0.001$ and $\Gamma = 0.5$.

A singular point, x^*, is an evolutionary stable strategy (ESS) if $\partial^2\lambda_{x_1}(x_2)/\partial x_2^2 < 0$ at $x_1 = x_2 = x^*$ (Metz *et al.* 1996), and from equation (2.3) it follows that the sign of $f''(x^*)$ determines whether a singular strategy x^* is an ESS. Therefore, for different shapes of the trade-off curve—for example, $f''(x) = 0$ (linear trade-off), $f''(x) < 0$ (concave trade-off) and $f''(x) > 0$ (convex trade-off)—different evolutionary outcomes can be expected.

In the case of a linear trade-off (figure 1), the PIP shows that the singular strategy x^* is convergent stable but not an ESS (figure 2*a*). Furthermore, the singular strategy is evolutionarily attainable, in other words it can invade other strategies when initially rare. Once the singular strategy has been established, all mutations are neutral and although mutual invasibility near the strategy is possible, the branching does not occur (Geritz *et al.* 1998).

In the case of a concave trade-off (figure 1), the PIP shows that the singular strategy x^* is an ESS, is convergence stable and that a pathogen with the phenotypic strategy x^* can invade nearby strategies when rare itself (figure 2*b*). Therefore, x^* is an evolutionary endpoint and is not invadable by any other mutant strategies. In this case an initially monomorphic pathogen population will stay monomorphic, evolving until the transmission on host 1 reaches x^*. This outcome is illustrated in figure 3*a* using numerical simulations described in Gudelj *et al.* (2004).

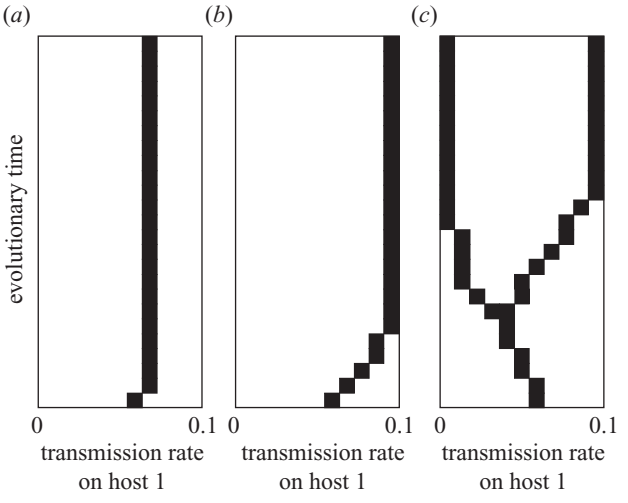

Figure 3. Simulations of the evolutionary dynamics of the transmission rate on host 1. (*a*) Concave trade-off, $f(x) = 0.12(x - 0.1)/(x - 0.12)$; (*b*) steep convex trade-off, $f(x) = -0.000\,04(x - 0.1)/(x + 0.000\,04)$; and (*c*) shallow convex trade-off, $f(x) = -0.04(x - 0.1)/(x + 0.04)$. Model parameters: $r_1 = 1$, $r_2 = 1$, $\mu = 0.001$ and $\Gamma = 0.1$.

In the case of a convex trade-off (figure 1), the PIP shows that two different evolutionary outcomes are possible. If, for small values of x, $f''(x)$ is sufficiently large (steep

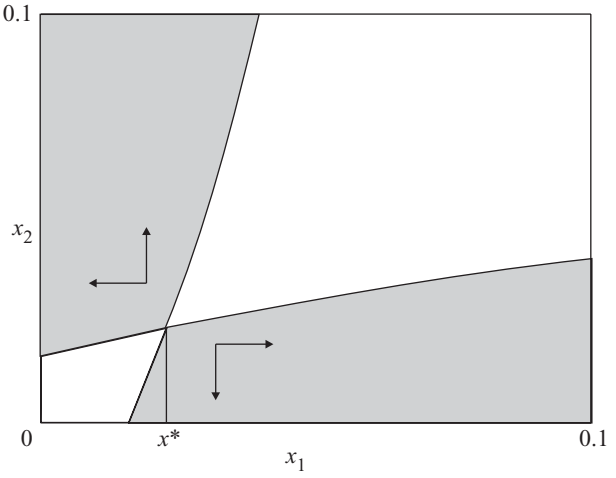

Figure 4. Evolution in a dimorphic population. The shaded area denotes sets of pairs (x_1, x_2), denoting pathogen transmission on host 1 and host 2, respectively, coexisting in a protected dimorphism. The singular strategy is denoted by x^*, while the model parameters are: $r_1 = 1, r_2 = 1.5, \mu = 0.001$ and $\Gamma = 0.5$.

convex) the singular point x^* is not an ESS, is not convergence stable and therefore acts as an evolutionary repeller (figure 2*c*). Therefore, depending on the value of the initial resident phenotypic trait, the evolutionary outcome will be a monomorphic population of pathogens only infective to host 1 or only infective to host 2 (figure 3*b*).

However, if, for small small values of x, $f''(x)$ is sufficiently small (shallow convex), the singular point x^* is not an ESS but is convergence stable, and in the vicinity of x^* a dimorphism can occur: such x^* is termed an evolutionary branching point (figure 2*d*). In this case an initially monomorphic population will approach the singular point and will undergo disruptive selection, becoming dimorphic and comprising two closely related resident phenotypic traits (see numerical simulations in figure 3*c*). We can show analytically (see Appendix A) that, through a series of small evolutionary steps, the two resident phenotypic traits will grow apart until they reach the boundary of the phenotypic domain Ω. On the boundary the two resident pathogen groups are completely host specialized, one infecting only host 1 and the other infecting only host 2. In this case we can also show (Appendix A) that if a dimorphic pathogen population consists of phenotypic traits that are completely host specialized, no mutant with non-zero transmission on both hosts is able to invade. The direction of evolution of each of the two residents is illustrated in figure 4. The shaded area represents a set of strategy pairs (x_1, x_2) that coexist in a protected dimorphism. Horizontal arrows represent the evolutionary direction of x_1, while vertical arrows represent the evolutionary direction of x_2. The final resting point of the dimorphic population is at the boundary of the phenotypic space, with each resident becoming host specialized and therefore present on only one host.

In the case of a sigmoidal (convex–concave) curve (see figure 1), the following two outcomes are possible. If $f''(x)$ is sufficiently small for small x (shallow convex–concave), essentially the same results are found as with the concave function (figure 2*b*). However, if $f''(x)$ is sufficiently large for small x (steep convex–concave), the PIP shows that two

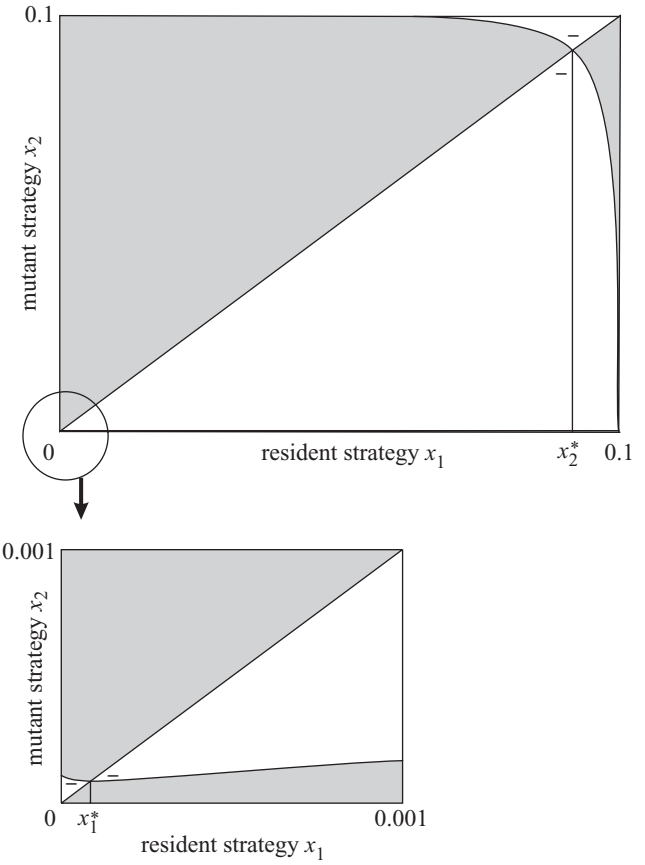

Figure 5. A PIP. The resident and mutant strategies are denoted by x_1 and x_2, respectively. The shaded areas indicate the combinations of x_1 and x_2 for which the fitness of the mutant, $\lambda_{x_1}(x_2)$, is positive. The singular strategy is denoted by x^* and the model parameters are $r_1 = 1, r_2 = 1.5$, $\mu = 0.001$ and $\Gamma = 0.5$. The trade-off curve is sigmoidal: for $0 \leqslant x \leqslant 0.04$, $f(x) = 0.0397 + 0.120610^{-4}/(x + 0.0002)$ and for $0.04 \leqslant x \leqslant 0.1$, $f(x) = 0.04045 + 0.275810^{-4}/(x - 0.1007)$.

singular strategies, an evolutionary repeller, x_1^*, and a continuously stable strategy, x_2^*, are present (figure 5). Therefore, depending on the value of the initial resident phenotypic trait, an initially monomorphic population will, through a series of small evolutionary steps, become either completely specialized on host 2 or infective to both hosts with transmission rates x_2^* and $f(x_2^*)$ on host 1 and host 2, respectively (see figure 6*a* for an illustration). Note that qualitatively similar results hold for sigmoidal (concave–convex) curves.

4. DISCUSSION

This paper considers the evolution of a pathogen population exposed to two different hosts and demonstrates that the outcomes strongly depend on the shape of the trade-off curve for the transmission rates on the different hosts. Evolutionary branching does not occur for linear, concave, steep convex and sigmoidal trade-off curves. In these cases, an initially monomorphic pathogen population remains monomorphic and its host range depends on the shape of the trade-off in the following ways. A generalist population occurs for both linear and concave trade-offs (see figure 2*a*,*b*). A steep convex trade-off leads to a specialist population (see figure 2*c*), whereas a sigmoidal trade-off leads to

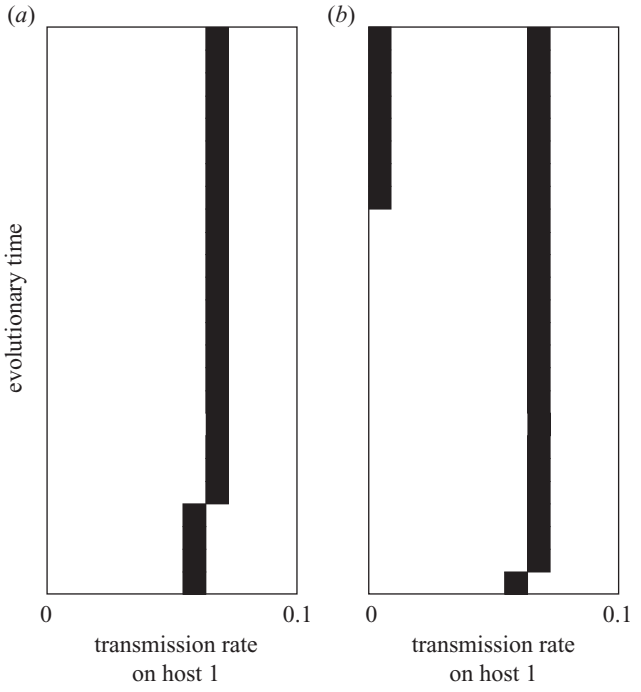

Figure 6. Simulations of the evolutionary dynamics of the transmission rate on host 1 for the sigmoidal trade-off $f = 50(0.001 - (2x - 0.1)^3)$. (*a*) Local mutations and (*b*) global mutations. Model parameters are $r_1 = 1$, $r_2 = 1$, $\mu = 0.001$ and $\Gamma = 0.1$.

either a generalist or a specialist population, depending on the value of the initial phenotypic trait (see figure 5).

We also found that the evolutionary branching of a monomorphic into a dimorphic population occurs only for a shallow convex trade-off curve (see figure 2*d*). In this case, the evolutionary outcome is a pathogen population consisting of two groups, each of which is specialized on one of the hosts. Examples of such divergent groups can be found in the *Blumeria* genus (Hiura 1978) where *B. graminis f.* sp. *hordei* is specialized on barley whereas *B. graminis f.* sp. *tritici* is specialized on wheat, and in the *Phytophthora* genus where *P. medicaginis* and *P. trifolii* are specialized on chickpea and clover, respectively.

Owing to its importance in epidemics, the pathogen transmission was chosen as the evolving trait and a trade-off relationship between the transmissions on different hosts was imposed. Regoes *et al.* (2000) considered the evolution of pathogen virulence using the community dynamics approach, in which the authors found that the presence of a trade-off relationship between the virulence on different hosts was not sufficient for multiple strain coexistence, even though two different host types were present in the system. Coexistence was possible only when virulence was correlated with transmission, and we conclude that transmission is crucial for the coexistence of multiple strains.

In this study the analysis rests on the use of the adaptive dynamics method. One shortcoming of this approach is that it assumes that mutations from clonal reproduction induce only small phenotypic changes (local mutations). This is somewhat simplistic because experimental studies demonstrate that the offspring of a plant pathogen can sometimes be phenotypically very different from its parent (*global mutations*). However, the results of computer simulations (Gudelj *et al.* 2004) show that the evolutionary outcomes do not qualitatively change when both global and local mutations are taken into account. Global mutations are expected to influence the outcome only in the sigmoidal (steep convex–concave) case where the following is anticipated. Instead of a monomorphic generalist or a monomorphic specialist population (see figure 5), a dimorphism of the two cases where a generalist and a specialist population coexist can be observed. This is possible because global mutations will eventually give rise to a phenotype that is situated on the opposite side of the repeller x_1^* (figure 5) to the resident phenotype. For an illustration of this outcome see figure 6*b*. Examples of such pathogen groups can be found within the *Botrytis* genus (Holliday 1989) where *B. cinerea* is a generalist whereas *B. fabae* is a specialist. It is important to note, however, that without the analytical advantages of the adaptive dynamics which clearly identify the importance of the trade-off shape, the computational study in Gudelj *et al.* (2004) lacks generality.

Classical evolutionary ecology (see Levins 1968) has also observed that convexity and concavity of trade-offs are important in determining the outcome of evolution. Contrary to the adaptive dynamics, the classical approach does not include environmental feedback and is based on a rather simplified view of the evolutionary processes, which rests on the assumption that the result of evolution is to realize an optimum strategy, a quantity that is maximized during the course of evolution. For example, the outcome of pathogen evolution is often predicted by maximizing the basic reproductive ratio, R_0, of that pathogen (Anderson & May 1982). Comparing the results presented in this paper with the predictions of the R_0 maximization we find that when the trade-off is concave, both approaches predict the evolution of a monomorphic generalist pathogen population. However, the differences arise when the trade-off is convex, and in this case the R_0 maximization predicts the evolution of a monomorphic specialist pathogen population specializing on the host with the highest value of r. Contrary to this, the adaptive dynamics method indicates that the presence of a dimorphic pathogen population consisting of two specialized groups is also possible. Note that the outcomes of the adaptive dynamics and the R_0 maximization approaches are in complete agreement only when $r_1 = r_2$.

There is an increasing number of theoretical studies indicating that trade-offs are an important component of those mechanisms that govern the evolution of not only pathogen but also host populations. While this paper demonstrates the importance of trade-offs in the evolution of pathogen populations, the results in Boots & Haraguchi (1999) and Bowers & Hodgkinson (2001) demonstrate similar trends in the evolution of host populations. Both Boots & Haraguchi (1999) and Bowers & Hodgkinson (2001) considered the evolution of host resistance to microparasitic infection, assuming a trade-off between host resistance to a pathogen and host intrinsic growth rate.

Formulation and implementation of experiments which could determine the shape of a particular trade-off relationship is a complex process, and although there have been some attempts to measure various trade-off relationships in host–pathogen systems for invertebrate and vertebrate host species (Anderson & May 1982; Boots & Haraguchi 1999),

there has been very little attempt to estimate trade-off relationships within plant–pathogen systems.

Understanding infection mechanisms of a particular plant–pathogen system may help to infer the shape of a trade-off curve. For example, mechanisms that increase the pathogen transmission on a particular host with very little initial cost but which become more costly as the pathogen transmission increases, would lead to progressive diminution of transmission costs (a concave trade-off). These mechanisms could be associated with changes in plant–pathogen relationships that have additive gene action.

Similarly, mechanisms that increase the pathogen transmission on a particular host and are costly to produce, but, once produced, increase the transmission over a large range at little additional cost, would lead to progressive diminution of transmission costs (a convex trade-off). For example, such a mechanism could relate to the changes in plant–pathogen gene-for-gene relationships (Crute 1994).

Therefore recent advances in molecular techniques (Idnurm & Howlett 2001; Van't Slot & Knogge 2002) could soon improve our understanding of biochemical processes that underline plant–pathogen trade-off relationships. In turn, this can aid the development of resistant crops that minimize evolutionary pressures on the pathogen to adapt to the resistant cultivar, or to adapt through evolutionary branching (emergence of new resistant strains). In the long term, such advances will help the development of sustainable agricultural systems.

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APPENDIX A

(a) *Dimorphic populations*

Suppose that an evolutionary branching near the singular point has occurred and that the pathogen population is in the new resident system

$$\frac{dH_1}{dt} = r_1 - \mu H_1 - x_1 P_1 H_1 - x_2 P_2 H_1,$$

$$\frac{dH_2}{dt} = r_2 - \mu H_2 - f(x_1) P_1 H_2 - f(x_2) P_2 H_2,$$

$$\frac{dP_i}{dt} = x_i P_i H_1 + f(x_i) P_i H_2 - \Gamma P_i, \qquad \text{(A 1)}$$

with phenotypic characteristics x_i, where $i = 1, 2$. Without loss of generality, we assume that $x_1 < x_2$. The above system (A 1), has a non-trivial coexistence steady state $(H_1^\dagger, H_2^\dagger, P_1^\dagger, P_2^\dagger)$, of the form

$$\left(\frac{\Gamma(f(x_2) - f(x_1))}{f(x_2)x_1 - x_2 f(x_1)}, \frac{\Gamma(x_1 - x_2)}{f(x_2)x_1 - x_2 f(x_1)}, P_1^\dagger, P_2^\dagger\right). \qquad \text{(A 2)}$$

A mutant pathogen with the phenotypic characteristic x_3 is introduced in a small neighbourhood of the resident phenotypic traits x_1 or x_2, and its fitness is denoted by

$$\lambda_{x_1, x_2}(x_3) = x_3 H_1^\dagger(x_1, x_2) + f(x_3) H_2^\dagger(x_1, x_2). \qquad \text{(A 3)}$$

We distinguish between the following two cases. The first case assumes that the phenotypic characteristic x_3, of the mutant pathogen is a small perturbation of the resident characteristic x_1, whereas the second case assumes that x_3 is a small perturbation of x_2.

(i) *Case I*

The mutant's phenotypic characteristic is of the form $x_3 = x_1 \pm \varepsilon$, for some small $\varepsilon > 0$. In this case using the standard Taylor's expansion, the mutant fitness, equation (A 3) becomes

$$\lambda_{x_1, x_2}(x_1 \pm \varepsilon) = \pm\varepsilon(H_1^\dagger + f'(x_1) H_2^\dagger),$$

for small ε.

(ii) *Case II*

The mutant's phenotypic characteristic is of the form $x_3 = x_2 \pm \varepsilon$, for some small $\varepsilon > 0$. Again, using the standard Taylor's expansion, the mutant fitness, equation (A 3) becomes

$$\lambda_{x_1, x_2}(x_2 \pm \varepsilon) = \pm\varepsilon(H_1^\dagger + f'(x_2) H_2^\dagger),$$

for small ε.

Therefore, for $i = 1, 2$,

$$H_1^\dagger + f'(x_i) H_2^\dagger = \frac{\Gamma(f(x_2) - f(x_1) + f'(x_i)(x_1 - x_2))}{f(x_2)x_1 - x_2 f(x_1)}, \qquad \text{(A 4)}$$

and using the mean value theorem there exits $\theta \in (x_1, x_2)$ such that

$$\frac{f(x_2) - f(x_1)}{x_2 - x_1} = f'(\theta). \qquad \text{(A 5)}$$

Recalling our assumptions that $x_1 < x_2$ and f is monotone decreasing function $(f' \leqslant 0)$, we can conclude that

$$f(x_2)x_1 - x_2 f(x_1) < 0. \qquad \text{(A 6)}$$

Because $f'' > 0$, it follows that f' is a monotone increasing function, in other words because $x_1 < \theta < x_2$ it follows that $f'(x_1) < f'(\theta) < f'(x_2)$, which when combined with equation (A 5) gives

$$f'(x_1) < f'(\theta) = \frac{f(x_2) - f(x_1)}{x_2 - x_1} < f'(x_2).$$

The above inequality together with equations (A 4) and (A 6) give

$$H_1^\dagger + f'(x_1) H_2^\dagger < 0 < H_1^\dagger + f'(x_2) H_2^\dagger,$$

which is used to conclude that

$$\lambda_{x_1, x_2}(x_1 - \varepsilon) > 0 \text{ and } \lambda_{x_1, x_2}(x_1 + \varepsilon) < 0, \qquad \text{(A 7)}$$

and similarly,

$$\lambda_{x_1, x_2}(x_2 - \varepsilon) < 0 \text{ and } \lambda_{x_1, x_2}(x_2 + \varepsilon) > 0. \qquad \text{(A 8)}$$

(b) *The case of host specialization*

Consider the host specialized system where $x_{\max} > 0$ denotes the transmission rate of a pathogen completely specialized to host 1, while $f(0)$ denotes the transmission rate of a pathogen completely specialized to host 2. In this case the resident system equation (A 1), with $x_1 = x_{\max}$, $f(x_1) = 0$, $x_2 = 0$, $f(x_2) = f(0)$, has a non-trivial locally stable steady state of the form $(\Gamma/x_{\max}, \Gamma/f(0), P_1^*, P_2^*)$. If a small mutant population that has a non-zero transmission rate on both hosts is introduced into the resident system, its fitness function will be

$$\lambda(x) = x\frac{\Gamma}{x_{\max}} + f(x)\frac{\Gamma}{f(0)} - \Gamma.$$

Because $f''(x) < 0$, it follows that $f(x) < f(0) - \frac{f(0)}{x_{\max}} x$ and hence $\lambda(x) < 0$.

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