Resistance May Be Futile: Dispersal Scales and Selection for Disease Resistance in Competing Plants

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Abstract

If a host species shares a pathogen with competing species, the disease may provide a net benefit. Selection for resistance will depend on the trade–off between the damage done by the disease and the positive effects resulting from infection of competitors. This paper presents a simple, spatially explicit model of a plant that shares a disease with a superior competitor. The phenotypic evolution of cost–free resistance is studied by using the method of pair approximation to analyze the small scale spatial structure of the interacting populations. Selection favors lower resistance when disease transmission is spatially local and the damage to the competitor is sufficient to outweigh the direct effects of infection. This suggests that local spatial structure may be critical in determining the coevolution of host–host–pathogen systems.

Introduction

When competing species share a common enemy, the interaction between direct competition for resources and apparent competition mediated by the predator or pathogen can have important ecological and evolutionary consequences (Park, 1948; Price *et al.*, 1986, 1988; Begon & Bowers, 1995; Grosholz, 1992; Yan, 1996; Alexander & Holt, 1998). For example, it is not uncommon for directly competing plant species to share a pathogen which can infect them both (Rice & Westoby, 1982; Clay, 1990). If a pathogen infects a host's competitors, it may directly harm that host while indirectly benefitting it by reducing competitive pressure. At the same time, coevolutionary forces acting on plants and their pathogens are thought to be strong; pathogen–specific resistance to infection is an important feature of plant evolution (Burdon, 1987; Mitchell–Olds & Bergelson, 2000; Richter & Ronald, 2000; Moran, 2002). The evolution of resistance may be influenced by the tradeoff between direct damage and indirect benefits that the pathogen confers to a host. A natural question is: when will the benefits exceed the damage done by the disease, thereby selecting for reduced resistance? This paper addresses this issue by presenting a model which predicts the evolution of disease resistance in a host that shares the pathogen with a superior competitor.

A number of theoretical studies have addressed the evolution of disease resistance in single host systems. If there is no cost of resistance, selection typically favors ever increasing resistance (Boots & Haraguchi, 1998), although Damgaard (1999) has shown that polymorphism for cost–free resistance may be maintained in a metapopulation. Despite the apparent selective advantage of resistance, every species remains susceptible to infection by some suite of pathogens. Several explanations for this have been proposed. First, resistance may come at a cost, such as reduced fecundity or lifespan (reviewed in Purrington, 2000; Zuk & Stoehr, 2002). In that case, tradeoffs between the costs and benefits of resistance may lead to selection for an intermediate resistance level and/or polymorphism (Antonovics & Thrall, 1994; Bowers *et al.*, 1994; Boots & Haraguchi, 1999). Second, selection for resistance may be countered by selection for virulence in the pathogen, leading to a coevolutionary "arms race" in which the host cannot achieve total resistance (Dawkins & Krebs, 1979; Mitchell–Olds &

Bergelson, 2000). This hypothesis is supported by the gene–for–gene system in which resistance alleles confer protection against pathogens with specific virulence alleles (Ellis *et al.*, 2000; Stahl & Bishop, 2000; Takken & Joosten, 2000). The interaction between these coevolutionary forces and the costs of resistance may lead to a type of "trench warfare" (Stahl *et al.*, 1999), in which resistance polymorphisms are maintained by spatial or temporal variation in selection.

Apparent competition between hosts that share a pathogen provides a third mechanism that may limit selection for disease resistance. One host may evolve low resistance if the presence of the disease is a net advantage because of the damage it does to a competitor (Rice & Westoby, 1982; Clay, 1990). This mechanism may be in effect whenever plant species interact both through apparent competition mediated by a pathogen, and through "real" competition via resource limitation or allelopathy. Because both real and apparent competition are thought to be common in natural plant communities (Connell, 1983; Connell, 1990), we conjecture that there are many systems in which both act simultaneously. However, this issue has received little attention, and thus direct support is scarce. Perhaps the best characterized examples are of root diseases in forests: soil borne fungal pathogens can spread between the root systems of multiple tree species which are likely competing. For example, the fungus *Heterobasidion annosum* is an important mortality factor for ponderosa pines (*Pinus ponderosa* Dougl.) and incense–cedars (*Calocedrus decurrens* (Torr.) Florin) in a mixed conifer forest (Rizzo *et al.*, 2000). Ponderosa pine appears to outcompete incense–cedar in the absence of disturbance, but differential mortality due to the disease can reverse this, with infected cedars outliving pines by 10–30 years. Other fungal pathogens of roots such as *Phytophthora*, *Pythium*, *Armillaria*, and *Phelinus* (Tainter & Baker, 1996) have the same potential to introduce apparent competition into direct competitive interactions in forests. Rice and Westoby (1982) argued that heteroecious rust fungi mediate apparent competition between directly competing host species. However, the extensive niche differentiation between hosts (typically, a tree is one host, while a fern, shrub, or grass is the other) makes the existence of direct competition between them doubtful in many cases.

Clay (1990) argued that closely related species are more likely to have overlapping niches and shared pathogens, suggesting that interactions between real and apparent competition may be most common between co-occurring relatives. Finally, parasitic plants may play a similar role to pathogens, by parasitizing competing host species and mediating apparent competition (Gibson & Watkinson, 1991; Matthies, 1996; Marvier, 1998).

This paper presents a model for the evolution of resistance in a host plant that shares a pathogen with a superior competitor. There are four important features of the model. First, it uses an evolutionarily stable strategy (ESS) analysis of phenotypic evolution. Although a great deal is known about the genetics of disease resistance in plants, a phenotypic approach allows us to focus on the structure of the ecological interactions without incorporating specific assumptions about the genetics. A number of studies on the evolution of resistance have used a phenotypic approach to generate robust predictions (Antononvics & Thrall, 1994; Bowers *et al.*, 1994; Boots & Haraguchi, 1998; Boots & Bowers, 1999). Omitting population genetics limits our ability to address the maintenance of polymorphisms in the population (an important aspect of resistance), but it clarifies the impact of the ecological interactions on evolutionary trends.

Second, the model does not incorporate direct costs of resistance. The previous theoretical studies of phenotypic evolution have shown that the selected level of resistance can depend critically on the costs associated with it, and there is extensive empirical evidence for costs of pathogen–specific resistance (Purrington, 2000; Zuk & Stoehr, 2002). By omitting costs from the model, we do not imply that they do not exist. Rather, we are studying in isolation a different force that acts on the evolution of resistance: an "ecological cost" (Strauss *et al.*, 1999) of resisting a pathogen that could serve as an ally. A fuller understanding of disease resistance will require the incorporation of both direct physiological costs and indirect ecological costs of resistance.

Third, we consider evolution only of a host; we do not incorporate coevolution of the pathogen. Our focus is on the selective pressures for resistance in a host; this is but one facet of the problem. There is simultaneous evolution in the pathogen (and other hosts),

with a constant feedback between these processes. However, a fully coevolutionary model would be dauntingly complex; we have the more limited goal of understanding how apparent competition affects the selective pressures on a host.

Fourth, the model is spatially explicit. A number of studies have shown that limited pathogen dispersal can have important consequences for the evolution of resistance and virulence. Large scale spatial structure can stabilize polymorphisms by decoupling coevolutionary processes across the landscape (Burdon & Thrall, 1999; Damgaard, 1999; Stahl *et al.*, 1999). At a smaller scale, spatial structure can determine the selective pressures acting on individuals, yielding ESS predictions that differ qualitatively from analogous nonspatial models (Rand *et al.*, 1995; van Baalen & Rand, 1998; Boots & Sasaki, 2000). It is this invidual–scale spatial structure that we focus on here. Rice & Westoby (1982) predicted that the dispersal scales of the hosts and pathogen stages would be critical in determining the evolutionary stability of heteroecious fungi systems. They argued that the fungus can only serve as a useful weapon for a host if spore dispersal from one host species to the other is sufficiently localized. Hosts that accomodate a pathogen with lower resistance must reap the benefits of reduced competition; this will not occur unless the pathogen primarily infects their neighbors. Thus, it is expected that small–scale spatial structure plays a key role in the evolution of resistance mediated by apparent competition.

We use the model to address the following general question: what level of resistance will be selected for in a plant that shares a pathogen with a superior competitor? Specifically, we examine how the ESS level of resistance in the "user" host depends on the following factors:

1. pathogen life history: we compare pathogens with strictly alternating host species with those that are transmitted from either host to either host;

2. dispersal scales of the pathogen and hosts: we compare local and global dispersal;

3. the level of damage caused by infection;

4. resistance by the other ("attacked") host.

Plant pathogens exhibit a wide variety of life histories, with the rate of transmission between host species dependent on a number of factors. For simplicity, we study two extreme cases: strictly alternating transmission, in which each host can only infect a host of the other species (as in certain heteroecious rust fungi), and arbitrary transmission, in which the rate of transmission depends only on the susceptible species, regardless of the infected species.

Our model is formulated as a stochastic, continuous–time process on a lattice (an interacting particle system). We analyze the model by using pair approximations, which incorporate local, pairwise spatial structure into a system of ordinary differential equations. The model is generic and the parameter values are chosen somewhat arbitrarily; thus, it is not intended to represent a particular set of species. Since we omit other aspects of the coevolutionary process, we cannot make any claims about the strength of selection due to apparent competition relative to other selective forces, such as direct costs of resistance. Rather, our focus is on how ecological and life history factors affect the selection for resistance. This provides a theoretical framework for understanding the role of apparent competition and spatial structure in the coevolution of host–host–pathogen systems.

Model

Each site in a square lattice is assumed to be in one of several states: E (empty), S_A or I_A (susceptible or infected attacked host), S_U or I_U (susceptible or infected user host). In addition, we study invasions by a new phenotype of the user host; we denote the susceptible and infected invaders S and I for simplicity. Each host and each pathogen stage either disperses locally (to the four nearest neighbors of a site) or globally (uniformly across the entire system). The attacked host is assumed to be competitively dominant; it reproduces by placing offspring on sites that are empty or occupied by the user host. For simplicity, we assume that the presence of a user host does not affect the probability of establishment by an attacked host. Conversely, user hosts reproduce only onto empty sites; they cannot colonize sites already occupied. Infection occurs when the pathogen is transmitted from an infected host to an appropriate susceptible host, and resistance fails. We assume that resistance is

a quantitative trait scaled to lie between 0 (no resistance) and 1 (total resistance). We also assume that the disease can increase the mortality rate of the hosts, but does not affect reproduction.

The model can be simulated on a computer as a spatially explicit stochastic process. However, the utility of such simulations is limited. It takes a great deal of computer time to explore parameter space, especially when evolution proceeds slowly. Moreover, it may be difficult to explain the outcomes of simulations without further analysis. Thus, we adopt the approach of studying differential equations that describe the dynamics of the populations. As we explain below, the differential equations provide exact results when dispersal is global, and useful approximations when it is local.

First consider the system when there is only one ("resident") phenotype of the user host. The probabilities that a randomly chosen site is in a particular state satisfy a set of ordinary differential equations. Let P_σ be the probability that a random site is in state σ, and $P_{\sigma|\sigma'}$ be the conditional probability that, given a site is in state σ', a randomly chosen neighbor is in state σ. Then by considering the possible changes to a state, and the rates at which they occur, we can derive equations for the rates of change of the various states. For example, consider colonization of an empty site by a susceptible attacked host. Suppose each such host produces locally dispersing offspring at rate β_A. Then any empty neighbor of an S_A site is colonized at rate $\frac{\beta_A}{4}$ (since there are four possible sites for the offspring to disperse to). Since the total number of empty neighbors of S_A sites is $4P_{E|S_A}$, this process increases the density of S_A sites at the rate $\beta_A P_{E|S_A} P_{S_A}$ (the first term in equation 1). On the other hand, if the offspring disperse globally and are produced at rate B_A, then colonization occurs at rate $B_A P_E P_{S_A}$. Incorporating all possible local and global interactions yields the resident density equations:

$$\begin{aligned}
\frac{dP_{S_A}}{dt} &= [\beta_A(P_{E|S_A} + P_{S_U|S_A} + P_{I_U|S_A}) + B_A(P_E + P_{S_U} + P_{I_U})]P_{S_A} \\
&\quad +[\beta_A(P_{E|I_A} + P_{S_U|I_A} + P_{I_U|I_A}) + B_A(P_E + P_{S_U} + P_{I_U})]P_{I_A} \\
&\quad -(1-\rho_A)[\gamma_{AA}P_{I_A|S_A} + \Gamma_{AA}P_{I_A} + \gamma_{UA}P_{I_U|S_A} + \Gamma_{UA}P_{I_U}]P_{S_A} - \mu_A P_{S_A} \qquad (1) \\
\frac{dP_{I_A}}{dt} &= (1-\rho_A)[\gamma_{AA}P_{I_A|S_A} + \Gamma_{AA}P_{I_A} + \gamma_{UA}P_{I_U|S_A} + \Gamma_{UA}P_{I_U}]P_{S_A} - \alpha_A\mu_A P_{I_A} \qquad (2)
\end{aligned}$$

$$
\begin{aligned}
\frac{dP_{S_U}}{dt} &= [\beta_U P_{E|S_U} + B_U P_E] P_{S_U} + [\beta_U P_{E|I_U} + B_U P_E] P_{I_U} \\
&\quad -[\beta_A (P_{S_A|S_U} + P_{I_A|S_U}) + B_A (P_{S_A} + P_{I_A})] P_{S_U} \\
&\quad -(1-\rho_U)[\gamma_{UU} P_{I_U|S_U} + \Gamma_{UU} P_{I_U} + \gamma_{AU} P_{I_A|S_U} + \Gamma_{AU} P_{I_A}] P_{S_U} - \mu_U P_{S_U} \qquad (3) \\
\frac{dP_{I_U}}{dt} &= (1-\rho_U)[\gamma_{UU} P_{I_U|S_U} + \Gamma_{UU} P_{I_U} + \gamma_{AU} P_{I_A|S_U} + \Gamma_{AU} P_{I_A}] P_{S_U} \\
&\quad -[\beta_A (P_{S_A|I_U} + P_{I_A|I_U}) + B_A (P_{S_A} + P_{I_A})] P_{I_U} - \alpha_U \mu_U P_{I_U} \qquad (4) \\
P_E &= 1 - P_{S_A} - P_{I_A} - P_{S_U} - P_{I_U}.
\end{aligned}
$$

Here, μ_A and μ_U are the density–independent death rates of uninfected attacked and user hosts, respectively; α_A and α_U are the factors by which infection increases host mortality – we refer to this as the damage done by the pathogen. The resistance levels of the hosts are given by ρ_A and ρ_U. The other parameters give rates of reproduction or transmission, and hence describe interactions between two sites; lower case parameters correspond to nearest–neighbor interactions, while upper case ones correspond to global dispersal. Thus, β_A is the rate at which the attacked host produces offspring which will disperse locally, while B_A is the rate at which it produces offspring which will disperse globally. The parameters $\gamma_{\sigma\sigma'}$ and $\Gamma_{\sigma\sigma'}$ give the transmission rates from a host of species σ to one of species σ'.

We assume throughout that the competitively dominant (attacked) host is much longer–lived than the user host. This assumption is only valid for a subset of the systems in which we are interested, but it does not change the model's qualitative predictions. We can rescale time by the lifespan of the organisms: in all the examples presented we set $\mu_U = 1$ and arbitrarily choose $\mu_A = 0.1$. Thus, uninfected user hosts have a mean lifespan of one time step, while attacked hosts have a lifespan of ten time steps. Note that with time rescaled in this way the system is nondimensional, since the population densities represent occupancy probabilities, lying between 0 and 1. We assume that each host species or pathogen stage has either completely local or completely global dispersal, so that for each interaction either the upper case or lower case parameter is zero. Finally, for alternating transmission we set $\gamma_{\sigma\sigma} = \Gamma_{\sigma\sigma} = 0$; i.e. transmission can only occur between different species of hosts. In the case of an arbitrarily transmitted pathogen, we set $\gamma_{\sigma\sigma'} = \gamma_{\sigma'\sigma'}$, assuming that the

transmission rate depends only on the susceptible species. In reality, the transmission rate will also depend on the rate of pathogen production by the infected species, but we do not include that complication.

If all interactions are global (the so–called "mean field" case), the terms for pairs of sites drop out and the resident density equations form a closed system. Otherwise, the equations contain the unknown conditional density terms $P_{\sigma|\sigma'}$. With a little more effort, we can write down ODEs for these terms; however, they include terms involving triplets of sites. We close the system at the level of pairs by assuming that $P_{\sigma|\sigma'\sigma''} = P_{\sigma|\sigma'}$; this is known as a pair approximation (Matsuda, 1992; Rand, 1999). Here, $P_{\sigma|\sigma'\sigma''}$ is the probability that, given a site is in state σ' and a randomly chosen neighbor is in state σ'', another randomly chosen neighbor will be in state σ. Thus, the pair approximation assumes that the state of one neighbor of a site does not depend on the states of the other neighbors. The resulting equations for the pair densities (Appendix 1) give an approximation to the local spatial structure of the system.

Once we have determined the resident densities from the mean field or pair approximation equations, we want to study the evolution of resistance by determining the ability of a different phenotype of the user host to invade. We assume that the invading phenotype differs from the resident only in its resistance, ρ'. The densities of the invading phenotype satisfy the following invasion equations:

$$\begin{aligned}
\frac{dP_S}{dt} &= [\beta_U P_{E|S} + B_U P_E]P_S + [\beta_U P_{E|I} + B_U P_E]P_I \\
&\quad -[\beta_A(P_{S_A|S} + P_{I_A|S}) + B_A(P_{S_A} + P_{I_A})]P_S - \mu_U P_S \\
&\quad -(1-\rho')[\gamma_{UU}(P_{I_U|S} + P_{I|S}) + \Gamma_{UU}(P_{I_U} + P_I) + \gamma_{AU}P_{I_A|S} + \Gamma_{AU}P_{I_A}]P_S \qquad (5) \\
\frac{dP_I}{dt} &= (1-\rho')[\gamma_{UU}(P_{I_U|S} + P_{I|S}) + \Gamma_{UU}(P_{I_U} + P_I) + \gamma_{AU}P_{I_A|S} + \Gamma_{AU}P_{I_A}]P_S \\
&\quad -[\beta_A(P_{S_A|I} + P_{I_A|I}) + B_A(P_{S_A} + P_{I_A})]P_I - \alpha_U\mu_U P_I. \qquad (6)
\end{aligned}$$

The success or failure of the invasion is determined by the dominant eigenvalue ($\lambda^*(\rho')$) of the invasion equations linearized about $P_S = P_I = 0$, and with the resident densities fixed at their equilibrium values obtained previously. When $\lambda^*(\rho') > 0$, the invasion succeeds and

selection favors the new phenotype. Of course, $\lambda^*(\rho_U) = 0$, since selection is neutral when the resident phenotype tries to invade itself.

The invasion equations include terms $P_{\sigma|S}$ and $P_{\sigma|I}$ that give the neighborhood structure of the invading population. In general, we expect the local structure of the invading phenotype to differ from that of the resident. Thus, we need to determine these conditional probabilities. Again, we can use pair approximation to write down ODEs for these terms (Appendix 2). As is commonly observed in this type of model (Matsuda, 1992; Brown, 2002), we find that the conditional probabilities for the invader go to equilibrium on a much faster timescale than the overall densities. This occurs because local interactions allow structure to develop at the local scale faster than at the global scale, especially when the invasion is developing slowly because of small phenotypic differences. Thus, we can solve the pair equations for equilibrium values of the conditional probabilities with the low density assumption $P_S = P_I = 0$; then we incorporate this spatial structure into the invasion equations as fixed parameters.

To determine the direction of phenotypic evolution, we use the following steps: compute the resident densities using equations 1–4 and Appendix 1; compute the local spatial structure of the invading phenotype using Appendix 2; find the largest eigenvalue of the invasion equations (5–6). Suppose $\rho^- < \rho_U < \rho^+$ are phenotypes that differ only slightly. Then typically we find that the invasion eigenvalues satisfy $\lambda^*(\rho^-) < 0 < \lambda^*(\rho^+)$ or $\lambda^*(\rho^-) > 0 > \lambda^*(\rho^+)$. In the first case, evolution leads to higher resistance; in the second case, it leads to lower resistance. An evolutionarily stable state is characterized by $\lambda^*(\rho^-) < 0$ and $\lambda^*(\rho^+) < 0$, i.e. the resident phenotype cannot be invaded. The direction and relative rate of evolution can be summarized in a term called the evolutionary flux (Rand *et al.*, 1994; Rand *et al.*, 1995):

$$\frac{\lambda^*(\rho^+) - \lambda^*(\rho^-)}{\rho^+ - \rho^-}. \tag{7}$$

In the limit of arbitrarily small differences in phenotypes, the flux can be thought of as describing the slope of the fitness landscape. Thus, a positive flux indicates that resistance will increase, while a negative flux indicates that resistance will decrease. At an ESS, the

flux is zero, passing from positive to negative as we increase resistance.

Results

The behavior of the model depends in a complex way on the full set of parameters. As would be expected, coexistence of both species and endemicity of the pathogen is only possible if the birth and transmission rates are sufficiently high. In the alternating case, there exists a threshold resistance level for each host, above which the pathogen cannot persist because successful infection is too rare. When the pathogen is transmitted arbitrarily between host species, it may or may not be able to persist in spite of total resistance by one species, depending on the other parameters.

In order to allow comparisons between different dispersal scales and pathogen life histories, we chose parameter values that yielded the same resident densities. As stated above, we chose death rates so that the user host is shorter–lived than the attacked host. We then chose birth rates so that, in the absence of the disease, the equilibrium densities of the attacked and user hosts were approximately 0.55 and 0.2, respectively. With no resistance and moderate levels of disease damage, we chose the transmission rates so that the disease was endemic, yielding approximate equilibrium densities of $(P_{S_A}, P_{I_A}, P_{S_U}, P_{I_U}) = (.1, .1, .5, .1)$. The presence of the disease thus reversed the relative abundance of the two hosts. For the disease damage values, we used $\alpha_U = 1.1$ always, while α_A varied from 2.0 to 2.4 as needed to match the equilibrium densities. Notice that at these damage levels, the pathogen increases the user host's mortality by 10% and the attacked host's mortality by 100% or more. However, because of the difference in background mortality rates between the two species, this yields an additive increase of 0.1 to the mortality of each. The disease removes individuals of both species at approximately the same rate, but the higher birth rate of the user host allows it overcome the added mortality and exploit the space cleared by it.

For these parameter values, the presence of the disease is advantageous to the user host. As a result, an increase in the level of resistance by the user host causes a decrease in its equilibrium density. The dependence of the equilibrium population levels on the user's resis-

tance is shown in Figure 1 for the mean field alternating case; other cases behave similarly. Notice that there is a threshold level of resistance at approximately $\rho_U = 0.6$, above which the disease vanishes and the resistance level has no effect. Typically, an increase in resistance by one host leads to lower infected populations of both hosts. However, we note in passing the counterintuitive result that under some circumstances, increasing ρ_U from zero initially increases the equilibrium level of P_{I_A}.

From the effect of user resistance on the equilibrium population levels, we might infer that under these conditions evolution will always lead to lower resistance by the user host. However, an ESS approach indicates that this is not the case. In the mean field case, selection always leads to higher resistance because a user phenotype with a higher resistance can always invade the system; a proof of this is given in Appendix 3. Thus, selection will lead to a user host that maximizes resistance, even though this causes its population to shrink. The intuitive reason for this was discussed in Rice & Westoby (1982): less resistant user hosts bear the price of increase mortality from infection, but they do not reap any greater reward that more resistant users. When transmission from the user to the attacked host occurs over long distances, there is nothing to prevent a higher–resistance phenotype from "cheating"; the damage done to the attacked hosts benefits all user hosts equally, regardless of their level of resistance. Because more resistant user hosts enjoy all of the benefits but bear less of the burdens of the disease, they always displace less resistant phenotypes. We can only prove this result in the mean field case, but we conjecture that it always holds when transmission of the pathogen from the user to the attacked host is global.

When this transmission is local, ESS analysis indicates that selection for reduced user resistance can occur. Figure 2a shows the evolutionary flux in user resistance as estimated from simulations of the full stochastic model (in the case of alternating hosts with local user dispersal and user–attacked transmission and global attacked host dispersal and attacked–user transmission). Here, selection leads to an intermediate level of resistance by the user (an ESS resistance between 0.2 and 0.25). Phenotypes with very low resistance can be invaded by more resistant phenotypes which experience lower mortality due to infection.

However, highly resistant phenotypes can be invaded by less resistant ones which experience less severe competition because of the presence of the disease. This phenomenon depends on the local spatial structure of the populations; to facilitate the analysis, we turn to the pair approximation equations.

Pair approximations correctly predict that selection can lead to lowered resistance in the user (Figure 2b). When the damage done to the user host (α_U) is sufficiently small, selection always favors lower resistance. As the damage increases, selection leads to an intermediate ESS: below this value resistance increases, while above it resistance decreases. Finally, for sufficiently high damage levels, the direct effect of infection outweighs the benefits and selection leads to a resistance level that drives the disease extinct (here, around $\rho_U = 0.45$). Unlike the mean field model, the pair approximation equations detect the fact that the different user phenotypes encounter different competitive pressures.

To the extent that pair approximation predicts selection for lower resistance, simulations indicate that it is conservative: lower resistance is favored over a wider set of parameters than predicted. For example, in Figure 2, with damage to the user $\alpha_U = 1.09$, the pair approximation predicts an ESS resistance of around 0.45, while simulations indicate that it lies between 0.2 and 0.25. This occurs because pair approximation underestimates the clustering of hosts. When dispersal is local, the presence of a conspecific nearby greatly increases the probability of finding another one, so that $P_{\sigma|\sigma'\sigma} >> P_{\sigma|\sigma'}$ when σ is rare. The strategy of lowered resistance takes advantage of the local buildup of infections, so that selection should be stronger than predicted by pair approximations. Corrections to the pair approximation to deal with this phenomenon have been developed (Sato *et al.*, 1994), though they increase the complexity of the analysis. We retain the usual pair approximation assumption, since we believe it correctly predicts the qualitative behavior of this evolutionary process. It should be kept in mind, however, that local dispersal will in general lead to a lower ESS resistance for the user than is predicted by pair approximation.

By studying the evolutionary fluxes, we can determine how the ESS level of resistance depends on various factors. The damage that the disease does to each host determines the

level of resistance that selection favors (Figure 3). As the pathogen becomes more lethal to the user host, the ESS resistance moves from complete nonresistance to the threshold level at which the disease dies out. When the pathogen becomes more damaging to either host, this threshold level of resistance decreases as the infectious period shrinks. Still, as the disease becomes more lethal to the attacked host, it becomes a more effective weapon, and selection favors lower resistance by the user host. The more damaging the disease is to the attacked host, the greater damage the user host can tolerate and still gain by lowering resistance.

A change in the resistance level of the attacked host also affects the ESS level of resistance by the user host (Figure 4). If the attacked host becomes more resistant, selection favors lower resistance by the user host. This suggests that the two hosts may engage in a kind of "arms race by proxy", with the user host lowering its own resistance to offset increased resistance by the other species. However, understanding this issue would require a broader ESS approach, in which we examined selection for each species' resistance and for the pathogen's virulence. Notice that although the user's ESS resistance decreases as the attacked host's resistance increases, the maximum level of damage that the user can sustain and still favor reduced resistance decreases. Thus, even as the user is selected to offset the other species' resistance by lowering its own, the range of damage over which this strategy works is shrinking. Although increased damage and increased attacked host resistance both lead to lower pathogen populations, they have different implications for the utility of the disease as a weapon by the user, and hence lead to different evolutionary trajectories for user resistance.

As we discussed above, the dispersal scale of the pathogen transmission from the user to the attacked host is a critical factor; selection only favors reduced resistance when this dispersal is local. The other dispersal scales in the system may also be important, since they determine the local spatial structure encountered by the user host. Figure 5 shows the effect of varying the dispersal scales of the attacked host and of attacked–user transmission while the other dispersal scales are kept local. Precise comparisons between these cases are not meaningful, since parameters were chosen to match resident densities approximately at an

arbitrary point ($\rho_U = 0$, $\alpha_U = 1.1$). However, it appears that the evolutionary advantage of low user resistance increases when more dispersal processes are local. Local dispersal increases spatial heterogeneity in the populations, strengthening selective forces that depend on local spatial structure.

Thus far, we have only considered local dispersal by the user host. Selection for lower resistance may also be possible when the user disperses globally; although the user will not be able to take advantage of the space cleared by infections, it will benefit from the reduced rate of displacement by competitively dominant neighbors. To investigate this case, we have to modify our approach slightly. In the low density limit, global dispersal implies that $P_{S|I} = P_{I|S} = 0$, so that we can no longer compute the ratio $\frac{P_{S|I}}{P_{I|S}} = \frac{P_S}{P_I}$ which appears in the invader equations (Appendix 2). However, this ratio is given by the eigenvector associated with the dominant invasion eigenvalue. Thus, we proceed by simultaneously solving for the equilibrium spatial structure and eigenvector of the invading phenotype (van Baalen & Rand, 1998). We find that the selection for lowered resistance can occur, but that it is much weaker than when user dispersal is local. For example, when user dispersal is global but all other processes are local, the ESS switches from nonresistance to maximal resistance when the damage to the user is only $\alpha_U = 1.003$ (c.f. Figure 5).

Protection from displacement offers a very small benefit to the user host in our system because of the assumption that the user's dynamics are much faster than the attacked host's. The main impact of the attacked host on the users is as a barrier to growth, rather than as a displacement threat. Under different assumptions about the plants' dynamics (for example, competition between annuals or between perennials), the relative importance of these aspects of competition will change. When competitive displacement is a sufficiently severe threat to the user host, selection for nonresistance could be strong even when the user disperses globally. Of course, when both hosts disperse globally, selection will not favor nonresistance, since there is no way for less resistant phenotypes to benefit from their infections.

The pair approximation approach fails in the case that disease transmission from user to attacked hosts is global, but all other processes are local. As we have argued, we do not

expect selection to favor reduced resistance in this case because infected users do not reap the rewards of transmission. However, the pair approximation equations predict very weak selection for nonresistance when $\alpha_U = 1.0$, with the ESS switching to complete resistance very quickly as this damage increases. The equations still satisfy the condition for consistency between the invader and resident phenotypes, $\lambda^*(\rho_U) = 0$, so this does not result from the low density assumptions. Rather, the pair approximation errs in predicting that the local spatial structure of the user depends on its resistance level even when transmission to the attacked host is global. This prediction is not supported by simulations, and is an anomaly of the pair approximation approach that we do not fully understand. In other dispersal cases, as we mentioned, the pair approximation's prediction of selection for lowered resistance is conservative when compared to simulations.

Finally, we consider the importance of the pathogen life cycle in determining the evolution of the user's resistance. Thus far, all examples have been based on a life cycle in which the pathogen alternates strictly between host species. Figure 6 compares the ESS resistance for an alternating pathogen with that obtained when the disease can be transmitted between any hosts. Recall that the parameters were chosen so that the resident densities match. Transmission between arbitrary hosts allows much easier spread of the disease, so that the transmission levels used in this case are much lower than those used in the corresponding alternating case. As a result, infected user hosts are less likely to infect neighboring attacked hosts when we use this basis of comparison. Consequently, the disease is a less potent weapon, and selection leads to higher levels of user resistance than in the alternating case. Of course, lowered resistance becomes a better strategy when transmission from user to attacked hosts increases. In fact, for the parameter values used here, transmission between arbitrary hosts at the same rate as used in the alternating case ($\lambda_{\sigma\sigma'} = 10$) leads to the extinction of the attacked host. Depending on the transmission rates and relative damage, the disease may be supported by either host at high enough levels to wipe out the other species. The dependence of the ESS on other parameters is qualitatively similar to the alternating case: increasing the damage done to the attacked host or that host's resistance leads to lower resistance by

the user, and the behavior is not sensitive to the dispersal scale of the attacked host. With transmission between arbitrary hosts, we assume that the pathogen has a single dispersal scale; selection for reduced user resistance is possible only if this dispersal is local.

In all of the cases studied, a successful invasion by a novel phenotype leads to the extinction of the resident phenotype. In addition, for each set of parameters we found only one resistance phenotype stable against invasions by neighboring phenotypes. Thus, the pair approximation equations indicate that polymorphism for resistance will not be maintained by the mechanism of apparent competition. Simulations of the stochastic model support this conclusion, although the time required for one phenotype to replace another is very long (typically, tens of thousands of generations) because the invasion eigenvalues are so small. Thus, while this mechanism does not itself lead to polymorphisms, the fact that it acts weakly and locally would allow other mechanisms to maintain them.

Discussion

We have modeled the selection for disease resistance in a plant that shares a pathogen with a competing species. Selection can favor lower resistance not only because of direct physiological costs of resistance, but because the disease may be a weapon that one host uses against the other. Under fairly generic conditions, lowering resistance by an inferior competitor led to a higher population in a nonspatial model. However, an ESS analysis showed that in this case evolution always leads to higher resistance, since invading phenotypes can cheat and take advantage of the resident phenotype's infection. Evolution only led to lower resistance if the disease transmission from user to attacked host was localized in space. This allows less resistant phenotypes to benefit from the disease by primarily infecting their own competitors.

The ESS level of resistance depended on the severity of the damage that the disease inflicted on each host. If infection of the user host was sufficiently benign, selection favored nonresistance. As the level of damage increased, higher resistance was favored; if the damage was too severe, selection favored total resistance. On the other hand, the greater the damage

the disease inflicted on the attacked host, the more potent a weapon it was, and selection favored lower resistance by the user. The level of resistance by the attacked host also impacted the evolution of resistance by the user. As the attacked host's resistance increased, the user was selected to offset this by lowering its own resistance. This has interesting implications for the coevolution of resistance and virulence in the system, with the possibility of a multilateral arms race.

The evolution of resistance was quite robust with respect to the pathogen life cycle and the dispersal scale of the attacked host. When we compared systems with approximately equal resident densities, we found that lowered resistance is favored over a similar range of disease damage. Thus, the tradeoff between the direct damage of the disease and the indirect benefit seems to depend in our model more on the overall plant and pathogen densities than on the details of the pathogen life cycle or on the dispersal scales of the attacked host and attacked–user transmission.

On the other hand, the evolution of resistance depended critically on the spatial scales of user host dispersal and user–attacked transmission. Local dispersal is necessary for the success of a non–resistance strategy because it takes advantage of spatial heterogeneity at the scale of individuals organisms. It allows different phenotypes to "see" different localized versions of the world, which is critical for the success of a strategy that would otherwise be overwhelmed by cheaters. The same mechanism has been invoked to explain evolutionary outcomes in several other systems (Rand *et al.*, 1995; van Baalen & Rand, 1998; Boots & Sasaki, 2000). In general, evolutionary trends must be influenced by differences in local population structures that are encountered by different phenotypes; in several systems, we have now seen that the qualitative outcomes of evolution are governed by this type of structure. Incorporating fine–scale spatial structure can lead to a model that is cumbersome to use; thus, approaches such as pair approximation are useful for streamlining the exploration of parameter space and explaining observed patterns.

Our conclusion that evolution may lead to reduced resistance by a plant that shares a pathogen with a superior competitor supports the feasibility of the mechanism that Rice

& Westoby (1982) invoked to explain ecological and phylogenetic patterns of heteroecious fungi. In order to complete their life cycle, some heteroecious fungi must sequentially infect two different host species (called the aecial and telial hosts, from the spore stages that infect them). While the fungi are highly host specific, usually unable to infect species closely related to their hosts, they are able to attack two different hosts which are often taxonomically distant. What allows them to do this? Rice & Westoby (1982) argued that the telial host is using the fungus as a weapon against a competitively superior aecial host. Their argument that this mechanism must be invoked to explain the existence of heteroecious fungi depends on the assumption that the same fungal genes are responsible for controlling infection and virulence/avirulence in both hosts. However, differences between the morphologies and infection mechanisms of the two spore stages suggest that the two infection processes are under the control of separate genes (McIntosh and Watson, 1982). Moreover, there is no *a priori* reason to expect that both hosts would target the same avirulence genes. Nevertheless, selection for resistance in one host may be mediated by the impact of the pathogen on the other host, provided that the hosts are directly competing. Even if apparent competition is not a necessary explanation for the persistence of heteroecy, it may still be an important factor in determining the evolution of resistance in some cases.

Our work establishes a theoretical framework for understanding the conditions under which competing plants that share a pathogen may undergo selection for lower disease resistance. It may be possible to extend the results further, to any system in which species compete both directly and through a common enemy. When both direct and apparent competition occur, a species will undergo selection on traits which determine its vulnerability to the predator or pathogen. A tradeoff can then occur between the direct damage of infection and the indirect benefits of apparent competition. In our system, spatial structure was essential in determing the evolutionary consequences of this tradeoff. There may be other types of localization that allow less "resistant" phenotypes to benefit from their strategy. For example, social interaction networks can determine the spread of diseases in animals (Keeling *et al.*, 1997; Keeling & Grenfell, 2000). If an individual can preferentially target

for infection its own competitors, selection may favor lowered resistance. In general, the coevolution of hosts and pathogens or predators and prey may depend not only on the direct effects of the pathogen or predator, but on its impact on other species in the system. The resistance strategy of the host or prey must then be interpreted in terms of a tradeoff between direct and indirect effects.

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Appendix 1

We present the pair approximation equations for the resident population densities. Let $P_{\sigma\sigma'}$ be the "pair density", the probability that a randomly chosen site is in state σ and a randomly chosen neighbor of it is in state σ'. Then:

$$P_{\sigma|\sigma'} = \frac{P_{\sigma\sigma'}}{P_{\sigma'}} \tag{8}$$

Note that $P_{\sigma\sigma'} = P_{\sigma'\sigma}$, but that in general $P_{\sigma|\sigma'} \neq P_{\sigma'|\sigma}$. The dynamics of the pairs can be obtained by considering interactions between the two sites and interactions with neighbors of them. These last interactions involve triplet terms of the form $P_{\sigma|\sigma'\sigma''}$. For example, consider again the colonization of an empty site by a susceptible attacked host. This event switches an S_AE pair to S_AS_A. If dispersal is local, the colonization can occur due to reproduction by the first S_A site or by any of the three other neighbors of the the empty site. Thus, local reproduction by S_A switches S_AE pairs to S_AS_A at the rate $\frac{\beta_A}{4}(1+3P_{S_A|ES_A})P_{S_AE}$. On the other hand, if dispersal is global, the offspring could have come from anywhere, and the rate of change due to this process is simply $B_AP_{S_A}P_{S_AE}$. After replacing the triplet terms with the ordinary pair approximation ($P_{\sigma|\sigma'\sigma''} = P_{\sigma|\sigma'}$), we obtain the pair density equations for the residents:

$$\begin{aligned}
\frac{1}{2}\frac{dP_{S_AS_A}}{dt} &= [\frac{\beta_A}{4}(1+3P_{S_A|E}+3P_{I_A|E}) + B_A(P_{S_A}+P_{I_A})]P_{S_AE} \\
&\quad +[\frac{\beta_A}{4}(1+3P_{S_A|S_U}+3P_{I_A|S_U}) + B_A(P_{S_A}+P_{I_A})]P_{S_AS_U} \\
&\quad +[\frac{\beta_A}{4}(1+3P_{S_A|I_U}+3P_{I_A|I_U}) + B_A(P_{S_A}+P_{I_A})]P_{S_AI_U} \\
&\quad -\{\mu_A + (1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A} + \Gamma_{AA}P_{I_A} \\
&\quad +\frac{3}{4}\gamma_{UA}P_{I_U|S_A} + \Gamma_{UA}P_{I_U}]\}P_{S_AS_A} \\
\frac{dP_{S_AI_A}}{dt} &= (1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A} + \Gamma_{AA}P_{I_A} + \frac{3}{4}\gamma_{UA}P_{I_U|S_A} + \Gamma_{UA}P_{I_U}]P_{S_AS_A} \\
&\quad +[\frac{\beta_A}{4}(1+3P_{S_A|E}+3P_{I_A|E}) + B_A(P_{S_A}+P_{I_A})]P_{I_AE} \\
&\quad +[\frac{\beta_A}{4}(1+3P_{S_A|S_U}+3P_{I_A|S_U}) + B_A(P_{S_A}+P_{I_A})]P_{I_AS_U}
\end{aligned} \tag{9}$$

$$
\begin{aligned}
&+[\frac{\beta_A}{4}(1+3P_{S_A|I_U}+3P_{I_A|I_U})+B_A(P_{S_A}+P_{I_A})]P_{I_AI_U}-\{\mu_A+\alpha_A\mu_A\\
&+(1-\rho_A)[\frac{\gamma_{AA}}{4}(1+3P_{I_A|S_A}+\Gamma_{AA}P_{I_A}\\
&+\frac{3}{4}\gamma_{UA}P_{I_U|S_A}+\Gamma_{UA}P_{I_U}]\}P_{S_AI_A} \qquad (10)\\
\frac{dP_{S_AS_U}}{dt} &= [\frac{3}{4}\beta_A(P_{S_A|E}+P_{I_A|E})+B_A(P_{S_A}+P_{I_A})]P_{S_UE}\\
&+[\frac{3}{4}\beta_A(P_{S_A|S_U}+P_{I_A|S_U})+B_A(P_{S_A}+P_{I_A})]P_{S_US_U}\\
&+[\frac{3}{4}\beta_A(P_{S_A|I_U}+P_{I_A|I_U})+B_A(P_{S_A}+P_{I_A})]P_{S_UI_U}\\
&+[\frac{3}{4}\beta_U(P_{S_U|E}+P_{I_U|E})+B_U(P_{S_U}+P_{I_U})]P_{S_AE}\\
&-\{\mu_A+\mu_U+\frac{\beta_A}{4}(1+3P_{S_A|S_U}+3P_{I_A|S_U}+B_A(P_{S_A}+P_{I_A})\\
&+(1-\rho_U)[\frac{3}{4}\gamma_{UU}P_{I_U|S_U}+\Gamma_{UU}P_{I_U}+\frac{3}{4}\gamma_{AU}P_{I_A|S_U}+\Gamma_{AU}P_{I_A}]\\
&+(1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A}+\Gamma_{AA}P_{I_A}\\
&+\frac{3}{4}\gamma_{UA}P_{I_U|S_A}+\Gamma_{UA}P_{I_U}]\}P_{S_AS_U} \qquad (11)\\
\frac{dP_{S_AI_U}}{dt} &= [\frac{3}{4}\beta_A(P_{S_A|E}+P_{I_A|E})+B_A(P_{S_A}+P_{I_A})]P_{I_UE}\\
&+[\frac{3}{4}\beta_A(P_{S_A|S_U}+P_{I_A|S_U})+B_A(P_{S_A}+P_{I_A})]P_{S_UI_U}\\
&+[\frac{3}{4}\beta_A(P_{S_A|I_U}+P_{I_A|I_U})+B_A(P_{S_A}+P_{I_A})]P_{I_UI_U}\\
&+(1-\rho_U)[\frac{3}{4}\gamma_{UU}P_{I_U|S_U}+\Gamma_{UU}P_{I_U}+\frac{3}{4}\gamma_{AU}P_{I_A|S_U}+\Gamma_{AU}P_{I_A}]P_{S_AS_U}\\
&-\{\mu_A+\alpha_U\mu_U+[\frac{\beta_A}{4}(1+3P_{S_A|I_U}+3P_{I_A|I_U})+B_A(P_{S_A}+P_{I_A})]\\
&+(1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A}+\Gamma_{AA}P_{I_A}\\
&+\frac{\gamma_{UA}}{4}(1+3P_{I_U|S_A})+\Gamma_{UA}P_{I_U}]\}P_{S_AI_U} \qquad (12)\\
\frac{1}{2}\frac{dP_{I_AI_A}}{dt} &= (1-\rho_A)[\frac{\gamma_{AA}}{4}(1+3P_{I_A|S_A})+\Gamma_{AA}P_{I_A}+\frac{3}{4}\gamma_{UA}P_{I_U|S_A}+\Gamma_{UA}P_{I_U}]P_{S_AI_A}\\
&-\alpha_A\mu_AP_{I_AI_A} \qquad (13)\\
\frac{dP_{I_AS_U}}{dt} &= (1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A}+\Gamma_{AA}P_{I_A}+\frac{3}{4}\gamma_{UA}P_{I_U|S_A}+\Gamma_{UA}P_{I_U}]P_{S_AS_U}\\
&+[\frac{3}{4}\beta_U(P_{S_U|E}+P_{I_U|E})+B_U(P_{S_U}+P_{I_U})]P_{I_AE}
\end{aligned}
$$

$$
\begin{aligned}
& -\{\alpha_A\mu_A + \mu_U + \frac{\beta_A}{4}(1 + 3P_{I_A|S_U} + 3P_{S_A|S_U}) + B_A(P_{S_A} + P_{I_A}) \\
& +(1-\rho_U)[\frac{\gamma_{AU}}{4}(1 + 3P_{I_A|S_U}) + \Gamma_{AU}P_{I_A} \\
& +\frac{3}{4}\gamma_{UU}P_{I_U|S_U} + \Gamma_{UU}P_{I_U}]\}P_{I_AS_U} \qquad (14)
\end{aligned}
$$

$$
\begin{aligned}
\frac{dP_{I_AI_U}}{dt} &= (1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A} + \Gamma_{AA}P_{I_A} + \frac{\gamma_{UA}}{4}(1 + 3P_{I_U|S_A}) + \Gamma_{UA}P_{I_U}]P_{S_AI_U} \\
& +(1-\rho_U)[\frac{\gamma_{AU}}{4}(1 + 3P_{I_A|S_U}) + \Gamma_{AU}P_{I_A} + \frac{3}{4}\gamma_{UU}P_{I_U|S_U} + \Gamma_{UU}P_{I_U}]P_{I_AS_U} \\
& -\{\alpha_A\mu_A + \alpha_U\mu_U + \frac{\beta_A}{4}(1 + 3P_{S_A|I_U} + 3P_{I_A|I_U}) \\
& +B_A(P_{S_A} + P_{I_A})\}P_{I_AI_U} \qquad (15)
\end{aligned}
$$

$$
\begin{aligned}
\frac{1}{2}\frac{dP_{S_US_U}}{dt} &= [\frac{\beta_U}{4}(1 + 3P_{S_U|E} + 3P_{I_U|E}) + B_U(P_{S_U} + P_{I_U})]P_{S_UE} \\
& -\{\mu_U + (1-\rho_U)[\frac{3}{4}\gamma_{UU}P_{I_U|S_U} + \Gamma_{UU}P_{I_U} + \frac{3}{4}\gamma_{AU}P_{I_A|S_U} + \Gamma_{AU}P_{I_A}] \\
& +\frac{3}{4}\beta_A(P_{S_A|S_U} + P_{I_A|S_U}) + B_A(P_{S_A} + P_{I_A})\}P_{S_US_U} \qquad (16)
\end{aligned}
$$

$$
\begin{aligned}
\frac{dP_{S_UI_U}}{dt} &= [\frac{\beta_U}{4}(1 + 3P_{I_U|E} + 3P_{S_U|E}) + B_U(P_{S_U} + P_{I_U})]P_{I_UE} \\
& +(1-\rho_U)[\frac{3}{4}\gamma_{UU}P_{I_U|S_U} + \Gamma_{UU}P_{I_U} + \frac{3}{4}\gamma_{AU}P_{I_A|S_U} + \Gamma_{AU}P_{I_A}]P_{S_US_U} \\
& -\{\mu_U + \alpha_U\mu_U + \frac{3}{4}\beta_A(P_{S_A|S_U} + P_{I_A|S_U} + P_{S_A|I_U} + P_{I_A|I_U}) \\
& +2B_A(P_{S_A} + P_{I_A}) + (1-\rho_U)[\frac{\gamma_{UU}}{4}(1 + 3P_{I_U|S_U}) + \Gamma_{UU}P_{I_U} \\
& +\frac{3}{4}\gamma_{AU}P_{I_A|S_U} + \Gamma_{AU}P_{I_A}]\}P_{S_UI_U} \qquad (17)
\end{aligned}
$$

$$
\begin{aligned}
\frac{1}{2}\frac{dP_{I_UI_U}}{dt} &= (1-\rho_U)[\frac{\gamma_{UU}}{4}(1 + 3P_{I_U|S_U}) + \Gamma_{UU}P_{I_U} + \frac{3}{4}\gamma_{AU}P_{I_A|S_U} + \Gamma_{AU}P_{I_A}]P_{S_UI_U} \\
& -\{\alpha_U\mu_U + \frac{3}{4}\beta_A(P_{S_A|I_U} + P_{I_A|I_U}) + B_A(P_{S_A} + P_{I_A})\}P_{I_UI_U}. \qquad (18)
\end{aligned}
$$

Since $P_\sigma = \sum_{\sigma'} P_{\sigma\sigma'}$, we can augment these equations with equations 1–4 for the singleton densities to obtain a closed system. The system is too complex to solve analytically, but numerical solution is straightforward and allows efficient exploration of parameter space. A more detailed explanation of the derivation of pair density equations can be found in, for example, Matsuda (1992) or Rand (1999).

Appendix 2

We present the conditional pair equations for the neighborhood structure of the invading phenotype at low density. First we find equations for pair densities involving S or I, by the same procedure used above. Now, the low density assumption means that $P_\sigma = P_{\sigma\sigma'} = 0$, when $\sigma \in S, I$. However, the conditional probabilities $P_{\sigma'|\sigma}$ are not necessarily small in this case. To obtain these quantities, we use the fact that

$$\frac{dP_{\sigma'|\sigma}}{dt} = \frac{1}{P_\sigma}\frac{dP_{\sigma'\sigma}}{dt} - \frac{P_{\sigma'|\sigma}}{P_\sigma}\frac{dP_\sigma}{dt} \tag{19}$$

to derive the dynamics of the conditional probabilities from those of the singleton and pair densities. Using the pair approximation and the low density assumption, we obtain:

$$\begin{aligned}
\frac{dP_{S_A|S}}{dt} &= [\frac{3}{4}\beta_A(P_{S_A|E} + P_{I_A|E}) + B_A(P_{S_A} + P_{I_A})]P_{E|S} \\
&+[\frac{3}{4}\beta_A(P_{S_A|S} + P_{I_A|S}) + B_A(P_{S_A} + P_{I_A})]P_{S|S} \\
&+[\frac{3}{4}\beta_A(P_{S_A|S_U} + P_{I_A|S_U}) + B_A(P_{S_A} + P_{I_A})]P_{S_U|S} \\
&+[\frac{3}{4}\beta_A(P_{S_A|I} + P_{I_A|I}) + B_A(P_{S_A} + P_{I_A})]P_{I|S} \\
&+[\frac{3}{4}\beta_A(P_{S_A|I_U} + P_{I_A|I_U}) + B_A(P_{S_A} + P_{I_A})]P_{I_U|S} \\
&+[\frac{3}{4}\beta_U(P_{E|S} + P_{E|I}\frac{P_I}{P_S}) + B_U P_E(1 + \frac{P_I}{P_S})]P_{S_A|E} \\
&-\{\mu_A + \frac{\beta_A}{4}(1 - P_{S_A|S} - P_{I_A|S}) \\
&+(1-\rho')[-\frac{\gamma_{AU}}{4}P_{I_A|S} - \frac{\gamma_{UU}}{4}(P_{I_U|S} + P_{I|S})] \\
&+(1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A} + \Gamma_{AA}P_{I_A} + \frac{3}{4}\gamma_{UA}P_{I_U|S_A} + \Gamma_{UA}P_{I_U}] \\
&+\beta_U(P_{E|S} + P_{E|I}\frac{P_I}{P_S}) + B_U P_E(1 + \frac{P_I}{P_S})\}P_{S_A|S}
\end{aligned} \tag{20}$$

$$\begin{aligned}
\frac{dP_{I_A|S}}{dt} &= (1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A} + \Gamma_{AA}P_{I_A} + \frac{3}{4}\gamma_{UA}P_{I_U|S_A} + \Gamma_{UA}P_{I_U}]P_{S_A|S} \\
&+[\frac{3}{4}\beta_U(P_{E|S} + P_{E|I}\frac{P_I}{P_S}) + B_U P_E(1 + \frac{P_I}{P_S})]P_{I_A|E} \\
&-\{\alpha_A\mu_A + (1-\rho')[\frac{\gamma_{AU}}{4}(1 - P_{I_A|S}) - \frac{\gamma_{UU}}{4}(P_{I_U|S} + P_{I|S})] \\
&+\frac{\beta_A}{4}(1 - P_{S_A|S} - P_{I_A|S}) + \beta_U(P_{E|S} + P_{E|I}\frac{P_I}{P_S})
\end{aligned}$$

$$+B_U P_E(1+\frac{P_I}{P_S})\}P_{I_A|S} \tag{21}$$

$$\begin{aligned}
\frac{dP_{S_U|S}}{dt} &= [\frac{\beta_U}{4}(1+3P_{S_U|E}+3P_{I_U|E})+B_U(P_{S_U}+P_{I_U})]P_{E|S} \\
&+[\frac{3}{4}\beta_U(P_{E|S}+P_{E|I}\frac{P_I}{P_S})+B_U P_E(1+\frac{P_I}{P_S})]P_{S_U|E} \\
&-\{\mu_U+(1-\rho_U)[\frac{3}{4}\gamma_{UU}P_{I_U|S_U}+\Gamma_{UU}P_{I_U}+\frac{3}{4}\gamma_{AU}P_{I_A|S_U}+\Gamma_{AU}P_{I_A}] \\
&+(1-\rho')[-\frac{\gamma_{AU}}{4}P_{I_A|S}-\frac{\gamma_{UU}}{4}(P_{I_U|S}+P_{I|S})] \\
&+\frac{3}{4}\beta_A(P_{S_A|S_U}+P_{I_A|S_U})+B_A(P_{S_A}+P_{I_A})-\frac{\beta_A}{4}(P_{S_A|S}+P_{I_A|S}) \\
&+\beta_U(P_{E|S}+P_{E|I}\frac{P_I}{P_S})+B_U P_E(1+\frac{P_I}{P_S})\}P_{S_U|S}
\end{aligned} \tag{22}$$

$$\begin{aligned}
\frac{dP_{I_U|S}}{dt} &= [\frac{3}{4}\beta_U(P_{E|S}+P_{E|I}\frac{P_I}{P_S})+B_U P_E(1+\frac{P_I}{P_S})]P_{I_U|E} \\
&+(1-\rho_U)[\frac{3}{4}\gamma_{UU}P_{I_U|S_U}+\Gamma_{UU}P_{I_U}+\frac{3}{4}\gamma_{AU}P_{I_A|S_U}+\Gamma_{AU}P_{I_A}]P_{S_U|S} \\
&-\{\alpha_U\mu_U+\frac{3}{4}\beta_A(P_{S_A|I_U}+P_{I_A|I_U})+B_A(P_{S_A}+P_{I_A})-\frac{\beta_A}{4}(P_{S_A|S}+P_{I_A|S}) \\
&+(1-\rho')[-\frac{\gamma_{AU}}{4}P_{I_A|S}+\frac{\gamma_{UU}}{4}(1-P_{I_U|S}-P_{I|S})] \\
&+\beta_U(P_{E|S}+P_{E|I}\frac{P_I}{P_S})+B_U P_E(1+\frac{P_I}{P_S})\}P_{I_U|S}
\end{aligned} \tag{23}$$

$$\begin{aligned}
\frac{dP_{S|S}}{dt} &= \frac{\beta_U}{2}P_{E|S}-\{\mu_U+(1-\rho')[\frac{\gamma_{UU}}{2}(P_{I_U|S}+P_{I|S})+\Gamma_{UU}P_{I_U} \\
&+\frac{\gamma_{AU}}{2}P_{I_A|S}+\Gamma_{AU}P_{I_A}]+\frac{\beta_A}{2}(P_{S_A|S}+P_{I_A|S})+B_A(P_{S_A}+P_{I_A}) \\
&+\beta_U(P_{E|S}+P_{E|I}\frac{P_I}{P_S})+B_U P_E(1+\frac{P_I}{P_S})\}P_{S|S}
\end{aligned} \tag{24}$$

$$\begin{aligned}
\frac{dP_{I|S}}{dt} &= \frac{\beta_U}{4}P_{E|I}\frac{P_I}{P_S}+(1-\rho')[\frac{3}{4}\gamma_{UU}P_{I_U|S}+\Gamma_{UU}P_{I_U}+\frac{3}{4}\gamma_{AU}P_{I_A|S}+\Gamma_{AU}P_{I_A}]P_{S|S} \\
&-\{\alpha_U\mu_U+\frac{3}{4}\beta_A(P_{S_A|I}+P_{I_A|I})+B_A(P_{S_A}+P_{I_A})-\frac{\beta_A}{4}(P_{S_A|S}+P_{I_A|S}) \\
&+(1-\rho')[-\frac{\gamma_{AU}}{4}P_{I_A|S}+\frac{\gamma_{UU}}{4}(1-P_{I_U|S}-P_{I|S})] \\
&+\beta_U(P_{E|S}+P_{E|I}\frac{P_I}{P_S})+B_U P_E(1+\frac{P_I}{P_S})\}P_{I|S}
\end{aligned} \tag{25}$$

$$\begin{aligned}
\frac{dP_{S_A|I}}{dt} &= [\frac{3}{4}\beta_A(P_{S_A|E}+P_{I_A|E})+B_A(P_{S_A}+P_{I_A})]P_{E|I} \\
&+[\frac{3}{4}\beta_A(P_{S_A|I_U}+P_{I_A|I_U})+B_A(P_{S_A}+P_{I_A})]P_{I_U|I}
\end{aligned}$$

$$
\begin{aligned}
&+[\frac{3}{4}\beta_A(P_{S_A|I}+P_{I_A|I})+B_A(P_{S_A}+P_{I_A})]P_{I|I}\\
&+[\frac{3}{4}\beta_A(P_{S_A|S_U}+P_{I_A|S_U})+B_A(P_{S_A}+P_{I_A})]P_{S_U|I}\\
&+[\frac{3}{4}\beta_A(P_{S_A|S}+P_{I_A|S})+B_A(P_{S_A}+P_{I_A})]P_{S|I}\\
&+(1-\rho')[\frac{3}{4}\gamma_{UU}(P_{I_U|S}+P_{I|S})+\Gamma_{UU}P_{I_U}+\frac{3}{4}\gamma_{AU}P_{I_A|S}+\Gamma_{AU}P_{I_A}]P_{S_A|S}\frac{P_S}{P_I}\\
&-\{\mu_A+\frac{\beta_A}{4}(1-P_{S_A|I}-P_{I_A|I})\\
&+(1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A}+\Gamma_{AA}P_{I_A}+\frac{\gamma_{UA}}{4}(1+3P_{I_U|S_A})+\Gamma_{UA}P_{I_U}]\\
&+(1-\rho')[\gamma_{UU}(P_{I_U|S}+P_{I|S})+\Gamma_{UU}P_{I_U}\\
&+\gamma_{AU}P_{I_A|S}+\Gamma_{AU}P_{I_A}]\frac{P_S}{P_I}\}P_{S_A|I} \qquad (26)\\
\frac{dP_{I_A|I}}{dt} &= (1-\rho_A)[\frac{3}{4}\gamma_{AA}P_{I_A|S_A}+\Gamma_{AA}P_{I_A}+\frac{\gamma_{UA}}{4}(1+3P_{I_U|S_A})+\Gamma_{UA}P_{I_U}]P_{S_A|I}\\
&+(1-\rho')[\frac{3}{4}\gamma_{UU}(P_{I_U|S}+P_{I|S})+\Gamma_{UU}P_{I_U}\\
&+\frac{\gamma_{AU}}{4}(1+3P_{I_A|S})+\Gamma_{AU}P_{I_A}]P_{I_A|S}\frac{P_S}{P_I}-\{\alpha_A\mu_A+\frac{\beta_A}{4}(1-P_{S_A|I}-P_{I_A|I})\\
&+(1-\rho')[\gamma_{UU}(P_{I_U|S}+P_{I|S})+\Gamma_{UU}P_{I_U}\\
&+\gamma_{AU}P_{I_A|S}+\Gamma_{AU}P_{I_A}]\frac{P_S}{P_I}\}P_{I_A|I} \qquad (27)\\
\frac{dP_{S_U|I}}{dt} &= [\frac{3}{4}\beta_U(P_{I_U|E}+P_{S_U|E})+B_U(P_{S_U}+P_{I_U})]P_{E|I}\\
&+(1-\rho')[\frac{3}{4}\gamma_{UU}(P_{I_U|S}+P_{I|S})+\Gamma_{UU}P_{I_U}+\frac{3}{4}\gamma_{AU}P_{I_A|S}+\Gamma_{AU}P_{I_A}]P_{S_U|S}\frac{P_S}{P_I}\\
&-\{\mu_U-\frac{\beta_A}{4}(P_{S_A|I}+P_{I_A|I})+\frac{3}{4}\beta_A(P_{S_A|S_U}+P_{I_A|S_U})+B_A(P_{S_A}+P_{I_A})\\
&+(1-\rho_U)[\frac{\gamma_{UU}}{4}(1+3P_{I_U|S_U})+\Gamma_{UU}P_{I_U}+\frac{3}{4}\gamma_{AU}P_{I_A|S_U}+\Gamma_{AU}P_{I_A}]\\
&+(1-\rho')[\gamma_{UU}(P_{I_U|S}+P_{I|S})+\Gamma_{UU}P_{I_U}\\
&+\gamma_{AU}P_{I_A|S}+\Gamma_{AU}P_{I_A}]\frac{P_S}{P_I}\}P_{S_U|I} \qquad (28)\\
\frac{dP_{I_U|I}}{dt} &= (1-\rho_U)[\frac{\gamma_{UU}}{4}(1+3P_{I_U|S_U})+\Gamma_{UU}P_{I_U}+\frac{3}{4}\gamma_{AU}P_{I_A|S_U}+\Gamma_{AU}P_{I_A}]P_{S_U|I}\\
&+(1-\rho')[\frac{\gamma_{UU}}{4}(1+3P_{I_U|S}+3P_{I|S})+\Gamma_{UU}P_{I_U}\\
&+\frac{3}{4}\gamma_{AU}P_{I_A|S}+\Gamma_{AU}P_{I_A}]P_{I_U|S}\frac{P_S}{P_I}
\end{aligned}
$$

$$
\begin{aligned}
& -\{\alpha_U\mu_U - \frac{\beta_A}{4}(P_{S_A|I} + P_{I_A|I}) + \frac{3}{4}\beta_A(P_{S_A|I_U} + P_{I_A|I_U}) + B_A(P_{S_A} + P_{I_A}) \\
& +(1-\rho')[\gamma_{UU}(P_{I_U|S} + P_{I|S}) + \Gamma_{UU}P_{I_U} \\
& +\gamma_{AU}P_{I_A|S} + \Gamma_{AU}P_{I_A}]\frac{P_S}{P_I}\}P_{S_U|I} \qquad (29) \\
\frac{dP_{I|I}}{dt} &= (1-\rho')[\frac{\gamma_{UU}}{2}(1 + 3P_{I_U|S} + 3P_{I|S}) + 2\Gamma_{UU}P_{I_U} + \frac{3}{2}\gamma_{AU}P_{I_A|S} \\
& +2\Gamma_{AU}P_{I_A}]P_{S|I} - \{\alpha_U\mu_U + \frac{\beta_A}{2}(P_{S_A|I} + P_{I_A|I}) + B_A(P_{S_A} + P_{I_A}) \\
& +(1-\rho')[\gamma_{UU}(P_{I_U|S} + P_{I|S}) + \Gamma_{UU}P_{I_U} \\
& +\gamma_{AU}P_{I_A|S} + \Gamma_{AU}P_{I_A}]\frac{P_S}{P_I}\}P_{I|I} \qquad (30) \\
\frac{dP_{S|I}}{dt} &= \frac{\beta_U}{4}P_{E|I} + (1-\rho')[\frac{3}{4}\gamma_{UU}(P_{I_U|S} + P_{I|S}) + \Gamma_{UU}P_{I_U} \\
& +\frac{3}{4}\gamma_{AU}P_{I_A|S} + \Gamma_{AU}P_{I_A}]P_{S|S}\frac{P_S}{P_I} \\
& -\{\mu_U - \frac{\beta_A}{4}(P_{S_A|I} + P_{I_A|I}) + \frac{3}{4}\beta_A(P_{S_A|S} + P_{I_A|S}) + B_A(P_{S_A} + P_{I_A}) \\
& +(1-\rho')[\frac{\gamma_{UU}}{4}(1 + 3P_{I_U|S} + 3P_{I|S}) + \Gamma_{UU}P_{I_U} + \frac{3}{4}\gamma_{AU}P_{I_A|S} + \Gamma_{AU}P_{I_A}] \\
& +(1-\rho')[\gamma_{UU}(P_{I_U|S} + P_{I|S}) + \Gamma_{UU}P_{I_U} \\
& +\gamma_{AU}P_{I_A|S} + \Gamma_{AU}P_{I_A}]\frac{P_S}{P_I}\}P_{S|I} \qquad (31)
\end{aligned}
$$

We obtain a closed system by including the resident equilibrium values and using:

$$\frac{P_I}{P_S} = \frac{P_{I|S}}{P_{S|I}}. \qquad (32)$$

By finding the equilibrium solution numerically, we obtain the local spatial structure of the invasion early in its development.

Appendix 3

We present a proof that selection cannot lead to lower user host resistance in the mean field case. Let J be the Jacobian matrix for the new phenotype invading at low density, i.e. equations 5–6 linearized around $P_S = P_I = 0$ and the resident populations at the equilibrium solution of equations 1–4. Then $J =$

$$\begin{bmatrix} B_U P_E^* - B_A(P_{S_A}^* + P_{I_A}^*) - \mu_U - (1-\rho')(\Gamma_{AU}P_{I_A}^* + \Gamma_{UU}P_{I_U}^*) & B_U P_E^* \\ (1-\rho')(\Gamma_{AU}P_{I_A}^* + \Gamma_{UU}P_{I_U}^*) & -B_A(P_{S_A}^* + P_{I_A}^*) - \alpha_U\mu_U \end{bmatrix}$$

Now, from equations 3–4, we know that the resident equilibrium populations satisfy:

$$B_U P_E^* - B_A(P_{S_A}^* + P_{I_A}^*) - \mu_U = (1-\rho_U)(\Gamma_{AU}P_{I_A}^* + \Gamma_{UU}P_{I_U}^*) - B_U P_E^* \frac{P_{I_U}^*}{P_{S_U}^*}, \tag{33}$$

and

$$B_A(P_{S_A}^* + P_{I_A}^*) + \alpha_U\mu_U = (1-\rho_U)(\Gamma_{AU}P_{I_A}^* + \Gamma_{UU}P_{I_U}^*)\frac{P_{S_U}^*}{P_{I_U}^*}. \tag{34}$$

After substituting these expressions into J and rearranging, we find:

$$det(J) = (\rho_U - \rho')(\Gamma_{AU}P_{I_A}^* + \Gamma_{UU}P_{I_U}^*)\left[(1-\rho_U)(\Gamma_{AU}P_{I_A}^* + \Gamma_{UU}P_{I_U}^*)\frac{P_{S_U}^*}{P_{I_U}^*} - B_U P_E^*\right]. \tag{35}$$

But from equation 33, we find that:

$$B_U P_E^* = (1-\rho_U)(\Gamma_{AU}P_{I_A}^* + \Gamma_{UU}P_{I_U}^*)\frac{P_{S_U}^*}{P_{I_U}^*} - (\alpha_U - 1)\mu_U\frac{P_{S_U}^*}{P_{S_U}^* + P_{I_U}^*}. \tag{36}$$

Therefore,

$$det(J) = (\rho_U - \rho')(\Gamma_{AU}P_{I_A}^* + \Gamma_{UU}P_{I_U}^*)\left[(\alpha_U - 1)\mu_U\frac{P_{S_U}^*}{P_{S_U}^* + P_{I_U}^*}\right]. \tag{37}$$

Provided that $\alpha_U \geq 1$ (i.e. infection shortens the host's lifespan), we see that $det(J)$ has the same sign as $\rho_U - \rho'$. Thus, if the invader has higher resistance than the resident, one eigenvalue is negative and the other is positive, so that the invasion succeeds. If the invader's resistance is lower than the resident's, both eigenvalues are positive or both are negative. Now,

$$tr(J) = (\rho' - \rho_U)(\Gamma_{AU}P_{I_A}^* + \Gamma_{UU}P_{I_U}^*) - B_U P_E^*\frac{P_{I_U}^*}{P_{S_U}^*} - B_A(P_{S_A}^* + P_{I_A}^*) - \alpha_U\mu_U, \tag{38}$$

so that $tr(J) \leq 0$ when $\rho' \leq \rho_U$. Thus, when the invader's resistance is lower than the resident's, both eigenvalues are negative and the invasion fails.

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Figure Captions

Figure 1: Equilibrium populations of user (a) and attacked (b) hosts as a function of user's resistance in the mean field alternating model. Increasing resistance lowers the user's population level, indicating that the disease provides a net benefit at the population level. Note the resistance threshold: the disease dies out if the resistance exceeds 0.6. Parameters are: $B_A = 0.215, B_U = 4.5, \Gamma_{AU} = 2.0, \Gamma_{UA} = 3.0, \alpha_A = 2.3, \alpha_U = 1.1, \rho_A = 0$.

Figure 2: Evolutionary flux in user's resistance. (a) Results from simulations of the full stochastic model, with standard error bars. An ESS occurs where the flux is zero. We ran 10 simulations on a 1000×1000 lattice for each resistance level. Invader's resistance was $\rho_U \pm 0.05$. After resident system reached equilibrium, invader was introduced by switching 0.5% of all sites to the susceptible invader state. Invasion eigenvalues were estimated by fitting an exponential curve to the total invader population for 400 time steps after introduction. Damage to user is $\alpha_U = 1.09$; other parameters are $B_A = .215, \beta_U = 6.3, \Gamma_{AU} = 1.7, \gamma_{UA} = 6.0, \alpha_A = 2.4, \rho_A = 0$. (b) Predictions by pair approximation for different levels of damage to user. Values of α_U for the curves are, from bottom to top: $1.0, 1.03, 1.06, 1.09$.

Figure 3: ESS user resistance as a function of damage done by the pathogen. Dashed lines give threshold resistance levels above which the disease dies out. Curves give ESS resistance for different levels of damage to the attacked host. Other parameters are as in Figure 2.

Figure 4: Relationship between ESS user resistance and the resistance level of the attacked host. Dashed lines give threshold user resistance levels; curves give ESS user resistance levels for different levels of attacked host resistance. Other parameters are as in Figure 2.

Figure 5: ESS user resistance for different dispersal scales of the attacked host and attacked–user transmission. For A and B, attacked host dispersal is local, with $\beta_A = .25$, $\beta_U = 5.0$, $\gamma_{UA} = 10.0$, and $\alpha_A = 2.0$. For A, $\gamma_{AU} = 10.0$ (local transmission); for B, $Gamma_{AU} = 2.0$

(global transmission). For C and D, attacked host dispersal is global, with $B_A = .215$, $\beta_U = 6.3$, and $\alpha_A = 2.4$. For C, $\gamma_{AU} = 5.0$ and $\gamma_{UA} = 5.5$; for D, $\Gamma_{AU} = 1.7$ and $\gamma_{UA} = 6.0$.

Figure 6: ESS user resistance for different pathogen life cycles. All dispersal is local, with $\beta_A = .25$, $\beta_U = 5.0$, and $\alpha_A = 2.0$. In the case of arbitrary transmission, $\gamma_{AU} = \gamma_{UU} = 1.5$ and $\gamma_{UA} = \gamma_{AA} = 1.45$. In the case of alternating transmission, $\gamma_{AU} = \gamma_{UA} = 10.0$.

Figures

Figure 1a:

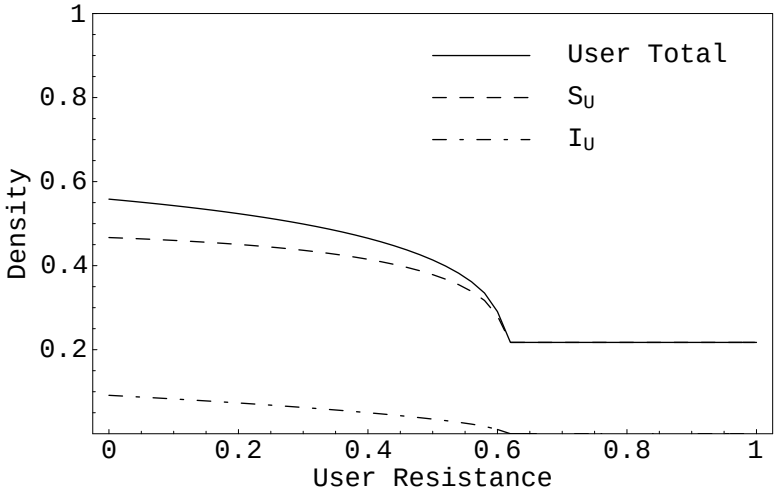

Figure 1b:

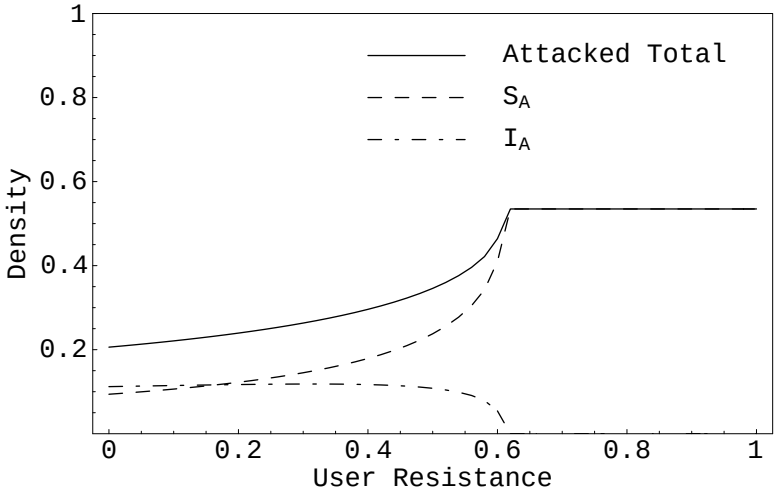

Figure 2a:

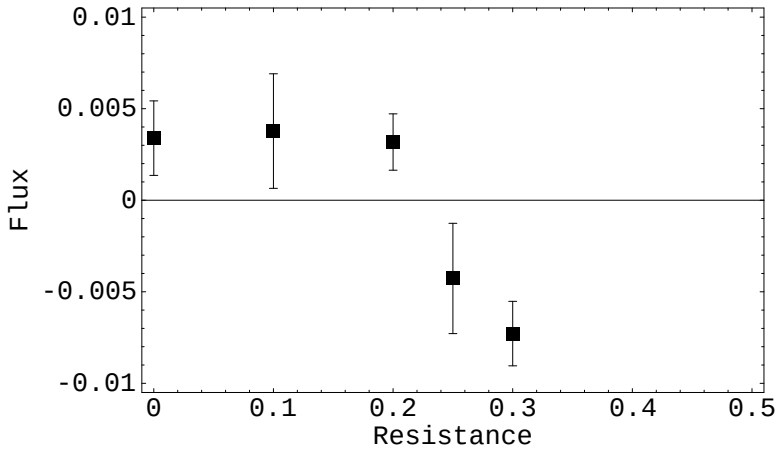

Figure 2b:

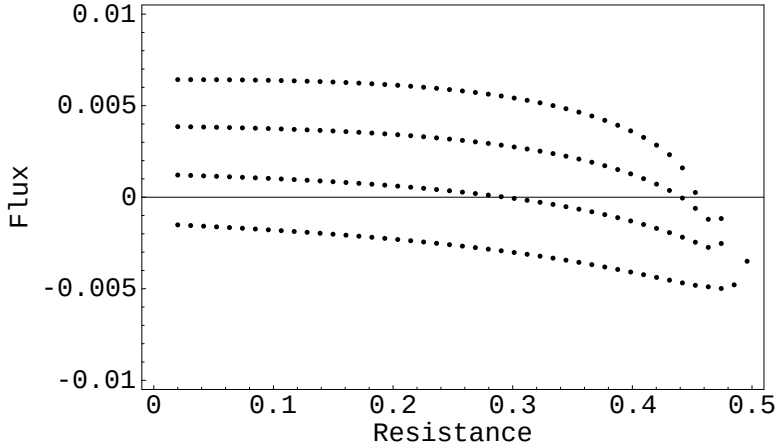

Figure 3:

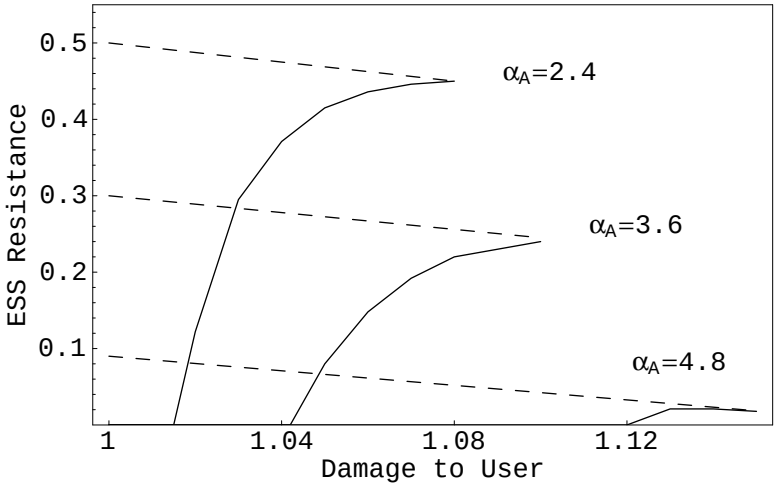

Figure 4:

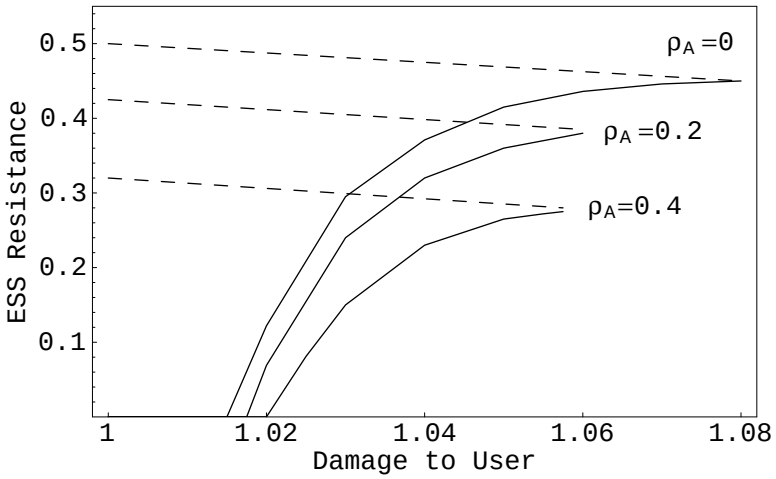

Figure 5:

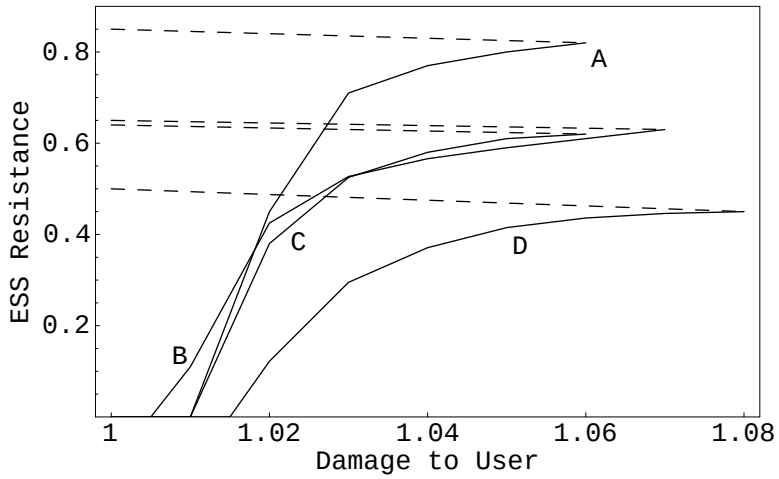

Figure 6: