

Apparent competition and recovery from infection

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Abstract

We use mathematical models to analyse how the recovery rate from infection influences the fitness of a host in a setting of interspecific competition. We show that sub-optimal immunity against pathogens can be advantageous for the host in the presence of cross-species infection. Weaker immunity allows the parasite to be used as a biological weapon, and this increases the fitness of the host relative to a competitor. A parameter region is observed in which the outcome of competition depends on the initial conditions. We extend this model and consider the dynamics in a spatial setting and find that the outcome depends on the migration rate of the host species. At low migration rates, coexistence of the host species is possible across space. For higher migration rates, the host species characterized by a lower recovery rate can invade the territory of its competitor. Finally, we study these dynamics in an evolutionary setting. Although a lower recovery rate from infection can increase the competitive ability of a species, we find that evolution maximizes the recovery rate and minimizes parasite burden. The models presented are related to the concept of apparent competition, and our results are discussed in relation to both theoretical and empirical studies.

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1. Introduction

Most living organisms are susceptible to pathogens. Many infections have a detrimental effect and reduce the fitness of the host. To counter pathogens, higher organisms have evolved immune systems. If an infection is detrimental for the host, improved immunity and lower pathogen prevalence can result in an increased host fitness, subject to possible metabolic and reproductive costs associated with maintaining an immune system. Thus, we can see a co-evolutionary race between pathogen infectivity and host resistance (Anderson, 1995; Boots and Bowers, 1999; Frank, 1991, 2000; van Baalen, 1998).

These arguments, however, consider the interaction between a host species and a pathogen in isolation. In the natural world, different host species interact with each other in a number of ways. For example, they can compete for shared resources. Competition can have a variety of outcomes, such as the exclusion of species.

Pathogens can play a major role in modulating the competitive interactions between host species. For example, pathogens can reduce the fitness of their hosts and reduce their competitive strength (Washburn et al., 1991; Gregory and Keymer, 1989). On the other hand, competition can be mediated by pathogens themselves. Even if two host species do not interact directly, exclusion of one species by another can be brought about by a pathogen which is shared. This concept is called apparent competition (Holt, 1977). Exclusion is the consequence of the pathogen becoming more abundant because it has a second host species. The species which survives is the one which can support higher pathogen loads in the population. The importance of apparent competition for structuring ecological assemblages has been demonstrated by experiment using a laboratory insect system (Hassell and Bonsall, 1997). Two host species shared a common parasitoid but were not allowed to interact directly. While both single host–parasitoid interactions were stable in separation, one species was excluded when the parasitoid was shared. Several examples of species exclusion in the wild have been attributed to apparent competition, where two host species are thought to share a parasite which is

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characterized by different degrees of pathogenicity in the two species. The host in which the parasite is less pathogenic can exclude the host in which the parasite is more pathogenic. Experimental data have been coupled with mathematical modeling to analyse these interactions (Hudson and Greenman, 1998; Greenman and Hudson, 1999, 2000; Tompkins et al., 2000, 2001, 2002).

According to these arguments, it might be beneficial for a host species to keep the pathogen load in the population at higher levels. In this paper, we build on these ideas and examine a more complex scenario. We assume that two host species share a resource and are in direct competition with each other. Each species has a native pathogen. Each pathogen can, however, also infect the other host species in which it is more pathogenic. Thus each species carries a native pathogen which can be used as a weapon against the competitor. We investigate how the recovery rate from infection influences the outcome of host competition. First, we study a basic ecological model to examine whether a reduced recovery rate from infection can be advantageous. We then extend this analysis to consider these dynamics in a spatial setting. Finally, we introduce mutation and evolution to the spatial model. We seek to understand in which direction the recovery rate from infection is expected to evolve.

2. Basic competition dynamics

2.1. The model

We consider a basic model which consists of four variables. Uninfected and infected hosts of species 1 (x_1 and y_1), as well as uninfected and infected hosts of species 2 (x_2 and y_2). The model is explained schematically in Fig. 1. Uninfected hosts reproduce, die, and become infected by the pathogen. Infected hosts are assumed not to reproduce. They may die or recover from infection. When hosts recover from infection, they are assumed to become susceptible again (SIS model). The model is given by the following set of differential equations which describe the development of the populations over time:

$$\begin{aligned} \dot{x}_1 = & [b_1 - d_1 - h_1(x_1 + x_2)]x_1 - \beta_1 x_1 y_1 \\ & - p_1 x_1 y_2 + \gamma_1 y_1, \end{aligned} \quad (1)$$

$$\dot{y}_1 = \beta_1 x_1 y_1 - (d_1 + \alpha_1 + \gamma_1)y_1 - q_1 y_1 y_2, \quad (2)$$

$$\begin{aligned} \dot{x}_2 = & [b_2 - d_2 - h_2(x_1 + x_2)]x_2 - \beta_2 x_2 y_2 \\ & - p_2 x_2 y_1 + \gamma_2 y_2, \end{aligned} \quad (3)$$

$$\dot{y}_2 = \beta_2 x_2 y_2 - (d_2 + \alpha_2 + \gamma_2)y_2 - q_2 y_2 y_1, \quad (4)$$

where b_i , d_i and h_i are the birth rate, the natural mortality, and the coefficient of density dependent

mortality of host species i ; β_i , γ_i , and α_i are the transmission rate, the recovery rate, and the pathogenicity (additional mortality) of pathogen species i in its native host.

In addition to these basic infection dynamics, we also assume that the pathogen of one host species may infect the competitor (cross-species infection). That is, pathogen 1 (y_1) may infect host species 2 (x_2), and pathogen 2 (y_2) may infect host 1 (x_1). It is further assumed that this cross-species infection results in more pathogenicity than infection with the native pathogen (because there is lack of adaptation). In fact, we assume an extreme scenario for simplicity: Cross-species infection kills the competitor instantaneously, and the competitor cannot pass on the pathogen. In model terms, cross-species infection kills the uninfected and infected host populations at rates p_i and q_i , respectively. Because we aim to concentrate on the effect of cross-species infection on the interactions between the two host species, we will only investigate the scenario where the host species are competitively equivalent in the absence of pathogens.

Some further notes regarding model assumptions. We make the assumption that cross-species infection kills the host. While such a scenario can be realistic for some emerging infections (e.g. brought about by zoonotic transmission), it need not be the case. Alternatively, hosts infected by the non-native pathogen may recover or even contribute to limited parasite transmission. Including these dynamics would result in a much more complex model which is not tractable. In addition, the fitness reduction of the competitor would be less significant. Since we are interested in the case where the fitness of the competitor is significantly reduced by cross-species infection, this simplification is valid. Other simplifications include that infected hosts are assumed not to reproduce, and that they do not contribute to density dependence in the growth terms. A more complicated model could include reproduction of infected hosts, contribution of infected to density dependence, and vertical transmission of the parasite. Numerical simulations, however, indicate that these complications do not qualitatively alter the cross-species infection dynamics which are subject of this paper. This is in part because the proportion of infected host individuals is relatively low.

2.2. Analysis of the model

In the following we describe the behavior of this model. This is done by a combination of analytical and numerical methods. Expressions for the more complicated equilibria and their stability are not given because they are too long and do not add to our understanding. In these cases, results are based on numerical simulations. Because we are interested in the effect of cross-species infection on the competition dynamics, we

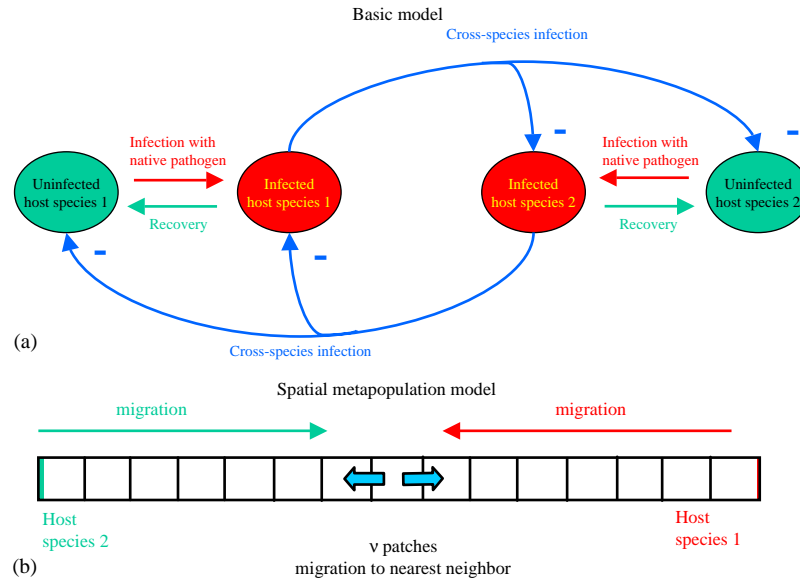


Fig. 1. (a) Schematic representation of the basic mathematical model. It is described and analysed in the text. (b) Schematic representation of the spatial model. We consider a one-dimensional metapopulation model which consists of v patches. In each patch, local dynamics occur according to the equations described in the text. Populations migrate to the two nearest neighboring patches. At the boundaries, migration occurs only in one direction. The diagram further shows how the model is simulated. We start with host species 1 at 2 at the respective ends of the space, and allow them to migrate.

assume that the two host species are competitively neutral in the absence of pathogens. That is, $b_1 = b_2$, $d_1 = d_2$, and $h_1 = h_2$. We further assume that the effect of cross-species infection is equivalent for the populations x and y ; that is, $p_i = q_i$. In addition, the killing rates are assumed to be identical for both host species. Because we aim to study the system under these particular assumptions, we will only present the outcomes and equilibria which are relevant in this context. While the system contains other equilibria additional to the ones presented here, they are not stable under the assumptions made and are not discussed. We define $K = (b - d)/h$ as the carrying capacity of either species of host. It is convenient to define the basic reproductive ratios, R_{01} and R_{02} , of pathogen species 1 and 2:

$$R_{01} = \beta_1 K / (d + \alpha_1 + \gamma_1),$$

$$R_{02} = \beta_2 K / (d + \alpha_2 + \gamma_2).$$

We observe the following outcomes.

- **Disease free equilibrium:** The pathogens are not maintained in the population. In this case, $y_1 = y_2 = 0$, and $x_1 + x_2 = K$, $K(1 - 1/R_{02}) < x_1 < K/R_{01}$. Because the host species are competitively neutral in the absence of pathogens, the sum of the two host species is kept at the carrying capacity K . The density of either host species must not exceed the threshold $x_1 < K/R_{01}$ and $x_2 < K/R_{02}$, in order to refuse the invasion of native pathogen species. The stable line segment defined above exists if $1/R_{01} + 1/R_{02} < 1$.

- **Species 1 wins and outcompetes species 2:** The native pathogen is maintained in the population. That is, $x_2 = 0$, $y_2 = 0$, and $x_1 = K/R_{01}$, $y_1 = [hK^2/(d + \alpha_1)](1/R_{01})(1 - 1/R_{01})$. This equilibrium is locally stable if $1 < R_{01} < p_2 K / (d + \alpha_2)$.
- **Species 2 wins and outcompetes species 1:** Again, the native pathogen is maintained in the population. That is, $x_1 = 0$, $y_1 = 0$, and $x_2 = K/R_{02}$, $y_2 = [hK^2/(d + \alpha_2)](1/R_{02})(1 - 1/R_{02})$. This equilibrium is locally stable if $1 < R_{02} < p_1 K / (d + \alpha_1)$.
- **Coexistence equilibrium:** Both species coexist and the respective pathogens are maintained ($x_1 > 0$, $y_1 > 0$, $x_2 > 0$, $y_2 > 0$). Equilibrium expressions are complex and not written out here. This outcome is observed if $R_{01} > p_2 K / (d + \alpha_2)$ and $R_{02} > p_1 K / (d + \alpha_1)$.

2.3. Recovery from infection and competition

We are interested in how the rate of recovery from infection, γ_i , influences the competitive ability of the hosts. The stability properties of the equilibria in dependence of the recovery rate for the two species (γ_1 and γ_2) are shown schematically in Fig. 2. The thresholds which separate different outcomes are derived from the stability conditions given in the last section, as explained in the figure. In the following we will ignore the disease free outcome, since we are interested in the effect of the pathogens. Coexistence of the two host species is only possible in a limited parameter region when the recovery rates of both species are low (see Fig. 2 for an exact definition of this parameter space).

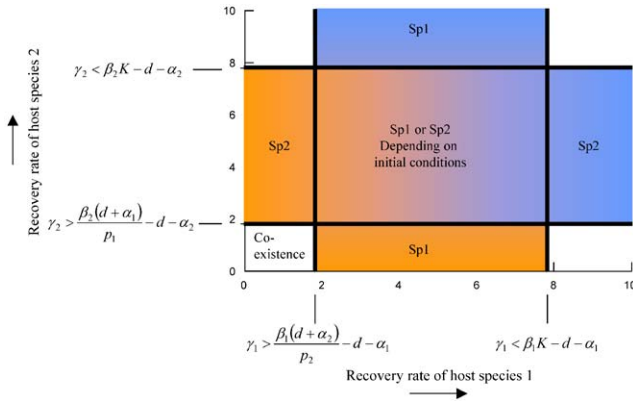


Fig. 2. The effect of the recovery rate from infection on the competition dynamics of two host species (Sp1 and Sp2) which share two species of parasites. In the different parameter regions, host species 1 can win, host species 2 can win, or both can win depending on the initial conditions. Which outcome is observed depends on the recovery rate from infection of both species (γ_1 , γ_2). The threshold conditions which separate the different outcomes are shown; they have been derived from the stability conditions for the equilibria presented in the text. The threshold expressions define exactly under which conditions the different outcomes are observed, and complement the verbal description in the text. In the parameter regions which are left blank, only the disease free outcome is stable, and this is not considered in this paper. Parameters were chosen as follows. $\beta_i = 1$; $p_i = 0.1$; $q_i = 0.1$; $r_i = 0.5$; $d_i = 0.1$; $\alpha_i = 0.1$; $b_i = 0.1$.

Otherwise, we observe the following pattern. If the recovery rate of species 1 lies below a threshold relative to that of species 2, species 2 wins. The cost of carrying the pathogen in the population outweighs the benefit. The expression for this threshold is given in Fig. 2. If the recovery rate of species 1 is high and lies above a threshold relative to species 2, species 2 wins again. This is because a high recovery rate keeps the pathogen at low levels, allowing for only little transmission to the competitor. The expression for this threshold is given in Fig. 2. If the recovery rate of species 1 is intermediate (between the thresholds cited above), it can win and outcompete species 2. The pathogen is prevalent enough to be used as a biological weapon, while it is not too costly for the native host. If the recovery rates of both species are intermediate (parameter space including the orange to blue gradient in Fig. 2), then two outcomes are stable: either species 1 wins, or species 2 wins, depending on the initial conditions. The higher the initial abundance of one species relative to the other, the more likely this species is to win and persist. In the region of bistability, the exact initial abundance of the host species required to win the competition depends in the recovery rate from infection. The higher the recovery rate, the higher the initial abundance required to outcompete the other host species. This is because a higher recovery rate compromises the use of the parasite as a weapon in host competition. The mathematical

conditions which define this parameter region are again specified in Fig. 2.

We would like to point out that the results presented in Fig. 2 are not dependent on specific simplifying assumptions made here, that is the absence of reproduction of infected hosts, or the lack of contribution to density dependence by infected hosts. We have explored more complex models which relax these simplifications by numerical simulations and found that the results remained qualitatively robust.

3. The effect of space

3.1. The model

Here we concentrate further on the parameter region where the outcome depends on initial conditions. Including space and migration into the model gives rise to further results. Space is formulated as a metapopulation model (Fig. 1b). We consider a one-dimensional space along which uninfected and infected hosts can migrate. In other words, we assume the existence of v patches. In each patch, local dynamics occur as described in the basic competition model explained in the last section. In addition, migration of hosts occurs to the nearest neighboring patches at a rate m . At the boundaries of this one-dimensional space, migration stops. In other words, at the boundaries migration occurs only in one direction.

This is a relatively simple spatial set-up. When considering the results of the model and the implications, it is important to keep in mind that a different and more complex spatial scenario might alter model behavior. In addition to the one-dimensional space, we have also explored a two-dimensional space or grid where migration occurs to the nearest neighboring patches. The basic results obtained for the one-dimensional space remained robust. However, many more possible assumptions regarding the spatial set-up and migration patterns exist. While a full exploration of these possibilities is beyond the scope of this study, this is important to keep in mind.

In the following we explore the dynamics in this spatial setting. While the results are quite simple and will not come as a surprise to ecologically minded readers, a description of this behavior is important because it forms the basis for the subsequent model which introduces mutations and explores the evolution of the recovery rate from infection.

3.2. Outcome of competition across space

We study the system by numerical simulations. Analytical results have not been obtained because of the complexity. We concentrate on the parameter region

in which the outcome of the dynamics depends on the initial conditions in the non-spatial model. As starting conditions we assume that species 1 is at equilibrium at one boundary, while species 2 is at equilibrium at the other boundary. We study the competition dynamics in dependence of the migration rate, m (Fig. 3). In the present context, we distinguish between two parameter regions, depending on the value of m . (i) If the migration rate, m , is low and lies below a threshold, the two species meet in the middle of the one-dimensional space, but cannot invade each others territory. In other words, they coexist across space. The reason is as follows. A low migration rate results in a low initial abundance of the invading species relative to that of the resident species in the patch occupied by the resident species. Under such initial conditions, invasion cannot occur. (ii) If the

migration rate, m , crosses the threshold, we find that the host species with the lower recovery rate invades the territory of its competitor and excludes it. This is because a higher migration rate ensures that the initial population size of the invading species is now higher relative to that of the resident species in the patch of the resident species. This larger invasion force tips the balance in favor of the invading species.

Analytical expressions for the migration rate threshold were not obtained because of the complexity of the system. The nature of these thresholds can, however, be understood easily by numerical simulations and from the properties of the non-spatial model. We examined the dynamics in the parameter regions in which the outcome depended on the initial conditions. Consider the interaction between a superior competitor (relatively

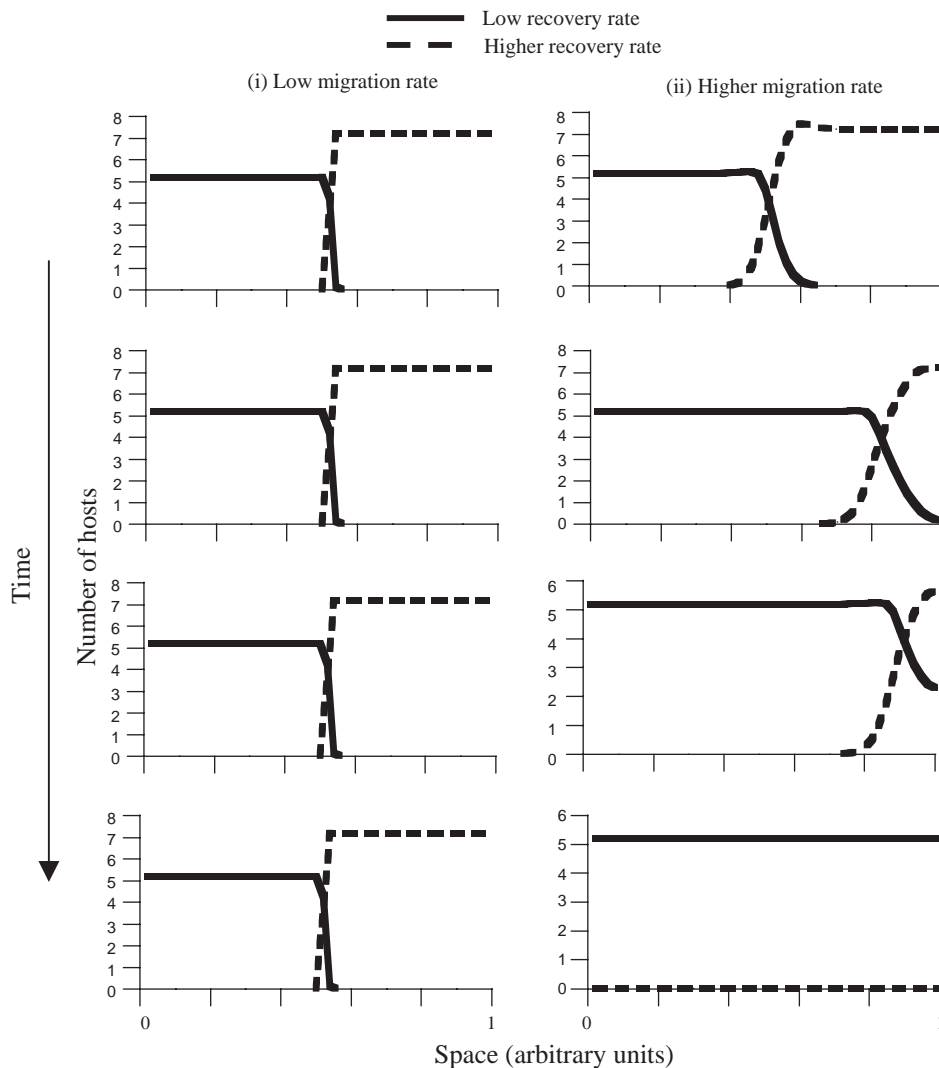


Fig. 3. Outcome of the competition dynamics in a spatial setting. The sequential graphs depict how the spatial distribution of the two host species develops over time. (i) Dynamics assuming a low migration rate. The graphs show an outcome in which the two host species coexist across space. That is, they cannot invade each others' territories. (ii) Higher migration rate. The species with the lower recovery rate invades the territory of its competitor and excludes it. Parameters were chosen as follows. $\beta_i = 1$; $p_i = 0.1$; $q_i = 0.1$; $r_i = 0.5$; $d_i = 0.1$; $\alpha_i = 0.1$; $b_i = 0.1$; $\gamma_1 = 7$; $\gamma_2 = 5$; $m = 0.000001$ for lower migration rate; $m = 0.0001$ for higher migration rate.

low recovery rate), and an inferior competitor (higher recovery rate). In the bistable parameter region, the superior competitor can only win if its initial abundance lies above a threshold relative to that of the inferior competitor. In the spatial model, the initial abundance of a species in a patch can be translated into the migration rate. Assume that we start with 100% prevalence of species 1 in patch 1, and 100% prevalence of species 2 in patch 2. Now, each species migrates to the other patch with a rate m . The invasion force corresponds to the initial abundance of the invading relative to the resident species, and is determined by the parameter m . The higher the value of m , the higher the initial abundance of the invading relative to the resident species. Therefore, as the migration rate crosses a threshold, the initial abundance of the invading species becomes sufficiently high so that it can take over if it is the superior competitor. Consequently, the exact migration rate threshold required for invasion depends in the same way on parameters as the initial conditions threshold in the non-spatial model. The threshold thus depends mainly on the advantage of the superior over the inferior competitor. The smaller this advantage, the higher the migration rate threshold required for invasion. In the current context, the main variable which determines competitive ability and thus the threshold is the recovery rate from infection, γ . The higher the recovery rate of the superior host species, the lower its competitive advantage, and the higher the migration rate threshold.

4. Evolutionary dynamics

4.1. The model

Now we study these dynamics from an evolutionary point of view. The above results have shown that a species with a lower recovery rate can outcompete a species with a higher recovery rate because the relative fitness of the competitor is reduced by cross-species infection. Here we ask in which direction the host recovery rate is expected to evolve. We start with the spatial model described above. This is the basis for exploring evolution. That is, on top of the spatial model, we introduce mutation. We assume that in each species, mutation can give rise to new variants which differ in the recovery rate. The different mutants of a given species are assumed to be in competition with each other. Thus, there is both intra- and inter-specific competition.

The model is described as follows. The local dynamics which occur in each patch are given by a system of differential equations which is based on the model presented in Section 2.1. The basic equations and assumptions are essentially the same. The difference is that the model considered here assumes the existence

of n variants of each species ($x_{1j}, y_{1j}, x_{2j}, y_{2j}$, where $j = 1, \dots, n$). The variants are assumed to compete for a shared resource. The variants are further assumed to differ in the recovery rate from infection, γ_{ij} . With a rate μ , a given variant can give rise to a new variant which is characterized by either a lower or a higher recovery rate. The local dynamics in each patch are therefore described as follows.

$$\begin{aligned} \dot{x}_{1j} = & \left[b_1 - d_1 - h_1 \left(\sum_{j=1}^n x_{1j} + \sum_{j=1}^n x_{2j} \right) \right] x_{1j} \\ & - \beta_1 x_{1j} \sum_{j=1}^n y_{1j} - p_1 x_{1j} \sum_{j=1}^n y_{2j} + \gamma_{1j} y_{1j} + \mu x_{1j-1} \\ & + \mu x_{1j+1} - 2\mu x_{1j}, \end{aligned} \quad (5)$$

$$\dot{y}_{1j} = \beta_1 x_{1j} \sum_{j=1}^n y_{1j} - (d_1 + \alpha_1 + \gamma_{1j}) y_{1j} - q_1 y_{1j} \sum_{j=1}^n y_{2j}, \quad (6)$$

$$\begin{aligned} \dot{x}_{2j} = & \left[b_2 - d_2 - h_2 \left(\sum_{j=1}^n x_{1j} + \sum_{j=1}^n x_{2j} \right) \right] x_{2j} + \gamma_{2j} y_{2j} \\ & - \beta_2 x_{2j} \sum_{j=1}^n y_{2j} - p_2 x_{2j} \sum_{j=1}^n y_{1j} + \gamma_{2j} y_{2j} + \mu x_{2j-1} \\ & + \mu x_{2j+1} - 2\mu x_{2j}, \end{aligned} \quad (7)$$

$$\begin{aligned} \dot{y}_{2j} = & \beta_2 x_{2j} \sum_{j=1}^n y_{2j} - (d_2 + \alpha_2 + \gamma_{2j}) y_{2j} \\ & - q_2 y_{2j} \sum_{j=1}^n y_{1j}. \end{aligned} \quad (8)$$

The local dynamics described above are incorporated into a spatial setting in the same way as in the previous section. That is, we consider a one-dimensional space which contains v patches. In each patch, the local dynamics occur according to the above equations. In addition, all populations migrate to the two neighboring patches at a rate m . At the boundaries, migration only occurs in one direction.

4.2. Evolution of the recovery rate

The evolutionary dynamics are studied by numerical simulations (Fig. 4). Within a given species, we find that a mutant with an increased recovery rate can always invade and outcompete a variant with a lower recovery rate. Thus, evolution will take a species towards stronger immunity until the recovery rate crosses a threshold and the pathogen cannot be maintained in the population anymore. Therefore, evolution leads to a loss of biological weapons. This can have the following consequences. If evolution increases the recovery rate of a given species, it can decrease its fitness relative to that

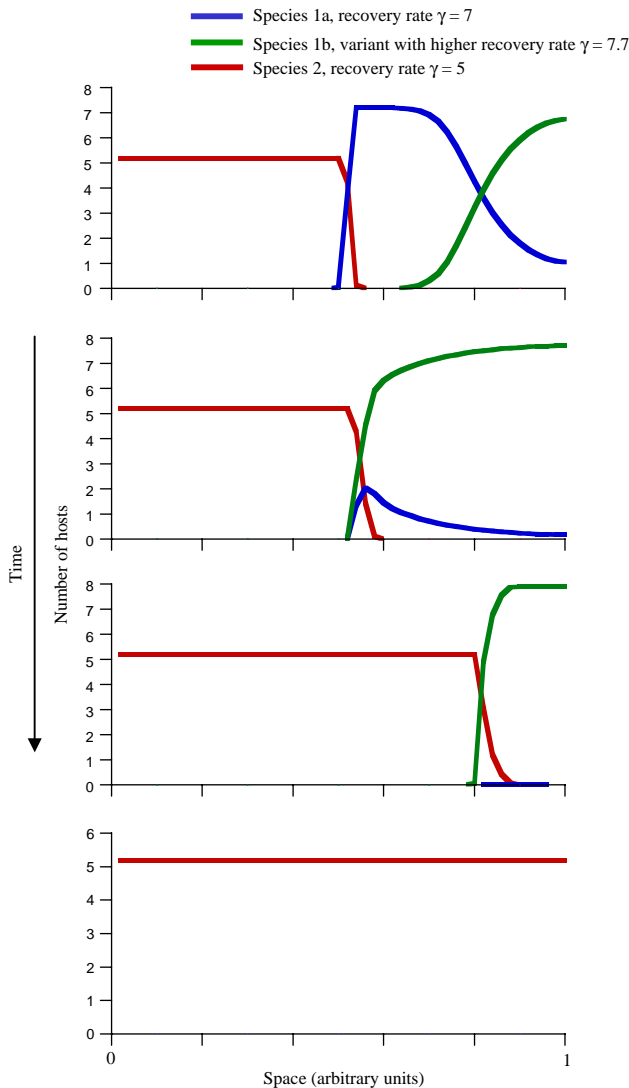


Fig. 4. Evolution of the recovery rate from infection. We assume that there are two variants of species 1 which differ in the recovery rate. Species 1a has a lower recovery rate than species 1b. Both variants of species 1, however, have a higher recovery rate than species 2. Species 1a can coexist with species 2 across space. However, species 1b outcompetes species 1a. Because species 1b has an elevated recovery rate from infection, it can then be excluded by species 2. Parameters were chosen as follows. $\beta_i = 1$; $p_i = 0.1$; $q_i = 0.1$; $r_i = 0.5$; $d_i = 0.1$; $\alpha_i = 0.1$; $b_i = 0.1$; $m = 0.0001$.

of a competitor. Consider two host species which can coexist across space and which mutate to give rise to variants which differ in their recovery rate. Assume that evolution increases the recovery rate of species 1 beyond a threshold relative to species 2. Now, species 2 can invade the territory of species 1 and drive it extinct (Fig. 4). Thus, evolution can take the species into a parameter region in which it is prone to competitive exclusion. While a lower recovery rate gives a species an advantage over a competitor, a higher recovery rate is selected for within a species. In other words, selection acts on the individual and not on the species.

5. Discussion

5.1. Parasites and host competition

In this paper we analysed mathematical models to investigate how the recovery rate from infection (immunity) influences the dynamics between parasites and hosts. We assumed that two host species are in competition with each other, and that both species carry parasites which can cause high pathogenicity upon cross-species infection. This gives rise to the following results.

- The basic ecological dynamics suggest that a relatively low rate of recovery from infection can give a host species a competitive advantage over a species with a higher recovery rate. While the parasite burden is costly for the host, the ability to transmit the parasite to the competing species, and hence to damage the competitor, can outweigh this cost. Therefore, a higher parasite burden allows the pathogen to be used as a biological weapon in host competition.
- The spatial model introduces new aspects to these dynamics. For low migration rates, it allows for coexistence of the two host species across space. The pathogen serves a defensive role and prevents invasion by the competitor. For higher migration rates, the host with the lower recovery rate drives its competitor extinct. The pathogen serves an offensive role and enables the host to invade new space.
- The evolutionary model suggest that evolution maximizes the recovery rate from infection and minimizes parasite burden. This is because selection maximizes the fitness of the individual and not of the group. Hence, evolution takes the species into a parameter region in which it is prone to be excluded by a competitor.

Having summarized the results derived in this paper and their implications, it is important to consider them in the context of model assumptions. Regarding the basic competition and infection dynamics, we have made a number of simplifying assumptions. For example, cross-species infection is assumed to result in instant death. This can indeed occur in nature if cross-species infection results in very high levels of pathogenicity, such as with ebola virus in humans. Alternatively, it could be assumed that cross-species infection results in disease which reduces the fitness of the host temporarily until recovery occurs. Of course, the less severe the effect of cross-species infection, the less it will benefit the population of hosts to maintain pathogens. That is, a low recovery rate from infection becomes less of an advantage in the context of inter-specific competition. Other simplifying assumptions of the basic model are that infected hosts do not contribute to the density dependence in the population growth term, and that they do not reproduce. We performed numerical simulations where these simplifying assumptions were

relaxed and found that the results considered here remained robust. The fraction of infected hosts does not reach high levels in biologically relevant regimes; in addition, infected hosts are assumed to recover with a rate γ , and the absence of reproduction during the phase of infection does not significantly impact the dynamics. In addition to these basic interactions, we also considered space, and the spatial model formed the basis for the considerations regarding the evolution of the recovery rate from infection. We assumed the existence of a one-dimensional space with migration. Much more complex spatial scenarios are possible. It is possible that specific spatial settings could favor the maintenance of infection, and therefore, evolution might not lead to the loss of biological weapons as suggested here. We have explored the evolutionary dynamics numerically in the setting of a two dimensional space, and the results described here remained robust. It should, however, be remembered that more intricate assumptions regarding space and migration could lead to different results.

Our analysis of the model has focused on the effect of the recovery rate from infection on the dynamics. This is because we were interested in immunity as a regulator of host competition. Parasite parameters which influence the basic reproductive ratio will also affect the outcome. Thus, an increased infectivity could enhance the competitive ability of a host species because it maintains a higher level of parasites which can be transmitted to a competitor. Evolution of viral parameters, however, is more complex in this setting and was not considered further.

Our modeling framework has similarities to the concept of apparent competition (Holt, 1977). This means that two or more host populations share a common enemy (such as a predator or parasite), and that competition is mediated only through this enemy. In such a scenario, the species which is most tolerant to the shared enemy can result in the exclusion of other species even in the absence of direct competition for resources. This effect of apparent competition has been recently demonstrated by experiments using a host–parasitoid system (Hassell and Bonsall). The effect of a shared parasite on the dynamics between two host species has been investigated by mathematical models (Hudson and Greenman, 1998; Greenman and Hudson, 1999, 2000). While some basic results of our analysis overlap with these studies, the framework presented here examined the effect of parasite-mediated competition between hosts from a different angle and in a different setting. We assumed the existence of two directly competing hosts which share two species of parasites. Each host is subject to infection with its native pathogen, and the native pathogen can kill the competitor via cross-species infection. This gave rise to a variety of interesting new results and allowed us to

study how the efficacy of immunity and recovery from infection influences the competitive strength of the host species. In addition, we explored how these ecological dynamics translate into an evolutionary setting. In the following we discuss empirical data in the context of our theoretical considerations.

5.2. Theory and field studies

This theoretical discussion has important implications for understanding observed patterns of species exclusion or coexistence in the wild. The most basic message is that the transmission of parasites across species may have to be taken into considerations in order to explain the outcome of competition dynamics. This conclusion has also been reached in a number of previous studies (Hudson and Greenman, 1998; Greenman and Hudson, 1999, 2000).

Perhaps, the best studied example is the competition between red squirrels (*Sciurus vulgaris*) and gray squirrels (*Sciurus carolinensis*) in the UK (Tompkins et al., 2002, 2003). When gray squirrels were introduced to the UK they invaded the territory of the resident red squirrels which consequently declined. Experimental data suggest that the replacement of red squirrels by gray squirrels could be caused by a parapoxvirus which was introduced to the UK together with the gray squirrel. Experiments have shown that parapoxvirus infection is highly pathogenic in red squirrels, whereas it does not have any detectable effect on the health of gray squirrels. In support of this hypothesis, gray squirrel seroprevalence to parapoxvirus correlates with the degree of decline of the red squirrel populations. The decline of red squirrels is most marked in English and Welsh populations, where gray squirrel seroprevalence to parapoxvirus is high. On the other hand, the negative impact of red squirrels is least apparent in Scottish and Irish populations where seroprevalence is zero.

Another intriguing example is the interaction between the ring-necked pheasant *Phasianus colchicus* and the gray partridge *Perdix perdix* (Tompkins et al., 2000, 2001). The pheasant population maintains an infection with the caecal nematode *Heterakis gallinarum*. While this parasite cannot be maintained in the partridge population because of low reproductive potential in this species (Tompkins et al., 2000), the pheasants can infect the partridges in which the infection is significantly more pathogenic. Empirical and theoretical studies suggest that this parasitic infection could be responsible for the decline of wild gray partridge populations in the UK which has been observed over the past 50 years (Tompkins et al., 2000, 2001).

These examples, however, represent situations in which a single parasite is shared between two species of hosts. One of the species carries the pathogen. It serves as a weapon to fight the second species which,

however, cannot retaliate in the same way. Therefore, these dynamics are asymmetric and are expected to result in the exclusion of one species. In the wild, however, exclusion of species is a relatively rare event. If both host species carry pathogens which can serve as weapons in competition, our mathematical models suggest that both exclusion and coexistence can be possible depending on the spatial setting and the migration rates of the species. In fact, if the migration rate is low, we can expect to observe that two species coexist and remain confined to a fraction of a given habitat, although they could in principle colonize the whole area. The fact that space can promote coexistence is a well-known ecological fact (Bonsall and Hassell, 2000). The reason for this behavior in our model is that mutual cross-species infection prevents host species from invading their respective territories. So far, detailed field or experimental studies addressing such dynamics have not been performed.

A more complex example in which cross-species infection plays a role in the competitive interactions of host species is the transmission of blood parasites between resident and migratory songbirds. Waldenström et al. (2002) studied the phylogeny of avian hemosporidian parasites, *Hemoproteus* and *Plasmodium*, in a number of African resident and European migratory songbird species. It was shown that host sharing and cross-infection was common both in *Hemoproteus* and in *Plasmodium*. The phylogenetic analysis indicated that transmission occurred both in Africa and Europe. Infection with these parasites can result in a significant fitness costs, especially when hosts are exposed to lineages for the first time and are not adapted to them. Therefore, resident birds in Africa may act as reservoirs for tropical avian blood parasites. These parasites can infect foreign migrants when reaching their African winter quarters and reduce their fitness. Thus, resident species of songbirds benefit from carrying the parasites because invasion of their habitat is costly for the migratory bird species. This could put pressure on the migrating species to select wintering habitats which do not significantly overlap with the territories of related resident bird species. Similarly, the migratory birds could carry parasites which can be transmitted to the resident species and help in the colonization of the wintering habitats.

6. Conclusion

The mathematical models described in this paper provide a framework to understand how cross-species infection can influence parasite–host dynamics. In particular, we have explored the relationship between the recovery rate from infection and the competitive ability of hosts. In the context of inter-specific competi-

tion we found that an intermediate or relatively low recovery rate from infection can give a host species an advantage over its competitor. In a spatial setting, the dynamics can result in a variety of outcomes. These include failure of invasion and thus coexistence of different host species in separate territories; or successful invasion and exclusion by a competitor characterized by less efficient immunity. We have used the model to discuss field data which show how cross-species infection can modulate host competition in relatively simple settings. In the light of theory, it would be interesting to perform more detailed ecological and epidemiological studies to examine the effect of cross-species infection on parasite–host dynamics. The role of parasites as biological weapons should not only be considered to explain species exclusion, but also to explain patterns of species coexistence and separation of habitats. Finally, the models suggest that the examples which show the use of parasites as weapons in host competition are only transient in time. Evolution is likely to minimize parasite burden which results in loss of these weapons. It is important to point out that all of these conclusions should be considered with the model assumptions in mind. Several simplifying assumptions have been made. While numerical simulations have shown that some of them do not significantly alter the basic results, the importance of the exact spatial set-up remains to be explored. More intricate patterns of species contact and migration might result in a situation where evolution does not maximize the recovery rate from infection. So far, we have not found such a scenario.

A particular application of these studies in the context of human health are zoonotic infections. Over 60 percent of pathogenic human infections have been found to be zoonotic (Taylor et al., 2001) and zoonotic infections are thought to be associated with emerging diseases (Daszak et al., 2000; McCarthy and Moore, 2000). Zoonotic infections include many different types of parasites, such as viruses, bacteria, protozoa, and helminths. Among viruses, prominent examples are ebola, influenza, and human immunodeficiency virus. In general, zoonotic infections emerge in humans following growth of the human population, mobility and migration of humans, and increased intrusion into the habitats of animals species (Mahy and Brown, 2000). Therefore, animal parasites which can be transmitted to humans and cause strong pathogenicity can be advantageous for the animal species because they can prevent habitat invasion.

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