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ORIGINAL PAPER

Invading with biological weapons: the role of shared disease in ecological invasion

Sally S. Bell · Andrew White · Jonathan A. Sherratt · Mike Boots

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Abstract Theory has been developed that examines the role of infectious disease in ecological invasions for particular natural systems. However, a general understanding of the role that shared disease may play in invasions is lacking. Here, we develop a strategic theoretical framework to determine the role of disease, in addition to competition, in ecological invasions and the expansion of species' spatial range. We investigate the effect of different disease parameters on the replacement time of a native species by an alien invader. The outcome is critically dependent on the relative effects that the disease has on the two species and less dependent on the basic epidemiological characteristics of the interaction. This framework is also used to investigate the effect of disease on the spatial spread of the invader. Our results show an interesting phenomenon where a wave of disease spreads through the landscape ahead of the wave of replacement.

Keywords Disease models · Spatial · Multi-species · Ecological invasions · Squirrelpox · Travelling waves

Introduction

The catastrophic damage to native communities of past introductions such as Nile perch (*Lates niloticus*) into Lake Victoria (Kolar and Lodge 2001) and the rapid spread of the European zebra mussel (*Dreissena polymorpha*) through North America (Lodge 1993; Pimentel et al. 2001; Vitousek et al. 1996) is well known. However, the rate at which human activity is introducing species, either accidentally or deliberately, into new habitats is still increasing (Prenter et al. 2004). Of course, the large majority of these organisms die out shortly after introduction, but those invasive species which establish themselves are recognised as a major international threat to native biodiversity (Kolar and Lodge 2001; Sala et al. 2000; Vitousek et al. 1997). In addition to these human-induced species introductions, current and predicted changes to the climate are likely to lead to significant shifts in species ranges which are also likely to threaten native systems (Dukes and Mooney 1999). In general, there are likely to be many factors which affect the success and rate of spread of invasive species, including differences in resource utilisation or life history characteristics between the invasive and native species. More recently, a number of studies have highlighted the role of infectious disease as an important determinant in native survival and invasive success (Daszak et al. 2000; Hudson and Greenman 1998; Prenter et al. 2004).

Commonly, when disease has been considered in the context of invasions, it has been as part of the enemy escape hypothesis. Here, the invasive species is thought to gain an advantage in its new environment by virtue of escaping its natural enemies, including virulent parasites as well as predators. For example, Aliabadi and Juliano (2002) found that when the invasive Asian tiger mosquito *Aedes albopictus* is released in North America, it initially experiences reduced infection by its gut parasite *Ascogregarina taiwanensis*. This escape gives it a small, but significant, competitive advantage over the native tree-hole mosquito *Ochlerotatus triseriatus* allowing it to expand its range more rapidly. It is also increasingly recognised that an invading species can gain an advantage by introducing a novel harmful disease to the native system. This scenario where the parasites acts as a "biological weapon" has been

S. S. Bell (✉) · A. White · J. A. Sherratt
Department of Mathematics and the Maxwell Institute for Mathematical Sciences, Heriot-Watt University, Edinburgh, EH14 4AS Scotland, UK
e-mail: s.bell@ma.hw.ac.uk

M. Boots
Department of Animal and Plant Sciences, University of Sheffield, Sheffield, S10 2TN, UK

an important factor in the replacement of the UK's only native crayfish, the white-clawed crayfish *Austropotamobius pallipes* throughout much of its range by the introduced North American signal crayfish *Pacifastacus leniusculus*. The white-clawed crayfish suffers both as a result of competition for resources from the larger and more aggressive signal crayfish and also the transmission of crayfish plague *Aphanomyces astaci* from the invading species. Signal crayfish are resistant to crayfish plague (Cerenius et al. 2003); however, it is lethal to white-clawed crayfish and has been responsible for mass mortality in many British crayfish populations (Bubb et al. 2004; Holdich 2003). Other examples include: the replacement of the pedunculate oak *Quercus robur* in the UK by the introduced Turkey oak *Quercus cerris* due to the impact of the knopper gall wasp *Andricus quercuscalicis* which causes huge acorn losses to the native species but has little effect on the introduced species (Hails and Crawley 1991); monogenean gill fluke *Nitzschia sturionis* which was introduced with the Caspian Sea sturgeon *Huso huso* in the 1930s has detrimentally affected the density of the Aral Sea sturgeon *Acipenser nudiventris* (Rohde 1984) and the expansion of the white-tailed deer *Odocoileus virginianus* in North America into territories occupied by moose *Alces alces* and caribou *Rangifer tarandus* which was aided by the meningeal worm *Parelaphostrongylus tenuis* which is carried by the white-tailed deer but lethal to the other species (Anderson 1972; Bergerud and Mercer 1989; Oates et al. 2000; Pybus et al. 1990). Perhaps the best known example is the decline of the UK's native red squirrels *Sciurus vulgaris* over the past 60 years as a result of the introduced North American grey squirrel *Sciurus carolinensis* (Lloyd 1983; Middleton 1930; Reynolds 1985). Red replacement was traditionally believed to result solely from the superior competitive ability of the greys (Okubo et al. 1989). However, recent evidence has revealed the existence of an infectious disease, squirrelpox, which is shared between the two species (Rushton et al. 2000) but is harmless (at least under laboratory conditions) to greys and lethal to reds (Tompkins et al. 2002). In models of the system, the inclusion of the effects of squirrelpox was necessary to explain the rapid replacement of red squirrels by greys in the UK (Rushton et al. 2006; Tompkins et al. 2003).

In all of the above examples, the invader gained an advantage through disease. However, there are also examples where disease can be advantageous to the native species. Hoogendoorn and Heimpel (2002) found that with ladybird beetles in North America; the native species *Coleomegilla maculata* (De Geer) suffers less from the parasitoid *Dinocampus coccinellae* (Schrank) when the alien species *Harmonia axyridis* (Pallas) is present. This lessens the competitive effects of the alien ladybird beetle and slows the rate of alien invasion. There is also evidence that infectious disease that is endemic in the native population may be highly pathogenic to invading species and therefore prevents the invader from establishing (Hilker et al. 2005; Petrovskii et al. 2005).

It is an open question, how the characteristics of particular parasite interactions affect the likelihood and rate of invasion by different species. Here, we develop a theoretical framework to understand how disease in combination with competition can affect the success of invasion, the time taken for a native species to be replaced by an invader and the spatial spread of the invading species. We will consider the impact of a shared disease on the dynamics of competing species under classical host–parasite frameworks. These frameworks are extended to include spatial spread by approximating dispersal as a diffusion process. The model formulations allow us to examine how disease affects the dynamics of invasion and, in particular, we address the question of how the presence of a shared disease affects the replacement time and spatial range over which a native species is replaced.

Methods

Below, we outline the framework for the full reaction-diffusion system. In the initial analysis, we set the diffusion coefficients to zero and consider the temporal dynamics only. The assumption is later relaxed when we consider the spatial spread of the invasive species. The classes of susceptible, S_i, and infected, I_i, individuals are represented by the following system of equations, where i=1, 2 with 1 representing the native species and 2 representing the alien invader

$$\frac{\partial S_1}{\partial t} = [a_1 - q_1(H_1 + c_2 H_2)](S_1 + f_1 I_1) - b_1 S_1 - \beta_{11} S_1 I_1 - \beta_{12} S_1 I_2 + \gamma_1 I_1 + D_1 \frac{\partial^2 S_1}{\partial x^2} \quad (1)$$

$$\frac{\partial I_1}{\partial t} = \beta_{11} S_1 I_1 + \beta_{12} S_1 I_2 - b_1 I_1 - \alpha_1 I_1 - \gamma_1 I_1 + D_1 \frac{\partial^2 I_1}{\partial x^2} \quad (2)$$

$$\frac{\partial S_2}{\partial t} = [a_2 - q_2(H_2 + c_1 H_1)](S_2 + f_2 I_2) - b_2 S_2 - \beta_{22} S_2 I_2 - \beta_{21} S_2 I_1 + \gamma_2 I_2 + D_2 \frac{\partial^2 S_2}{\partial x^2} \quad (3)$$

$$\frac{\partial I_2}{\partial t} = \beta_{22} S_2 I_2 + \beta_{21} S_2 I_1 - b_2 I_2 - \alpha_2 I_2 - \gamma_2 I_2 + D_2 \frac{\partial^2 I_2}{\partial x^2} \quad (4)$$

where $H_1 = S_1 + I_1$ and $H_2 = S_2 + I_2$.

We assume all parameters are non-negative and a_i represents the maximum reproduction rate, b_i the natural mortality rate, c_i the competitive affect of species i on the other species and β_{ij} the disease transmission coefficient from species j to i. (In this study, we assume that $\beta_{ij}=\beta$ for all i and j, but see Tompkins et al. (2003) for an assessment of different within- and between-species transmission rates. We assume density-dependent (mass action) infection dynamics (Bowers and Turner 1997; Tompkins et al. 2003) but see Saenz and Hethcote (2006) for a similar model with frequency-dependent transmission.) We assume a positive carrying capacity, K_i, which is related to susceptibility to crowding, q_i, since $K_i = (a_i - b_i)/q_i$. The model assumes that infected individuals experience disease-induced mortality at rate α_i. Infecteds may recover back to susceptibility at rate γ_i and infecteds experience only a proportion, f_i, of the fecundity of a susceptible host; $f_i \in [0,1]$. The diffusion coefficients, D_1 and D_2, approximate random movement for each of the species; we assume that dispersal is not affected by the disease.

By manipulating the infection parameters, the model Eqs. 1 to 4 can represent different classical disease frameworks. If $\gamma_i>0$, the model represents an SIS framework, whereas if $\gamma_i=0$ it represents an SI framework. It is also of interest to examine the effects of infection on fecundity in these frameworks. These range from a castrating parasite ($f_i=0$) to one in which disease has no effect on fecundity ($f_i=1$). We will investigate the role of disease on invasion under these different scenarios.

Results

Temporal results

We examine a non-spatial framework by setting the diffusion coefficients to zero ($D_i=0$). The model framework is similar to that analysed in detail by Bowers and Turner (1997) but we additionally include the possibility of the parasite reducing the fecundity of infected hosts (see also Begon et al. 1992; Greenman and Hudson 1997; Holt and Pickering 1985; Malchow et al. 2008; Saenz and Hethcote 2006). The key difference between previous studies that consider ecological interaction and infectious disease and the current study is our focus on determining whether disease increases or decreases the time taken for an invader to replace the native population. A summary of the steady states and their stability properties can be found in "Appendix 1" and are analogous to the findings of Bowers and Turner (1997). In this study we examine the invasion of an alien species into a purely susceptible native species at its carrying capacity, $(S_1, I_1, S_2, I_2) = (K_1, 0, 0, 0)$. As stated, our focus is to understand how the disease affects the replacement time of the native species by the invader. To achieve this, we assume that the non-disease parameters in Eqs. 1 to 4 are equal for the native and invading species except that the invader has a superior competitive ability, $c_2>c_1$. In the absence of the disease, the native will be replaced and the population will be transformed to the steady state containing a purely susceptible alien species at its carrying capacity $(0, 0, K_2, 0)$ (Fig. 1). The replacement time is measured as the time taken for the native population to fall below 0.1% of its carrying capacity (which in Fig. 1 is when t=70.4 time units). To examine how the inclusion of disease alters the replacement time, we use the same non-disease parameters as in Fig. 1 and compare the replacement time for competition-mediated replacement to that for competition-and-disease-mediated replacement in which the population is transformed to the endemic disease equilibrium $\left(0, 0, S_2^*, I_2^*\right)$. In this way, we examine whether disease increases or decreases the replacement time of the native species for a range of disease parameters.

Effects of disease-induced fecundity loss The effect on the replacement time when both species suffer equal fecundity loss as a result of the infection for a variety of combinations of the other disease parameters is shown in Fig. 2a. When there is no disease-induced mortality and no fecundity loss for infecteds, the replacement time is the same as in the absence of disease. As the loss of fecundity due to infection increases (f_i decreases from 1 to 0), the replacement time increases. This trend occurs since fecundity loss leads to an overall lower growth rate for the invader and therefore it takes longer for the invader to increase in number and oust

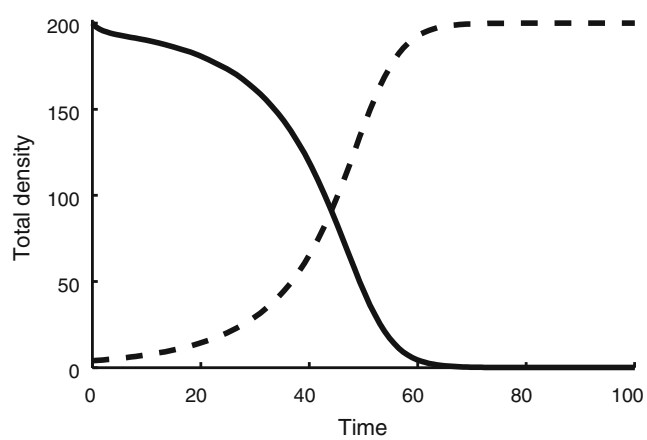

Fig. 1 Density of the native (*solid line*) and alien (*dashed line*) species over time in the absence of disease. Initially, the native species is at its carrying capacity and the invader is introduced at low density. The alien species replaces the native species and reaches its carrying capacity. Parameters are: $a_1=a_2=1$, $b_1=b_2=0.4$, $K_1=K_2=200$, $c_1=0.9$ and $c_2=1.5$. These results were produced using MATLAB ODE45 which is based on an explicit Runge-Kutta (4, 5) formula

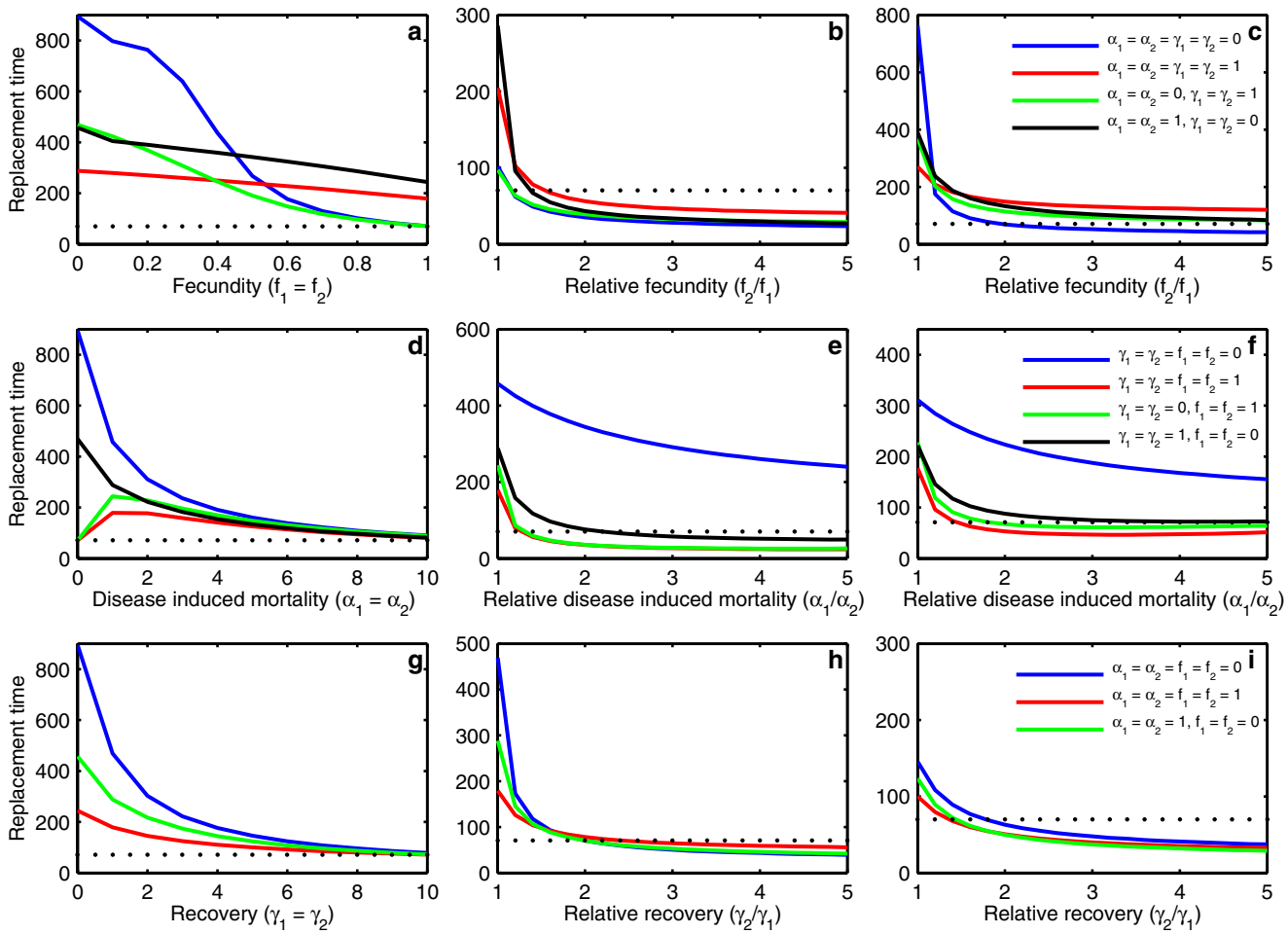

Fig. 2 The effect of different disease parameters on competition-and-disease-mediated replacement time: **a**–**c** disease-induced fecundity loss; **d**–**f** disease-induced mortality; **g**–**i** recovery from infection. For each of these parameters, we look at combinations of presence–absence of the other two disease parameters as detailed in the key for each row of plots. In **a**, **d** and **g**, the native and alien have equal values for the disease parameter, while in the remaining plots the native suffers a relative disadvantage. The *dotted line* represents the replacement time when disease is absent. Parameters common to every plot are: a_1=a_2=1, b_1=b_2=0.4, K_1=K_2=200, c_1=0.9, c_2=1.5 and β_{ij}=0.06. In addition, we fix the following parameters: in **b** f_2=0.8, **c** f_2=0.2, **e** α_2=1, **f** α_2=2, **h** γ_2=1 and **i** γ_2=5. The results are qualitatively similar for a wide range of parameters that satisfy the conditions necessary for the invader to have a competitive advantage. All results were produced using MATLAB ODE45 which is based on an explicit Runge-Kutta (4, 5) formula

the established native population (Fig. 2a). When there is mortality due to the disease (α_i>0), the replacement time is increased when compared to competition-only for all levels of fecundity loss. When fecundity loss is low, disease-induced mortality increases replacement time compared to when it is absent. In contrast, when fecundity loss is high, disease-induced mortality reduces replacement time (compared to when disease-induced mortality is absent) since here infected individuals, which contribute little to the overall growth rate of the invading species, are removed more rapidly. The effect of recovery is to reduce the replacement time at all levels of fecundity when compared to the appropriate results for the presence or absence of disease-induced mortality (Fig. 2a). The clear general trend is that if disease has the same effect in reducing the native and invading species fecundity the replacement time increases.

We next examine the effect on replacement time when the disease-induced reduction in fecundity is more severe for the native than the invading species. If the invading species has a 'low' level of fecundity loss, then the disease will increase replacement time (compared to in the absence of disease) if the relative fecundity loss of the native is small but reduce replacement time once the relative fecundity advantage of the invading species exceeds a threshold (Fig. 2b). This threshold increases if individuals can recover to susceptibility (as, once recovered, the disease-induced relative costs are not realised) and if the

disease induces additional mortality (as the death of infected individuals also negates the fecundity advantage of the invading species). The threshold also increases if the invading species has a 'high' level of fecundity loss (Fig. 2c) and here even a high relative fecundity advantage may not be sufficient for the replacement time to be less than in the absence of disease.

Effects of disease-induced mortality We consider increases in disease-induced mortality when it is equal in both species ($\alpha_1=\alpha_2$) for a variety of combinations of the other disease parameters (Fig. 2d). If the parasite castrates both species ($f_i=0$), an increase in disease-induced mortality reduces replacement time, but it can never be less than that for competition-only replacement (Fig. 2d). Here, the increase in disease-induced mortality acts to reduce the prevalence of an infection and so the castrating effect of the parasite becomes less apparent. If the parasite has no effect on fecundity ($f_i=1$), the replacement time initially increases as disease-induced mortality reduces the overall density and therefore lowers total reproduction. As disease-induced mortality increases further, replacement time reaches a maximum and then decreases and tends to but is never less than that for competition-only replacement. The approach of the diseased replacement time to the non-diseased time occurs because, when disease-induced mortality is high, infected individuals are removed so quickly that the model system behaves in a similar manner to the competition-only case. As the fecundity loss increases (f_i changes from 1 to 0), the replacement time at $\alpha_1=\alpha_2=0$ increases and the curves change between the two cases. The effect of recovery is to reduce the replacement time at all levels of disease-induced mortality for all the cases (and is similar to the response shown in Fig. 2a).

Next, we consider when the native suffers higher disease-induced mortality than the invading species. If the parasite is castrating and there is no recovery, the replacement time is always increased even if the native suffers high disease-induced mortality compared to the invader (Fig. 2e). With recovery present, if the advantage of the invader (in terms of lower disease-induced mortality) is small, the disease will again increase replacement time, but if the relative advantage of the invader exceeds a threshold the replacement time can be reduced compared to competition-only. This threshold is lower if the parasite is non-castrating as this allows higher reproduction into the susceptible class and therefore faster growth of the invading species. The trend is observed if the underlying level of disease-induced mortality of the invading species is increased but the threshold values at which the disease acts to reduce the replacement time are increased and in some circumstance a high relative advantage for the invader may not be sufficient to reduce the replacement time below that of competition-only (Fig. 2f).

Effects of recovery from disease If both species have an equal recovery rate from the disease ($\gamma_1=\gamma_2$), the replacement time decreases as the recovery rate increases but replacement is never faster than competition-only (Fig. 2g). If the relative advantage of the invader in terms of recovery exceeds a threshold, then the replacement time can be lower than for competition-only (Fig. 2h,i). When the disease is castrating (or leads to a 'large' reduction in fecundity), the recovery advantage is particularly important, as recovery acts as a route back to full fecundity.

Generality of temporal results Above, we consider the effects of disease on replacement time when the invader is a superior competitor, but the disease can also have a significant effect when the native species is the superior competitor (and therefore the competition alone would eradicate the invader). The disease can allow a competitively inferior invader to replace a native species (this occurs when criterion 21 is satisfied, see "Appendix 1"). When the parasite is castrating, the invader requires a high recovery rate to negate its inferior competitive ability (α_i can affect the speed of replacement but cannot alone allow invasion). For a non-castrating parasite, replacement requires the invader to suffer sufficiently lower mortality due to disease (differences in recovery can affect the replacement time but alone cannot allow invasion). In general, the qualitative trends in replacement time for changes in disease parameter values are as outlined in Fig. 2. (Although not the focus of this study, the invading species could coexist with the native, see "Appendix 1" for relevant criteria. The disease impacts on the time taken to coexistence in a similar manner to the one outlined above.) It is also possible to have situations where the disease acts to prevent invasion, even if the native is an inferior competitor, providing the native has sufficiently better recovery when $f_i=0$ or sufficiently lower mortality when $f_i>0$ (see criterion 18 in "Appendix 1").

The temporal results highlight the importance of infection in determining the outcome and time required for an invading species to replace a native species. The replacement time for alien species to invade are shortest when the invader has better recovery than the native species, lower mortality from the disease and a greater reproduction rate when infected. However, a disease introduced by an invading species may not reduce the replacement time. The general message is that a shared disease carried by an invading species may be detrimental to the invaders attempts to replace the native if the disease has a similar effect on both species even if it is more 'harmful' to the

native. Only when the invading species has a sufficient relative advantage does the disease assist in reducing the replacement time.

Spatial spread

We now extend our analysis to investigate how the temporal findings for the replacement of a native species extend to a spatial model framework. Our aim is to investigate the spatial spread and replacement when an invading species is introduced at one location into a disease-free native population. Again, we compare results for competition-mediated and competition-and-disease-mediated replacement.

We consider a situation in which the alien species has a competitive advantage in the absence of disease. In competition-only replacement, a travelling wave sweeps across the landscape, transforming the population from the native carrying capacity in front of the wave to a population of invaders only, at their carrying capacity, behind the wave. In an extensive programme of numerical simulations, we have found that, when disease is present, a rapid "wave of disease" spreads across the landscape, followed by a slower "wave of replacement". The wave of disease spreads through the native population (in the absence of the invading species) and transforms the native population from its disease-free to its endemic population level. This is followed by the wave of replacement in which the invading species replaces the native species, leaving the invading species at its endemic population level. Figure 3a shows the spatial replacement of the native species when there is no disease present and Fig. 3b shows the spatial replacement when there is disease present. For these parameter values, the temporal model predicts that the replacement of the native species will be faster when the disease is included. In line with this, the invading wave moves faster when the disease is present. For parameter values for which the disease would slow the replacement of the native species in the temporal system, our results indicate that the spatial replacement is also slower than in the absence of disease.

The three types of wave described above are all transition fronts, with a locally unstable steady state ahead of the front and a locally stable steady state behind. Such transition fronts are well understood for scalar reaction-diffusion equations. Providing that the local dynamics satisfy some simple conditions, there are wave front solutions for any value of the wave speed above a critical minimum value. Moreover, initial conditions of the type relevant in any ecological application (specifically, having sufficiently fast decay rate) lead to a wave travelling at this minimum speed. Reviews of the theory of travelling waves for scalar reaction-diffusion equations in ecological contexts are given in the books by Kot (2001), Murray (2002), Petrovskii and Li (2006) and de Vries et al. (2006). For systems of reaction-diffusion equations, the analogous theory of transition wave fronts is much less complete. In a few cases, it has been shown that the actual wave speed arising from localised initial conditions corresponds to a minimal speed that can be calculated in a manner analogous

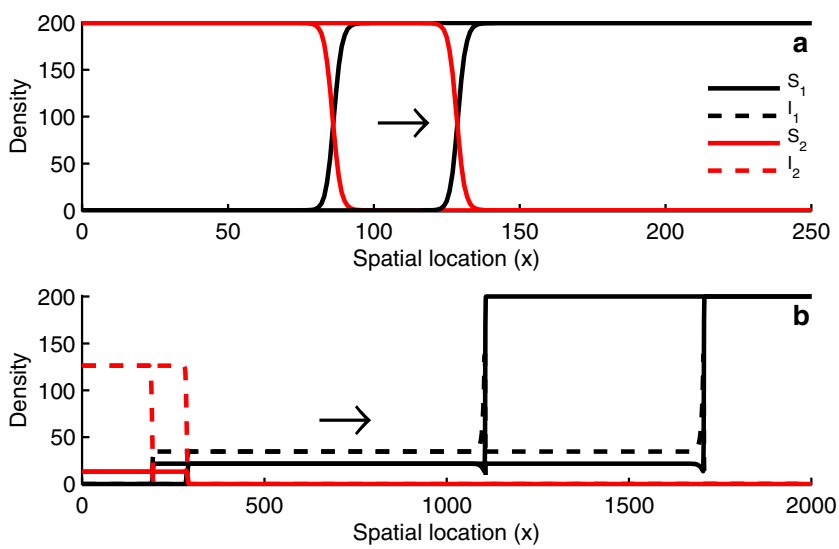

Fig. 3 Density of the native (S_1, I_1) and alien (S_2, I_2) species across the spatial landscape. In **a**, competition-mediated spatial replacement is shown and **b** competition-and-disease-mediated spatial replacement at time points 400 and 600, respectively. The parameters are: $f_1=f_2=1$, $a_1=a_2=1$, $b_1=b_2=0.4$, $K_1=K_2=200$, $c_1=0.9$, $c_2=1.5$, $\alpha_1=0.7$, $\alpha_2=0.2$, $\gamma_1=\gamma_2=0.2$, $\beta_{ij}=0.06$ and $D_i=0.18$. In a temporal model, these parameters result in a decrease in replacement time when disease is present. Similarly, in the spatial model, we see the invading species spreading further across the landscape when disease is present (**b**) compared to when it is absent (**a**). Note that in **b** the wave of replacement occurs behind the wave of disease. These observed values were produced using a semi-implicit Crank-Nicolson method

to that used for scalar equations (Lewis et al. 2002; Li et al. 2005; Weinberger et al. 2002, 2007). Moreover, this approach to calculating the wave speed has been proven successful in a great many other cases, although underlying theory is lacking. However, an important caveat is that there are some systems for which the actual speed is significantly faster than the minimal speed (Hosono 1998).

We have calculated the critical wave speed for the three types of wave that occur in our solutions; details of the calculations are given in "Appendix 2". The critical wave speed for competition-only-mediated replacement, θ_C, is given by $\theta_C = 2\sqrt{D_2(a_2 - b_2)(1 - c_1K_1/K_2)}$. The critical wave speeds for the waves of disease and of replacement, denoted by θ_D and θ_R, respectively, are given by $\theta_D = 2\sqrt{D_1(\beta_{11}K_1 - \alpha_1 - b_1 - \gamma_1)}$ and $\theta_R = \sqrt{2D_2\left(A + \Delta + \sqrt{(A+\Delta)^2 - 4A\Delta + 4EB}\right)}$ (A, B, Δ and E are defined in "Appendix 2"). For the parameters used in Fig. 3, the values of these speeds are θ_C=0.2, θ_R=0.5 and θ_D=2.8. These match very closely with the numerical simulations plotted in Fig. 3, and this is true for a wide range of other parameters for which $\theta_D > \theta_R$. When $\theta_D < \theta_R$, one might expect that the wave of replacement would "catch up with" the wave of disease, leading to the formation of a single combined wave front. However, in numerical simulations, we observe different behaviour, namely that, while the wave of replacement still travels at speed θ_R, the wave of disease travels at a speed faster than θ_D and indeed faster than θ_R (illustrated in Fig. 4). This is reminiscent of the behaviour observed by Hosono (1998) in competition-only models; a detailed understanding of what determines the actual wave speed is lacking even in that much simpler case and is therefore beyond the scope of the present paper. The only exception that we found to this behaviour was in the case D_1=0; then, a wave of disease is not possible, and the invasion occurs via a single travelling wave, moving at speed θ_C. Apart from in this very special case, our results always show that a rapid wave of disease spreads through the native population, with the actual invasion of the alien population occurring more slowly. This occurs regardless of whether disease acts to increase or decrease the replacement time.

The spatial investigation has shown that, when the disease decreases the temporal replacement time, it also results in a faster spatial wave of replacement. We also investigated whether disease can change the spatial range over which the alien species can invade. To examine this, we considered a heterogeneous spatial landscape in which the carrying capacity of the invader decreases across the

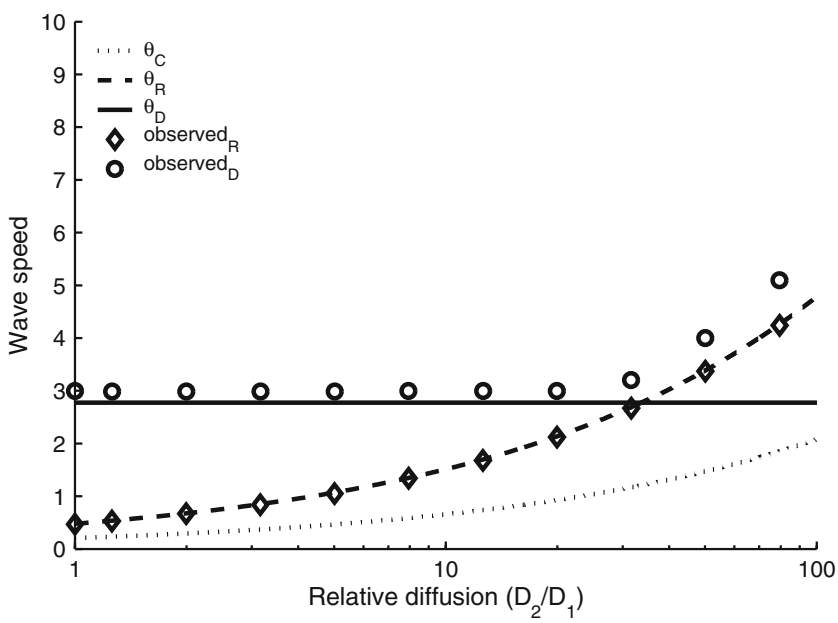

Fig. 4 Effect of relative diffusion on observed and critical wave speeds. Critical minimum wave speeds are calculated for the three types of waves seen in our solutions (competition-only wave (θ_C), wave of disease (θ_D) and wave of replacement (θ_R)) for a range of relative diffusion rates (D_2/D_1). In this scenario, the alien species invades and replaces the native species with disease present. This results in two waves, a wave of disease followed by a wave of replacement. When $\theta_D > \theta_R$, the observed wave speeds seen in numerical simulations match very closely with the critical wave speeds. If $\theta_D < \theta_R$, the observed wave of replacement (observed$_R$) still travels at speed θ_R; however, the observed wave of disease (observed$_D$) travels at a speed faster than θ_D. The parameters used are: f_1=f_2=1, a_1=a_2=1, b_1=b_2=0.4, K_1=K_2=200, c_1=0.9, c_2=1.5, α_1=0.7, α_2=0.2, γ_1=γ_2=0.2, β_{ij}=0.06 and D_i=0.18. The "observed" results come from numerical solutions of the equations using a semi-implicit Crank-Nicolson method. For larger ratios of the diffusion coefficients, the numerical simulations are relatively time consuming as they require a large spatial domain

spatial landscape while the carrying capacity of the native species increases. In the absence of the disease, the wave of invasion causes replacement of the native population. However, as this wave spreads across the landscape, it begins to slow and eventually stops (Fig. 5a). The wave halts as the competitive advantage of the invader is countered by its inferior carrying capacity. Figure 5b shows the outcome when the disease is included. The wave of replacement is again observed, with the wave speed slowing as the invader progresses across the landscape, but the disease allows the wave to progress further.

Discussion

In this study, we have considered a strategic theoretical framework to investigate the role of a shared disease, in addition to competition for resources, in the invasion of novel organisms and the expansion of their spatial range. We have shown that disease can increase or decrease the time taken for an invading species to replace an established native population with the outcome critically dependent on the relative effects that the disease has on the two species and less dependent on the basic epidemiological characteristics of the interaction. Disease may also allow the invasion of a poorer competitor that otherwise would have been excluded by the native species. A shared disease may benefit an invading species by allowing it to expand over a larger spatial region. When this occurs, a wave of disease spreads through the native population in advance of the invading species. This phenomenon may have important management and conservation implications.

Of great conservation concern is the situation where a shared disease can aid the invasion of an exotic species. The extinction of native red squirrel by the introduced grey in much of England and Wales has highlighted the role that disease may play in speeding up the replacement process (Rushton et al. 2006; Tompkins et al. 2003). We have shown that a shared disease is most likely to aid the invasion of a species if the native suffers higher disease-induced mortality, a lower level of fecundity due to infection and a lower rate of recovery compared to the invading species. These characteristics closely match those of the squirrel system in the UK. Squirrelpox virus appears to have little effect on the mortality or fecundity of grey squirrels and greys appear to make a full recovery from infection. Red squirrels however suffer high mortality from the virus, do not reproduce when infected and do not recover from infection (Tompkins et al. 2002). As such, the greys benefit from all of the factors that allow disease to increase the speed of invasion.

The results for the temporal replacement of a native species can be extended to understand the spatial spread of invasion. When a disease reduces the temporal replacement time (compared to the absence of disease), this translates into a faster wave of replacement in the spatial framework

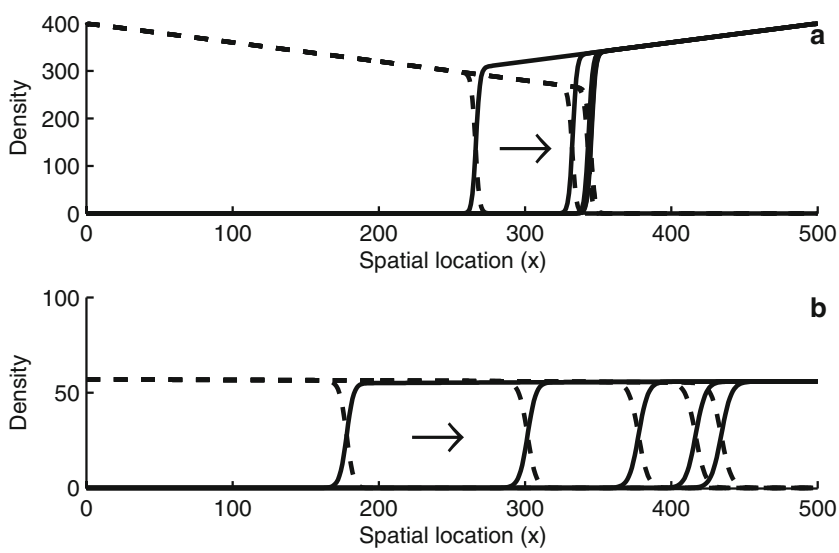

Fig. 5 Total density of the native (*solid line*) and alien (*dashed line*) species across a heterogeneous landscape. The heterogeneity is created by altering their carrying capacities; the invader's is 400 at x=0 and decreases linearly to 200 at x=500 while the native's is 200 at x=0 and increases linearly to 400 at x=500. In **a**, the disease is absent and the competitive advantage, which is modified by the difference in carrying capacity, allows the native to halt the spread of the invader at approximately x=350. In **b**, the disease is present and the invading species can spread further across the landscape and is halted at approximately x=450 (note the change in scale on the y-axis). The time points of each wave are 800, 1,600, 2,400, 3,200 and 4,000 in both plots (although in **a** the final plot is the final three time plots effectively superimposed). The parameters used are $f_1=f_2=0$, $a_1=a_2=1$, $b_1=b_2=0.4$, $c_1=0.9$, $c_2=1.5$, $\alpha_1=1.1$, $\alpha_2=1.0$, $\gamma_1=1.0$, $\gamma_2=1.1$, $\beta_{ij}=0.06$ and $D_i=0.18$. These results were produced using a semi-implicit Crank-Nicolson method

(and increased temporal replacement time relates to a slower wave of replacement). This correlation emphasises how informative relatively straightforward temporal models can be. Our spatial results also highlight an important phenomenon. When a diseased population invades a landscape composed of a disease-free native population, the initial response is for a wave of disease to sweep through the native population, reducing the population to its endemic level. The wave of replacement of the invading species travels well behind the wave of infection. Importantly, this phenomenon is observed even when the wave of replacement is slowed down by the presence of disease. There is some evidence that this may occur in natural systems. Reynolds (1985) catalogued the replacement of red squirrels by greys in East Anglia between 1960 and 1981. He reported that diseased red squirrels where found well in advance of grey squirrels being reported at a particular spatial location. At the time, this was used as evidence to dismiss disease being linked to the subsequent replacement of red squirrels. Our study suggests that such observations may be a direct result of the invasion of a disease-carrying species. From a conservation point of view, the emergence of the disease in a protected native population before the invader has reached the area may indicate the imminent replacement of the conserved species. This could be used as an early warning system to implement emergency conservation efforts.

Disease can also allow an invading species to increase the spatial range of replacement. The boundary between species often arises due to niche separation whereby in its own niche a species can out-compete another species. However, the shared disease can act to remove the competitive advantage of a species within its niche resulting in its replacement. Grey squirrels have still to invade some regions of Scotland and this is partly believed to be due to habitat characteristics that favour reds over greys. Conservation efforts are also being used to provide red squirrel refuges in such suitable habitat. Our study indicates that such conservation efforts should also consider the role of squirrelpox virus as this may spread beyond grey-squirrel-occupied areas and allow greys to invade regions which would otherwise be unsuitable. From a conservation point of view, the manipulation of habitat may not be enough to prevent the spread of the invasive species since the prevention of the disease is also crucial.

If an invading species has sufficient advantage due to the disease, it can replace a native species even if the invader is an inferior competitor. Disease, however, does not always benefit invading species. Hoogendoorn and Heimpel (2002) show that for ladybird beetle populations in North America; the native species suffers less from a shared parasitoid when the alien species is present. This reduces the competitive effects of the alien ladybird beetle, allowing the native an extra advantage and slowing the alien invasion. If the native suffers less harm from a disease, then this can allow the native to repel a potential invasion even if the native is an inferior competitor (Hilker et al. 2005; Petrovskii et al. 2005). A shared disease can increase the replacement time when disease characteristics are similar for the native and invading species even when an invading species has a competitive advantage. This emphasises that detailed epidemiological studies are needed when we want to predict the impact of disease in natural communities. A virulent disease may increase replacement time as often it is the relative effects of the disease on the native and alien species that are important. Furthermore, it is not just the lethal effects that are important. Sub-lethal effects on fecundity can have a pronounced influence on the outcome of the interaction. It is increasingly recognised that sub-lethal fecundity effects, rather than mortality effects, can drive the population dynamics of natural systems (Boots and Norman 2000; Dobson and Hudson 1986; Hudson et al. 1998). Our work emphasises that they may also be crucial to invasion dynamics. Therefore, disease may be crucial to conservation efforts even if it does not result in large mortality since less obvious and less studied effects on reproduction may be more important.

There are several examples where disease has played an important role in the successful invasion on non-native species. In the UK, the native white-clawed crayfish suffers very high disease-induced mortality, with no recovery, while the invasive signal crayfish are resistant (Bubb et al. 2004; Cerenius et al. 2003; Holdich 2003). If we consider these characteristics under our framework, the relatively higher mortality suffered by the native as a result of the disease decreases replacement time compared to competition-only. More detailed information on the system regarding fecundity, recovery and competition would allow us to gain better insight into this invasion but our framework highlights that the disease should be considered in conservation strategies to save the white-clawed crayfish. In North America, the invasion of white-tailed deer has been aided by the transmission of a meningeal worm which is lethal to caribou (Anderson 1972; Oates et al. 2000; Pybus et al. 1990). One of the conservation strategies has been to reintroduce caribou; however, in regions where infected white-tailed deer are present, these reintroductions have been unsuccessful (Bergerud and Mercer 1989). This emphasises the detrimental effect disease can have on conservation efforts. A further example is the replacement of the native pedunculate oak with Turkey oak in the UK. The Turkey oak is aided in this replacement by the detrimental effects of the knopper gall wasp on the pedunculate oak; the sexual generation of gall wasp develops in Turkey oak but causes little harm. However, during the agamic generation, knopper galls develop which

distort the growing acorns of pedunculate oak and can greatly reduce fecundity (Hails and Crawley 1991). The results from our general framework show that replacement time can be lower than competition-only when the native suffers a relative reduction in fecundity compared to the invading species. This is the case here, with infection giving the Turkey oak an advantage over the native oak species. These effects should be considered in conservation strategies for the pedunculate oak.

In summary, disease can reduce replacement time and speed up the spatial replacement of native species providing that invading species suffers sufficiently less 'harm' from the disease. Conservation efforts to protect native species should consider the role of disease as it can spread in advance of invading species and allow the non-native species to extend the spatial range over which it can invade.

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Appendix 1

In this appendix, we present a summary of the steady states and their stability properties of the temporal model. These results are analogous to the equilibrium and stability analysis described in detail in Bowers and Turner (1997).

The temporal model has no spatial movement; the relevant equations are found by setting D_1=D_2=0 in the full model (Eqs. 1 to 4 in the main text), giving

$$\frac{dS_1}{dt} = [a_1 - q_1(H_1 + c_2H_2)](S_1 + f_1I_1) - b_1S_1 - \beta_{11}S_1I_1 - \beta_{12}S_1I_2 + \gamma_1I_1 \quad (5)$$

$$\frac{dI_1}{dt} = \beta_{11}S_1I_1 + \beta_{12}S_1I_2 - b_1I_1 - \alpha_1I_1 - \gamma_1I_1 \quad (6)$$

$$\frac{dS_2}{dt} = [a_2 - q_2(H_2 + c_1H_1)](S_2 + f_2I_2) - b_2S_2 - \beta_{22}S_2I_2 - \beta_{21}S_2I_1 + \gamma_2I_2 \quad (7)$$

$$\frac{dI_2}{dt} = \beta_{22}S_2I_2 + \beta_{21}S_2I_1 - b_2I_2 - \alpha_2I_2 - \gamma_2I_2 \quad (8)$$

where H_1=S_1+I_1 and H_2=S_2+I_2. There are seven equilibrium points obtained from setting the right-hand side of Eqs. 5–8 equal to zero.

$$(S_1, I_1, S_2, I_2) = (0, 0, 0, 0), \quad (9)$$

$$(K_1, 0, 0, 0) = \left(\frac{a_1 - b_1}{q_1}, 0, 0, 0\right), \quad (10)$$

$$(S_1^*, I_1^*, 0, 0) = \left(\frac{b_1 + \alpha_1 + \gamma_1}{\beta_{11}}, \frac{\Psi_1 + \sqrt{\Psi_1^2 + \Omega_1}}{2q_1f_1\beta_{11}}, 0, 0\right), \quad (11)$$

$$(0, 0, K_2, 0) = \left(0, 0, \frac{a_2 - b_2}{q_2}, 0\right), \quad (12)$$

$$(0, 0, S_2^*, I_2^*) = \left(0, 0, \frac{b_2 + \alpha_2 + \gamma_2}{\beta_{22}}, \frac{\Psi_2 + \sqrt{\Psi_2^2 + \Omega_2}}{2q_2f_2\beta_{22}}\right), \quad (13)$$

$$(S_1^+, 0, S_2^+, 0) = \left(\frac{c_2K_2 - K_1}{c_1c_2 - 1}, 0, \frac{c_1K_1 - K_2}{c_1c_2 - 1}, 0\right), \quad (14)$$

$$\left(\widehat{S}_1, \widehat{I}_1, \widehat{S}_2, \widehat{I}_2\right) \quad (15)$$

where in the steady state (Eq. 15), the values are algebraically complicated and therefore omitted for brevity. The steady state defined in Eq. 11 holds when f_1>0 with Γ_1= α_1+b_1+γ_1, Ψ_1=$(a_1f_1-b_1-\alpha_1)\beta_{11}-q_1f_1\Gamma_1-q_1\Gamma_1$ and $\Omega_1 = 4q_1^2\Gamma_1(\beta_{11}K_1 - \Gamma_1)$. The steady state defined by Eq. 13 holds when f_2>0; where Γ_2, Ψ_2 and Ω_2 are equivalent to Γ_1, Ψ_1 and Ω_1, respectively, with the subscript 1 changed to 2. In the case when f_1=0, I_1^* in Eq. 11 becomes $I_1^* = [\Gamma_1(\beta_{11}K_1 - \Gamma_1)]/[\beta_{11}(\beta_{11}K_1 + \Gamma_1)]$. If f_2=0, I_2^* in Eq. 13 is equivalent to that for I_1^* with the subscript 1 changed to 2.

The trivial equilibrium is unstable (since we assume a_1> b_1 and a_2>b_2). For the other equilibrium points, we will give a brief description of their stability conditions. These are calculated using standard linear stability analysis (see, for example, Murray 2002) and the mathematical software package Maple for algebraic manipulation. At the steady state (K_1, 0, 0, 0), the native is at its carrying capacity and

the invader is not present. This is always feasible and is stable if the following two conditions hold

$$c_1K_1 - K_2 > 0 \quad \text{(native has a competitive advantage)}, \tag{16}$$

$$R_0(1) = \frac{K_1\beta_{11}}{\Gamma_1} < 1 \quad \text{(the disease cannot invade the native species)} \tag{17}$$

where $\Gamma_1=\alpha_1+b_1+\gamma_1$ represents the total removal from infection for the native species. At $\left(S_1^*, I_1^*, 0, 0\right)$, the native is at its endemic level and the invader is not present. This is feasible if $R_0(1)>1$; when feasible, it is stable if the following condition holds

$$\left(a_2 - b_2 - q_2c_1\left(S_1^* + I_1^*\right)\right) + \left(\left(\beta_{21}I_1^*\right)/\Gamma_2\right) \left(f_2a_2 - b_2 - f_2q_2c_1\left(S_1^* + I_1^*\right) - \alpha_2\right) < 0. \tag{18}$$

This condition represents the fact that the fitness of the alien species is negative.

At (0, 0, K_2, 0), the invader is at its carrying capacity and the native is not present. This is always feasible and is stable if the following two conditions hold

$$c_2K_2 - K_1 > 0 \quad \text{(invader has a competitive advantage)}, \tag{19}$$

$$R_0(2) = \frac{K_2\beta_{22}}{\Gamma_2} < 1 \quad \text{(the disease cannot invade the alien species)} \tag{20}$$

where $\Gamma_2=\alpha_2+b_2+\gamma_2$.

At $\left(0, 0, S_2^*, I_2^*\right)$, the invader is at its endemic levels and the native is not present. This is feasible if $R_0(2)>1$, and when feasible it is stable if the following condition holds

$$\left(a_1 - b_1 - q_1c_2\left(S_2^* + I_2^*\right)\right) + \left(\left(\beta_{12}I_2^*\right)/\Gamma_1\right) \left(f_1a_1 - b_1 - f_1q_1c_2\left(S_2^* + I_2^*\right) - \alpha_1\right) < 0 \tag{21}$$

This condition represents the fact that the fitness of native species is negative.

At $\left(S_1^+, 0, S_2^+, 0\right)$, the native and alien species are coexisting with no disease present. This steady state is feasible and stable if the following five conditions hold

$$c_1K_1 - K_2 < 0, \tag{22}$$

$$c_2K_2 - K_1 < 0, \tag{23}$$

$$c_1c_2 - 1 < 0, \tag{24}$$

$$\left(\beta_{11}S_1^+ - \Gamma_1\right) + \left(\beta_{22}S_2^+ - \Gamma_2\right) < 0, \tag{25}$$

$$\left(\beta_{11}S_1^+ - \Gamma_1\right)\left(\beta_{22}S_2^+ - \Gamma_2\right) - \beta_{12}\beta_{21}S_1^+S_2^+ > 0. \tag{26}$$

The final equilibrium, $\left(\widehat{S}_1, \widehat{I}_1, \widehat{S}_2, \widehat{I}_2\right)$, represents all the classes having positive densities and both the native and invader coexisting with the parasite. We do not discuss the stability of this steady state here but see Bowers and Turner (1997) and Greenman and Hudson (1997) for a detailed steady state and stability analysis for a similar model. We remind readers that in this study we focus on a set-up in which native species is initially at its carrying capacity with no disease (K_1, 0, 0, 0). Parameters are chosen such that when the invader is introduced it has a competitive advantage and replaces the native, with the invader attaining its carrying capacity (0, 0, K_2, 0) in the absence of disease or its endemic steady state $\left(0, 0, S_2^*, I_2^*\right)$ when disease is present.

Appendix 2

In this appendix, we calculate the critical minimum wave speeds for the three types of travelling wave that are described in the spatial spread section of the paper. The minimum wave speed corresponds to a change from complex to real for the eigenvalues that govern the decay of the travelling wave solution to the steady state ahead of the wave. This approach is based on established theory for scalar reaction-diffusion equations, which is reviewed in Kot (2001), Murray (2002), Petrovskii and Li (2006) and de Vries et al. (2006). For each of the three types of travelling wave, we begin by rewriting the relevant equations as a system of first-order differential equations. These can then be linearised about the steady state ahead of the wave to find the relevant eigenvalues for calculating the minimum wave speed.

Competition-only

The "competition-only" wave has $I_1=I_2=0$; substituting this into the full model (Eqs. 1 to 4 in main text) gives

$$\frac{\partial S_1}{\partial t} = [a_1 - b_1 - q_1(S_1 + c_2S_2)]S_1 + D_1\frac{\partial^2 S_1}{\partial x^2} \tag{27}$$

$$\frac{\partial S_2}{\partial t} = [a_2 - b_2 - q_2(S_2 + c_1S_1)]S_2 + D_2\frac{\partial^2 S_2}{\partial x^2}. \tag{28}$$

To investigate travelling wave solutions, we consider $S_i(x, t)=S_i(x-\theta t)$ where $\theta>0$ is the wave speed. We use $Z=x-\theta t$ to denote the travelling wave variable. Substituting these solution forms into Eqs. 27 and 28 and denoting differentiation with respect to Z by prime gives

$$-\theta S_1' = [a_1 - b_1 - q_1(S_1 + c_2 S_2)]S_1 + D_1 S_1'' \quad (29)$$

$$-\theta S_2' = [a_2 - b_2 - q_2(S_2 + c_1 S_1)]S_2 + D_2 S_2''. \quad (30)$$

Using $\dot{S}_1 = S_1'$ and $\dot{S}_2 = S_2'$, Eqs. 29 and 30 can be written as a system of four first-order ordinary differential equations. These equations have four equilibrium points:

$$\left(S_1, \dot{S}_1, S_2, \dot{S}_2\right) = (0,0,0,0),\ (K_1,0,0,0),\ (0,0,K_2,0) \text{ and } \left(S_1^+, 0, S_2^+, 0\right),$$

where $S_1^+ = (c_2K_2 - K_1)/(c_1c_2 - 1)$ and $S_2^+ = (c_1K_1 - K_2)/(c_1c_2 - 1)$.

In the "competition-only" wave, the native species is at its carrying capacity until the alien species invades, so the equilibrium in front of the wave will be (K_1, 0, 0, 0). Linearising the travelling wave equations about (K_1, 0, 0, 0) gives a Jacobian matrix with the following four eigenvalues

$$\lambda_{1,2} = \frac{-\theta \pm \sqrt{\theta^2 + 4D_2(a_2 - b_2)\left(\frac{c_1K_1}{K_2} - 1\right)}}{2D_2},$$

$$\lambda_{3,4} = \frac{-\theta \pm \sqrt{\theta^2 + 4D_1(a_1 - b_1)}}{2D_1}.$$

λ_3 and λ_4 are always real since $\theta^2+4D_1(a_1-b_1)>0$; one is positive and the other is negative. λ_1 and λ_2 are either both real and negative, or a complex conjugate pair with a negative real part; the condition for them being real is

$$\theta \geq \theta_C = 2\sqrt{D_2(a_2 - b_2)\left(1 - \frac{c_1K_1}{K_2}\right)}.$$

This suggests that the competition-only wave will move with speed θ_C.

Wave of disease

The "wave of disease" is the wave seen ahead of the wave of replacement, in which the native species is reduced from its carrying capacity to its endemic state. This transition occurs without direct involvement from the alien species, so that the relevant equations are given by setting $S_2=I_2=0$ in the full model (Eqs. 1 to 4 in main text), giving

$$\frac{\partial S_1}{\partial t} = (a_1 - q_1(S_1 + I_1))(S_1 + f_1 I_1) - b_1 S_1 - \beta_{11} S_1 I_1 + \gamma_1 I_1 + D_1 \frac{\partial^2 S_1}{\partial x^2} \quad (31)$$

$$\frac{\partial I_1}{\partial t} = \beta_{11} S_1 I_1 - b_1 I_1 - \alpha_1 I_1 - \gamma_1 I_1 + D_1 \frac{\partial^2 I_1}{\partial x^2}. \quad (32)$$

To investigate travelling waves, we look for a solution of the form $S_1(x, t)=S_1(x-\theta t)$ and $I_1(x, t)=I_1(x-\theta t)$ where $\theta>0$ is the wave speed. As before, we define the wave variable $Z=x-\theta t$ and denote differentiation with respect to Z by prime. Therefore, Eqs. 31 and 32 become

$$-\theta S_1' = (a_1 - q_1(S_1 + I_1))(S_1 + f_1 I_1) - b_1 S_1 - \beta_{11} S_1 I_1 + \gamma_1 I_1 + D_1 S_1'' \quad (33)$$

$$-\theta I_1' = \beta_{11} S_1 I_1 - b_1 I_1 - \alpha_1 I_1 - \gamma_1 I_1 + D_1 I_1''. \quad (34)$$

Using $\dot{S}_1 = S_1'$ and $\dot{I}_1 - I_1'$, Eqs. 33 to 34 can be written as a system of four first-order ordinary differential equations. We are considering a situation in which the native species is at its carrying capacity ahead of the wave, with no disease and no invader present, and so we linearise this system of equations about the steady state (S_1, $\dot{S}_1$, I_1, $\dot{I}_1$)=(K_1, 0, 0, 0). The resulting Jacobian matrix has the following four eigenvalues

$$\lambda_{1,2} = \frac{-\theta \pm \sqrt{\theta^2 - 4D_1(\beta_{11}K_1 - b_1 - \alpha_1 - \gamma_1)}}{2D_1},$$

$$\lambda_{3,4} = \frac{-\theta \pm \sqrt{\theta^2 + 4D_1(a_1 - b_1)}}{2D_1}.$$

λ_3 and λ_4 are real since $\theta^2+4D_1(a_1-b_1)>0$; one is positive and the other is negative. λ_1 and λ_2 are either both real and negative, or a complex conjugate pair with a negative real part; the condition for them being real is

$$\theta \geq \theta_D = 2\sqrt{D_1(\beta_{11}K_1 - \alpha_1 - b_1 - \gamma_1)}.$$

This suggests that the wave of disease will move with speed θ_D, which is critically dependent on the basic reproductive number of the disease $R_0(1)$ (as defined in Eq. 17 in "Appendix 1").

Wave of replacement

The "wave of replacement" is the wave seen behind the wave of disease, in which the invading species replaces the native species. This transition involves both species and thus we must consider all four of the model equations (Eqs. 1 to 4 in main text). To investigate travelling waves, we look for a solution of the form $S_1(x, t)=S_1(x-\theta t)$, $I_1(x, t)=I_1(x-\theta t)$, $S_2(x, t)=S_2(x-\theta t)$ and $I_2(x, t)=I_2(x-\theta t)$ where $\theta>0$ is the wave speed.

As before, we define the wave variable $Z=x-\theta t$ and denote differentiation with respect to Z by prime. Therefore, Eqs. 1 to 4 become

$$-\theta S_1' = [a_1 - q_1(H_1 + c_2H_2)](S_1 + f_1I_1) - b_1S_1 - \beta_{11}S_1I_1 - \beta_{12}S_1I_2 + \gamma_1I_1 + D_1S_1'' \quad (35)$$

$$-\theta I_1' = \beta_{11}S_1I_1 + \beta_{12}S_1I_2 - b_1I_1 - \alpha_1I_1 - \gamma_1I_1 + D_1I_1'' \quad (36)$$

$$-\theta S_2' = [a_2 - q_2(H_2 + c_1H_1)](S_2 + f_2I_2) - b_2S_2 - \beta_{22}S_2I_2 - \beta_{21}S_2I_1 + \gamma_2I_2 + D_2S_2'' \quad (37)$$

$$-\theta I_2' = \beta_{22}S_2I_2 + \beta_{21}S_2I_1 - b_2I_2 - \alpha_2I_2 - \gamma_2I_2 + D_2I_2''. \quad (38)$$

Using $\dot{S}_i = S_i'$ and $\dot{I}_i = I_i'$, Eqs. 35–38 can be rewritten as a system of eight first-order equations. As before, we will examine the stability of the equilibrium point in front of the wave. We are considering a situation in which the native species has already been reduced to its endemic state by the disease (via the "wave of disease") and there is no invader present, so we linearise about (S_1, $\dot{S}_1$, I_1, $\dot{I}_1$, S_2, $\dot{S}_2$, I_2, $\dot{I}_2$)=($S_1^*, 0, I_1^*$, 0, 0, 0, 0, 0). The four equations obtained from Eqs. 37 and 38 decouple from the four equations obtained from Eqs. 35 and 36.

For realistic travelling wave solutions, we require the four eigenvalues obtained from these decoupled equations to be non-oscillatory (if they have a negative real part). The other four eigenvalues have zero components for S_2 and I_2 and therefore do not impose any restrictions on population densities being positive in the travelling wave. For notational simplicity, we define

$$A = a_2 - q_2c_1H_1^* - b_2 - \beta_{21}I_1^*,$$
$$B = \left(a_2 - q_2c_1H_1^*\right)f_2 + \gamma_2,$$
$$E = \beta_{21}I_1^*,$$
$$\Delta = -b_2 - \alpha_2 - \gamma_2.$$

The relevant part of the resulting Jacobian has the following four eigenvalues

$$\lambda_{1,2} = \frac{-\theta \pm \sqrt{\theta^2 - 2D_2\left(A + \Delta + \sqrt{(A+\Delta)^2 - 4A\Delta + 4EB}\right)}}{2D_2},$$
$$\lambda_{3,4} = \frac{-\theta \pm \sqrt{\theta^2 - 2D_2\left(A + \Delta - \sqrt{(A+\Delta)^2 - 4A\Delta + 4EB}\right)}}{2D_2}.$$

We are concerned with parameter values for which the native species, in its endemic state, is unstable to the introduction of the alien species. The condition for this is $(a_2 - b_2 - q_2c_1(S_1^* + I_1^*)) + (\beta_{21}I_1^*)/(\alpha_2 + b_2 + \gamma_2)$ $(f_2a_2 - b_2 - f_2q_2c_1(S_1^* + I_1^*) - \alpha_2) > 0$ (Eq. 18 in "Appendix 1"), which is equivalent to $EB - A\Delta > 0$. This implies that the eigenvalues λ_3 and λ_4 are real with one positive and the other negative. λ_1 and λ_2 are either both real and negative or a complex conjugate pair with a negative real part; the condition for them being real is

$$\theta \geq \theta_R = \sqrt{2D_2\left(A + \Delta + \sqrt{(A+\Delta)^2 - 4A\Delta + 4EB}\right)}.$$

This suggests that the wave of replacement will move with speed θ_R.

References

Aliabadi BW, Juliano SA (2002) Escape from gregarine parasites affects the competitive interactions of an invasive mosquito. Biol Invasions 4:283–297

Anderson RC (1972) The ecological relationships of meningeal worm and native cervids in North America. J Wildl Dis 8:304–310

Begon M, Bowers RG, Kadianakis N, Hodgkinson DE (1992) Disease and community structure: the importance of host-regulation in a host–host–pathogen model. Am Nat 139:1131–1150

Bergerud AT, Mercer WE (1989) Caribou introductions in Eastern North America. Wildl Soc Bull 17:111–120

Boots M, Norman R (2000) Sublethal infection and the population dynamics of host–microparasite interactions. J Anim Ecol 69:517–524

Bowers RG, Turner J (1997) Community structure and the interplay between interspecific infection and competition. J Theor Biol 187:95–109

Bubb DH, Thom TJ, Lucas MC (2004) Movement and dispersal of the invasive signal crayfish *Pacifastacus leniusculus* in upland rivers. Freshw Biol 49:357–368

Cerenius L, Bangyeekhun E, Keyser P, Soderhall I, Soderhall K (2003) Host prophenoloxidase expression in freshwater crayfish is linked to increased resistance to the crayfish plague fungus, *Aphanomyces astaci*. Cell Microbiol 5:353–357

Daszak P, Cunningham AA, Hyatt AD (2000) Emerging infectious diseases of wildlife—threats to biodiversity and human health. Science 287:443–449

De Vries G, Hillen T, Lewis M, Muller J, Schoenfisch B (2006) A course in mathematical biology: quantitative modelling with mathematical and computational methods. Society for Industrial and Applied Mathematics, Philadelphia

Dobson AP, Hudson PJ (1986) Parasites, disease and the structure of ecological communities. Trends Ecol Evol 1:11–15

Dukes JS, Mooney HA (1999) Does global change increase the success of biological invaders? Trends Ecol Evol 14:135–139

Greenman JV, Hudson PJ (1997) Infected coexistence instability with and without density dependent regulation. J Theor Biol 185:345–356

Hails RS, Crawley MJ (1991) The population dynamics of an alien insect: *Andricus quercuscalicis* (Hymenoptera: Cynipidae). J Anim Ecol 60:545–561

Hilker FM, Lewis MA, Seno H, Langlais M, Malchow H (2005) Pathogens can slow down or reverse invasion fronts of their hosts. Biol Invasions 7:817–832

Holdich D (2003) Ecology of the white-clawed crayfish. Conserving Nature 2000 Rivers Ecology Series No. 1. English Nature, Peterborough

Holt RD, Pickering J (1985) Infectious disease and species coexistence: a model of Lotka-Volterra form. Am Nat 126:196–211

Hoogendoorn M, Heimpel GE (2002) Indirect interactions between an introduced and a native ladybird beetle species mediated by a shared parasitoid. Biol Control 25:224–230

Hosono Y (1998) The minimal speed of traveling fronts for a diffusive Lotka-Volterra competition model. Bull Math Biol 60:435–448

Hudson PJ, Greenman J (1998) Competition mediated by parasites: biological and theoretical progress. Trends Ecol Evol 13:387–390

Hudson PJ, Dobson AP, Newborn D (1998) Prevention of population cycles by parasite removal. Science 282:2256–2258

Kolar CS, Lodge DM (2001) Progress in invasion biology: predicting invaders. Trends Ecol Evol 16:199–204

Kot M (2001) Elements of mathematical ecology, vol. l. Cambridge University Press, Cambridge

Lewis MA, Li B, Weinberger HF (2002) Spreading speed and linear determinacy for two-species competition models. J Math Biol 45:219–233

Li B, Weinberger HF, Lewis MA (2005) Spreading speeds as slowest wave speeds for cooperative systems. Math Biosci 196:82–98

Lloyd HG (1983) Past and present distribution of red and grey squirrels. Mammal Rev 13:69–80

Lodge DM (1993) Biological invasions: lessons for ecology. Trends Ecol Evol 8:133–137

Malchow H, Petrovskii S, Venturino E (2008) Spatiotemporal patterns in ecology and epidemiology: theory, models, simulations. Chapman & Hall/CRC Press, Boca Raton

Middleton AD (1930) Ecology of the American gray squirrel in the British Isles. Proc Zoolog Soc Lond 2:809–843

Murray JD (2002) Mathematical biology I: an introduction, 3rd edn. Springer, Berlin

Oates DW, Sterner MC, Boyd E (2000) Meningeal worm in deer from western Nebraska. J Wildl Dis 36:370–373

Okubo A, Maini PK, Williamson MH, Murray JD (1989) On the spatial spread of the grey squirrel in Britain. Proc R Soc Lond B Biol Sci 238:113–125

Petrovskii SV, Li B (2006) Exactly solvable models of biological invasion. Chapman & Hall/CRC, London

Petrovskii SV, Malchow H, Hilker FM, Venturino E (2005) Patterns of patchy spread in deterministic and stochastic models of biological invasion and biological control. Biol Invasions 7:771–793

Pimentel D, McNair S, Janecka J, Wightman J, Simmonds C, O'Connell C, Wong E, Russel L, Zern J, Aquino T, Tsomondo T (2001) Economic and environmental threats of alien plant, animal, and microbe invasions. Agric Ecosyst Environ 84:1–20

Prenter J, MacNeil C, Dick JTA, Dunn AM (2004) Roles of parasites in animal invasions. Trends Ecol Evol 19:385–390

Pybus MJ, Samuel WM, Welch DA, Wilke CJ (1990) Parelaphostrongylus andersoni (Nematoda: Protostrongylidae) in white-tailed deer from Michigan. J Wildl Dis 26:535–537

Reynolds JC (1985) Details of the geographic replacement of the red squirrel (*Sciurus vulgaris*) by the grey squirrel (*Sciurus carolinensis*) in Eastern England. J Anim Ecol 54:149–162

Rohde K (1984) Ecology of marine parasites. Helgol Mar Res 37:5–33

Rushton SP, Lurz PWW, Gurnell J, Fuller R (2000) Modelling the spatial dynamics of parapoxvirus disease in red and grey squirrels: a possible cause of the decline in the red squirrel in the UK? J Appl Ecol 37:997–1012

Rushton SP, Lurz PWW, Gurnell J, Nettleton P, Bruemmer C, Shirley MDF, Sainsbury AW (2006) Disease threats posed by alien species: the role of a poxvirus in the decline of the native red squirrel in Britain. Epidemiol Infect 134:521–533

Saenz RA, Hethcote HW (2006) Competing species models with an infectious disease. Math Biosci Eng 3:219–235

Sala OE, Chapin FSL, Armesto JJ, Berlow E, Bloomfield J, Dirzo R, HuberSanwald E, Huenneke LF, Jackson RB, Kinzig A, Leemans R, Lodge HA, Oesterheld M, LeRoy Poff N, Sykes MT, Walker BH, Walker M, Wall DH (2000) Global biodiversity scenarios for the year 2100. Science 287:1770–1774

Tompkins DM, Sainsbury AW, Nettleton P, Buxton D, Gurnell J (2002) Parapoxvirus causes a deleterious disease in red squirrels associated with UK population declines. Proc R Soc Lond B Biol Sci 269:529–533

Tompkins DM, White AR, Boots M (2003) Ecological replacement of native red squirrels by invasive greys driven by disease. Ecol Lett 6:189–196

Vitousek PM, D'Antonio CM, Loope LL, Westbrooks R (1996) Biological invasions as global environmental change. Am Sci 84:468–478

Vitousek PM, D'Antonio CM, Loope LL, Rejmanek M, Westbrooks R (1997) Introduced species: a significant component of human-caused global change. N Z J Ecol 21:1–16

Weinberger HF, Lewis MA, Li B (2002) Analysis of linear determinacy for spread in cooperative models. J Math Biol 45:183–218

Weinberger HF, Lewis MA, Li B (2007) Anomalous spreading speeds of cooperative recursion systems. J Math Biol 55:207–222