Disease, habitat fragmentation and conservation

Hamish McCallum^{1*} and Andy Dobson²

¹Department of Zoology and Entomology, The University of Queensland, Brisbane 4072, Australia

²Ecology and Evolutionary Biology, Princeton University, Princeton, NJ 08544-1003, USA (andy@eno.princeton.edu)

Habitat loss and the resultant fragmentation of remaining habitat is the primary cause of loss of biological diversity. How do these processes affect the dynamics of parasites and pathogens? Hess has provided some important insights into this problem using metapopulation models for pathogens that exhibit 'S-I' dynamics; for example, pathogens such as rabies in which the host population may be divided into susceptible and infected individuals. A major assumption of Hess's models is that infected patches become extinct, rather than recovering and becoming resistant to future infections. In this paper, we build upon this framework in two different ways: first, we examine the consequences of including patches that are resistant to infection; second, we examine the consequences of including a second species of host that can act as a reservoir for the pathogen. Both of these effects are likely to be important from a conservation perspective. The results of both sets of analysis indicate that the benefits of corridors and other connections that allow species to disperse through the landscape far outweigh the possible risks of increased pathogen transmission. Even in the commonest case, where harmful pathogens are maintained by a common reservoir host, increased landscape connectance still allows greater coexistence and persistence of a threatened or endangered host.

Keywords: habitat loss; metapopulation; pathogens; conservation; reservoir species

1. INTRODUCTION

Habitat loss is widely considered to be the most important factor causing species extinctions (Sih *et al.* 2000). Primarily, theoretical discussions about the effects of habitat loss on endangered species have been based on the metapopulation paradigm: as patches become smaller and more separated, there are increased rates of extinction and decreased rates of recolonization (e.g. Hanski 1997). The potential significance of parasites and pathogens in species decline is now acknowledged (McCallum & Dobson 1995). However, apart from Hess (1996) and more recently Gog *et al.* (2002), the effect of habitat fragmentation on the dynamics of pathogens has received relatively little attention. In a pathogen that infects only an endangered species, the naive view would be that fragmentation should decrease risks to endangered species from pathogens for two reasons. First, almost all pathogens have a threshold host population beneath which they cannot persist. If a population is subdivided into smaller subpopulations, then some or all of the populations may be below the threshold, whereas the single unfragmented population may be above the threshold. Second, if an exotic (introduced) pathogen does become established in a subpopulation, fragmentation may prevent it from spreading to the remaining populations. Third, we note that the social systems of many vertebrate species cause them to be naturally 'fragmented' into social groups into which pathogens are either introduced by infected immigrants, or by contact with individuals from infected groups. The incubation periods, virulence and transmission rates of pathogens infecting these species are likely to have evolved in ways that allow them to persist in fragmented popu-

lations. Habitat fragmentation is likely to reduce the dispersal rates of individuals between social groups; this will initially slow the rate of disease spread. However, it will simultaneously exert selection pressure on the pathogen to evolve ways of persisting in a more fragmented host population. In general, these will have to reduce the impact of the pathogen on the host, if the pathogen is to persist.

For a variety of reasons, this naive view is a considerable oversimplification. There are two broad classes of host-pathogen interactions that need to be considered. First, the pathogen threatening a 'target' endangered species may be specific to the particular host in the environment in question. Alternatively, there may be one or more reservoir species on which the pathogen has much less of an impact than the potentially endangered host species.

An example of a host-specific pathogen of conservation concern is the chlamydial infection sometimes believed to be threatening koala populations in Australia (Augustine 1998; Melzer *et al.* 2000; Phillips 2000). In such single-host-single-pathogen situations, the key consideration with regard to habitat fragmentation is how transmission between host individuals might be affected by fragmentation. As noted above, one would expect that fragmentation should decrease between-patch transmission in almost all circumstances. Effectively, increased fragmentation should act to 'quarantine' infected patches, diminishing the impact of the pathogen on the population as a whole.

Within-patch transmission may be quite different. In most host-pathogen models, transmission is assumed to occur via 'mass action', at a rate βSI , where S and I are the susceptibles and infecteds, respectively, and β is the transmission parameter. This transmission term has become the topic of considerable recent debate (e.g. de Jong *et al.* 1995; McCallum *et al.* 2001), and its nature

*Author for correspondence (hmccallum@zen.uq.edu.au).

has major implications for the effect of fragmentation on disease dynamics. If S and I refer to 'numbers' of susceptible and infected hosts (as they are, for example, in the classic papers of Anderson & May 1979; May & Anderson 1979), one would expect fragmentation to reduce the probability of a susceptible host encountering an infected host. At some point, the population size of each patch would be reduced to such an extent that the pathogen was no longer able to persist. Habitat destruction, at least in the initial stages, is likely to cause an increase in the local density of patch-dependent animals, as they crowd into the remaining suitable habitat. If S and I refer to the 'densities' of susceptible and infected hosts per unit area, one thus might expect that within-patch transmission should increase following fragmentation, also increasing the impact of the pathogen upon its host population.

Several authors, particularly de Jong *et al.* (1995) have suggested that a transmission term of the form βSI is often not appropriate in natural populations. Instead, they argue that the transmission should depend on the 'proportion' of hosts that are infected, and thus should occur at a rate $\beta SI/N$, where N is the total population size. This assumes that hosts, in general, make the same number of contacts with other hosts, whatever the population size in the patch or local density, and is known as frequency-dependent transmission. According to this assumption, habitat fragmentation would have no direct effect on within-patch transmission.

Finally, fragmentation might affect other aspects of disease biology than transmission. Habitat fragmentation is frequently thought to increase stress on animals through displacement and crowding. Stress may affect the immune response of the host, increasing the possibility that infection with a pathogen will lead to clinical disease. For example, White & Timms (1994) surveyed the same population of koalas at two different times, looking both for the presence of the chlamydia pathogen (using cell culture) and for clinical signs of disease caused by the pathogen. At both times, the prevalence of chlamydial infection was similar (*ca.* 50%), but clinical signs increased more than threefold (from 14% to 47%). They proposed that this may have been due to increased stress. Whatever the reason, this example illustrates that infection with a pathogen and overt clinical disease are two different things.

(a) *Models of single-host infections in metapopulations*

Hess (1996) developed a model for pathogens in a fragmented host population. The model was closely based on the classical Levins (1969) metapopulation structure. Patches could either be infected, empty or susceptible, but there was no recovery. Infection increased the extinction rate of patches. The key result was that there were four possible equilibrium states, attained in the following order, with increasing migration rate and all other parameters held constant.

- (i) Extinction. Neither host nor pathogen can persist.
- (ii) Host can persist on some patches, but the pathogen cannot. Proportion of patches occupied increases with increased migration rate.
- (iii) Endemic disease. Host and pathogen coexist, but

disease is not present on all occupied patches. The total proportion of occupied patches decreases.

- (iv) Pandemic disease. Host and pathogen coexist, but all occupied patches are infected. The proportion of occupied patches increases monotonically.

On the basis of this model and some simulation models, Hess concluded that 'movement of individuals among populations is a conservation strategy that may carry a greater risk than is recognized commonly', although he did acknowledge that his models showed that some movement between populations was necessary to prevent metapopulation extinction.

A limitation of Hess's analytic model is that it did not include a recovered class, or indeed any form of recovery at all: once patches had infection introduced into them, the patch remained infected until local host extinction occurred. This is an assumption that does not apply in many instances of diseases infecting spatially subdivided populations. For example, there is a whole literature on 'fade-outs' of measles in human communities of varying sizes (e.g. Bolker & Grenfell 1995). This is essentially a stochastic phenomenon: even in human communities of many thousands of individuals, the number of infected individuals remaining after an epidemic is so small that the disease becomes locally extinct. Following a fade-out, the proportion of susceptible individuals in the population is so low that the disease cannot successfully invade that same patch for some time until the proportion of susceptible individuals in the population has built up. The extent to which fade-outs occur in single-species infections in spatially subdivided animal populations is unclear. One example where a pathogen caused conservation problems in a subdivided population, but did not subsequently persist, is the phocine distemper epidemic that occurred in North Sea harbour seal populations (Heide-Jorgensen & Härkönen 1992; Swinton *et al.* 1998). If patches each contain a relatively small number of hosts, as will be the case for many endangered species, fade-outs are particularly likely to occur.

(b) *Infections involving reservoir hosts*

The preceding discussion assumes that the pathogen in question affects only the endangered species. Whilst this is obviously the simplest situation and a good place to start, almost all examples of diseases leading to declines in endangered species have involved multiple host infections (Cleaveland *et al.* 2002; Gog *et al.* 2002). One host (or often a group of host species) serves as a reservoir, in which the pathogen is maintained with a less detrimental impact than that on the 'target' endangered host species. One particularly important consequence of the existence of a reservoir host is that a positive force of infection can be maintained on the endangered host species as its density declines towards extinction. This is in contrast to single-pathogen–single-host models with density-dependent transmission, in which there is almost always a threshold host density, beneath which the pathogen cannot persist. This means that, in a deterministic model, the pathogen itself cannot drive the host extinct (although the host population may be reduced to such a low level that stochastic factors or Allee effects drive it to extinction).

The presence of a reservoir host can potentially lead to

three different scenarios in the presence of habitat fragmentation. First, the reservoir may exist only in the matrix around the patches occupied by the endangered species. This would be the case if domestic stock were the reservoir species for the pathogen in question, and reserves from which stock were excluded constituted the habitat patches for the endangered species. Simonetti (1995) describes a possible example of this sort of interaction. The tapeworm *Cysticercus tenuicollis* is highly pathogenic to the Andean deer *Hippocamelus bisulcus*. Domestic stock act as a reservoir for *C. tenuicollis*. In this situation, increased habitat fragmentation is unequivocally bad for the endangered species, as it will increase the rate of contact the target has with the reservoir. Even if the pathogen would not be maintained in populations of the target species alone (as might be the case if the pathogenicity was very high), habitat fragmentation might maintain a high force of infection across the patch boundaries and hence lead to decline or extinction of the endangered species.

Second, the reservoir may occupy the same patch type as the endangered 'target' species, although the pattern of patch occupancy might potentially be different, whether for stochastic reasons, or because extinction and colonization rates vary as a function of patch size, separation and quality. This case requires detailed consideration, which follows in the next section of the paper.

Finally, the reservoir may occupy both the matrix and the patches. In terms of the dynamics of the disease, this last case is unlikely to differ substantially from infection in a spatially homogeneous population.

2. A SINGLE-HOST-SPECIES MODEL WITH RECOVERED PATCHES

Assume that there are N available patches, where N is large. These patches are identical in terms of the carrying capacity for the host, and distances from each other, but may be in four states at time t .

- (i) Empty patches. Hosts are not present.
- (ii) Occupied patches in which the proportion of susceptible hosts is sufficiently high that the disease can invade. Represented by $s(t)$.
- (iii) Patches in which infection is present. Represented by $i(t)$.
- (iv) Occupied patches, in which the proportion of resistant hosts is sufficiently high that the disease cannot persist. Represented by $r(t)$.

Following Hess's model closely, assume that the rate of extinction of patches of each class can be represented as x_s, x_i, x_r , respectively, where all rates are positive, and $x_i > x_s$ and $x_i > x_r$. Furthermore, assume that migrants leave patches of all types at a rate m . If a migrant from a susceptible or resistant patch enters an empty patch, that patch becomes colonized with susceptible hosts. If an infected host enters an empty patch, the patch becomes colonized with infected hosts, whereas if an infected host migrates to a susceptible patch, that patch has a probability δ of becoming infected. Resistant or susceptible hosts entering patches other than empty ones do not change patch status, and infected hosts entering resistant patches do not affect the status of the patch. Further

assume that infected patches recover from infection at a rate γ , and that resistance is lost, with patches joining the susceptible class, at a rate λ . If $\gamma = 0$, Hess's model is recovered.

Defining the dynamics in terms of the proportions of all patches occupied by each type of host (i.e. $S = s/N$, $I = i/N$, $R = r/N$), rescaling time in units of the extinction rate of susceptible patches, so that $\tau = x_s t$, and using upper-case parameters to represent rates relative to x_s , the metapopulation dynamics can be represented by the following equations:

$$\frac{dS}{d\tau} = S[M(1 - S - I - R) - 1 - \delta MI] + R(\Lambda + M(1 - S - I - R)), \quad (2.1)$$

$$\frac{dI}{d\tau} = I[M(1 - S - I - R) - X_i + \delta MS - \Gamma], \quad (2.2)$$

$$\frac{dR}{d\tau} = \Gamma I - R[X_r + \Lambda]. \quad (2.3)$$

The variables and parameters in equations (2.1)–(2.3) are summarized in table 1. These equations have the following equilibria.

(a) Host extinction $S = 0$, $I = 0$, $R = 0$

In the absence of the disease, $I = 0$ and $R = 0$. In this case, equation (2.1) becomes

$$\frac{dS}{d\tau} = S[M(1 - S) - 1]. \quad (2.4)$$

So a necessary condition for metapopulation persistence is $M > 1$, recovering the condition for persistence in a classic disease-free metapopulation that the colonization rate must exceed the extinction rate (Levins 1969).

(b) Disease free equilibrium $S > 0$, $I = 0$, $R = 0$

Setting equation (2.4) to equal zero, and denoting the equilibrium value of S as S^*

$$S^* = 1 - \frac{1}{M}. \quad (2.5)$$

This equilibrium, with $I = 0$ and $R = 0$, will be stable unless the pathogen can invade the system (see equation (2.6) below).

(c) Endemic disease $S > 0$, $I > 0$, $R > 0$

The condition for disease invasion into a population consisting of susceptible patches can be obtained from conditions for the right-hand side of equation (2.2) being more than zero, substituting in S^*

$$M > 1 + \frac{X_i - 1 + \Gamma}{\delta}. \quad (2.6)$$

This is always greater than the threshold migration rate for maintenance of susceptible patches, as one would expect, provided that the extinction rate of hosts in infected patches exceeds that for hosts in susceptible patches. It is also monotonically decreasing in δ : the higher the transmission rate, the more likely the disease is to be maintained.

Hess's model also has a potential equilibrium with pandemic disease ($S = 0$, $I > 0$). This equilibrium exists in

Table 1. Parameter and variable definitions for the single-host model (equations (2.1)–(2.3)).

symbol	meaning (upper-case parameters relative to extinction rate on susceptible patches)
N	total number of patches
s, S	number or proportion of patches containing susceptible hosts
i, I	number or proportion of patches containing infected hosts
r, R	number or proportion of patches containing resistant hosts
x_s	extinction rate of susceptible host patches
x_i, X_i	extinction rate of infected host patches
x_r, X_r	extinction rate of resistant host patches
m, M	colonization rate of empty patches connectance
δ	proportion of immigrants from infected patches that cause infection on arrival in a susceptible patch
γ, Γ	rate of recovery from infection of infected patches
λ, Λ	rate of loss of resistance from patches
τ	time scaled in units of x_s

Figure 1. The proportion of patches occupied at equilibrium in equations (2.1)–(2.3) as a function of the migration rate M and extinction rate of infected patches X_i . Other parameters are: $\delta = 0.8$, $X_r = 1.0$, $\Gamma = 1.0$, $\Lambda = 2.0$.

Hess’s formulation with no resistant class, but will not in the current model, because if $S = 0$ in equation (2.1), $dS/dt > 0$, provided $R > 0$. Likewise, if $R = 0$ in equation (2.3), $dR/dt > 0$, provided $I > 0$. Thus, any equilibrium with disease present will have patches of all three types of infection status (and some empty patches).

Figure 1 shows a numerical solution of the equilibrium for equations (2.1)–(2.3), for varying values of the migration rate M and extinction rate of infected patches X_i . As Hess noted for his model, there is a small decrease in the equilibrium proportion of patches occupied when the migration rate increases sufficiently that the pathogen can persist in the metapopulation. For all parameter combinations we have investigated, this decrease is slight, and as the migration rate increases further, the proportion of occupied patches again begins to increase.

Our extension of Hess’s (1996) model indicates that increasing connectivity between patches is unlikely to lead to conservation problems in cases where the pathogen is specific to the endangered species. Indeed, if the extinction rate of infected patches is very high, the benefit of high connectance permits rapid recolonization of patches that have become extinct through infection. The benefits of this are likely to counterbalance the cost of connectance that transfers infection into susceptible patches. Ideally, one would want to maintain connectance for uninfected

hosts, whilst reducing it for infected hosts. This would be equivalent to reducing δ in our model, whilst maintaining M at a high level. In highly managed metapopulations (e.g. zoo populations) this might be possible, but the model indicates that reducing connectance for all classes of hosts in a free-living population is not a sensible strategy.

3. A METAPOPULATION MODEL WITH A RESERVOIR HOST

Assume now that there are two host species: a reservoir (species 1), and an ‘endangered’ target (species 2). Species 2 is highly susceptible to a pathogen carried by species 1, to the extent that it cannot persist in a patch in the presence of an infected population of species 1. Species 2 is potentially ‘endangered’ by the disease: it is not necessarily rarer or present on fewer patches than the reservoir if infection is absent. Assume that there are N available patches, where N is large. These patches are identical, but may be in one of seven states at time t .

- (i) Empty patches. Hosts are not present.
- (ii) Patches occupied only by susceptible hosts of species 1 (reservoirs). Represented by $s_1(t)$.
- (iii) Patches occupied only by susceptible hosts of species 2 (endangered). Represented by $s_2(t)$.
- (iv) Patches occupied by susceptible hosts of both species. Represented by $s_{12}(t)$.
- (v) Patches in which infection is present (these will contain reservoirs only). Represented by $i(t)$.
- (vi) Patches occupied by resistant hosts (necessarily reservoirs only). Represented by $r(t)$.
- (vii) Patches occupied by resistant reservoir hosts and susceptible hosts of species 2. Represented by $r_2(t)$.

(a) Reservoir hosts

Following Hess’s model, assume that the rate of extinction of patches of each class of reservoir host can be represented as x_s, x_i, x_r respectively, where all rates are positive, and $x_i > x_s$ and $x_i > x_r$. Furthermore, assume that reservoir migrants of all types leave patches they occupy at a rate m_1 . If a susceptible migrant from a particular patch type enters an empty patch, that patch becomes colonized with hosts of that appropriate type. Infected host migrants (necessarily reservoirs) produce an infected

patch. If infected hosts enter a patch containing susceptible reservoirs, that patch has a probability δ of becoming infected. Resistant hosts entering patches already containing reservoirs do not change patch status, and infected hosts entering resistant patches do not affect the status of the patch. Furthermore, assume that infected patches recover from infection at a rate γ , and that resistance is lost, with patches joining the susceptible class, at a rate λ .

(b) *Endangered species*

All patches containing endangered species produce emigrants at a rate m_2 . Endangered species go extinct on patches they occupy at a rate x_e , whether they are alone or co-occupy the patch with susceptible or resistant reservoirs. They immediately become extinct if the reservoirs on their patch become infected, or if an infected immigrant arrives.

Defining the dynamics, as before, in terms of the proportions of all patches occupied by each type of host ($S_1 = s_1/N$, $I = i/N$, $R = r/N$ etc.), rescaling time in units of x_e , the extinction rate of reservoirs and representing rate parameters measured in units of x_e with upper-case symbols, the following equations embody the assumptions above.

$$\frac{dS_1}{d\tau} = M_1(S_1 + S_{12} + R + R_2)E - S_1 - \delta M_1 I S_1 + \lambda R - M_2(S_2 + S_{12} + R_2)S_1 + X_e S_{12}, \quad (3.1)$$

$$\frac{dS_2}{d\tau} = M_2(S_2 + S_{12} + R_2)E - X_e S_2 - \delta M_1 I S_2 - M_1(S_1 + S_{12} + R + R_2)S_2 + S_{12} + X_e R_2, \quad (3.2)$$

$$\frac{dS_{12}}{d\tau} = M_1(S_1 + S_{12} + R + R_2)S_2 + M_2(S_2 + S_{12} + R_2)S_1 - \delta I M_1 S_{12} - (1 + X_e)S_{12} + \lambda R_2, \quad (3.3)$$

$$\frac{dI}{d\tau} = I[M_1 E - X_e + \delta M_1(S_1 + S_2 + S_{12}) - \Gamma], \quad (3.4)$$

$$\frac{dR}{d\tau} = -R(X_r + \lambda) + \Gamma I + X_e R_2 - M_2(S_2 + S_{12} + R_2)R, \quad (3.5)$$

$$\frac{dR_2}{d\tau} = M_2(S_2 + S_{12} + R_2)R - (X_r + X_e)R_2 - \lambda R_2. \quad (3.6)$$

Here, $E = 1 - S_1 - S_2 - S_{12} - I - R - R_2$ is the proportion of empty patches. The variables and parameters in equations (3.1)–(3.6) are summarized in table 2.

(i) *All patches extinct*

The system may be invaded by reservoir hosts if

$$M_1 > 1. \quad (3.7)$$

This is, of course, the identical condition to invasion of the system by a species considered in isolation. Likewise, the endangered species may invade if

$$M_2 > X_e. \quad (3.8)$$

As the two hosts do not interfere with each other in the absence of infection, these conditions are independent. Which species enters first depends on the relative immigration and extinction rates.

At any disease-free equilibrium, the total number of patches occupied by the reservoir is

$$S_1^* + S_{12}^* = 1 - \frac{1}{M_1}, \quad (3.9)$$

and those occupied by the endangered species will satisfy

$$S_2^* + S_{12}^* = 1 - \frac{X_e}{M_2}. \quad (3.10)$$

Hence,

$$S_{12}^* = \left(1 - \frac{1}{M_1}\right) \left(1 - \frac{X_e}{M_2}\right), \quad (3.11)$$

$$S_1^* = \left(1 - \frac{1}{M_1}\right) \frac{X_e}{M_2}, \quad (3.12)$$

and

$$S_2^* = \left(1 - \frac{X_e}{M_2}\right) \frac{1}{M_1}. \quad (3.13)$$

(ii) *Equilibria with the pathogen present*

The pathogen cannot invade a system when only the endangered species is present, as the endangered species cannot support a pathogen population. The condition for successful pathogen invasion remains the same as it is in the single-host model, namely

$$M_1 > 1 + \frac{X_e - 1 + \Gamma}{\delta}. \quad (3.14)$$

Understanding the behaviour of the system with the pathogen present requires numerical solutions for particular parameter values in the region of interest. Unlike the single-host pathogen model in equations (2.1)–(2.3), in some circumstances high rates of connectance between patches may lead to extinction of the endangered host. For example, figure 2a shows where the movement rate of the endangered species is less than that of the reservoir, the extinction rate in the absence of infection of the endangered species exceeds that of the reservoir, and the rate of recovery from infection is similar to the patch extinction rate. In this scenario, the level of connectance that is necessary to maintain the endangered species is greater than that for the reservoir, and there is a relatively narrow range of connectance values in which the two host species and the pathogen can coexist.

Extinction of the endangered species at high levels of connectance is not, however, inevitable in this model. For example, figure 2b shows a case where the endangered species can exist at lower levels of connectance than can the reservoir species, because of a higher colonization rate, M_e . In this case, the proportion of patches occupied by the endangered species declines with connectance once the pathogen can persist, but the endangered species can persist for a wide range of connectance values. Similarly, the endangered species might persist at lower levels of connectance than the reservoir, because of a low extinction rate. In both these cases, the endangered species is essentially operating as a fugitive species, persisting on patches vacated by the pathogen through extinction.

High rates of recovery from infection may also lead to persistence of the endangered species at high connectance levels. For example, figure 2c shows a slight increase in endangered species prevalence at high connectance values, despite a concomitant increase in the overall proportion

Table 2. Parameter and variable definitions for the two-host model (equations (3.1)–(3.6)).

symbol	meaning (upper-case parameters relative to extinction rate of susceptible reservoir hosts from patches)
N	total number of patches
s_1, S_1	number or proportion of patches containing only susceptible reservoir hosts
s_2, S_2	number or proportion of patches containing only susceptible endangered hosts
s_{12}, S_{12}	number or proportion of patches containing both susceptible reservoir and endangered hosts
i, I	number or proportion of patches containing infected hosts that are necessarily reservoirs
r, R	number or proportion of patches containing only resistant reservoir hosts
r_2, R_2	number or proportion of patches containing resistant reservoir hosts and susceptible endangered hosts
x_s	extinction rate of susceptible reservoir host from patches
x_i, X_i	extinction rate of infected reservoir host patches
x_r, X_r	extinction rate of resistant reservoir host from patches
x_e, X_e	extinction rate of endangered hosts from patches
m_1, M_1	colonization rate of susceptible reservoirs (connectance)
m_2, M_2	colonization rate of endangered species
M_e	colonization rate of endangered species relative to rate of reservoirs
δ	proportion of immigrants from infected patches that cause infection on arrival in a susceptible patch
γ, Γ	rate of recovery from infection of infected patches
λ, Λ	rate of loss of resistance from patches
τ	time scaled in units of x_s

Figure 2. Equilibria of the two-host model (equations (3.1)–(3.6)), as a function of connectance between patches M_1 . The migration rate of endangered hosts M_2 is scaled relative to the migration rate of reservoirs, such that $M_2 = M_1 M_e$, where M_e is the proportionality constant. In each figure, the total proportion of patches occupied by reservoir hosts, whether susceptible, infected or resistant, is shown as a solid line, the proportion of patches occupied by endangered hosts is shown as a dotted line, the proportion of patches occupied by infected hosts is shown as a dashed line, and the proportion of patches occupied by resistant hosts is shown as a dotted and dashed line. (a) $\delta = 0.8$, $X_i = 4.0$, $X_r = 1.0$, $X_e = 2.0$, $M_e = 0.5$, $\Gamma = 1.0$, $\Lambda = 2.0$; (b) $\delta = 0.8$, $X_i = 4.0$, $X_r = 1.0$, $X_e = 2.0$, $M_e = 3.0$, $\Gamma = 1.0$, $\Lambda = 2.0$; (c) $\delta = 0.8$, $X_i = 2.0$, $X_r = 1.0$, $X_e = 2.0$, $M_e = 0.5$, $\Gamma = 5.0$, $\Lambda = 5.0$; and (d) $\delta = 0.9$, $X_i = 2.0$, $X_r = 1.0$, $X_e = 2.0$, $M_e = 0.5$, $\Gamma = 1.0$, $\Lambda = 1.0$.

of patches infected by the pathogen. It appears that the increasing proportion of patches occupied by resistant reservoir populations is providing a refuge for the endangered species.

Finally, for some combinations of parameter values, there are no connectance values that allow the endangered species to persist, if the pathogen is present. Figure 2*d* shows an example where equation (3.14) is satisfied at lower levels of connectance than is equation (3.8), and no stable equilibrium with both hosts present exists.

The model has a number of potential equilibria.

4. DISCUSSION

Corridors between remnant habitat patches have often been suggested as a conservation strategy (Bennett 1990; Hobbs 1992; Dobson *et al.* 1999). The strongest argument in favour of this is that, prior to anthropological disturbance, the landscapes in which most species evolved were more completely connected. Hess (1994) has argued that corridors are not without risks, where infectious disease threats exist. In the first part of this paper, we refute this claim. For the case considered by Hess (1996, 1994), where a pathogen affects only an endangered species, our model that considers host recovery indicates that the adverse consequences of pathogen movement between patches are largely counterbalanced by the benefits of increased colonization of patches that would otherwise become extinct. Furthermore, conservation problems caused by pathogens that are specific to only an endangered species are relatively unusual. More commonly, there is a reservoir species or species complex. Gog *et al.* (2002) have recently modelled this situation by assuming that there is an external 'propagule rain' onto the patches occupied by the endangered species. This broadly corresponds to the situation where the reservoirs occupy the matrix around the patches. Their conclusion was that, although occupancy fraction may decline with increased host movement and a small amount of external pathogen input, there is a critical level of pathogen input beyond which occupancy fraction is a monotonically increasing function of the host movement rate.

We have therefore extended Hess's model to examine cases where the pathogen may infect both an endangered species and a more common reservoir host, and the two host types have similar patch requirements. The costs and benefits of maintaining connectance via corridors between habitat patches are more complex, once additional reservoir species using the same patches become involved. In any species which follows the metapopulation paradigm to the extent that long-term persistence depends on a balance between local extinction and recolonization, some degree of connection between habitat patches is needed, whatever the characteristics of a potential pathogen and its hosts. If an exotic pathogen or reservoir host is introduced into the system, there may be no level of connectance that permits persistence of an endangered species through a balance between patch extinction and colonization (corresponding to figure 2*d*). This situation is likely to arise when the endangered species has limited ability to disperse and therefore fails to colonize either abandoned patches or those occupied by previously infected and now resistant reservoir hosts. For these species, strict quaran-

tine and controlled movement of disease-free individuals between patches are the only possible conservation strategies.

All our numerical results show that the proportion of patches occupied by the endangered species declines once connectance is sufficient for the pathogen to persist. Given that we have assumed that the endangered species cannot be maintained on patches in the presence of the pathogen, this is not surprising. The result that is rather more interesting is that a species can be maintained in a metapopulation, together with a lethal pathogen, at high levels of connectance between patches (see figure 2*b,c*). This can occur because the endangered species colonizes patches that have become vacant through pathogen-induced extinction of the reservoir host (figure 2*b*) and persists until the patch is again infected. In this situation, the endangered species is essentially acting as a fugitive species. The endangered species can also persist by colonizing patches containing resistant reservoir populations (figure 2*c*). Such patches are refugia from the pathogen, for as long as they maintain their resistant status. Resistant patches act as refugia because we assume that infection does not become established on a patch containing resistant reservoir hosts. Empirical evidence suggests that transmission for many pathogens is towards the frequency-dependent end of the density-dependence to frequency-dependence continuum (McCallum *et al.* 2001). This means that a reservoir host entering a patch containing both resistant reservoir hosts and endangered hosts will have most of its contacts with the resistant hosts, and an epidemic is unlikely to result.

We have chosen to present the results of the model as functions of M_1 , the 'connectance' between patches, as a major management question is whether to maintain or establish corridors and connections between existing patches. Another possibility is to examine the same problem from the perspective of habitat destruction: under what conditions will habitat loss increase or decrease pathogen threats to endangered species? Nee & May (1992) developed a model of competition between two species in a metapopulation, and showed that as habitat loss, represented as a proportion h of the patches originally present, increased, an inferior competitor might increase relative to a superior competitor. This might occur if the inferior 'weedy' species had a higher rate of colonization than the superior competitor, even if the superior competitor always drove the weedy species to extinction on patches where both occurred. This system has obvious parallels to our model, with the pathogen acting as the agent of competition, its presence preventing coexistence of the two host species on the same patch. Dytham (1995) developed an explicitly spatial cellular analogue of Nee & May's competition model, showing that the effect of habitat loss on coexistence depended greatly on its spatial arrangement. Almost certainly, our pathogen-mediated model would behave similarly, but this is a topic for future research.

May (1994) briefly discusses predator-prey dynamics in a classic metapopulation, as a function of habitat loss. As is the case with our model in equations (2.1)–(2.3), habitat loss will cause the loss of the predator 'pathogen', before the loss of the prey 'host', causing an increase in prey patch occupancy at some level of patch loss. More

recently, Bascompte & Solé (1998) have extended May's model, in particular by developing a spatially explicit version. Their conclusions remain broadly the same.

It is very easy to present our results as a function of the proportion of patches remaining after habitat loss. In a simple metapopulation model, patch loss has the effect of decreasing the rate of recolonization, whilst keeping the patch-specific extinction rate constant. Our 'connectance' parameter is thus equivalent to the proportion of suitable patches remaining. Formally, we can replace 1 with h in the expression for E , the proportion of empty patches used in equations (3.1), (3.2) and (3.4), and we can rescale the variables as proportions of the remaining available patches (e.g. define $S'_i = S_i/h$). Thus, the x -axis now depicts the proportion of original habitat remaining, whence each panel of figure 2*a–d* can then be interpreted as showing the effect of progressive habitat loss from any starting level of connectance on the horizontal axis.

Habitat loss may therefore permit the existence of a host that is unable to persist in a metapopulation because it is highly susceptible to a pathogen it shares with another species (see figure 2*a*, commencing with an initial connectance of about 10), even if the habitat loss is not sufficient to eliminate the pathogen entirely. However, habitat loss, even in the presence of a lethal pathogen, will not necessarily have this effect (see figure 2*b,c*, in which the endangered host is maintained for a wide range of connectance, and is eliminated only at low levels of connectance (or equivalently, high levels of habitat loss)).

Models with two hosts sharing a common pathogen in a non-spatial environment have been discussed in a number of papers (e.g. Holt & Pickering 1985; Bowers & Begon 1991; Begon & Bowers 1995*a,b*). The emphasis in these has been in determining conditions for the coexistence of both host species in the presence of the shared pathogen. In general, the main conclusion is similar to that for coexistence in Lotka–Volterra competition models: infected members of each species must have a greater impact on their own species than on the other. If the reverse is true, contingent elimination, depending on the starting values, may occur. If the interaction is asymmetrical, so that an infected member of species 1 has a greater impact on species 2 than on its own species, but infected members of 2 have a greater impact on their own species than on species 1, the inferior species will always be eliminated. Our model is highly asymmetrical, but the spatial subdivision permits coexistence in some circumstances.

Our model is, of course, a very crude caricature of a two-host pathogen interaction in a spatially subdivided environment. It relies on a number of simplifying assumptions. First, it assumes that both host and parasite function as classical metapopulations, maintained in a dynamic equilibrium by a balance between extinction and colonization. How commonly spatially subdivided populations can adequately be described by the metapopulation paradigm is debated (e.g. Harrison 1994; Hanski 1997). Second, the model ignores within-patch dynamics. This means that we assume that when reservoir or endangered hosts colonize a patch, numbers reach carrying capacity on a time-scale that is fast relative to patch colonization and extinction rates. Similarly, we assume that the pathogen reaches equilibrium prevalence with patches suf-

ficiently fast that the time since infection is not an important variable.

Finally, the model results presented also deal with equilibrium at the metapopulation level. If the equilibria presented in figures 1 and 2 are reached only after transient large amplitude oscillations, then they may provide a very misleading impression of the likelihood of extinction in the system, as host extinction might occur for stochastic reasons at points of low host abundance. Another aspect of transient behaviour that cannot be included in a simple metapopulation model such as ours is the possibility of synchronous epidemics across many patches at high levels of connectance. Transient behaviour depends on the values at which a simulation is commenced, as well as the equations themselves. Perhaps the most probable scenario is that an exotic pathogen would be introduced into a system that was close to equilibrium in the disease-free state. Time-dependent solutions commencing at this initial state, with the pathogen introduced to a small proportion of patches, approached equilibrium with rapid damping and little overshoot, at least for the parameter range represented in figure 2.

Accepting these caveats, our models suggest several conclusions about managing connectivity in the face of disease threats. If a pathogen is specific to the host species in question, the models indicate that pathogen transmission should not be a crucial factor in deciding whether to maintain corridors between habitat patches. Whilst the pathogen may spread via corridors, the benefit of permitting recolonization of vacant patches counterbalances the cost of disease spread. If there is a reservoir species, upon which the pathogen has a less detrimental effect than on the target, the situation becomes less straightforward.

The worst-case scenario, in which the pathogen can be maintained at lower levels of connectance than the endangered species, may exist if the endangered species is prone to local extinction in the absence of infection and is a poor colonizer. In addition, the pathogen would need to be highly infectious to the reservoir ($\delta \approx 1$) and relatively benign in the reservoir.

If the endangered species can be maintained in the system at lower levels of connectance than the pathogen, there will be an optimal level of connectance, at which the proportion of patches occupied by the endangered species is maximized. Not surprisingly, this level is a connectance just insufficient to allow the pathogen to persist. In reality, the approximations made in this model, together with difficulties in parameter estimation and stochasticity, mean that aiming for some optimal level of connectance will not be an easy conservation strategy. An important practical consideration is whether it is better to err on the side of too little or too much connectivity between patches.

The two-host model results indicate that too much connectance may lead to a pathogen driving an endangered species to extinction under a specific set of conditions. Namely, if the endangered species has a higher extinction rate and colonization rate than the reservoir; and the pathogen is persistent in the reservoir patches. Pathogen persistence in the reservoir is associated with low parasite-induced extinction and low rates of loss of infection from infected patches. Extinction and colonization rates are difficult to measure directly, but if the endangered species occupies a smaller proportion of suitable patches than the

reservoir, in the absence of infection, this indicates low colonization or high extinction rates relative to the reservoir.

Nevertheless, erring on the side of too much connectance is advisable if the endangered species is more vagile than the reservoir, or if infection in reservoir patches is transient. Whilst the proportion of patches occupied by the endangered species will decline at high connectance, the species will nevertheless persist, whereas it will always become extinct at low levels of connectance.

This paper had its genesis whilst Hamish McCallum was on study leave at Princeton University. We are grateful for the hospitality of the Department of Evolution and Ecology, Princeton University. This research is partially supported by Australian Research Council grant DP0210375. This paper was improved with the suggestions made by two anonymous referees and by discussions with members of the NCEAS working group on Disease and Conservation.

REFERENCES

- Anderson, R. M. & May, R. M. 1979 Population biology of infectious diseases. Part I. *Nature* **280**, 361–367.
- Augustine, D. J. 1998 Modelling *Chlamydia*–koala interactions: coexistence, population dynamics and conservation implications. *J. Appl. Ecol.* **35**, 261–272.
- Bascompte, J. & Solé, R. V. 1998 Effect of habitat destruction in a prey–predator metapopulation model. *J. Theor. Biol.* **195**, 383–393.
- Begon, M. & Bowers, R. G. 1995a Beyond host–pathogen dynamics. In *Ecology of infectious diseases in natural populations* (ed. B. T. Grenfell & A. P. Dobson), pp. 478–509. Cambridge University Press.
- Begon, M. & Bowers, R. G. 1995b Host–host–pathogen models and microbial pest control: the effect of host self-regulation. *J. Theor. Biol.* **169**, 275–287.
- Bennett, A. F. 1990 *Habitat corridors. Their role in wildlife management and conservation*. Melbourne, Australia: Department of Conservation and Environment.
- Bolker, B. & Grenfell, B. 1995 Space, persistence and dynamics of measles epidemics. *Phil. Trans. R. Soc. Lond. B* **348**, 309–320.
- Bowers, R. G. & Begon, M. 1991 A host–host–pathogen with free-living infective stages, applicable to microbial pest control. *J. Theor. Biol.* **148**, 305–329.
- Cleaveland, S., Hess, G. R., Dobson, A. P., Laurenson, M. K., McCallum, H. I., Roberts, M. G. & Woodroffe, R. 2002 The role of pathogens in biological conservation. In *The ecology of wildlife diseases* (ed. P. J. Hudson, A. Rizzoli, B. Grenfell & H. Heesterbeek), pp. 139–150. Oxford University Press.
- de Jong, M. C. M., Diekmann, O. & Heesterbeek, H. 1995 How does transmission of infection depend on population size? In *Epidemic models: their structure and relation to data* (ed. D. Mollison), pp. 84–94. Cambridge University Press.
- Dobson, A. P. (and 13 others) 1999 Landscape viability equals landscape connectivity: reconnecting fragmented landscapes. In *Continental conservation: scientific foundations of regional reserve networks* (ed. M. Soule & J. Terborgh), pp. 129–170. Washington, DC: Island Press.
- Dytham, C. 1995 The effect of habitat destruction pattern on species persistence: a cellular model. *Oikos* **74**, 340–344.
- Gog, J., Woodroffe, R. & Swinton, J. 2002 Disease in endangered metapopulations: the importance of alternative hosts. *Proc. R. Soc. Lond. B* **269**, 671–676. (DOI 10.1098/rspb.2001.1667.)
- Hanski, I. 1997 Metapopulation dynamics: from concepts and observations to predictive models. In *Metapopulation biology: ecology, genetics and evolution* (ed. I. Hanski & M. E. Gilpin), pp. 69–91. London: Academic.
- Harrison, S. 1994 Metapopulations and conservation. In *Large-scale ecology and conservation biology* (ed. R. M. May & N. R. Webb), pp. 111–128. Oxford, UK: Blackwell Scientific.
- Heide-Jorgensen, M. P. & Härkönen, T. 1992 Epizootiology of the seal disease in the eastern North Sea. *J. Appl. Ecol.* **29**, 99–107.
- Hess, G. 1996 Disease in metapopulation models: implications for conservation. *Ecology* **77**, 1617–1632.
- Hess, G. R. 1994 Conservation corridors and contagious disease: a cautionary note. *Conserv. Biol.* **8**, 256–262.
- Hobbs, R. J. 1992 Role of corridors in conservation: solution or bandwagon? *Trends Ecol. Evol.* **7**, 389–392.
- Holt, R. D. & Pickering, J. 1985 Infectious disease and species coexistence: a model of the Lotka–Volterra form. *Am. Nat.* **126**, 196–211.
- Levins, R. 1969 Some demographic and genetic consequences of environmental heterogeneity for biological control. *Bull. Entomol. Soc. Am.* **15**, 237–240.
- McCallum, H., Barlow, N. & Hone, J. 2001 How should transmission be modelled? *Trends Ecol. Evol.* **16**, 295–300.
- McCallum, H. I. & Dobson, A. P. 1995 Detecting disease and parasite threats to endangered species and ecosystems. *Trends Ecol. Evol.* **10**, 190–194.
- May, R. M. 1994 The effects of spatial scale on ecological questions and answers. In *Large-scale ecology and conservation biology* (ed. P. J. Edwards, R. M. May & N. R. Webb), pp. 1–17. Oxford: Blackwell Scientific.
- May, R. M. & Anderson, R. M. 1979 Population biology of infectious diseases. Part II. *Nature* **280**, 455–461.
- Melzer, A., Carrick, F., Menkhurst, P., Lunney, D. & St John, B. 2000 Overview, critical assessment, and conservation implications of koala distribution and abundance. *Conserv. Biol.* **14**, 619–628.
- Nee, S. & May, R. M. 1992 Dynamics of metapopulations: habitat destruction and competition coexistence. *J. Anim. Ecol.* **61**, 37–40.
- Phillips, S. S. 2000 Population trends and the koala conservation debate. *Conserv. Biol.* **14**, 650–659.
- Sih, A., Johnson, B. G. & Luikart, G. 2000 Habitat loss: ecological, evolutionary and genetic consequences. *Trends Ecol. Evol.* **15**, 132–134.
- Simonetti, J. A. 1995 Wildlife conservation outside parks is a disease-mediated task. *Conserv. Biol.* **9**, 454–456.
- Swinton, J., Harwood, J., Grenfell, B. T. & Gilligan, C. A. 1998 Persistence thresholds for phocine distemper virus infection in harbour seal *Phoca vitulina* metapopulations. *J. Anim. Ecol.* **67**, 54–68.
- White, N. A. & Timms, P. 1994 *Chlamydia psittaci* in a koala (*Phascolarctos cinereus*) population in southeast Queensland. *Aust. Wildl. Res.* **2**, 141–147.

As this paper exceeds the maximum length normally permitted, the authors have agreed to contribute to production costs.