

Disease in endangered metapopulations: the importance of alternative hosts

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Conventional applications of metapopulation theory have suggested that increasing migration between patches is usually good for conservation. A recent analysis by Hess has pointed out a possible exception to this: when infectious disease is present, migration may promote disease spread and therefore increase local extinction. We extend Hess's model to discuss this problem: when infections have spilled over from more abundant alternative hosts. This is often the case for species of conservation concern, and we find that Hess's conclusions must be substantially modified. We use deterministic analytic and stochastic numerical approaches to show that movement between patches will rarely have a negative impact, even when the probability of external infection is low.

Keywords: metapopulation; spillover; reservoir; disease model

1. INTRODUCTION

Over the past decade, metapopulation dynamics has come to have an increasingly important role in conservation planning. Theory predicts that movement between subpopulations should increase the number of patches that are occupied at equilibrium, the genetic effective size of the metapopulation and the time delay to metapopulation extinction (Hanski & Gilpin 1997). For this reason, many conservationists have favoured measures—such as construction of corridors, or active translocation of individuals—which encourage migration between patches.

In a recent paper, however, Hess (1996) suggested a negative aspect to such migration. Infectious disease represents a serious extinction risk to threatened species, having the capacity to cause populations to crash suddenly, as well as slowing their recovery from perturbations (Woodroffe 1999). Using a metapopulation model, Hess (1996) showed that, by facilitating the spread of disease between subpopulations, migration could increase the probability of metapopulation extinction and reduce patch occupancy. We believe that this conclusion is likely to be misleading for many wild populations and demonstrate the reason in a simple modification of Hess's model.

The analytic model developed by Hess assumes that susceptible subpopulations become infected only by receiving immigrants from elsewhere within the metapopulation (Hess 1996). By contrast, most of the infections that have threatened wildlife populations with extinction have 'spilled over' from other, more abundant hosts (table 1; Woodroffe 1999; McCallum & Dobson 1995). We have modelled this phenomenon by altering the Hess model, to include a background force of infection. Our intention is not to evaluate the likelihood of such a spillover, but to explore the relationship between within-population migration rates and disease in those populations for which spillover occurs.

2. THE MODEL

Like the Hess model, our model contains a proportion, S , of susceptible host patches (containing a host population, but no disease), and I , of infected host patches (containing a host population with disease present). Susceptible and infected populations become extinct at rates x_S and x_I , respectively ($x_S < x_I$), with migration occurring from both susceptible and infected patches at a rate m . On arrival at a susceptible patch, an infected disperser infects the resident population with probability δ ; thus, infection spreads at a migration-dependent rate $m\delta IS$. In addition, and as an extension to the analytic Hess model, susceptible populations may become infected from an outside source, at a rate g , similarly to the 'propagule rain' concept analysed by Gotelli (1991). Such an external force of infection was included in the simulation models of Hess, although set at a very low level, to initiate the epidemic. The possible events in the model are given in table 2.

3. DETERMINISTIC ANALYSIS

First, we investigate the effects of this extra term in the context of a deterministic system. We use the following mean field model for the proportion of patches occupied in the different states, thus:

$$\dot{S} = mS(1 - I - S) - x_S S - m\delta IS - gS,$$

$$\dot{I} = mI(1 - I - S) - x_I I + m\delta IS + gS.$$

From this, we find the stable equilibria (Appendix A) and use these to plot equilibrium total occupancy rates against migration rates in figure 1. When $g = 0$ (i.e. in the Hess model) and for some range of g , occupancy rates initially increase with migration rate, then decrease as infection becomes established and a greater proportion of patches experience the higher extinction rates associated with infection. Eventually, occupancy increases once more, as all patches become infected and migration acts only to promote host population persistence. It is the non-monotonicity of this curve that underlies Hess's con-

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Table 1. Examples of wildlife populations which have been threatened by infectious disease. The majority of cases involve generalist pathogens which have apparently 'spilled over' from other, more abundant, host species. Note that a well-documented case of infection transmitted between populations by translocation (Castle & Christensen 1990) involved a pathogen believed to cause only subclinical infections, which are likely to have little or no effect upon population viability.

threatened host	pathogen	impact of pathogen	probable source	references
African wild dog (<i>Lycaon pictus</i>)	rabies	population extinction	domestic dogs	Gascoyne <i>et al.</i> (1993), Kat <i>et al.</i> (1995)
bighorn sheep (<i>Ovis canadensis</i>)	scabies, <i>Pasteurella</i>	population extinctions and crashes	domestic sheep	Jessup <i>et al.</i> (1991, 1995)
several African antelope species	rinderpest	population extinctions and crashes	domestic cattle	Plowright (1982)
African lion (<i>Panthera leo</i>)	canine distemper virus	population crash	domestic dogs	Roelke-Parker <i>et al.</i> (1996)
black-footed ferret (<i>Mustela nigripes</i>)	canine distemper virus	population crash	unknown; coyotes and badgers suspected	Thorne & Williams (1988)
Ethiopian wolf (<i>Canis simensis</i>)	rabies	population crash	domestic dogs	Sillero-Zubiri <i>et al.</i> (1996)
harbour seal (<i>Phoca vitulina</i>)	phocine distemper virus	population crash	harp seals	Swinton <i>et al.</i> (1998)
Baikal seal (<i>Phoca sibirica</i>)	canine distemper virus	population crash	domestic dogs	Grachev <i>et al.</i> (1989)
desert tortoise (<i>Gopherus agassizii</i>)	<i>Mycoplasma</i>	population crash	pet tortoises	Jacobson (1993)
grey wolf (<i>Canis lupus</i>)	rabies	high mortality	arctic and red foxes	Brand <i>et al.</i> (1995)
mountain gorilla (<i>Gorilla gorilla</i>)	measles	high mortality	man	Hastings <i>et al.</i> (1991)
grey wolf (<i>Canis lupus</i>)	canine parvovirus	pup mortality, hindering population recovery	domestic dogs (historically)	Mech & Goyal (1995)
Mednyi arctic fox (<i>Alopex lagopus semenovi</i>)	mange	pup mortality, hindering population recovery	domestic dogs	Goltsman <i>et al.</i> (1996)

Table 2. Possible events and corresponding rates for the model. This reduces to the analytic model of Hess (1996) when $g = 0$.

event	change in state	rate
a susceptible patch dies out	$(S, I) \rightarrow (S - 1, I)$	$x_S S$
an infected patch dies out	$(S, I) \rightarrow (S, I - 1)$	$x_I I$
susceptibles colonize an empty patch	$(S, I) \rightarrow (S + 1, I)$	$mS(N - I - S)/N$
infecteds colonize an empty patch	$(S, I) \rightarrow (S, I + 1)$	$mI(N - I - S)/N$
a susceptible patch becomes infected	$(S, I) \rightarrow (S - 1, I + 1)$	$m\delta I S/N + gS$

clusion that encouraging migration between patches may increase, rather than decrease, the probability of metapopulation extinction. This non-monotonicity is continued for a range of g . For greater values of g we see an entirely monotonic curve meaning that increasing movement rates only increases occupancy in this model.

To gain some sense of the magnitude of the critical value of g , we define ε to be $(1 - x_S/x_I)$. This is the expected reduction in patch persistence time that occurs when infection is present. We find our curve becomes monotonic at $g = x_S \varepsilon^2 / (1 - \varepsilon^2)$ in terms of this new parameter. To give an example, suppose that the disease reduces the life expectancy of a patch by one-third; under these conditions the occupancy fraction is monotonic whenever the chance of external infection is greater than one-eighth of the chance of extinction for a susceptible

patch. These spontaneous extinction rates are typically low and our analysis indicates that critical rates of external infection are an order of magnitude still smaller. Thus, the negative effects of increasing movement rates on patch occupancy are eliminated in parameter ranges possibly encountered in natural systems.

This shows that the conclusion from Hess's deterministic model is sensitive to a perturbation by the inclusion of an independent force of infection. This suggests that the exact form of this force is unlikely to be important. We would expect qualitatively the same results, were we to impose some structure such as only half the patches being subject to this risk of external infection, because of spatial structure in alternative hosts, for example, or if we were to link this risk to movement rates (for example if we made g proportional to m).

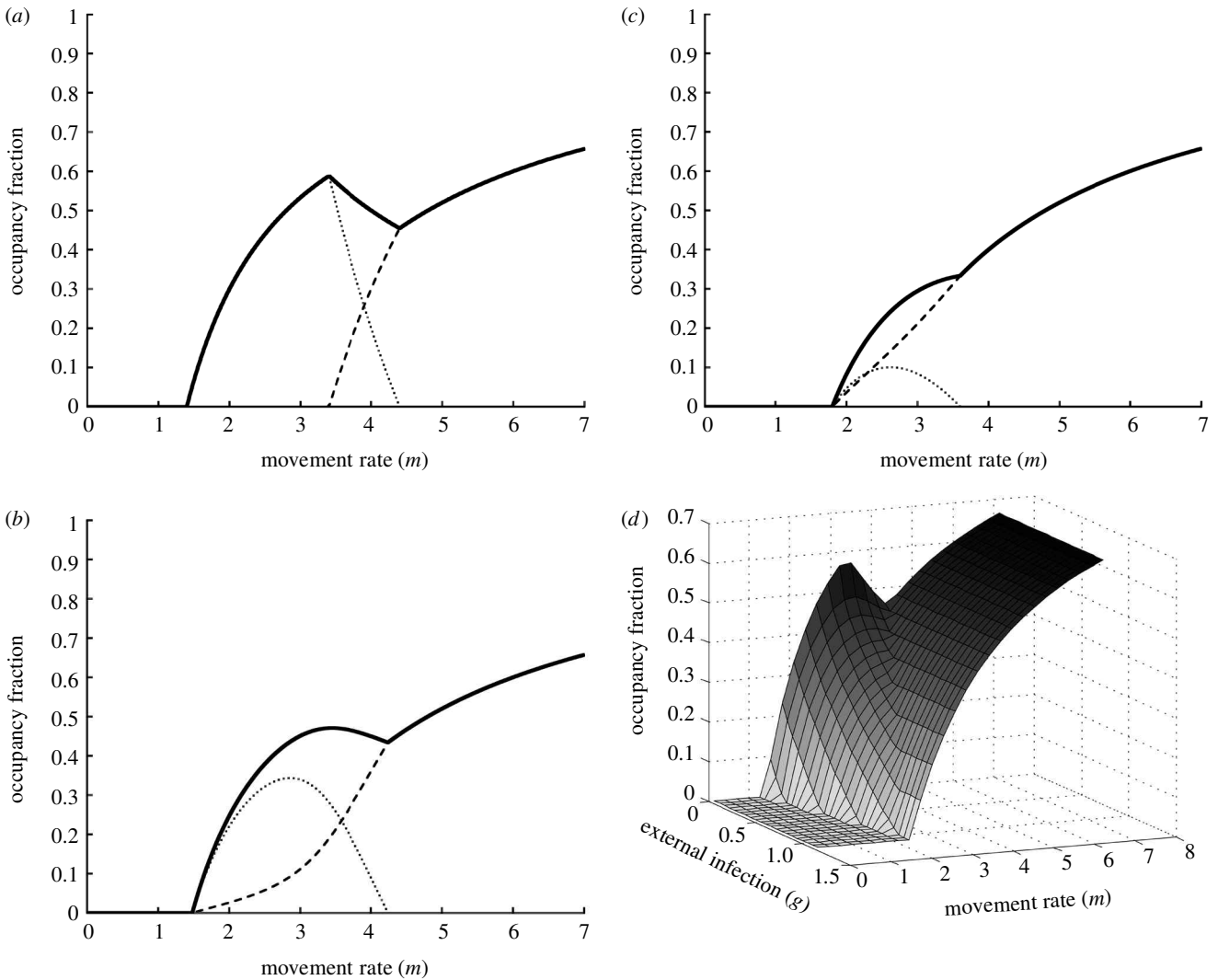


Figure 1. Occupancy fractions as a function of movement rates (m) calculated from the analytic expressions in Appendix A for the cases: (a) $g = 0$, the Hess model with no external infection: occupancy is non-monotonic in m , (b) $g = 0.08$, small background infection: occupancy is non-monotonic in m , (c) $g = 0.4$, larger background infection: occupancy is monotonic in m , and (d) occupancy fraction against both movement and external infection rates. (Dotted lines, susceptible; dashed lines, infected and solid lines, total rate). We have used the same parameters as Hess: $x_s = 1.4$, $x_I = 2.4$, $\delta = 0.5$, $g = 0$. For these values, $g^* = 0.2941$.

4. STOCHASTIC MODEL

The results of this deterministic analysis can only yield occupancy fractions, but for many conservation purposes, extinction rates may be a more appropriate measure of the costs and benefits of movement between subpopulations. To confirm that occupancy fractions are a reasonable proxy for this measure, and more importantly to test the robustness of our results, we now move to a stochastic model based on the same underlying model processes.

The pair of coordinates (S, I) completely characterizes the present state. The rates of transition between states are given in table 2. Rather than simply simulating a number of realizations of this system, this paper makes use of the Markov properties of this system. We may easily construct the explicit continuous time Markov transition matrix for these rates (Grimmett & Stirzaker 1992). This will be a large (of the order $N^2 \times N^2$), sparse (six non-zero entries per row) matrix. We can show that extinction is the unique absorbing state and also that all states communicate with it. Thus, the transition matrix has a unique

zero eigenvalue that corresponds to extinction and all other eigenvalues have a strictly negative real part. The asymptotic rate of extinction is the largest real part among these eigenvalues.

In formulating our deterministic model, we assumed that a high occupancy fraction in the deterministic model corresponded with a high chance of survival over a long time-scale; our stochastic model is designed to test this. To compare with the deterministic results, figure 2 shows the results in the form of probability of survival over a given time. The results from the stochastic model broadly support the conclusions of the deterministic approach, demonstrating in figure 2 the non-monotonicity in m at low g , which fades out to leave a monotonic curve for g around the critical value of g , calculated from the deterministic model. In the special case of $g = 0$, the subset of states with no sites infected is absorbing and the quasi-stationary state of the system is disease free. Hence, for $g = 0$, there is always monotonicity in the expected survival probabilities. In this respect, and for this special case, the deterministic and stochastic models disagree.

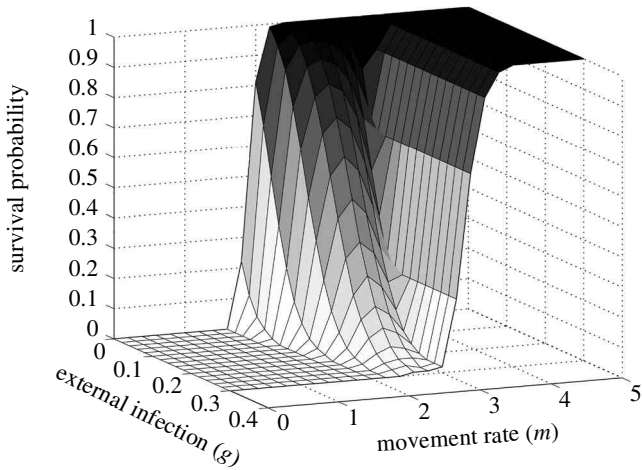


Figure 2. Survival probability against movement and external infection rates. Using a 100-patch model (hence a 5151×5151 transition matrix) and other parameters as in figure 1, we find the second largest eigenvalue of the transition matrix for a range of movement and external infection rates. Multiplying by 100 then exponentiating gives the survival probability over a time unit of 100; survival probabilities over other periods scale accordingly.

5. CONCLUSIONS

Using both a deterministic and a stochastic approach, we have shown that the simple metapopulation model with disease is sensitive to a perturbation that introduces infection from outside the system at probability g . We have also demonstrated that the threshold value of g at which such a perturbation becomes important is small, in comparison with the probability that a healthy patch becomes extinct, which we assume to be low. Thus, where subpopulations risk infection through contact with alternative hosts, as well as by immigration of infected conspecifics, the net effect of increased migration will often be entirely positive. If there is much background infection disease is already widespread and increasing movement rates will

have little further negative impact. It is intuitively clear that at high external infection rates, the benefits will always outweigh the costs of an increased movement rate. We emphasize that what our analysis demonstrates is that it is also true at surprisingly low levels of background, and that low levels of background infection are to be expected in many natural systems.

These results suggest that, for wild populations in which a high proportion of infections ‘spill over’ from alternative host species, migration between patches is likely to promote, rather than reduce, patch occupancy. Thus, corridors that encourage movement are likely to be beneficial in this context, although of course extinction rates in the presence of external infection are always higher than in its absence. However, Hess’s model may be more applicable to the captive situation, where transfer of animals between captive facilities, or between captive and wild populations, are a known cause of disease outbreaks (table 3). Detection of infected animals intended for translocation or release (e.g. Karesh & Cook 1995) indicates that veterinary screening and quarantine procedures (e.g. Cooper 1989; Woodford & Rossiter 1994) are effective in limiting disease transmission between populations, as predicted by Hess (1996), and must be incorporated into translocation strategies.

There are other complexities in the relationship between migration rates and disease. Highly virulent pathogens may prevent the carrier completing migration; the stresses of migration may act particularly on infected animals, thus modifying the disease profile of a migrating group; and the effect of any pathogen brought into a new population will depend heavily on the degree of physiological stress that the population is under.

An important caveat to this analysis is the possibility that measures that encourage migration between patches may also increase contact between threatened species and alternative hosts. If alternative hosts occur primarily outside reserves (as would be expected if reservoir hosts are domestic species), then corridors might increase contact

Table 3. Examples of captive populations (size n) which have been threatened with extinction by infectious disease. Some cases have been caused by movement of conspecifics between populations; other infections originate in other host species.

threatened host	pathogen	impact of pathogen	probable source of infection	references
black-footed ferret (<i>Mustela nigripes</i>)	canine distemper virus	100% mortality ($n = 6$)	introduced conspecific	Thome & Williams (1988)
rock rattlesnake (<i>Crotalus lepidus</i>)	ophidian paramyxovirus	89% mortality ($n = 9$)	introduced conspecific	Jacobson (1993)
cheetah (<i>Acinonyx jubatus</i>)	coronavirus	43% mortality ($n = 42$)	introduced conspecific	O’Brien <i>et al.</i> (1985), Wilson <i>et al.</i> (1994)
Rothchild’s mynah (<i>Leucospar rothchildii</i>)	avian pox	40% mortality ($n = 15$)	wildlife entering enclosure	Landolt & Kocan (1976)
Argentine tortoise (<i>Geochelone chilensis</i>)	herpesvirus	55% mortality ($n = 2200$)	other species in same enclosure	Jacobson (1993)
Arabian oryx (<i>Oryx leucoryx</i>)	<i>Mycobacterium bovis</i>	25% mortality ($n = 57$)	probably other species in enclosure	Flamand <i>et al.</i> (1994)
pink pigeon (<i>Nesoenas mayeri</i>)	herpesvirus	100% mortality ($n = 4$)	other species used as foster parents	Snyder <i>et al.</i> (1985)
golden lion tamarin (<i>Leontopithecus rosalia</i>)	hepatitis	43% mortality ($n = 7$)	domestic mice used as food	Montali <i>et al.</i> (1993)
pygmy marmoset (<i>Cebuella pygmaea</i>)	hepatitis	71% mortality ($n = 7$)	domestic mice used as food	Montali <i>et al.</i> (1993)

between host species by increasing the reserves' perimeter–area ratios. Such 'edge effects' would compound other extinction risks associated with exposure to reserve borders (Woodroffe & Ginsberg 1998). This possibility might be investigated by adding an external force of infection to the spatially explicit simulations proposed by Hess (1996). Our analyses emphasize the need for conservation managers to take an ecological approach to disease control, involving the management of reservoir as well as threatened host species (Woodroffe 1999).

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APPENDIX A

This gives the stable equilibrium for different values of g and m for the system of equations in § 3.

For $m \leq g + x_S$ and $m \leq x_I$:

$$S = 0, I = 0.$$

For $g + x_S \leq m \leq m_1 - g/\delta$ (implying $g \leq x_I - x_S$):

$$S = \frac{m_1 - m + 2g + \delta(m - x_S) - (1 + \delta)\sqrt{\xi}}{2m\delta},$$

$$I = \frac{m - m_2 - 2g + \sqrt{\xi}}{2m\delta}.$$

And for $m_1 - g/\delta \leq m$ and $x_I \leq m$:

$$S = 0, I = 1 - x_I/m,$$

where

$$m_1 = x_I + (x_I - x_S)/\delta,$$

$$m_2 = x_S + (x_I - x_S)/\delta,$$

$$\xi = (m - m_2)^2 + 4g(x_I - x_S)/\delta.$$

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