LETTER

Ecological replacement of native red squirrels by invasive greys driven by disease

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Abstract

Although a parapoxvirus harmful to red squirrels is present in UK squirrel populations it has not been considered a major cause of red squirrel decline, and replacement by the introduced grey squirrel, mainly because diseased individuals are rarely observed. By developing a generic model we show that parapoxvirus is likely to have played a crucial role in the red squirrel decline even though the prevalence of infection is low. Conservationists are quite rightly concerned with the invasion of exotic organisms such as the grey squirrel. Our work emphasizes that they, along with other ecologists, should pay particular attention to pathogens, even when they occur at low prevalence.

Keywords

Ecological replacement, emerging infectious disease, grey squirrel, parapoxvirus, red squirrel, *Sciurus*.

INTRODUCTION

The importance of disease in shaping human populations is clear cut (Anderson & May 1991), especially in cases where novel pathogens have been introduced by invading populations [for example, the conquest of the New World by European adventurers in the sixteenth century (Diamond 1997)]. However, although parasites and pathogens can clearly also affect wildlife host dynamics (Hudson *et al.* 1998; Tompkins & Begon 1999), they are still often disregarded unless clinical symptoms are widely documented in the population and are seen to be a major cause of mortality (Holmes 1982; Tompkins *et al.* 2002a). This is a fallacy – low visibility of disease need not indicate low importance. Since the early modelling work of Anderson & May (1978) it has been clear that pathogens do not need to be highly prevalent in order to have regulatory effects on their host populations [see Anderson (1995) for a worked example]. Furthermore, recent work has shown how the interplay between interspecific infection and other factors, such as competition or predation, can be crucial in determining host community structure (Bowers & Turner 1997). To illustrate these points, we apply the Bowers & Turner (1997) model to a species of great conservation importance in the UK, the red squirrel (*Sciurus vulgaris*). Our aim is to investigate whether disease has played a role in its decline since the introduction (and subsequent range expansion) of the grey squirrel (*S. carolinensis*) at the turn of the twentieth century (Middleton 1930; Shorten 1954; Lloyd 1983).

Since its introduction into the UK, the grey squirrel has 'replaced' the red squirrel throughout most of England and Wales, and in parts of Scotland and Ireland (Bryce 1997; O'Teangana *et al.* 2000). However, while evidence suggests that a squirrel parapoxvirus was introduced into the UK with the grey squirrel (Scott *et al.* 1981; Sainsbury & Gurnell 1995; Duff *et al.* 1996; Sainsbury & Ward 1996), its role in the red squirrel decline is believed to be minor as diseased red squirrels are rarely observed outside highly localized epidemics (Reynolds 1985). The traditional explanation is that competition with the grey squirrel (principally over food resources) is the primary cause of decline (MacKinnon 1978; Okubo *et al.* 1989; Kenward & Holm 1993; Bryce *et al.* 2001), with disease impacting only at an individual or local scale (Reynolds 1985). Two recent lines of evidence demonstrate, however, that the parapoxvirus may be important at the population scale. First, simulation models indicate that competition alone cannot account for the rate and pattern of red squirrel decline observed (Rushton *et al.* 1997). Secondly, experimental infections (conducted on adult individuals) have shown that the virus causes a deleterious disease in the red squirrel while having no detectable effect on grey squirrel health (Tompkins *et al.* 2002b). Parapoxvirus, for which the grey squirrel is implicated as a reservoir host (Sainsbury *et al.* 2000), may thus be the missing element from our understanding of the UK red squirrel decline and its ecological replacement by the introduced grey squirrel.

METHODS

To investigate the role of parapoxvirus in the red squirrel decline, the two-host/shared microparasite model of Bowers & Turner (1997) was modified to make it specifically applicable to the squirrel/virus system. The focus in the present study was different from that of Bowers & Turner (1997), whose aim was to determine the criteria for different population equilibria. Here, we investigated the time taken to transform the disease-free red squirrel population equilibrium to either the disease-free grey squirrel population equilibrium (when considering competition-mediated replacement) or the grey squirrel population equilibrium with endemic infection (when considering competition/infection-mediated replacement). A spatial model of the squirrel/virus system was then developed by linking a grid of patches containing the temporal model equations by dispersal. Specifically, we tested whether the spatial model could replicate the dynamics of the well-documented expansion of grey squirrels and decline of red squirrels that occurred in Norfolk (England) from 1960 to 1982, for which data are available on the presence or absence of the two species in 5-km grid squares (Reynolds 1985). A previous study which attempted to include parapoxvirus in an existing competition model could not account for the observed replacement detailed in the Reynolds (1985) data (Rushton et al. 2000). Furthermore, the red squirrel decline in the best-fit model obtained was caused by neither competition nor disease, but was due to the base rate of red squirrel fecundity being lower than the base rate of red squirrel mortality.

The dynamics of susceptible and infected red ($S_R + I_R$) and grey ($S_G + I_G$) squirrels are represented by a system of ordinary differential equations, with infected reds dying at rate α and infected greys recovering (to R_G) at rate γ:

$$\frac{dS_G}{dt} = [a_G - q_G(H_G + c_R H_R)]H_G - bS_G - \beta S_G(I_G + I_R) \tag{1}$$

$$\frac{dI_G}{dt} = \beta S_G(I_G + I_R) - bI_G - \gamma I_G \tag{2}$$

$$\frac{dR_G}{dt} = \gamma I_G - bR_G \tag{3}$$

$$\frac{dS_R}{dt} = [a_R - q_R(H_R + c_G H_G)]H_R - bS_R - \beta S_R(I_R + I_G) \tag{4}$$

$$\frac{dI_R}{dt} = \beta S_R(I_G + I_R) - (\alpha + b)I_R \tag{5}$$

H_G represents the total population of grey squirrels ($S_G + I_G + R_G$) and H_R represents the total population of red squirrels ($S_R + I_R$). The two species have the same rate of adult mortality (b) but different rates of maximum reproduction (a_R, a_G) and different carrying capacities (K_R, K_G). This could potentially lead to different susceptibilities to crowding (q_R, q_G) as $q = (a - b)/K$. Both intraspecific crowding and interspecific competition (see below) were modelled as causing density-dependent effects on reproduction but not on adult mortality. At least two lines of evidence justify this. First, Kenward et al. (1998) documented negative correlations between squirrel density and squirrel productivity (but not adult survival) for both species while, second, Wauters et al. (2000) documented reduced red squirrel recruitment (but no effect on adult mortality) in the presence of grey squirrels.

Several sources agree on an adult mortality value of c. 0.40 year^{-1} for both species (Gurnell 1987; Wauters et al. 2000) while Leslie matrices presented in Okubo et al. (1989), summarizing information from a range of relevant studies, indicate a difference in the average intrinsic net growth rates of both species (0.82 year^{-1} for grey squirrels and 0.61 year^{-1} for red squirrels). These values equate to average rates of maximum reproduction (maximum based on the habitat, but before competitive and other interactions between species) of 1.2 year^{-1} for grey squirrels and 1.0 year^{-1} for red squirrels, incorporating both adult fecundity and juvenile survival. Carrying capacities for the two species were calculated, as per Rushton et al. (1997), on the basis of published estimates of core range size. This is preferable to directly observing density from the field, where values obtained may already be depressed by interspecific interactions. For the case specifically under consideration (Norfolk, 1960–1982), woodland cover (suitable habitat for squirrels) was relatively stable at c. 6.5% with a 50/50 mix of coniferous and broadleaf trees (Reynolds 1985; Rushton et al. 1997). Based on core range areas of 2.3 ha per individual, a species packing factor of 1.1 (allowing for the fact that individual core ranges in squirrels can overlap), and a scaling factor for the carrying capacity of red squirrels in broadleaf habitat of 0.6, the carrying capacities for grey and red squirrels in one 5-km grid square were c. 80 and 60, respectively. The lower carrying capacity and average rate of maximum reproduction for the red squirrel reflects the lower efficiency with which this species utilizes food resources (specifically acorns) in broadleaf habitat (Kenward & Holm 1993). The competitive effect of grey squirrels on red squirrels is denoted by c_G, whilst that of red squirrels on grey squirrels is denoted by c_R. These values have been calculated by Bryce et al. (2001) from considerations of daily energy expenditure and food consumption. A value of 1.65 for c_G indicates that the presence of one grey squirrel exerts a crowding pressure (mediated by competition over food) on a red squirrel population that is equivalent to the presence of 1.65 red squirrels, while the value for c_R (0.61) is the inverse of this.

It was assumed that parapoxvirus is transmitted at the same rate (β) both within and between each squirrel species, because the likely route of infection is via environmental contamination (Rushton *et al.* 2000), and field evidence suggests that habitat partitioning between the two species does not occur (Kenward *et al.* 1998; Wauters *et al.* 2000). As surveys indicate that the average seroprevalence against parapoxvirus in English and Welsh grey squirrel populations (where the red squirrel is almost extinct) is 74% (Sainsbury *et al.* 2000), the rate of virus transmission was determined from simulation of the model as the value (0.7 year^{-1}) which resulted in 74% recovered individuals (R_G) in a population of grey squirrels alone. Parameter values for the mortality rate of infected red squirrels due to disease (26 year^{-1}) and the recovery rate of infected grey squirrels from disease (13 year^{-1}) were obtained from the disease dynamics of the experimental infections documented in Tompkins *et al.* (2002b). The fact that antibodies to parapoxvirus have only been detected in red squirrels found dead with clinical symptoms (Sainsbury *et al.* 2000), and the observation that all experimentally infected red squirrels are debilitated to the point at which mortality in the wild is almost a certainty (Tompkins *et al.* 2002b), indicate that recovery does not occur in this species.

Two variants of the non-spatial model were run by simulating the introduction of two grey squirrels either with or without parapoxvirus (to represent competition with and without infection, respectively) into a virus-free population of red squirrels at their carrying capacity. The parameter values used in this study ensure a switch from the virus-free red squirrel equilibrium to the virus-free grey squirrel equilibrium (if two greys without the virus are initially introduced) or the grey squirrel equilibrium with endemic infection (if two greys with the virus are initially introduced).

The investigation and comparison of the time taken for the switch between these equilibriums is the novel purpose of this work.

The spatial extension of the model was developed to approximate the set-up in the study of Reynolds (1985) in which the county of Norfolk is divided into 5-km grid squares. To achieve this we generated an 11 × 11 grid in which the population dynamics in each grid square were represented by the temporal model (eqns 1–5), with the same parameters as in the non-spatial analysis. The grid cells were then linked by dispersal. All grid squares were initially occupied by red squirrels at the carrying capacity, and two grey squirrels either with or without the parapoxvirus were introduced to two grid squares (again representing competition with or without infection, respectively). The temporal dynamics were numerically solved in each grid square for one model year before a fraction (μ) of each squirrel species was allowed to disperse to the eight nearest neighbouring cells (we imposed reflective boundary conditions). This process was then repeated for subsequent years. The dispersal fraction used (0.2) was approximated as half the maximum dispersal fraction calculated by the method of Okubo *et al.* (1989) using the parameter estimates for the grey squirrel intrinsic growth rate and carrying capacity outlined above. All parameter values are summarized in Table 1.

RESULTS

The non-spatial model demonstrated clearly that the addition of disease speeds up both the rate of decline of red squirrels and the rate at which grey squirrel numbers increase. Figure 1(a) illustrates the rate of ecological replacement that is generated when interspecific competi-

Table 1 Parameter estimates for the two-host/shared microparasite model. Carrying capacity and virus transmission are based on a 5-km square area in which woodland cover (suitable habitat for squirrels) is 6.5% with a 50/50 mix of coniferous and broadleaf trees

| Parameter	Symbol	Value	Reference
Both species			
Natural mortality rate	*b*	0.4	Gurnell (1987)
			Wauters *et al.* (2000)
Rate of virus transmission	β	0.7	This study
Dispersal fraction	μ	0.2	Okubo *et al.* (1989)
Red squirrel			
Maximum reproductive rate	a_R	1.0	Okubo *et al.* (1989)
Carrying capacity	K_R	60	Rushton *et al.* (1997)
Mortality rate due to virus	α	26	Tompkins *et al.* (2002b)
Competitive effect on grey	c_R	0.61	Bryce *et al.* (2001)
Grey squirrel			
Maximum reproductive rate	a_G	1.2	Okubo *et al.* (1989)
Carrying capacity	K_G	80	Rushton *et al.* (1997)
Recovery rate from virus	γ	13	Tompkins *et al.* (2002b)
Competitive effect on red	c_G	1.65	Bryce *et al.* (2001)
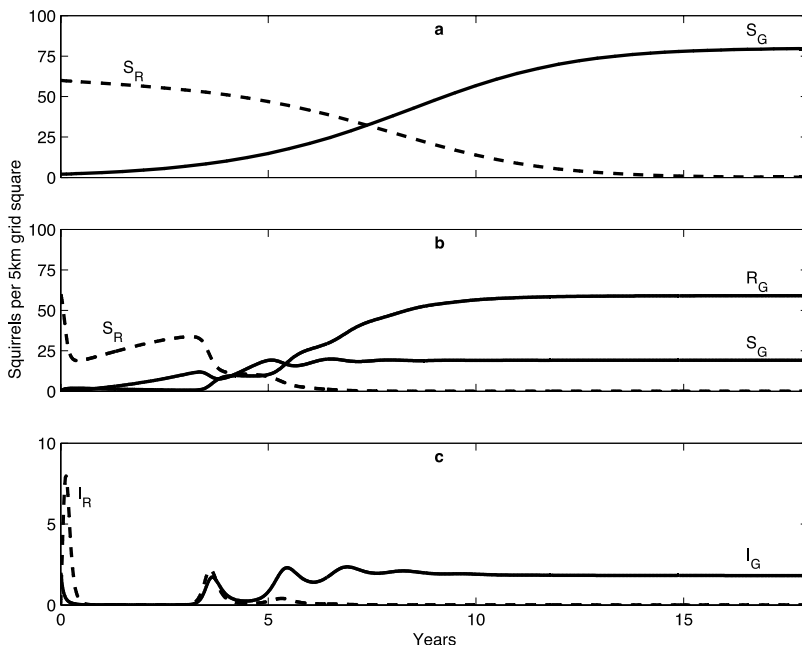
Figure 1 Results from the model eqns 1–5 showing the ecological replacement of red squirrels by grey squirrels. Solid and dashed lines represent grey and red squirrel densities, respectively. Different classes within the red and grey classification are labelled on each graph. In the competition-only model (a), the rate of red squirrel decline is relatively slow with complete replacement by the greys occurring after 15 years. In the competition and infection model (b and c) complete replacement of the red squirrel by the grey occurs within 6 years. Note (b) and (c) are from the same model output but the density scale in (c) has been expanded so the infected density classes can be observed clearly.

tion is considered in the absence of parapoxvirus. When both interspecific competition and parapoxvirus are considered (Fig. 1b,c), the importance of the disease in causing a more rapid replacement of the red squirrel population is clear.

The rate of ecological replacement generated by the spatial model, incorporating both interspecific competition and parapoxvirus, is very close to that seen in the actual Norfolk data (Fig. 2a,b). Without the virus, it is obvious that natural rates of competition alone cannot explain the red squirrel decline and the grey squirrel expansion that was observed (Fig. 2c).

Sensitivity analyses conducted on the non-spatial model indicate that our results are relatively insensitive to changes in parameter values (certainly within the ranges known from squirrel biology). Figure 3(a) illustrates the time taken for red squirrel replacement to occur for different levels of interspecific competition both with and without the virus, showing clearly that for competition alone to be responsible for the rate of decline observed, its strength would need to be considerably higher than that which is currently thought to occur. Figure 3(a) also demonstrates that the impact of the virus is relatively insensitive to the level of competition. In addition, analysis of the red squirrel replacement time over a range of grey squirrel carrying capacities (Fig. 3b) and birth rates (Fig. 3c) shows clearly that a doubling of either of these parameters is insufficient to reduce the time for competition-mediated replacement to that achieved with competition/infection-mediated replacement (using the best estimates for the other parameters). Furthermore, even if the rates of virus transmission differ within and between

species – for which there is no evidence – the disease is still important within a wide range of parameter values (Fig. 3d).

A sensitivity analysis of the spatial model when the dispersal fraction parameter is varied is shown in Fig. 4. The difference between the replacement time with competition-only and that with competition and infection is near constant for all values of the dispersal fraction. This indicates that differences between replacement times in the two set-ups (with and without the virus) are generated by the temporal dynamics. The replacement time is also relatively insensitive to a doubling or halving of the dispersal rate.

DISCUSSION

This study indicates that parapoxvirus has potentially played a crucial role in the UK red squirrel decline. While the model incorporating only interspecific competition in the absence of parapoxvirus could not account for the rate and pattern of replacement by the grey squirrel that has occurred in Norfolk, the model incorporating both factors produced a very close fit to the available data (Fig. 2). Our output from the competition-only model is in line with observations from the previous studies of Okubo *et al.* (1989) and Rushton *et al.* (1997), who also noted an unrealistically slow rate of replacement of red squirrels by grey squirrels under the levels of competition that have been recorded in the wild. The biological explanation for the enhanced, disease-induced, replacement is intuitive. Upon the invasion of infected grey squirrels into a disease-free population of red squirrels, the rate of infection of red squirrels is high as
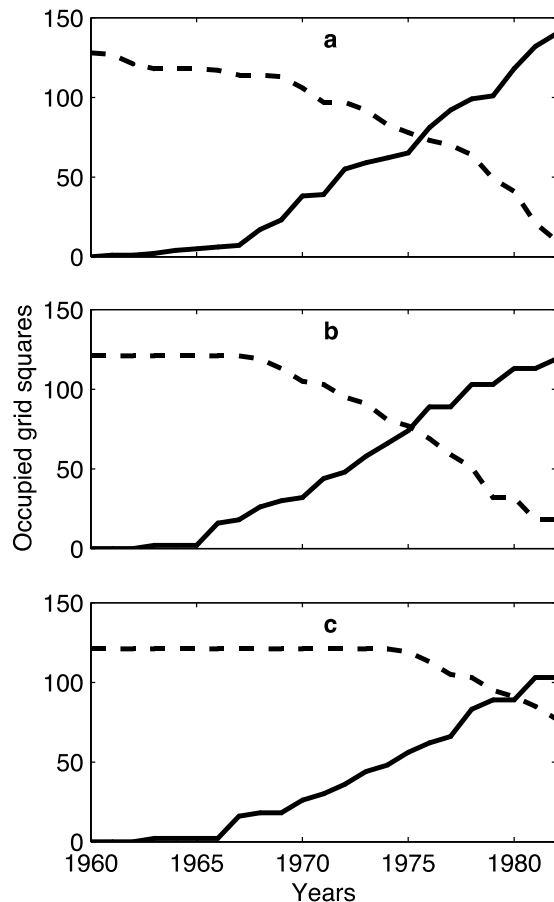
Figure 2 Results from the spatial model compared with the actual red squirrel decline (dashed line), and grey squirrel expansion (solid line), observed in Norfolk (England) from 1960 to 1982 (Reynolds 1985). The actual number of 5-km grid squares occupied by red and grey squirrels over this period is shown in (a). The output of the competition and infection model (b) closely fits the actual ecological replacement observed. The output from the competition-only model (c) shows a slower decrease in the number of grid squares occupied by red squirrels.

a large number of susceptible individuals are initially present (Fig. 1c). This induces the red squirrel population crash, reducing the crowding pressure (mediated by competition over food) on the invading grey squirrels which can subsequently increase in numbers more rapidly than in the competition-only scenario. Our model therefore indicates that the ecological replacement of red by grey squirrels in the UK can only be understood in light of an interaction between competition and parapoxvirus. In addition, the prevalence of infection in our model is low (Fig. 1c), although the role of disease is critical in determining the dynamics observed. This fits the expected pattern (Anderson & May 1978), and highlights how observational studies may incorrectly disregard the role of disease.

Although our model clearly indicates that competition alone (affecting reproduction) between the two squirrel species could not have caused the rate and pattern of replacement observed in Norfolk, there may be additional competitive interactions that have occurred in other parts of the UK. Specifically, in years of good acorn crops it is possible for grey squirrels to reach very high densities that can destroy entire hazel crops (an important food resource for red squirrels), and therefore reduce red squirrel survival (Kenward & Holm 1993; Kenward et al. 1998). Such effects are unlikely to have occurred during the ecological replacement in Norfolk because, while coniferous habitat occurred in large plantations at this time, most broadleaf habitat was relatively fragmented (Reynolds 1985). Under such circumstances, the density boost to grey squirrels of good acorn crops would be limited.

The sensitivity analyses conducted here clearly demonstrate that the fundamental message of this study (that disease is likely to have played a role in the replacement of red by grey squirrels in the UK) is not dependent on the parameters chosen for the model. Most importantly, our model suggests that even if the competitive effect of grey on red squirrels is indeed greater, parapoxvirus still greatly accelerates the process of replacement. The combination of using the best objective parameter estimates available and the fit of the model to actual data using these parameters, gives much credence to the conclusion that parapoxvirus has been a crucial factor in the UK red squirrel decline. This result has wide-ranging implications for the conservation of the remaining UK red squirrel population. Specifically, over the last few decades, the planting of a large coniferous forest has connected groups of forest fragments in the north of England with those in southern Scotland. This defragmentation of habitat is generally considered a good thing as it has resulted in a substantial genetic mixing of Scottish and English squirrel populations (Hale et al. 2001). However, as parapoxvirus has yet to be recorded in Scotland (Sainsbury et al. 2000), and increased habitat connectivity can also increase the risk of pathogen transmission (Hess 1996; Rodriguez & Torres-Sorando 2001), such defragmentation may ultimately do more harm than good. Hence, the conservation implications of this landscape management practice – if such habitat change increases the potential for parapoxvirus transmission – clearly need to be reassessed.

Invasions of exotic species and their subsequent impact on native flora and fauna are key issues in conservation biology. Understanding the mechanisms that underpin these invasions is clearly of central importance, and therefore stands as one of the major challenges of ecological research. As demonstrated here, pathogens may play a greater role than is often currently thought. Complacency in the livestock industry with regard to disease matters has lead to problems such as the recent Bovine Spongiform
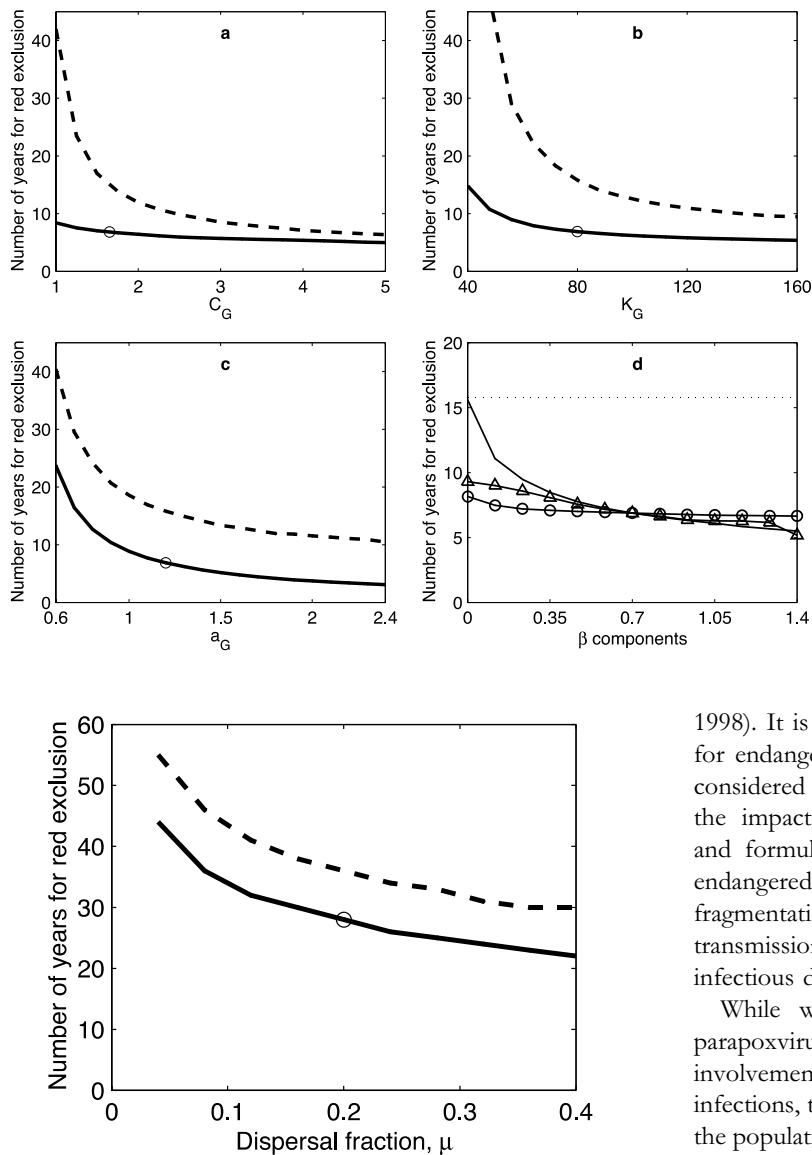
Figure 3 Time for the red squirrel population to reach 1% of K_R, using eqns 1–5, for both the competition-only simulation (dashed line) and the competition and infection simulation (solid line). The parameters are as in Table 1, except (a) C_G (and $C_R = 1/C_G$) is varied, (b) K_G is varied, (c) a_G is varied, and (d) β components are varied. For (d), β was decomposed to represent the separate intraspecific and interspecific interactions (β$_{RG}$, solid line; β$_{GR}$, circles; β$_{RR}$, triangles), i.e the term containing beta in eqn 1 becomes $-\beta_{GG}S_G I_G - \beta_{GR}S_G I_R$ and the exact term but with the opposite sign appears in (2), and the term in eqn 4 becomes $-\beta_{RR}S_R I_R - \beta_{RG}S_R I_G$ and with the opposite sign in (5). The circles in (a)–(c) represent the parameters used in simulations for Figs 1 and 2 (Table 1).

Figure 4 Results from the spatial model comparing the number of years for red squirrel exclusion, in both the competition-only simulation (dashed line) and competition and infection simulation (solid line), with the dispersal fraction, μ. All parameters are as in Table 1 with the circle indicating the value of μ used in simulations for Figs 1 and 2.

Encephalopathy and foot and mouth outbreaks. One may argue that such complacency would be even more costly to wildlife conservation. Unlike the recent foot and mouth epidemic in the UK, where culled livestock could be replaced from outside sources, such replenishment is not so easy to carry out when wildlife species are lost. For example, in the case of the red squirrel, releases of individuals into areas of the UK where the species has already disappeared have not resulted in re-introduction (Kenward & Hodder

1998). It is thus essential, not only for the red squirrel but for endangered species in general, that disease matters are considered more thoroughly than at present when assessing the impact of species introduction and habitat change, and formulating management plans. Actually maintaining endangered species in a state of relative population fragmentation, thus limiting opportunities for pathogen transmission, may even be desirable in instances where infectious disease is deemed to be a major threat.

While we have demonstrated the potential role of parapoxvirus in the UK red squirrel decline, its actual involvement is still to be proven. For most microparasite infections, the best available evidence for a regulatory role at the population scale of their hosts is obtained by monitoring the natural (and human induced) introduction of pathogens into areas where host populations can be carefully monitored and subsequent reductions in density detected (Tompkins *et al.* 2002a). Such a 'natural experiment', providing a test for the role of parapoxvirus, will occur if the defragmentation of forest habitat does indeed allow the virus to spread from northern England to southern Scotland. Any increase in the rate of ecological replacement coinciding with such an event (demonstrable as the appearance of either clinical disease in the resident red squirrels or antibodies to parapoxvirus in the resident grey squirrels) would provide strong evidence for a role of the virus in the ongoing red squirrel decline, as long as other factors (habitat structure, emigration and immigration rates) could be shown to remain constant. No change in the rate of replacement would prove that the virus was not involved at the population scale.

Although mathematical models are unable to prove the role of specific factors in determining host population dynamics, they are invaluable tools in modern ecology and conservation biology. It is increasingly being recognized that wildlife populations exhibit a large degree of context dependency, with their dynamics often determined by interactions between a variety of both biotic and abiotic factors (Blaustein & Kiesecker 2002). Under such circumstances the use of models like the one presented here, to tease apart complex interactions and identify mechanisms that may not be intuitively obvious, is a fundamental way in which our understanding can be increased. Such models can then provide the basis for the design and implementation of experimental work to provide the necessary proof (see, for example, Hudson *et al.* 1998).

ACKNOWLEDGEMENTS

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