

# Disease persistence and apparent competition in a three-host community: an empirical and analytical study of large-scale, wild populations

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## Summary

1. We investigated the effects of three types of host on the persistence of a tick-borne virus, using the grouse–hare–deer–tick–louping ill virus system of upland Britain. Each host differed in its interaction with the vector and pathogen. Grouse amplify virus only, deer amplify vector only and hares amplify both. Grouse alone suffer high virus-induced mortality.
2. An analytical model of the system was parameterized using empirical data from two wild populations with different community structures. By changing relative host densities we examined the conditions under which the virus would persist and considered the possibility of parasite-mediated competition between hosts.
3. Although deer alone and grouse alone were unable to maintain louping ill virus, a deer–grouse community usually allowed virus persistence because grouse transmitted virus while deer maintained the tick population. Since virus reduces grouse populations this is a type of apparent competition, and is unusual because deer do not amplify the virus.
4. At very high deer densities, the opposite effect could occur, whereby virus died out because of ‘wasted’ infected tick bites on deer, that do not transmit virus (the dilution effect).
5. In a hare–grouse two-host system virus usually persisted because hares amplified both the vector and virus (through non-viraemic transmission). Thus, apparent competition may occur between mountain hares and grouse.
6. The addition of a third host type increased the likelihood of disease persistence. Hares added to the deer–grouse system rendered the dilution effect unlikely because of additional virus amplifiers. Deer added to the hare–grouse system meant virus almost always persisted because they amplified the vector.

*Key-words:* louping ill virus, model, mountain hares, red deer, red grouse.

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## Introduction

While it is axiomatic that most pathogens reduce the survival or fecundity of their hosts, only recently have field studies started to demonstrate that they play an important role in determining the dynamics of natural

populations (Hudson, Dobson & Newborn 1998). In simple single-host systems, the final outcome of a parasite–host interaction is determined by the virulence and transmission efficiency of the parasite. In multi-host systems the dynamics can be completely different: apparent competition between hosts may operate through the action of shared parasites and cause changes in community structure (Holt 1977; Holt & Pickering 1985; Holt & Lawton 1994; Bonsall & Hassell 1997, 1999; Hudson & Greenman 1998). In

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general, the more susceptible host species may become eliminated because the parasite increases in the less susceptible reservoir host (e.g. Tompkins, Draycott & Hudson 2000). Alternatively, the pathogen may suppress the population of the competitively dominant host, thereby reducing direct competition with the subordinate hosts and resulting in increased biodiversity (Sih *et al.* 1985).

There has been an increasing theoretical interest in parasite-mediated apparent competition in recent years (Yan 1996; Bowers & Turner 1997; Greenman & Hudson 1997, 1999; Norman *et al.* 1999) following the initial modelling of this by Holt & Pickering (1985). Both theoretical and empirical studies of vector-borne pathogen-mediated apparent competition have concentrated primarily on systems with two types of hosts, such as the tick-transmitted Lyme disease system (Ostfeld 1997; Ostfeld & Keesing 2000a,b) that involves white-tailed deer (*Odocoileus virginianus* L.), and white-footed mice (*Peromyscus leucopus* L.). However, little attention has been given to more complex host communities such as the interesting scenario of a three-host vector–pathogen community, which is important when many vectors, especially ticks, are generalist feeders using a range of host species.

Community structure can affect the persistence of the disease just as diseases can affect community structure. Indeed, for the improvement of human health, farming and conservation one of the principal challenges in epidemiology is to determine how variations in host community structure influences the dynamics of diseases (van Buskirk & Ostfeld 1995; Giardina *et al.* 2000; Ostfeld & Keesing 2000a,b). In this study, we apply both a theoretical and empirical approach to explore how a three-host community affects the dynamics of a tick-borne virus, louping ill, in wild host populations. What is important to disease dynamics is not the number of host species *per se*, but the types of host that differ in their reservoir competence (e.g. Ostfeld & Keesing 2000a). For tick-borne diseases in particular, there can be a variety of host types amplifying the vector, the pathogen, or both, and pathogen transmission may be viraemic or non-viraemic (Labuda *et al.* 1993; Jones *et al.* 1997). There is a need to understand the precise role that these different host types play in disease persistence. More specifically, then, this study addresses the question: how does community structure and host type influence the persistence of louping ill virus, and does this result in apparent competition between hosts?

Louping ill virus is transmitted by the sheep tick (*Ixodes ricinus* L.) and causes 78% mortality in infected red grouse (*Lagopus lagopus scoticus* Lath.) and variable mortality, depending on farming practices, in sheep (*Ovis aries* L.; Reid 1975; Reid *et al.* 1978; Hudson 1992). Larval and nymph stages of the sheep tick feed on a large range of vertebrate species, whereas adults require a large mammalian host (for detailed life history see Lees & Milne 1951). The significance of

this life cycle is that grouse alone cannot sustain tick populations because they rarely host adult ticks. Although *trans*-stadial transmission of louping ill virus occurs in ticks, *trans*-ovarial transmission has not been recorded in *I. ricinus*, so unengorged larval ticks are not infected (Hudson *et al.* 1997). Hence, only nymphs and adult ticks carry virus.

Within the community of hosts that support louping ill virus only sheep and red grouse produce a sufficient viraemia to infect ticks (Reid & Doherty 1971; Reid 1975), although sheep can be effectively removed as hosts by vaccination against louping ill virus and the use of acaricides (Laurenson *et al.* 2000). Mountain hares (*Lepus timidus* L.) can be instrumental in the persistence of louping ill virus (Hudson *et al.* 1995; Norman *et al.* 1999) since they permit the transmission of virus non-viraemically between cofeeding ticks (Jones *et al.* 1997). Cofeeding trials conducted on rabbits (*Oryctolagus cuniculus* L.), small mammals and red deer (*Cervus elephus* L.) found no evidence of either viraemic or non-viraemic transmission (Jones *et al.* 1997; Gilbert *et al.* 2000). However, red deer may be important in the system because, being large and abundant, they can feed a significant proportion of the tick population (e.g. Gilbert *et al.* 2000).

Thus, in upland Britain there are three major types of tick host that vary according to the potential for amplifying louping ill virus. First, grouse that transmit the virus viraemically, but alone cannot maintain the vector population. Secondly, red deer that do not amplify virus either through viraemic or non-viraemic routes but maintain large vector populations. Thirdly, mountain hares that can both transmit virus non-viraemically and feed all stages of the tick population. As such, we can predict that grouse alone and deer alone should not be able to sustain the virus. However, there remains the interesting possibility that in combination their effect may cause the basic reproductive rate of the virus to exceed unity, and louping ill will persist. Moreover, when mountain hares are added to the system, can this non-viraemic transmission host alter the population dynamics of the grouse through apparent competition? We examine these possibilities by using both empirical field data and an analytical framework, and ask how changes in the relative densities of the different types of host influence disease persistence.

## Methods

Our approach was to develop, first, an analytical model for the tick-louping ill virus system where red grouse, mountain hares and red deer were the hosts; secondly, to parameterize the model using field data and previously published estimates; thirdly, to validate the model by comparing the predicted values of grouse densities and louping ill virus sero-prevalences with empirical data; and finally, to use the model to predict whether virus persists under different host combinations and densities.

MODEL OF THE GROUSE-HARE-DEER-TICK-  
LOUPEING ILL SYSTEM

The dynamics of infection in the total grouse population ( $G$ ) can be described by changes in susceptible ( $G_s$ ), infected ( $G_i$ ) and immune ( $G_z$ ) grouse densities; in the total tick population ( $T$ ) by describing infected ( $T_i$ ) and susceptible ( $T_s$ ) tick densities. We assumed that, for each scenario examined, deer ( $D$ ) and mountain hare ( $H$ ) densities are at equilibrium through control by game keepers. We changed the densities of each host in the model in order to explore different scenarios that reflect variations between areas. The dynamics of the system were described by the following equations:

$$\frac{dG_s}{dt} = A + (a_g - sG)G - b_g G_s - \beta_1 T_i G_s$$

$$\frac{dG_i}{dt} = \beta_1 T_i G_s - (\alpha + b_g + \gamma) G_i$$

$$\frac{dG_z}{dt} = \gamma G_i - b_g G_z$$

$$\frac{dT_s}{dt} = (a_T - S_T)T(\beta_3 H + \beta_4 D) - b_T T_s - \beta_2 T_s G_i - \beta_3 T_s H - \beta_4 T_s D$$

$$\frac{dT_i}{dt} = \theta H T_s + \beta_2 T_s G_i - b_T T_i - \beta_3 T_i H - \beta_4 T_i D$$

where  $A$  is the grouse immigration rate, which reflects the immigration that occurs to replace grouse in areas where virus causes significant mortality (Hudson 1992; Hudson *et al.* 1997);  $a_g$  is the fecundity of grouse;  $s$  is a measure of the density dependence acting on the birth rate of the grouse;  $b_g$  is the death rate of the grouse;  $\alpha$  is the pathogen-induced death rate and  $\gamma$  is the rate at which grouse recover into the immune class. The  $\beta$  terms are combinations of tick biting and transmission rates (e.g. Sandberg, Awerburg & Spielman 1992) and are linear functions (see Dye & Williams 1995).  $\beta_1$  is the rate at which infected ticks (nymphs) bite susceptible grouse and infect them;  $\beta_2$  is the rate at which susceptible ticks (larvae and nymphs) bite infected grouse and become infected;  $\beta_3$  is the rate at which adult female ticks bite hares and then reproduce; and  $\beta_4$  is the rate at which adult female ticks bite deer and then reproduce.  $\theta$  is the non-viraemic transmission coefficient and represents the rate at which susceptible ticks become infected through cofeeding with infected ticks on hares. For the tick equations  $a_T$  is the tick birth rate;  $s_T$  is a measure of the density-dependent constraints acting on tick birth rates and  $b_T$  is the tick death rate. These parameters are explained in more detail below.

Equilibrium stability analyses of this model show that there are two important biological thresholds, one which allows the ticks to persist ( $R_{o,ticks} > 1$ ) and one which allows the disease to persist ( $R_{o,virus} > 1$ ) where  $R_o$  is the reproductive rate of the pathogen or vector, such that:

$$R_{o,ticks} = \frac{a_T(\beta_3 H + \beta_4 D)}{b + \beta_3 H + \beta_4 D} \text{ and}$$

$$R_{o,virus} = \frac{\theta H K_T \Gamma + \beta_1 \beta_2 K_T K_g}{\Gamma(b_T + \beta_3 H + \beta_4 D)}$$

where  $\Gamma = \alpha + b + \gamma$ ,  $K_g$  is the carrying capacity of grouse, given by the positive root of  $sK_g^2 - rK_g - A = 0$  where  $r$  is the grouse population growth rate (i.e.  $a_g - b_g$ ), and  $K_T$  is the carrying capacity of ticks, given by:

$$K_T = \frac{(a_T - 1)(\beta_3 H + \beta_4 D) - b_T}{s_T(\beta_3 H + \beta_4 D)}$$

To capture the effects of different combinations of hosts on disease persistence the model was run under various scenarios. First, with varying densities of grouse and deer only; secondly, with grouse and hares only; thirdly, with varying densities of grouse and hares but with a constant deer density; and fourthly, with varying densities of all three hosts.

PARAMETER ESTIMATION

The model's parameters were estimated using previously published data and empirical data collected during this study (summarized in Tables 1 and 2). Each parameter is expressed per individual host or vector per month and/or per km<sup>2</sup> unless stated otherwise.

Study sites

Data were collected from four Scottish estates, each with different densities of the three relevant hosts (Table 1). At site M1 (in Morayshire), keepers have reduced mountain hare numbers from 8 km<sup>-2</sup> in 1993 to 0 in 1998. We used the 1993 data assuming that numbers of grouse and hares were at equilibrium at that time. There were no red deer on this estate, and in 1993 the grouse density was low. The second estate, in Caithness (site C) had a very high red deer density, a low grouse density, and no mountain hares. The third estate, in Perthshire (site P), had intermediate densities of all three hosts, and the fourth estate (site M2 in Morayshire) was similar in community structure to site M1. Sites M1 and C were used to obtain parameters for the model, and sites M2 and P for validation of the model.

At all four study sites grouse and hare densities were estimated by conducting counts on 1-km<sup>2</sup> plots with

**Table 1.** Empirical data from each study site on each host, used to generate expected results from the model. Grouse carrying capacity ( $K_g$ ) was estimated from the highest numbers in July counted on the estate. For site M1 this was in 1998, site C 1998, site P 1987 and site M2 1996

Site	Grouse carrying capacity (km <sup>-2</sup> )	Hare density (km <sup>-2</sup> )	Deer density (km <sup>-2</sup> )
M1	130	8	0
C	30	0	10
P	140	4	1
M2	93	6	0

**Table 2.** Comparison of empirical data with values predicted from the model for sites M1 and C which were used to parameterize the model, and for sites M2 and P which were used as validation sites. The predicted values are those produced when  $\beta = 0.0002$ ,  $\theta = 3.9 \times 10^{-7}$  and  $s_T = 0.00139$ . Empirical data were collected in 1993 for site M1, 1998 for site C 1985–2000 for breeding grouse at site P and 1993–96 and 1999 for seroprevalence at site P 1992–96 for breeding grouse at site M2 and 1998 for seroprevalence at site M2

Site	Equilibrium density of breeding grouse (km <sup>-2</sup> )		Seroprevalence in young grouse (%)	
	Predicted	Empirical	Predicted	Empirical
Site M1	9.0	9.5	80.0	81.8
Site C	29.4	24.1	0.5	0.0
Site P	23.6	11.0–52.0	41.7	7.1–26.1
Site M2	17.6	14.6–40.0	52.0	46.0

trained pointer dogs using standard methods (Hudson & Newborn 1995), and red deer density was estimated from known resident deer numbers in a known area.

Louping ill virus prevalence was measured by taking blood samples from shot red deer, mountain hares and young grouse, and then conducting standard haemagglutination-inhibiting antibody (HIA) tests on the sera (Clark & Casals 1958).

Ticks were counted on live red grouse chicks in June and July, on recently shot red deer from July to September, and on recently shot mountain hares in August. Hares were placed in sealed bags immediately after death to contain any ticks that might leave the dead host but this was not possible for deer, so tick counts on deer were a minimum. Larvae and nymphs were not easy to count accurately on red deer due to their large body size and the large numbers of ticks they carry. However, since hare and deer are both large mammal hosts that share the same habitat and locations, we assumed that tick life stage ratios on deer were similar to those on hares. Therefore, to estimate larvae and nymph tick biting rates on red deer, we extrapolated from the known adult tick counts on deer, using larvae: nymph: adult ratios counted on mountain hares at site M1.

Each site was assumed to have identical parameter values except where indicated, and except in their relative host densities (Table 1).

#### *Per capita grouse birth rate ( $a_g$ )*

Grouse production varied between areas and between years but, on average, they produce four young per pair per year (Hudson 1992), equivalent to 0.167 per month per individual (assuming a 1 : 1 sex ratio).

#### *Per capita grouse death rate ( $b_g$ )*

Approximately 35% of the grouse population on a managed grouse moor remain alive at the end of the year (Hudson 1992). Assuming populations die exponentially over time,  $0.35 = e^{-12b}$  then  $b$  is 0.087, i.e. this fraction of a grouse dies per month to gain the death rate for a year.

#### *Grouse immigration rate ( $A$ )*

At site C the grouse density was very low, and neighbouring areas carried very few grouse, so we assumed the immigration rate to be zero. At sites M1, M2 and P, however, which have louping ill virus and are therefore 'sink' rather than 'source' grouse populations, immigration almost certainly occurred from elsewhere (Hudson *et al.* 1997) and was necessary in order to account for the high level of virus prevalence observed in the field. Using the known grouse equilibrium densities from site M1, we estimated  $A$  such that the proportions of immune, susceptible and infected grouse were correct. From this,  $A = 1.94$ , and is assumed the same for sites M2 and P.

#### *Per capita grouse death rate due to disease ( $\alpha$ )*

Laboratory studies suggest that grouse die approximately 6 days after louping ill virus infection (Reid 1975; Reid *et al.* 1978). In other words, every 6 days the death rate is 1, so the equivalent over a 30-day month is 5. This applies only to those grouse that become infected and die and another parameter is needed for those that recover ( $\gamma$ ).

#### *Per capita grouse recovery rate from disease ( $\gamma$ )*

Laboratory experiments have shown that up to 80% of grouse die when infected with louping ill virus (Reid 1975; Reid *et al.* 1978). Therefore, four times more grouse die than recover from the disease, so the recovery rate is  $\alpha/4 = 1.25$ .

#### *Density-dependent constraints acting on grouse ( $s$ )*

We have used the standard simple logistic form of density dependence acting on the birth rate (e.g. Bowers, Begon & Hodgkinson 1993), which is related intrinsically to grouse numbers through the equation  $sK_g^2 - rK_g - A = 0$ . For each scenario explored,  $s$  is calculated to give the observed  $K_g$ ;  $r$  and  $A$  do not vary between sites.

*Per capita tick reproductive rate ( $a_T$ )*

Female *I. ricinus* generally produce 3–6000 eggs, although hatching success is unknown (Wilson 1994). However, to provide a relatively arbitrary estimate, we assumed an egg survival rate similar to that of *I. dammini* larvae, which is an estimated one-third (Schulze *et al.* 1986) to one-fifth (Carey, Krinsky & Main 1980). Hence, we estimated that 1000 larvae could be produced per female in a year; per month this is 83.33.

*Per capita tick death rate ( $b_T$ )*

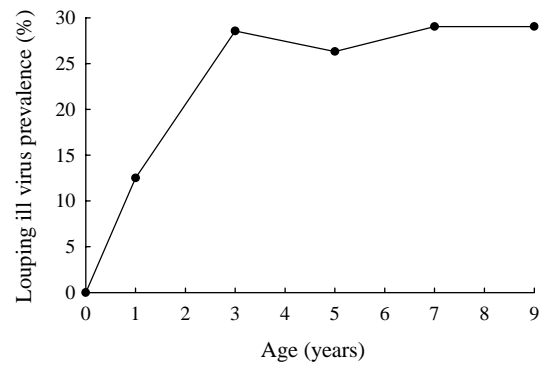
Each tick stage can live for 1 year (e.g. Lees & Milne 1951), and we assumed a tick will die if it fails to feed after this time. Per month, therefore, the death rate of ticks that do not feed is  $1/12 = 0.0833$ .

*Density-dependent constraints acting on ticks ( $s_T$ )*

High tick densities may cause higher tick mortality and lower female tick fecundity (from reduced engorgement: Wilson *et al.* 1990) due to more intense grooming by hosts or increased competition for prime attachment sites. Thus, density dependence is likely to act on both tick birth and death rates. However, for mathematical simplicity, we used a density dependence function which acts on the birth rate and fits the standard logistic form (e.g. Bowers *et al.* 1993). Due to a lack of empirical information, we used the model to estimate a value for  $s_T$  such that predicted host densities and virus prevalences fitted the empirical data for sites M1 and C. Table 2 shows these empirical data and the values predicted by the model when  $s_T = 0.00139$ .

*Transmission constant ( $\beta$ )*

This is the biting rate of infected nymphs on grouse. We used a crude method to estimate  $\beta$  from data of disease prevalence and tick biting rates on deer and grouse at site C as follows. The gradient of age prevalence curves can be used as an estimate of the force of infection in natural populations (Hudson & Dobson 1997; McCallum 2000). In red deer hinds at site C this is estimated to be 0.1 per year, or 0.0083 per month (Fig. 1). To convert this into the infected nymph biting rate on grouse, we need the ratio of tick bites on deer to nymph bites on grouse. For simplicity, and because we do not know how much more infective adult ticks are than nymphs, we made a null assumption that adults and nymphs have the same infectivity. Tick counts on 98 stags between July and September in 1998 and 1999 showed that a mean of 82 adults and 345 nymphs, i.e. 427 potentially infectious ticks bite deer at any one time. Tick counts on 11 grouse chicks in July at the same site revealed that a mean of 9.3 nymphs bite grouse at any given time (adults generally do not bite birds). This gives a deer : grouse tick biting ratio of  $427/9.3 = 45.9$ . Therefore, the estimated infected tick biting rate on



**Fig. 1.** Age-louping ill virus seroprevalence curve of 99 red deer hinds at site C. The force of infection, used in the derivation of the transmission coefficient,  $\beta$ , is the gradient of the curve, and in this case is approximately 0.1 per year, or 0.0083 per month.

grouse per month is  $0.0083/45.9 = 0.0002$ . Since it is known to be difficult to estimate  $\beta$  accurately in natural populations (McCallum 2000) we also used, as an alternative method, the model to provide a value for  $\beta$  (as for  $s_T$ ). Since there are four tick biting/transmission parameters ( $\beta_1$ ,  $\beta_2$ ,  $\beta_3$  and  $\beta_4$ ), we assumed that they were linearly related, i.e. ratios of larvae: nymphs were assumed to be constant between different hosts. The estimate for  $\beta$  with these assumptions was again 0.0002.

*Transmission rate from infected ticks to grouse ( $\beta_1$ )*

This is the rate at which susceptible grouse are bitten by infected nymphs and is taken as  $\beta_1 = \beta$ .

*Transmission rate from infective grouse to ticks ( $\beta_2$ )*

This was estimated from the biting rate of susceptible larvae and nymphs on grouse chicks at site C in 1998 and 1999. There was a mean of 9.3 nymphs and 81.3 larvae per chick ( $n = 18$  chicks) so for every nymph there were 8.75 larvae. Since  $\beta$  is the biting rate of infected nymphs we relate all other  $\beta$  parameters to it such that, for nymphs + larvae  $(1 + 8.75)$  biting a grouse,  $\beta_2 = 9.75\beta$ .

*Rate at which adult ticks bite hares and reproduce ( $\beta_3$ )*

This can be considered one of the tick (rather than virus transmission) parameters along with  $a_T$ ,  $b_T$  and  $s_T$ . Assuming that all fed adult ticks reproduce,  $\beta_3$  was estimated from counts of adult female ticks feeding on 30 mountain hares at site M1 in August 1993, and compared to the nymph biting rate on grouse in the same year at the same site ( $\beta$ ). A mean of 16.53 adult ticks was counted per hare, compared with 9.0 nymphs per grouse chick. Therefore,  $\beta_3 = 1.84\beta$ .

*Rate at which adult ticks bite deer and reproduce ( $\beta_4$ )*

As for  $\beta_3$ , this can be considered a part of the tick

parameters.  $\beta_4$  was estimated from counts of adult female ticks feeding on red deer stags at site C in 1998, and related to the nymph biting rate on grouse in the same year at the same site ( $\beta$ ). A mean of 82 adult ticks was counted per stag ( $n = 19$  stags), compared with 9.3 nymphs per grouse chick, so  $\beta_4 = 8.82\beta$ .

#### Non-viraemic transmission coefficient ( $\theta$ )

This is the probability that an uninfected tick co-feeding with an infected tick on a mountain hare will become infected through non-viraemic transmission. This cannot be estimated from current empirical information; thus, we used the model, with  $\beta = 0.0002$ , to provide a value for  $\theta$  such that predicted host densities and virus prevalences matched the empirical data of sites M1 and C (Table 2). Using this method,  $\theta = 3.9 \times 10^{-7}$ .

#### Validation data

To provide some measure of the validity of the model, we generated predicted values of the equilibrium densities of grouse and virus sero-prevalences, and compared these with empirical data from site P and site M2, which were not used for parameterizing the model.

## Results

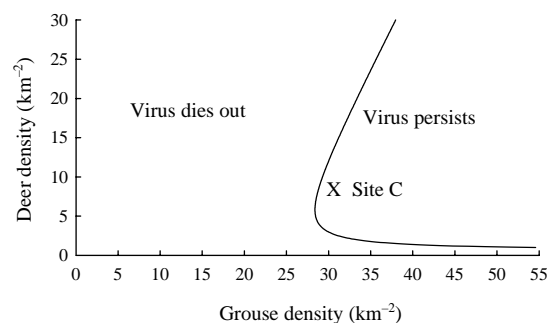
### PREDICTIONS FROM THE MODEL

#### Validation data

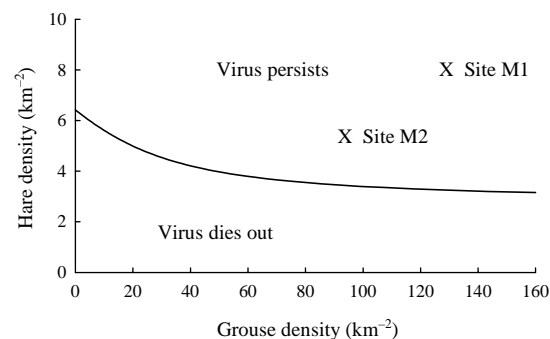
Sites M2 and P (which were not used to parameterize the model) were used as an indication of the model's validity, and comparisons of empirical data (from a range of years) with values predicted from the model are shown in Table 2. The model predicted values for the equilibrium density of grouse within the empirical range of both sites M2 and P. The predicted value of virus seroprevalence in young grouse was close to the empirical value for site M2, but over-estimated for site P. This discrepancy suggests that our assumption that the parameters are similar between sites may not always be true. For example, grouse immigration rates may differ between sites, depending on the relative densities of grouse on adjacent populations. Tick life history and grouse and tick density dependent regulatory factors may differ between sites due to differences in microclimate and habitat. However, changing the parameter values does not alter the qualitative pattern of persistence (i.e. the shape of the  $R_0 = 1$  curve in Figs 2–5), but merely changes the quantitative host densities (i.e. the values on the axes in Figs 2–5).

#### Pathogen persistence with grouse and deer only

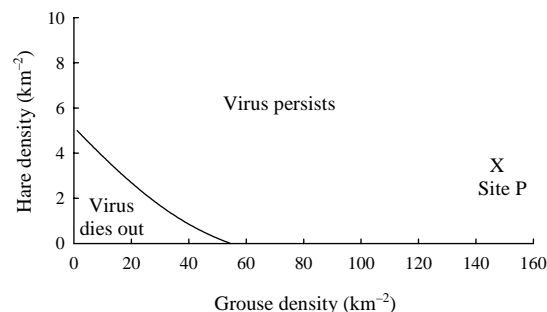
Where grouse and red deer were the only hosts several combinations of host densities could lead to the virus persisting or dying out (Fig. 2). At low deer densities



**Fig. 2.** Louping ill virus persistence in the model scenario of grouse and red deer as the only community hosts. The line represents  $R_0 = 1$ , such that within the curve virus persists, and outside it virus dies out. The position in parameter space of site C is indicated.

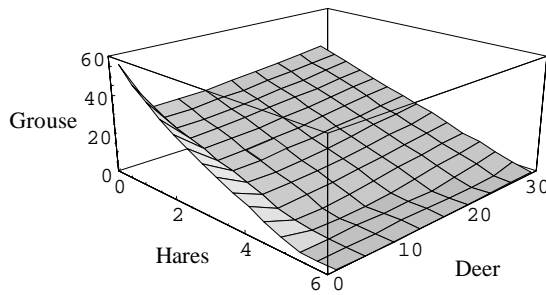


**Fig. 3.** Persistence of louping ill virus estimated from the model of grouse and hares in the absence of red deer. The line represents  $R_0 = 1$ , such that above the curve virus persists, and below it virus dies out. The positions in parameter space of sites M1 and M2 are indicated.



**Fig. 4.** Persistence of louping ill virus from the model of constant red deer density in relation to changing hare and grouse densities in a three-host-type community. The line represents  $R_0 = 1$ , such that above the curve virus persists, and below it virus dies out. The position in parameter space of site P is indicated.

the virus could not persist, since there were then too few hosts for adult ticks to maintain the tick population. At extremely high deer densities the virus tended to die out due to a 'dilution' effect caused by infected ticks 'wasting' bites on deer, an incompetent transmission host. However, at more realistic deer densities the disease was likely to persist due to the combined effects of deer amplifying the tick population and grouse transmitting virus.



**Fig. 5.** Three-dimensional representation of the plane of  $R_0 = 1$  for a range of densities of red deer, mountain hare and red grouse ( $k_g$ ). Louping ill virus persists above the  $R_0 = 1$  plane, and dies out below it.

#### *Pathogen persistence with grouse and hares only*

In the case of a two-host community of grouse and hares the model predicted that, with low hare densities, virus could not persist since grouse cannot sustain the tick population. However, in the absence of grouse a density of more than six hares  $\text{km}^{-2}$  could still sustain the disease through non-viraemic transmission between cofeeding ticks (Fig. 3).

#### *Pathogen persistence in a three-host-type community*

In the presence of all three types of host, the virus almost always persisted (Fig. 4). Only very low densities of both grouse and hares resulted in the disease dying out due to low rates of transmission. Even in the absence of hares the disease persisted because the deer maintained the tick population while the grouse transmitted the virus. In the absence of grouse a density of at least five hares  $\text{km}^{-2}$  could maintain the disease through non-viraemic transmission. This emphasizes the importance of red deer in the role of primary vector host (compare Figs 3 and 4).

Finally, we allowed all three hosts to vary in density and explored how louping ill virus could persist under a wide range of realistic relative host densities, and represented the results on a 3-dimensional graph (Fig. 5). An increase in hares corresponded with an increase in the likelihood of virus persistence, as indicated by more parameter space above the persistence threshold,  $R_0 = 1$  in Fig. 5. At zero densities of deer and hares virus always died out even with high numbers of grouse, as shown by all parameter space being below the persistence threshold at this point. Tracking the  $R_0 = 1$  threshold from this point through increasing deer density shows that virus persistence is predicted to be most likely at a deer density of  $5 \text{ km}^{-2}$ , and becomes less likely thereafter through the dilution effect.

## Discussion

Using an empirical model of the louping ill virus system of upland Britain, we asked how changes in the relative densities of three types of host influenced

pathogen persistence, and whether apparent competition could exist between hosts. Hosts amplified either vector only (deer), virus only (grouse) or both (mountain hares).

Chaneton & Bonsall (2000) suggested that parasite-mediated apparent competition is almost always asymmetrical between hosts. In our hare–grouse two-host-type system, hares were detrimental to grouse because they not only increased vector numbers, but also amplified virus through non-viraemic transmission. In other words, parasite-mediated apparent competition is deemed to occur between mountain hares and grouse and there is extreme asymmetry caused by louping ill virus, which kills up to 80% of infected grouse but has no apparent impact on hares. We also suggest the presence of apparent competition between red deer and grouse in Scotland (again with extreme asymmetry). This is an interesting and unconventional type of parasite-mediated competition where one host does not transmit the pathogen itself but amplifies only the vector population. As a result, the susceptible host population becomes damaged by the pathogen because of the increase in vector numbers. This may occur in many more vector-borne disease systems, particularly with tick species that are non-specific in their hosts.

Ostfeld & Keesing (2000a) looked at how Lyme disease (caused by the spirochaete *Borrelia burgdorferi*) risk altered with different numbers of host species across regions of the United States. They found that, with increased species richness of small terrestrial mammals, there was a decrease in disease risk, i.e. there was a dilution effect. However, with increased species richness of birds, the opposite occurred, and they termed this increase in disease risk a ‘rescue effect’. In the louping ill virus system, the addition of a third host (either hares to the deer–grouse host community or deer to the hare–grouse host community) completely changed the likelihood of virus persistence. Hares added to the deer–grouse system rendered the dilution effect of extreme deer densities impossible because they amplified the virus. The addition of deer to the hare–grouse system meant virus almost always persisted because they amplified the vector; only with very low densities of both hares and grouse could virus die out, due to insufficient virus amplifiers. In the louping ill virus system therefore it seems that increased species richness results in the rescue effect of increased disease prevalence. Van Buskirk & Ostfeld (1995) found that the dilution effect on *Borrelia* prevalence was due to increased numbers of incompetent hosts and, conversely, that the rescue effect was due to increased numbers of competent hosts.

This fits well with our findings as well as highlighting an interesting point about vector-borne diseases. We observed the rescue effect with an increase in a competent host (mountain hares), which is expected. However, we also observed the rescue effect with an increase in a completely incompetent host (red deer). Conversely, deer could also, when at very high densities,

help decrease louping ill (and, as a result, increase grouse numbers) by diluting the pathogen (the dilution effect; see also Norman *et al.* 1999). It is one of the interesting facets of vector-borne diseases that this initially counter-intuitive effect can be exhibited: the virus is not amplified by the deer, but the vector is. It is because of this facet of vector-borne diseases and the asymmetric nature of the host-vector-pathogen interactions that red deer in the deer-grouse two-host community can have, bizarrely, both a dilution and a rescue effect on louping ill virus. Hence, deer can both compete apparently with grouse and help increase grouse numbers, depending on relative host density. This finding is likely to be universal where different hosts interact with the vector and pathogen differently in this way, such that the less susceptible host can have two opposing effects on the more susceptible host.

Analytical models, by necessity, are simplistic and make assumptions. Clearly, our assumption that most parameters are identical between sites is not necessarily true, as demonstrated by the discrepancy between the predicted and empirical value of seroprevalence at one of the validation sites. In addition, the model assumes that the host densities are at equilibrium, even though both grouse and hare populations in Scotland fluctuate (e.g. Flux 1970; Watson *et al.* 1973; Moss, Watson & Parr 1996). The lack of knowledge about tick dynamics and density dependent factors was a limitation since these could be estimated only from the model itself. However, we stress that changing the values of these parameters does not change the patterns of disease persistence (i.e. the shapes of the  $R_0 = 1$  curves), merely the quantitative host densities. Hence, our general conclusions about the patterns of louping ill virus persistence are not altered, and we feel that the model adequately captures the essence of the louping ill virus system and produces biologically realistic results.

In conclusion, we have used an empirical model to describe a three-host-type vector-pathogen system in large-scale wild populations. We have suggested a novel type of parasite-mediated apparent competition which highlights the importance of considering community members that do not transmit the pathogen, and we emphasize the impact a third host can have on disease dynamics.

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