



## Persistence of Tick-borne Virus in the Presence of Multiple Host Species: Tick Reservoirs and Parasite Mediated Competition

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Tick-borne viruses in tropical and temperate parts of the world have a significant impact on human, livestock and wildlife hosts both directly, through mortality/morbidity, and economically. Since the ticks have multiple life stages and can utilize a large range of host species our understanding of the dynamics of these infections is often not clear. In this paper we consider the impact of a population which is a tick host but non-viraemic on one which is both a tick host and viraemic. We present two simple deterministic models and use joint threshold density curves to illustrate the basic reproductive ratios of both the ticks and the virus. We find that the non-viraemic hosts can have considerable impact on the viraemic host. Either they amplify the tick population and cause the virus to persist, or they dilute the infection and cause it to die out. A general model framework is presented here but a special case of this model describes the red grouse-hare-Louping-ill system.

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### 1. Introduction

Tick-borne viruses cause significant mortality, morbidity and economic loss to human, livestock and wildlife hosts in the tropical and temperate parts of the world (Sonenshine & Mather, 1994). Ticks exhibit a range of characteristics that have made them particularly successful agents for virus transmission and more specifically agents for cross-species transmission. They feed on a wide range of species, all ixodid ticks have three stages which, once infected, usually remain so through

each of its life stages, and the virus can be transmitted trans-ovarially.

Tick vectors have been implicated as important routes for parasite mediated competition where one host may act as a reservoir of infection, pass this infection via the tick to a more vulnerable host which then suffers reduced survival and population size (Hudson & Greenman, 1998). Such effects are not directly dependent on the density of the second host species so it is possible for the second host to be excluded by the indirect effects of the first host acting as a reservoir. Indeed it may be possible for one host to simply sustain the tick population, not even amplifying the pathogen, and, by keeping the vector

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population large, lead to transmission within a second host species that is subsequently excluded. In this respect the tick itself acts as the reservoir of the virus. Counter to this, a host that does not amplify the pathogen could act as a "pathogen sink" and result in the net loss of pathogen from the second host that may be large enough for the pathogen to go extinct.

Clearly, tick-borne pathogens are significant and the wide range of transmission routes, potential hosts and tick life stages can make the dynamics difficult to predict and so limit the efficacy of control strategies. In this paper, we use simple deterministic analytical models to examine the effect of interactions of shared hosts on the persistence and dynamics of tick-borne pathogens. While we develop a generalized model to examine these questions, we also consider the Louping-ill virus system specifically when it infects red grouse (*Lagopus lagopus scoticus*), which produce a viraemic response and mountain hares (*Lepus timidus*) which do not. It should be noted that the generalized model describes temperate (and not tropical) systems.

We address two questions:

1. Does the presence of a tick host, which does not amplify the pathogen, influence the impact of the pathogen on a viraemic host that suffers from the infection?
2. What conditions determine the persistence of pathogen in this system?

The paper falls into three sections. First, a general description is given of the biology on which these models are based with references to the Louping ill system. Second, a generalized tick-host model without the tick-borne pathogen is constructed. Third, the pathogen is incorporated into the model. Particular attention is paid to the joint threshold density curves produced by these models.

### Biological Framework

The model is based on a tick life cycle that develops from the egg through two immature stages (larvae and nymph) to the adult stage. Each immature stage requires a blood meal from a suitable vertebrate host before developing to

the next stage and the adult female requires a blood meal before producing eggs. Adult females are restricted to large mammals whilst the immature stages can also feed on smaller warm-blooded vertebrates. Once a questing tick locates a suitable host, it generally feeds on the host in an area where it cannot be removed easily through grooming or preening. The tick life cycle usually takes 3–4 years.

Low tick burdens do not have any clear impact on host growth or survival (e.g. Louping ill, Hudson, 1986, 1992). However, since ticks transmit a range of virulent pathogens they can cause significant morbidity and mortality of the hosts. Pathogens are transmitted inter-stadially, so once an immature stage is infected the subsequent stages can transmit the pathogen to a susceptible host. There is assumed to be no trans-ovarial transmission in ticks in this simple model. In general, transmission to ticks occurs when the concentration of virus in the blood reaches a threshold; this is known as viraemic transmission.

While the route of viraemic transmission is, in these models, considered the principal route of infection, non-viraemic routes are possible. In several systems it has been demonstrated that tick hosts which do not produce a viraemic response, will permit non-viraemic transmission between co-feeding ticks (Jones *et al.*, 1997; Randolph *et al.*, 1996). However, in order to build an understanding of the basic system, non-viraemic transmission through co-feeding will not be included in the models. The models consider just two types of tick host, a host species with a viraemic response and a second host that simply acts as a host for the ticks.

A particular case presented here is the Louping-ill virus. This is transmitted by the sheep tick, *Ixodes ricinus*, and causes appreciable mortality in sheep and red grouse (*Lagopus lagopus scoticus*) (Reid, 1976). This is an interesting system since it includes a domestic host (sheep) in which infection can be reduced through vaccination and acaricide treatment and a wild host (red grouse) which can suffer high mortality but may act as an amplifier of the virus. Complications arise when we consider that other wild hosts (notably the mountain hare *Lepus timidus* or red deer *Cervus elephas*) can act as important hosts

for the tick but are not viraemic hosts for the virus. In practice, sheep are usually removed from the system through vaccination and acaricide treatments, leaving hares as non-viraemic hosts and grouse as viraemic hosts.

**Model 1—Tick Dynamics Without the Virus**

This model considers two tick host species (in the Louping-ill system hares and grouse). These hosts are assumed to be at constant densities. Incorporating density dependence in the host dynamics does not alter the findings from this study.

An important element of the model is the rate at which each tick stage feeds on each host species. These feeding rates are defined as the average number of ticks (of the given stage) that feed on an average host in a unit of time and increase with the size of the tick population. In these first models we take the tick feeding rate as  $\beta V$ , where  $V$  represents the density of either larvae, nymphs or adults and  $\beta$  is a measure of the probability of a tick finding and then feeding on an average host per unit time. The number of ticks which pass from one stage to the next is the product of the number which feed on an average host (the tick feeding rate) and the number of hosts ( $\beta V H$ ). This is similar to the function used in the modelling of the ticks which transmit lyme disease by Sandberg *et al.* (1992). Mount & Haile (1989) in their model of Rocky Mountain Spotted Fever considered a development rate of the form  $\beta V^p H^q$  where  $p$  and  $q$  are constants. Since we do not have data to support this relationship we start here with the simplest function.

Model 1 is based on the life cycles of ticks and is described by the three coupled differential equations given below

$$\begin{aligned} \frac{dL}{dt} = & (\beta_5 D_5 H_1 A + \beta_6 D_6 H_2 A)(a - s_T T) \\ & - bL - \beta_1 H_1 L - \beta_2 H_2 L, \end{aligned} \tag{1}$$

$$\begin{aligned} \frac{dN}{dt} = & \beta_1 D_1 H_1 L + \beta_2 D_2 H_2 L - bN \\ & - \beta_3 H_1 N - \beta_4 H_2 N, \end{aligned} \tag{2}$$

$$\begin{aligned} \frac{dA}{dt} = & (\beta_3 D_3 H_1 N + \beta_4 D_4 H_2 N - bA \\ & - \beta_5 H_1 A - \beta_6 H_2 A. \end{aligned} \tag{3}$$

Here  $L$ ,  $N$  and  $A$  are larval, nymph and adult densities, respectively, and  $T$  is the total tick density ( $L + N + A$ );  $H_1$  is the density of host 1 (hares in the Louping-ill system) and  $H_2$  is the density of host 2 (grouse in the Louping-ill system). The per capita birth rate of larval ticks per adult tick is  $a$ ;  $s_T$  is a measure of the density-dependent constraints on the birth of ticks;  $b$  is the per capita natural death rate of the ticks and is assumed to be the same for each stage. The  $D_i$ 's are the proportion of ticks that feed and moult successfully. The probability of an average larva feeding on an average host 1 is  $\beta_1$ ;  $\beta_2$  is the probability of an average larva feeding on a host 2;  $\beta_3$  the probability of a nymph feeding on a host 1;  $\beta_4$  the probability of a nymph feeding on host 2;  $\beta_5$  and  $\beta_6$  are the probabilities of an adult female feeding on hosts 1 and 2, respectively, and subsequently producing eggs that hatch. All of the parameters,  $\beta$ , are defined per unit time. It should be noted that, in the case when host 2 are grouse, then  $\beta_6 = 0$  since adult ticks do not generally feed on grouse.

There are two possible equilibria in this model:  $(0, 0, 0)$  where no ticks are sustained, and  $(L^*, N^*, A^*)$ , where positive numbers of ticks can be maintained. The algebraic formulae for  $L^*$ ,  $N^*$  and  $A^*$  are found by setting the right-hand sides of eqns (1–3) equal to zero and then solving for  $L$ ,  $N$  and  $A$ . In this model, the ticks have no impact on the host and we are interested in the circumstances under which the tick population would decline to zero or settle to a biologically relevant equilibrium. The standard Jacobian method shows that the ticks die out (the origin is stable) if

$$\begin{aligned} & (b + \beta_1 H_1 + \beta_2 H_2)(b + \beta_3 H_1 + \beta_4 H_2) \\ & (b + \beta_5 H_1 + \beta_6 H_2) > (\beta_1 D_1 H_1 + \beta_2 D_2 H_2) \\ & (\beta_3 D_3 H_1 + \beta_4 D_4 H_2)(\beta_5 D_5 H_1 + \beta_6 D_6 H_2)a \end{aligned} \tag{4}$$

whereas they settle to  $(L^*, N^*, A^*)$  when this inequality is reversed (in this case the densities

can be shown to be non-negative). This has a rather obvious biological interpretation that states that if the product of the losses from each tick stage is greater than the product of the gains to each tick stage, then the ticks will die out; if not, they will persist.

Equation (4) essentially means that if the basic reproductive ratio of the ticks ( $R_{o,ticks}$ ), is less than one then the tick numbers will decrease,  $R_{o,ticks}$  can be derived directly from eqns (1–3). If we initially consider one larva, then the basic reproductive ratio of the ticks is the average number of the next generation larvae that this produces. On average one larvae will live for  $1/(b + \beta_1 H_1 + \beta_2 H_2)$  units of time [eqn (1)] and the probability of it becoming a nymph is  $(\beta_1 D_1 H_1 + \beta_2 D_2 H_2)/(b + \beta_1 H_1 + \beta_2 H_2)$  [eqns (1) and (2)]. Each nymph lives for  $1/(b + \beta_3 H_1 + \beta_4 H_2)$  units of time [eqn (2)] and has a probability  $(\beta_3 D_3 H_1 + \beta_4 D_4 H_2)/(b + \beta_3 H_1 + \beta_4 H_2)$  of becoming an adult [eqns (2) and (3)]. Each adult lives for  $1/(b + \beta_5 H_1 + \beta_6 H_2)$  years [eqn (3)] and produces  $(\beta_5 D_5 H_1 + \beta_6 D_6 H_2)a/(b + \beta_5 H_1 + \beta_6 H_2)$  larvae [eqns (3) and (1)]. Therefore, the total number of larvae produced by the one initial larvae is the product of these three terms and

$$R_{o,ticks} = \frac{(\beta_1 D_1 H_1 + \beta_2 D_2 H_2)(\beta_3 D_3 H_1 + \beta_4 D_4 H_2)(\beta_5 D_5 H_1 + \beta_6 D_6 H_2)a}{(b + \beta_1 H_1 + \beta_2 H_2)(b + \beta_3 H_1 + \beta_4 H_2)(b + \beta_5 H_1 + \beta_6 H_2)}.$$

If we plot the curve  $R_{o,ticks} = 1$  in the  $H_1$ – $H_2$  plane (for given values of all the parameters), then we produce a joint threshold curve for the ticks [Fig. 1(a)] which separates combinations of host 1 and 2 densities for which the ticks will survive from those for which they will die out (applied to directly transmitted diseases in Bowers & Turner, 1997). Given the density of host 2 in an area and with estimates of the  $\beta$  values, we can predict the density to which host 1 must be reduced in order for the ticks (and therefore the virus) to die out. This curve will be analysed in more detail in the discussion.

## Model 2. Tick and Viraemic Host Dynamics with the Virus

In model 2 we incorporate the virus and let host 1 ( $H_1$ ) be a non-viraemic host that suffers no

ill effects from the infection while host 2, ( $H_2$ ) is a viraemic host that suffers mortality. The second host is now classified as either susceptible ( $H_{2s}$ ), infected ( $H_{2i}$ ) or immune ( $H_{2z}$ ). The ticks are also divided into those that are susceptible ( $s$ ) and infected ( $i$ ). The non-viraemic host is kept at constant density while density-dependent regulation is included in the birth rates of both the viraemic host ( $H_2$ ) and the tick populations. The equations for the system are now as follows:

$$\frac{dL}{dt} = (\beta_5 D_5 H_1 + \beta_6 D_6 H_2)(A_i + A_s)(a - s_T T) - bL - \beta_1 H_1 L - \beta_2 H_2 L, \quad (5)$$

$$\frac{dN_i}{dt} = \beta_2 D_2 H_{2i} L - bN_i - \beta_4 H_2 N_i - \beta_3 H_1 N_i, \quad (6)$$

$$\frac{dN_s}{dt} = \beta_2 D_2 (H_{2z} + H_{2s}) L + \beta_1 D_1 H_1 L - bN_s - \beta_4 H_2 N_s - \beta_3 H_1 N_s, \quad (7)$$

$$\frac{dA_i}{dt} = \beta_4 D_4 H_2 N_i + \beta_4 D_4 H_{2i} N_s + \beta_3 D_3 H_1 N_i - bA_i - \beta_5 H_1 A_i - \beta_6 H_2 A_i, \quad (8)$$

$$\frac{dA_s}{dt} = \beta_4 D_4 (H_{2z} + H_{2s}) N_s + \beta_3 D_3 H_1 N_s - bA_s - \beta_5 H_1 A_s - \beta_6 H_2 A_s, \quad (9)$$

$$\frac{dH_{2s}}{dt} = (a_2 - s_2 H_2) H_2 - b_2 H_{2s} - \beta_7 H_{2s} N_i - \beta_8 H_{2s} A_i, \quad (10)$$

$$\frac{dH_{2i}}{dt} = \beta_7 H_{2s} N_i + \beta_8 H_{2s} A_i - \Gamma H_{2i}, \quad (11)$$

$$\frac{dH_{2z}}{dt} = \gamma H_{2i} - b_2 H_{2z}. \quad (12)$$

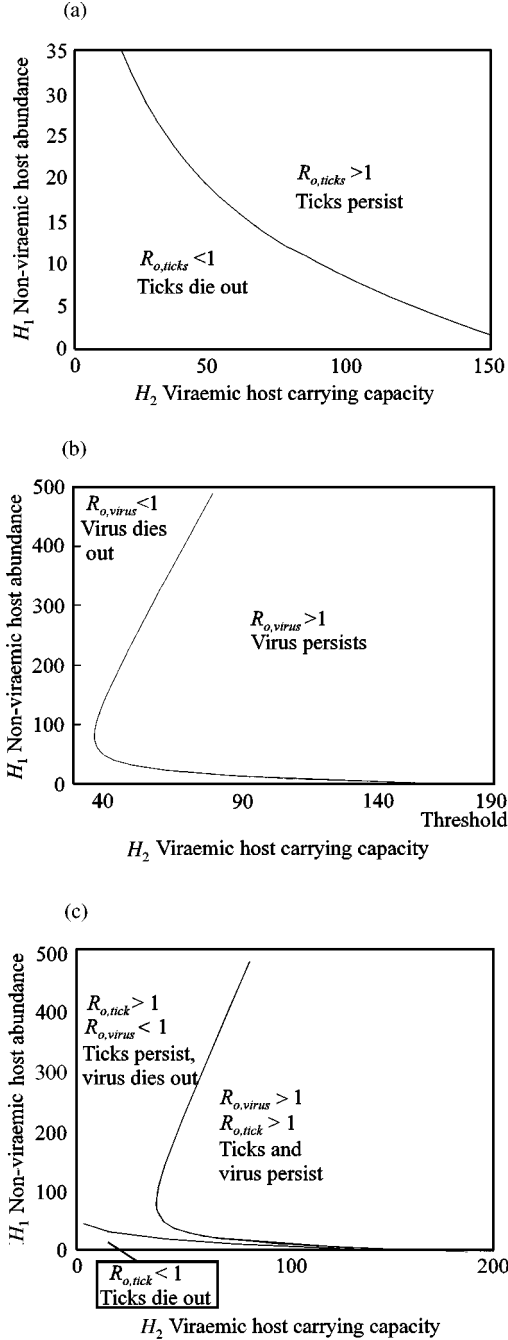


FIG. 1. (a) Graph to show the joint threshold curve for ticks, i.e.  $R_{o,ticks} = 1$ , in non-viraemic-viraemic host space. Parameter values:  $\beta_1 = 0.0002$ ;  $\beta_2 = 0.0005$ ;  $\beta_3 = 0.011$ ;  $\beta_4 = 0.0005$ ;  $\beta_5 = 0.001$ ;  $\beta_6 = 0.001$ ;  $b = 1$ ;  $a = 1500$ . (b) Graph to show the joint threshold curve for the pathogen, i.e.  $R_{o,virus} = 1$ , in non-viraemic-viraemic host space. Parameters as above with  $\beta_7 = 0.0005$ ;  $\beta_8 = 0.001$ ;  $s_T = 0.0001$ ;  $\alpha = 100$ ;  $b_g = 3$ ;  $\gamma = 5$ ;  $d_i = 0.9$ . (c) Graph to show both the pathogen and the tick joint threshold curves together.

The parameters are similar to those described in the previous model with  $\beta_7$ , the probability per unit time of a viraemic host being bitten and infected by an infectious nymph and  $\beta_8$ , the probability per unit time of a viraemic host being bitten and infected by an infectious adult. The per capita birth rate for viraemic hosts is  $a_2$ ;  $b_2$  is the per capita natural death rate of viraemic hosts;  $s_2$  is a measure of the density-dependent constraints on the viraemic host birth rate;  $\alpha$  is the rate at which viraemic hosts die from the disease and  $\gamma$  is the rate at which they recover to immunity. The rate at which infectious hosts are lost either through death or recovery is expressed as  $\Gamma = b_2 + \alpha + \gamma$ . In the Louping-ill system  $\beta_6 = \beta_8 = 0$  since adult ticks do not feed on grouse.

For any given non-viraemic host density, there are four biologically possible equilibria. The first has no ticks or viraemic hosts present and is never stable. The second has viraemic hosts and no ticks,  $(0, 0, 0, 0, 0, K, 0, 0)$ , where  $K = r_2/s_2$  is the carrying capacity of the viraemic hosts. This is stable if and only if

$$(b + \beta_1 H_1 + \beta_2 K)(b + \beta_3 H_1 + \beta_4 K)(b + \beta_5 H_1 + \beta_6 K) > (\beta_1 D_1 H_1 + \beta_2 D_2 K)(\beta_3 D_3 H_1 + \beta_4 D_4 K)(\beta_5 D_5 H_1 + \beta_6 D_6 K)a;$$

in other words if, and only if,  $R_{o,ticks} < 1$ .

The third possible equilibrium has viraemic hosts, ticks but no virus,  $(L^*, 0, N_s^*, 0, A_s^*, 0, K, 0, 0)$ , which is feasible and stable if and only if

$$(b + \beta_1 H_1 + \beta_2 K)(b + \beta_3 H_1 + \beta_4 K)(b + \beta_5 H_1 + \beta_6 K) < (\beta_1 D_1 H_1 + \beta_2 D_2 K)(\beta_3 D_3 H_1 + \beta_4 D_4 K)(\beta_5 D_5 H_1 + \beta_6 D_6 K)a;$$

and

$$\frac{(b + \beta_3 H_1 + \beta_4 K)\beta_4 D_4 N_s^* \beta_8 K + \beta_2 D_2 L^* \beta_8 K(\beta_3 D_3 H_1 + \beta_4 D_4 K) + (b + \beta_5 H_1 + \beta_6 K)\beta_7 K \beta_2 D_2 L^*}{\Gamma(b + \beta_3 H_1 + \beta_4 K)(b + \beta_5 H_1 + \beta_6 K)} < 1.$$

The first of these inequalities is  $R_{o,ticks} > 1$ ; the second involves the basic reproductive ratio of the pathogen and is  $R_{o,virus} < 1$ .

$R_{o,virus}$  can be derived directly from eqns (5–12). If ticks and hosts are both present but the pathogen is absent then the equilibrium will be  $(L^*, 0, N_s^*, 0, A_s^*, 0, K, 0, 0)$ . If one infected host is added then it will remain an infected host for  $1/\Gamma$  units of time [eqn (11)] and produce  $\beta_2 D_2 L^*/\Gamma$  infected nymphs [eqns (11) and (6)] and  $\beta_4 D_4 N_s^*/\Gamma$  infected adults [eqns (11) and (8)]. Each infected adult tick lives for  $1/(b + \beta_5 H_1 + \beta_6 K)$  units of time [eqn (8)] and produces  $\beta_8 K/(b + \beta_5 H_1 + \beta_6 K)$  infected hosts [eqn (11)]. The infected nymphs live for  $1/(b + \beta_3 H_1 + \beta_4 K)$  units of time [eqn (6)] and produce  $\beta_7 K/(b + \beta_3 H_1 + \beta_4 K)$  infected hosts [eqn (11)] and  $(\beta_3 D_3 H_1 + \beta_4 D_4 K)/(b + \beta_3 H_1 + \beta_4 K)$  infected adults [eqn (8)] which in turn produce  $\beta_8 K/(b + \beta_5 H_1 + \beta_6 K)$  infected hosts as discussed above. We therefore have three terms to add together to get

$R_{o,virus} =$

$$\frac{\beta_4 D_4 N_s^* \beta_8 K}{\Gamma(b + \beta_5 H_1 + \beta_6 K)} + \frac{\beta_2 D_2 L^* \beta_7 K}{\Gamma(b + \beta_3 H_1 + \beta_4 K)} + \frac{\beta_2 D_2 L^* \beta_8 K (\beta_3 D_3 H_1 + \beta_4 D_4 K)}{\Gamma(b + \beta_5 H_1 + \beta_6 K)(b + \beta_3 H_1 + \beta_4 K)}.$$

Both of the equations for the  $R_{o,s}$  can be represented by joint threshold curves as described in the previous model. If we take  $R_o = 1$  in each case and plot  $H_1$  against  $K$  for a chosen set of parameter values, then two curves are produced. The first one, seen previously, determines the combined densities of hosts which have to be present for the ticks to persist [Fig. 1(a) and (c)]. The second determines the densities of viraemic and non-viraemic hosts that must be present for the virus to persist [Fig. 1(b) and (c)]. Since the  $R_o$  equations include  $K$  rather than  $H_2$ , we are considering the density of the viraemic host that the environment must be able to sustain (the carrying capacity) for the ticks or pathogen to persist, not the current abundance.

When  $R_{o,ticks} > 1$  and  $R_{o,virus} > 1$ , no other equilibria are possible, so we assume that the

fourth possibility in which hosts, ticks and virus all persist, given by  $(L^+, N_i^+, N_s^+, A_i^+, A_s^+, H_{2s}^+, H_{2i}^+, H_{2z}^+)$ , must be stable or replaced by stable cycles. This is an assumption that is commonly made with similar models (e.g. Begon *et al.*, 1992, Bowers & Begon, 1991) and is supported by numerical simulations. It is neither biologically nor mathematically possible to have the virus present but not the ticks.

## Discussion

Parasite-mediated apparent competition can be an important driving force influencing biodiversity (Bonsall & Hassell, 1997). Transmission of pathogens between species can result in the more susceptible species being excluded although this very much depends on relative rates of between species transmission and the relative impact of the pathogen on the respective host species (Holt & Pickering, 1985). In reality, parasite-mediated competition is unlikely to operate via directly transmitted pathogens since individuals do not frequently come into contact with individuals from other species. However, pathogens with long-lived free-living stages or pathogens transmitted by vectors that feed on a broad range of hosts are quite capable of causing parasite mediated competition (Hudson & Greenman, 1998). This study has shown that if a tick population is amplified by a host which does not transmit the disease then the threshold density of viraemic hosts which has to be present for pathogen persistence is effectively lowered.

Within this study we addressed two main questions:

1. Does the presence of a tick host, which does not amplify the pathogen, influence the impact of the pathogen on a viraemic host that suffers from the infection?
2. What conditions determine the persistence of the pathogen in this system?

We examined these questions by producing a detailed model and describing the joint threshold curves. The models included a simple term to describe density dependence.

The fact that two threshold curves exist, one for the ticks and one for the virus is of interest

and is different to the threshold curves produced for directly transmitted pathogens (Bowers & Turner, 1997). Of particular significance is the shape of the curves. The joint threshold curve for the ticks [Figs 1(a) and (c)] identifies the joint conditions for  $R_o = 1$  between the densities of viraemic and non-viraemic hosts. As such the curve can be used to estimate the density to which the hosts would have to be reduced for the ticks to die out. Since both hosts are able to maintain the ticks alone there are threshold densities of each host, above which ticks can persist in the absence of the other host. These are where the curve cuts the axes. In the Louping-ill system the curve does not cross the grouse axis since adult ticks do not feed on the grouse. Hence the ticks cannot persist in the absence of an adult tick host, usually a large mammal.

With the parameters chosen, the joint threshold curve for the virus [Fig. 1(b) and (c)] “turns over”, and each carrying capacity of the viraemic host ( $K$ ) identifies two points where  $R_{o,virus} = 1$  for the non-viraemic host density ( $H_1$ ). The lower section of the curve identifies the threshold where there are just sufficient ticks for virus persistence and cuts the axis where the virus can be maintained by the viraemic host alone. This curve shows that parasite mediated, apparent competition is acting on the viraemic host population since below the point where the curve crosses the viraemic host axis the disease will only persist in the presence of the non-viraemic host species. The upper boundary for the threshold curve identifies the point where the relatively large densities of non-viraemic hosts dilute the virus through “wasted bites” of the ticks. At this point more virus is being lost through the ticks biting the non-viraemic host than is being gained in the system through the amplification and transmission in the viraemic hosts. At moderate densities of the non-viraemic hosts there is a net gain for the virus since there are both sufficient non-viraemic hosts to keep the tick population relatively high and the proportion of viraemic hosts within the population is sufficiently large to keep the virus circulating. Numerical simulations have shown that this qualitative behaviour holds for a broad range of parameter values. Altering parameters moves the curve rather than changing its shape. However, there are also para-

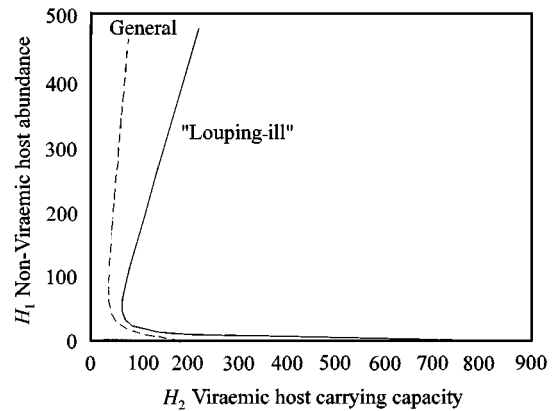


FIG. 2. Graph to compare the pathogen joint threshold curve for a virus which behaves like Louping-ill, i.e. with  $\beta_6 = \beta_8 = 0$ , with the general curve.

meter values for which the curve does not turn over.

In the Louping-ill system, the adult ticks rarely bite the grouse so  $\beta_6 = \beta_8 = 0$ . In this situation, for all other parameters the same, the joint threshold curve for the virus is further to the right than for the  $\beta_8$  positive. This means that there has to be more grouse available for the pathogen to persist (Fig. 2). In this case, the grouse cannot sustain the tick population without a second species present that plays host to the adult stages of the tick and thus the curve never reaches the viraemic host axis (Fig. 2).

These simple models provide some indication of the interactions occurring in the system and the sensitivity of virus persistence to the abundance of the non-viraemic host population. This is currently being examined through field experiments in the Louping-ill system although there are other complications that may be significant. Non-viraemic transmission (Jones *et al.*, 1997) and the role of lambs in viral amplification (Laurenson *et al.*, unpublished data) could be important features within this system. However, this work has shown the importance of an alternative host for the tick on the dynamics of an infection. In general, joint threshold curves are of particular interest if we are considering two host systems in which the carrying capacity of one host can be manipulated in order to eradicate the pathogen. The shape of these curves may provide important information about the interactions of

the hosts. For example, it may be possible to quantify the dilution effect which is difficult to measure in the field (Matuschka & Spielman, 1992).

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