ISRAEL JOURNAL OF ECOLOGY & EVOLUTION, Vol. 56, 2010, pp. 393–416
DOI: 10.1560/IJEE.56.3-4.393

APPARENT COMPETITION AND VECTOR–HOST INTERACTIONS

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ABSTRACT

Infectious disease influences the dynamics of host populations and the structure of species communities via impacts on host demography. Species that share infectious diseases are well-known to interact indirectly through the process of apparent competition, but there has been little attention given to the role of vectors in these indirect interactions. Here we explore how vector-borne disease and host-vector interactions can drive apparent competitive interactions. We show that different facets of the ecology associated with vector-host-host interactions affect the structure of the three-species assemblage. Crucially, the patterns associated with invasion of alternative hosts, the spread of the infectious disease by the vector, and the dynamics of the community interactions are influenced by the mode of transmission. We highlight the role of alternative hosts on disease amplification, dilution and magnification and discuss the results with reference to recent developments in apparent competition and community structure.

Keywords: basic reproductive number; coexistence; indirect interactions; intrinsic growth rate; invasion dynamics; non-linear dynamics; vector-borne disease.

INTRODUCTION

It is now widely appreciated that pathogens can affect the population dynamics of host populations (Anderson and May, 1978, 1979; May and Anderson, 1978, 1979), and with this has emerged a growing realization that pathogens can more broadly influence the structure of species communities and even the functioning of ecosystems (e.g., Price et al., 1986; Thomas et al., 2005). Over sixty years ago, Haldane (1949; see Lederberg, 1999 for a commentary on this work) argued that infectious disease is a driving force in natural selection and that "a non-specific parasite is a potent competitive weapon" in the struggle for existence. More recently, the idea that shared natural enemies, such as parasites, can influence the structure of species communities has been intensely explored

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Received 27 April 2009, accepted July 20, 2010.

(e.g., Holt and Lawton, 1994; Chaneton and Bonsall, 2000). Theory now predicts that, in presence of shared parasites (and in the absence of countervailing regulatory factors such as strong intraspecific competition), the host that suffers increased levels of mortality from parasitism or reduced population growth rate due to lower fecundity will be excluded from a community (Holt, 1977, 1984; Holt and Pickering, 1985). This effect is widely known as apparent competition.

Apparent competition is a pervasive force in the structure of ecological communities (Holt and Lawton, 1994; Chaneton and Bonsall, 2000). When considering pathogens or parasites, the dynamical action of a shared natural enemy (tacitly a generalist) leads to a state of emergent specialization on the host (or hosts) best able to resist infection (either through heightened immune defences, lower parasite-induced mortality, or higher host birth rates). If the natural enemy can be maintained at sufficient levels, it can severely affect host species and exclude those host species with low immune responses, high parasite-induced deaths, or a low reproductive capacity to replenish losses due to parasitism. In effect, the presence of alternative hosts can destabilize otherwise persistent single host–single parasite interactions and thereby lead to the loss of host diversity (Bonsall and Hassell, 1997, 1998, 1999). The role of apparent competition in disease interactions arising from directly transmitted parasites has been widely explored in the theoretical ecology literature (e.g., Getz and Pickering, 1983; Holt and Pickering, 1985; Begon et al., 1992; Hudson and Greenman, 1998; Greenman and Hudson, 1999, 2000; Rudolf and Antonovics, 2005). Similarly, there are an increasing number of empirical studies exploring apparent competition due to directly transmitted parasites (e.g., the poxvirus carried by grey squirrels as a contributory factor in the decline of red squirrels in the UK, Sainsbury et al., 2008). However, the role of this indirect effect in vector-borne disease has received rather less attention. Our aim here is to begin to redress this imbalance.

While many animal and plant diseases are vector-borne and cause large scale epidemics—including yellow fever, dengue, West Nile virus, and malaria – few empirical studies have highlighted the importance of shared vectors as potential agents of apparent competition or other indirect interactions. But there are some plausible examples. For instance, apparent competition mediated by tick-transmitted Lyme disease infecting white-tailed deer (*Odocoileus virginianus*) and white-footed mice (*Peromyscus leucopus*) is believed to affect host persistence and thereby community structure (LoGiudice et al., 2003). In this system, the short-tailed shrew (*Blarina brevicauda*) appears to act as a "rescue host", able to maintain the infection when other competent hosts are rare. In another relatively well-understood example, in UK upland habitats, the red grouse (*Lagopus lagopus scoticus*) is infected by a tick-borne flavivirus. This virus, which leads to louping ill and is transmitted by sheep tick vectors, causes high levels of mortality in infected grouse. The sheep ticks also feed on a wide range of alternative hosts including hare and deer. Gilbert et al. (2001) explored how a complex multi-host–vector–pathogen interaction is established in which disease is amplified in grouse and hares, and vectors increase only on deer and hares. The disease is non-viraemic in deer and hares but induces increased mortality in grouse. Different patterns of community structures of hare, grouse, deer, and ticks can affect the persistence of the virus. For instance, a

combination of deer and grouse can maintain persistent virus infections, as deer support ticks and grouse amplify the disease, provided deer densities are low. Although this will lead to apparent competition from deer onto grouse, a sufficient increase in deer density will conversely dilute the effects of louping ill. This is expected to occur as deer draw off ticks and thereby indirectly protect grouse. Focusing on the hare–grouse interaction, apparent competition is expected to occur, but in an asymmetric fashion, with grouse being harmed relatively more (as the virus is non-viraemic in hares). Invasion of additional host types can thus affect both the amplification and dilution of disease and alter the strength of species interactions (Gilbert et al., 2001).

More recently, Borer et al. (2009) have demonstrated that the prevalence of aphid-vectored plant viruses in Californian shrublands can be influenced by the presence of vertebrate consumers (e.g., mule deer). While mule deer do not directly affect infection rates on plants, they do increase the risk of disease by altering the structure of the plant community and allowing the plants most susceptible to disease to prosper. Alternative hosts have also been implicated in the spread and persistence of plague (*Yersinia pestis*) in prairie dogs in the Great Plains (Webb et al., 2006; Stapp et al., 2009). Empirical and theoretical studies have shown that while infectious fleas are an important focus of plague infection, disease transmission from flea vector to prairie dog host is insufficient to explain plague outbreaks and the epizootics frequently observed in prairie dogs. It is plausible that an alternative host of the pathogen provides a reservoir, sustaining plague between epizootics. However, in this system, evidence to date suggests that it is the vector rather than the pathogen which is sustained on an alternative host (Webb et al., 2006), one that is not competent at amplifying the pathogen. Apparent competition in this case emerges because vector abundance is boosted by the presence of these alternative hosts, which indirectly facilitate extermination of prairie dog colonies via plague outbreaks. In other situations, reservoir hosts may prove important in sustaining the pathogen itself. In either case, alternative mammalian hosts may be necessary for the persistence of plague. This establishes the potential for strong shared indirect competitive interactions at both local and regional scales. Two comparable examples of vector-mediated apparent competition are avian malaria in Hawaii and tsetse flies in Africa. In Hawaii, avian malaria is restricted in native land birds to high elevations, above the flight zone of an introduced mosquito (van Riper et al., 1986), while in Africa the tsetse fly, which transmits trypanosomes to both humans and domestic stock, is believed to have affected domestic cattle and human distributions. In particular, this vector has restricted both cattle and high-density human settlements (which depend upon draft animals in agriculture) to high elevations in Ethiopia (e.g., Baumgartner et al., 2008).

Based on such examples, it is anticipated that disease vectors could mediate apparent competition in two distinct ways. First, even in the absence of disease transmission, bites by a vector to obtain a blood meal might have detrimental consequences by reducing host condition, affecting individual performance and lifetime fitness, and, more broadly, population growth. It has been argued that mosquitoes in the high Arctic have negative effects on caribou, for instance, tormenting them sufficiently to influence their propensity to move among habitats (Toupin et al., 1996). Mosquitoes have been implicated in

high rates of egg loss and adult mortality in an Arctic seabird, the Brünnich's guillemot (*Uria lomvia*) (Gaston et al., 2002), and high densities of ticks can lead to fatal anaemia in hosts (Mehlhorn and Armstrong, 2001, p. 571). If vector population size is sensitive to the abundance of hosts, then the presence of alternative hosts could lead to apparent competition occurring directly because of the impact of the vector itself, even without the pathogen it transmits.

Second, the action of a shared pathogen mediated through vector transmission might also increase host morbidity and mortality, leading to apparent competition. Conversely, alternative hosts could reduce infection by drawing off attacks by vectors, if these hosts neither increase vector numbers nor sustain pathogen growth. If one or the other of these effects occurs, the qualitative nature of the indirect interaction between hosts may change with shifts in host absolute and relative abundances.

Thus, when there are multiple hosts available to a vector (and for a pathogen it transmits), there is the potential interplay of three distinct effects on disease prevalence (Keesing et al., 2006; Peixoto and Abramson, 2006): a dilution effect (if alternative hosts are incompetent to sustain the pathogen), an amplification effect (if alternative hosts are competent), and a magnification effect (if alternative hosts can boost the number of vectors). The net effect of one host upon another is then an emergent property of all three of these. As such, indirect interactions between hosts due to vectors are expected to be richly complex.

Here, we aim to explore, theoretically, some of these effects of vector-borne disease on the interaction between two hosts. We begin by outlining the mathematical framework we use and emphasize the different transmission processes that might be appropriate to understand the spread of vector-borne disease. We show how both disease transmission and recruitment to the susceptible host populations can affect the persistence of species in the community and the potential for apparent competition. We show that in some circumstances, simple models of vector-borne diseases in multiple host systems can display complex dynamics. Finally, in the discussion we highlight the importance of these results, in light of recent work on vector-borne diseases, for understanding the effects of species diversity on disease spread and prevalence.

MATHEMATICAL FRAMEWORK

To begin to explore the role of apparent competition and indirect effects of vector-borne diseases, we develop a theoretical framework describing the interaction between two different hosts (denoted by H and J) infected with a pathogen carried by a vector (F) (Fig. 1). As we are interested in the numerical response of the vector itself to each host, we need to include an explicit equation for changes in vector abundance, rather than simply assuming a generic term for frequency-dependent transmission between the two hosts. Moreover, for each species in the system we assume an open population, for which there can be recurrent immigration from an external source (whose dynamics are not explicitly tracked); the dynamics of closed populations emerge as a limiting case. This recurrent immigration is often appropriate in open, local populations, measured

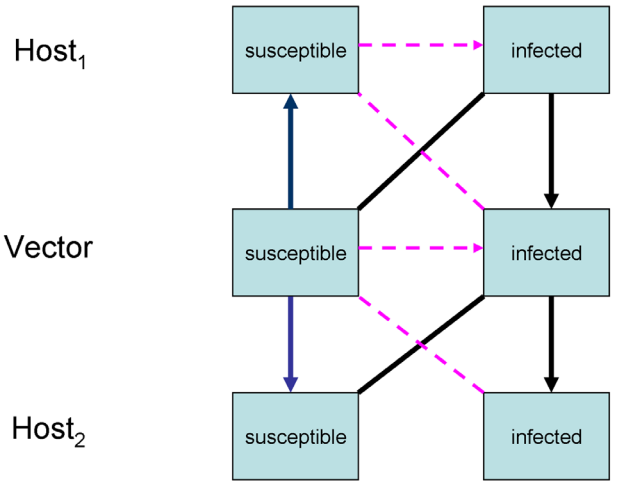

Fig. 1. Schematic diagram of the two-host–one-vector interaction. Apparent competition can be mediated by two mechanisms: through the effects of biting reducing susceptible host fitness (blue lines) or through the effects of disease transmission (pink and black lines).

at spatial scales that are small relative to the spatial scale of overall recruitment in the regional population.

HOST DYNAMICS

Each host population is divided into susceptible (H_s, J_s) and infected (H_i, J_i) classes. The dynamics of host H are described by:

$$\frac{dH_s}{dt} = \lambda_H + r_H H_s(t) f_H(H) - \beta_{F_i H_s} F_i(t) H_s(t) - \mu_H H_s(t) \quad (1)$$

$$\frac{dH_i}{dt} = \beta_{F_i H_s} F_i(t) H_s(t) - (\alpha_H + \mu_H) H_i(t) \quad (2)$$

where λ_H is the rate of immigration of susceptible hosts, r_H is the local birth rate of the host population (by susceptible individuals only—infected individuals are assumed to be functionally sterile), $f_H(H)$ is a density-dependent function acting on the birth rate of susceptible hosts (as a function of total host density $H = H_s + H_i$), β_{FiHs} is the transmission rate of the pathogen from infected vectors (F_i) to susceptible hosts (H_s) (which can be a function of densities; see below), μ_H is the background per capita mortality rate (plus emigration, if any) of hosts, and α_H is the extra pathogen-induced mortality rate in infected hosts. The dynamics of the second host type (J) are described by a similar set of expressions (with J substituted for H in the above equations).

VECTOR DYNAMICS

The vector population is also divided into susceptible (F_s) and infected (F_i) classes. Its dynamics are given by:

$$\frac{dF_s}{dt} = \lambda_F + r_F F_s(t) f_F(F) - F_s(t)\left[\beta_{F_s H_i} H_i(t) + \beta_{F_s J_i} J_i(t)\right] - \mu_F F_s(t) \tag{3}$$

$$\frac{dF_i}{dt} = F_s(t)\left[\beta_{F_s H_i} H_i(t) + \beta_{F_s J_i} J_i(t)\right] - \mu_F F_i(t) \tag{4}$$

where λ_F is the immigration rate of susceptible vectors (F_s), r_F is the birth rate of the vector population (which here is assumed to be independent of bite rate), $f_F(F)$ is the density-dependent function acting on vector birth rate, β_{FsHi} and β_{FsJi} are the transmission rates of the pathogen to susceptible vectors from infected hosts of types H_i and J_i, respectively, and μ_F is the mortality rate of vectors. Parallel with the host model, we assume that there can be immigration from an external source, and emigration is incorporated into the mortality terms. Unlike for the hosts, we assume that the pathogen does not have adverse effects on vector survival. Below, we will consider a simple model in which vector dynamics does depend on host abundance.

Transmission

Two important aspects of vector-borne disease transmission are the density of vectors per host and the probability that a bite transmits disease (Ross, 1910; MacDonald, 1957). In the presence of alternative hosts, transmission can be described in a number of different ways. We highlight three distinct forms of disease transmission:

1. Density-dependent transmission. Simple mass action based on the number of bites per unit time per vector per host (*a*) and the probability that a bite transmits the disease (*b*):

$$\beta = ab \tag{5}$$

2. Specialist frequency-dependent transmission. Scaled transmission based on the abundance of vectors per focal host type (assuming that biting behaviour of vector depends on only one host species, but are indifferent to classes within this species):

$$\beta = \frac{ab}{H_s + H_i} \tag{6}$$

This mode of transmission might occur in a frequency-dependent fashion on just one host species, or where each host species experiences its own level of bites and probability that a bite transmits disease (i.e., transmission in host *J* depends upon an expression like Eq. 6, but with *J* substituted for the *H* in the denominator). Both mechanisms could in principle pertain to particular host-vector systems. The first could arise if the vector attacks both hosts indiscriminately, but requires a certain number of bites per day on a particular host (after which it stops feeding), because

of an essential nutrient which only that host species supplies. The second mode of transmission could arise if hosts are found in distinct habitats or are active at different times of the day.

3. Generalist frequency-dependent transmission. Scaled transmission based on the density of vectors across all hosts (assuming the vector is indifferent to the two hosts):

$$\beta = \frac{ab}{H_s + H_i + J_s + J_i}. \quad (7)$$

As noted above, different behavioral assumptions about the interactions between vectors and hosts can lead to the above forms of transmission. For instance, if vectors have a fixed number of bites they can deliver in their lifetime, and are successful at delivering all of them, then these bites will be diluted among all accessible host classes within a mosquito generation (leading to case [3]—Eq. 7). Conversely, if vectors can deliver bites in direct proportion to the number of hosts available, then case [1] (Eq. 5) for transmission may be more appropriate. More broadly, one might expect the number of bites per unit time, per vector, to show a saturating functional response to the number of hosts available, and vectors may also preferentially attack certain species or classes (which could be represented mathematically by weighting factors in the denominators of Eqs. 6 and 7) so the above expressions encapsulate limiting cases of a broad range of vector biologies.

The full model for both hosts and the vector is a six-dimensional system, which is potentially highly nonlinear. We analyse some important limiting cases and illustrative examples that shed light on disease dynamics and the effects of apparent competition on community structure and assembly using invasion analyses and numerical simulations of the model (Eqs. 1–4).

RESULTS

FORCE OF INFECTION AND R_0

A fundamental concept in infectious disease ecology is the basic reproductive number, R_0, which is defined as the "average number of secondary cases produced by an average infectious individual, introduced into a totally susceptible population" (e.g., Anderson and May, 1991). R_0 in a sense is the expected lifetime reproductive success of an infection, measured by the number of infections it produces itself over its "lifetime", taking into account both mortality and recovery from infection. Diekmann et al. (1990), in an important study, demonstrated how one can derive R_0 for a heterogeneous population, which in our case is comprised of a mixture of interacting vectors and host species. One starts with a matrix based on the "who acquires infection from whom" (WAIFW) matrix, which compactly describes the force of infection experienced by and exerted by each host species (Diekmann et al., 1990). Dobson (2004) provides a useful exposition, with worked examples, of this methodology for the case of multiple host species. Here we extend his approach by explicitly accounting for vector dynamics.

We can represent the flow of infections in our two-host–one-vector model schemati-

cally as:

$$\text{From} \begin{bmatrix} H \to H & H \to F & H \to J \\ F \to H & F \to F & F \to J \\ J \to H & J \to F & J \to J \end{bmatrix} \quad \text{To} \tag{8}$$

where each entry is a measure of the rate of infection from one class to the other. As Diekmann et al. (1990) showed, one first constructs a matrix (which following Dobson, 2004 we call G) where the ij^{th} entry describes the per capita rate of infection of susceptible individuals in class j per infected individual in class i (when the infection is rare) divided by the per capita loss rate of individuals in class i. Then the dominant eigenvalue of this matrix provides a formula for R_0 of the infection.

We assume that the two host species and the vector are initially present and at equilibrium, in the absence of infection. With these assumptions, the relevant G matrix is as follows:

$$G = \begin{pmatrix} 0 & \dfrac{\beta_{FsHi} F_s^*}{\mu_H + \alpha_H} & 0 \\ \dfrac{\beta_{FiHs} H_s^*}{\mu_F} & 0 & \dfrac{\beta_{FiJs} J_s^*}{\mu_F} \\ 0 & \dfrac{\beta_{FsJi} F_s^*}{\mu_J + \alpha_J} & 0 \end{pmatrix} \tag{9}$$

where parameters are as defined previously, F_s^*, H_s^*, and J_s^* are the abundances of susceptible vectors and hosts at equilibrium.

So that the steps leading toward the results given below are clear, one constructs the G matrix as follows (following Diekmann et al., 1990). As we are assuming that all infections are mediated by the vector, with no direct transmission within or among hosts, or vector-to-vector, the diagonals and corner terms are zero. The remaining non-zero entry in the first row describes the flow of infections from an infected individual of host species H to susceptible vectors. There are F_s^* susceptible vectors, and the instantaneous rate of infection, per vulnerable vector, by an infected individual of species H is β_{FsHi}. This individual is expected to live $1/(\alpha_H + \mu_H)$ time units. The total number of individual susceptible vectors the host H should infect over its lifetime is thus this quantity multiplied by $\beta_{FsHi} F_s^*$. The other non-zero elements are found in a comparable way. The G matrix thus summarizes the total number of infections generated by each class of individuals, over their respective lifetimes (or period of infectivity if there is acquired immunity). As noted by Dobson (2004), a measure of the total force of infection can be found by summing over rows and columns of the transmission matrices (without the denominators expressing loss rates). The total forces of infection ***by*** vectors $\beta_{FiHs} H_s^* + \beta_{FiJs} J_s^*$ and ***on*** vectors $\beta_{FsHi} F_s^* + \beta_{FsJi} F_s^*$ are the predominant drivers of infection in this host-pathogen system. It is important to note that the choice of a transmission function (Eqs. 5–7) will affect the total forces of infection. With density-dependent

transmission, the force of infection is linear with the abundance of each host, and with vector abundance. At the other extreme, with generalist frequency dependence, rare hosts will have little impact on the force of infection while conversely abundant hosts will weaken infections through communities of species via diluting attacks by the shared vector.

Diekmann et al. (1990; see also Dobson 2004) argue that the dominant eigenvalue of the *G*-matrix is equivalent to R_0, which is usually defined as the total number of infections generated by an infected individual introduced into an entirely susceptible host population. If, for instance, one considers the biological meaning of the basic reproduction number, one should measure expected reproductive success among equivalent life-history stages; this will be a power of the eigenvalue of *G*, rather than the eigenvalue itself. In our case, comparable life stages of the pathogen would be say vector-to-vector, and one would need to compute the eigenvalue associated with this block matrix (e.g., vector-to-vector) to ascertain the number of secondary infections in vectors, generated by a primary infected vector.

VECTOR DISEASE SPREAD

Understanding the spread of a vector-borne infectious disease can be determined in a number of different ways (Anderson and May, 1991). Evaluating R_0 permits one to determine whether or not an infection can spread at all when rare, which of course is essential; the infection goes extinct, for instance, if $R_0 < 1$. But to gauge the actual *rate* at which it spreads or declines, quantitatively, requires other approaches (Anderson and May, 1991), which typically are mathematically more challenging. The correspondence between R_0 and growth rate can be very loose; a high R_0 in principle could correspond either to a very rapid growth rate, or to a sluggish growth rate (e.g., the latter might occur if there is a low transmission rate, but hosts have a very long intrinsic lifespan, the disease has no impact on mortality, and there is no recovery from infection). Analogously, in age-structured demography, lifetime reproductive success

$$R_0 = \int_0^\infty l(x)m(x)dx$$

where $l(x)$ is survivorship from birth to age x, and $m(x)$ is birth rate at that age can be a highly inadequate measure of population growth r which one finds from the Euler equation,

$$1 = \int_0^\infty e^{-rx} l(x)m(x)dx \,.$$

The intrinsic growth rate r is a better measure of fitness than is R_0. The proper analogue of the demographic intrinsic growth rate r in infectious disease epidemiology, we suggest, is not the standard R_0. The equivalent measure, at least in some circumstances, is the dominant eigenvalue of a linearized version of the dynamical model. Here, we adopt this standard and well-defined eigenvalue approach (Pielou, 1977; Klug and Bonsall, 2007,

2010; Bonsall and Mangel, 2009) to determine the initial rate of spread of an infection when it is still rare, but has been present sufficiently long that initial transients have damped away. We assume (as we did in deriving matrix *G*) that the disease is initially rare, and all the species it needs to spread (susceptible vectors and hosts) are at their respective disease-free equilibria.

The two approaches, of course, converge in their conclusions when one considers the issue of pathogen persistence versus extinction, and we will mainly focus on this limiting case. But it useful when possible to have in hand explicit formulations for the growth rate itself, which can provide insight into situations where one is concerned with rates of spread or decline and not simply the qualitative *fact* of spread or decline. Moreover, although epidemiology has typically focused on expressions for R_0, in community ecology it has been more customary to consider *per capita* growth rates for species during community assembly. This latter approach is clearly more appropriate for understanding how indirect interactions such as apparent competition affect the structure of host and vector-borne disease communities.

Before considering the indirect interaction between the alternative hosts, it is useful to consider the basic interaction between just one host and vector. After a few infected vectors or hosts are introduced, the pathogen spreads (after a transient phase) if the asymptotic rates of increase of infectious vectors and hosts are greater than zero (i.e., $\frac{dF_i}{dt} > 0$ and $\frac{dH_i}{dt} > 0$). More explicitly, the rate of increase of the infection when rare is determined from the following subset of the general model:

$$\frac{dF_i}{dt} = F_s^* \beta_{F_s H_i} H_i(t) - \mu_F F_i(t) \tag{10}$$

$$\frac{dH_i}{dt} = \beta_{F_i H_s} F_i(t) H_s^* - (\alpha_H + \mu_H) H_i(t) \tag{11}$$

where all parameters are as defined in Eqs. 1–4, and F_s^* and H_s^* are the equilibrium abundances of vectors and hosts, respectively, in the absence of disease. Under mass action transmission the rate of spread of disease can be obtained from these linearized equations, which have the following Jacobian:

$$\begin{pmatrix} -\mu_F & \beta_{FsHi} F_s^* \\ \beta_{FiHs} H_s^* & -(\mu_H + \alpha_H) \end{pmatrix}. \tag{12}$$

Solving for the eigenvalues of this matrix gives:

$$\omega = \frac{-(\mu_H + \alpha_H + \mu_F) \pm \sqrt{(\mu_H + \alpha_H + \mu_F)^2 - 4\left((\mu_H + \alpha_H)\mu_F - \beta_{F_i H_s} \beta_{F_s H_i} H_s^* F_s^*\right)}}{2} \tag{13}$$

where β_{FsHi} and β_{FiHs} are the transmission rates of infection from infected hosts to susceptible vectors and from infected vectors to susceptible hosts, respectively. The dominant eigenvalue is the one with the largest real part, and determines the ultimate rate of spread

of the infection, after a transient period.

In epidemiology, as noted above it is customary to discuss pathogen persistence and extinction in terms of the basic reproduction number R_0. In terms of R_0, the necessary condition for disease spread is $R_0 > 1$; in terms of the above formalism, this inequality is equivalent to one of the eigenvalues having a positive real part. If we consider the upper-left submatrix of Eq. 9, which is the matrix G for the vector and host H alone, its dominant eigenvalue is:

$$q = \sqrt{\frac{F_S^* \beta_{F_s H_i}}{\mu_H + \alpha_H} \frac{H_S^* \beta_{F_i H_s}}{\mu_F}}. \tag{14}$$

On the right hand side, the first part of this expression is the product of the expected lifetime of an infected host and the number of susceptible vectors encountered and infected per unit time. Comparably, the second part is the product of the lifetime of an infected vector and the number of susceptible hosts encountered and infected per unit time. Thus, this expression can be viewed as the (square root of) the number of (say) of secondary hosts infected, per primary infected host (as noted above, if one defines R_0 over a complete cycle, say for transmission from a primary host to a secondary host, one should consider the square of the quantity q).

The disease spreads when $\omega > 0$, or equivalently $R_0 > 1$. From Eq. 13, $\omega > 0$ (and $q > 1$) occurs if the product of susceptible host and vector densities is greater than the product of the mortalities acting on vectors and hosts scaled by the transmission rates of the disease:

$$F_S^* H_S^* > \frac{(\mu_H + \alpha_H)\mu_F}{\beta_{F_i H_s} \beta_{F_s H_i}}. \tag{15}$$

So the usual threshold criterion of a minimal host density for pathogen persistence for a single host species is replaced by a more complex criterion including densities of both the host and the vector. The multiplicative nature of the above invasion criterion reflects the fact that dynamically the vector and host can be viewed as "alternating hosts", in that a pathogen has to pass through both of them to complete its life cycle. For density-dependent transmission, as noted in Holt et al. (2003) (and Case 1 for transmission—Eq. 5), this means that invasion of the pathogen tends to be particularly sensitive to whichever species (host or vector) is rarer. Spread of the disease is affected by six factors: the transmission of the disease from infected vectors to susceptible hosts, the transmission of the disease from infected hosts to susceptible vectors, the equilibrium densities of hosts and vectors in the absence of disease, and the death rates of vectors and infected hosts. This makes intuitive sense; with density-dependent transmission, R_0 increases with increasing density of susceptible vectors and hosts, and declines as the bite rate or the probability of transferring disease fall. Likewise, increasing the death rate of vectors decreases the number of hosts they can expect to infect before dying.

For purely frequency-dependent transmission within both the vector and a single host (Case 2—Eq. 6), pathogen invasion occurs if:

$$\frac{F_S^*}{H_S^*} > \frac{(\mu_H + \alpha_H)\mu_F}{a^2 bb'} \tag{16}$$

where b is the probability that a bite transmits disease from the host to vector, and b' is the probability that a bite transmits disease from vector to host. Increasing vector abundance, or reducing vector mortality, both make it more likely an infection will become established. In contrast, for the host, comparable changes are expected to go in opposite directions; reduced mortality of infected hosts enhance pathogen establishment, but higher host numbers can hamper disease establishment.

The basic reproduction number ($R_{0\text{all}}$) for the full three-species system (two hosts—one vector) is obtained by finding the dominant eigenvalues of the full matrix in Eq. 9, which gives:

$$R_{0\text{all}} = \sqrt{\frac{F_s^*}{\mu_F}\left(\frac{\beta_{F_i H_s}\beta_{F_s H_i} H_s^*}{\mu_H + \alpha_H} + \frac{\beta_{F_i J_s}\beta_{F_s J_i} J_s^*}{\mu_J + \alpha_J}\right)}. \tag{17}$$

The square of this quantity is equal to the sum of the (squared) basic reproductive numbers for each host species with the vector, taken separately. In a three-dimensional space, a surface describes the boundary of vector and host abundances that permit the infection to spread in the community.

Spread of the disease into a community of three species (two hosts and one vector) is again strongly affected by species abundance, at least if transmission is density-dependent. Often, the presence of alternative hosts for the vector (and pathogen) favors the spread of the disease (Fig. 2). For density-dependent transmission (Eq. 5) and frequency-dependent transmission determined by focal host abundance (Eq. 6), the threshold vector density that allows the disease to spread ($R_0 > 1$) is an order of magnitude lower when alternative hosts are available.

For a given community structure (1-host or 2-host-species system), disease invasion is more likely to occur given density-dependent transmission (Eq. 5), as the threshold density of vectors is lower (Figs. 2a,c and 2b,d). However, if there is frequency-dependent transmission, these conclusions must be altered. Now, after substitution into Eq. 17, we find that the basic reproductive number in the two-host community is:

$$R_{0\text{all}} = \frac{ab}{(H_s^* + J_s^*)}\sqrt{\frac{F_s^*}{\mu_F}\left(\frac{H_s^*}{\mu_H + \alpha_H} + \frac{J_s^*}{\mu_J + \alpha_J}\right)}. \tag{18}$$

On the right hand side of this expression, the term outside the square root sign describes a dilution effect due to vectors spreading out attacks or bites among alternative hosts. Whether the diverse host community sustains a higher, or lower, reproductive number for the pathogen depends upon if the factors sustaining vector numbers outweigh this dilution effect.

Moreover, the dilution effect is stronger overall for whichever host species has the higher intrinsic mortality, or pathogen-induced death rate. R_0 is then dominated by the

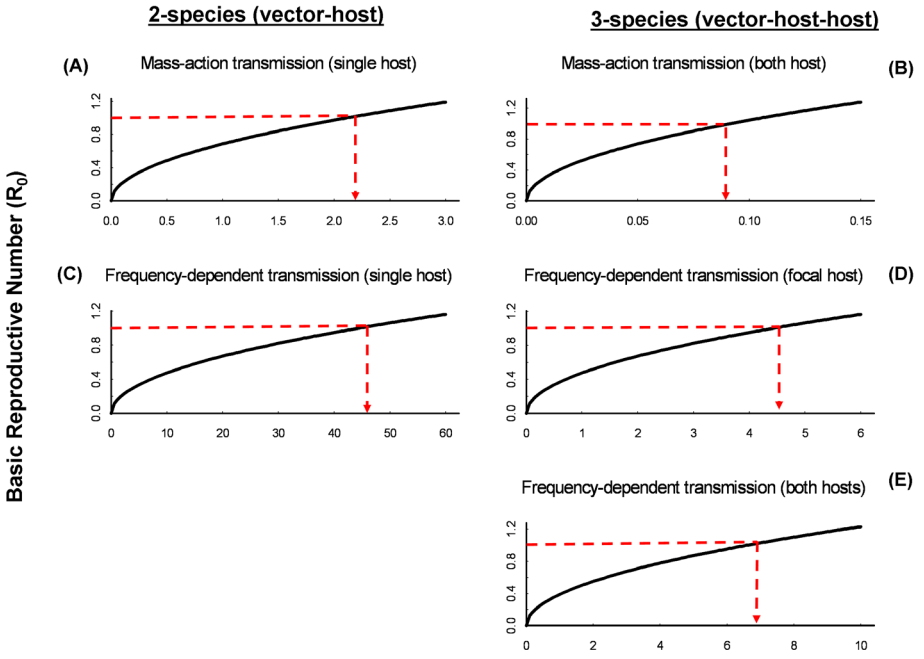

Fig. 2. Basic reproductive number (R_0) as function of susceptible vector density for a one host – one vector and a two host–one vector interaction under the different modes of transmission (Eqs. 5–7). (A) Host–vector interaction under mass action transmission; (B) Two host–one vector interaction under mass action transmission; (C) Host–vector interaction under frequency-dependent transmission based on focal host abundance; (D) Two host–vector interaction under frequency-dependent transmission based on focal host abundance; (E) Two host–vector interaction under frequency-dependent transmission based on total host abundance. Red dashed line denotes the threshold vector density when $R_0 = 1$. (Parameter values: $a = b = 0.15$, $\alpha_H = 0.9$. $\alpha_J = 0.01$, $\mu_F = 1.0$, $\mu_H = \mu_J = 0.1$, $H^* = 21$, $J^* = 51$).

host with the lower intrinsic mortality, and lower mortality impact of the pathogen.

Consider further a host (say species *J*) which dies instantly upon being infected. In this case, the expression for the basic reproductive number becomes:

$$R_{0\text{all}} = \frac{ab}{(H_s^* + J_s^*)}\sqrt{\frac{F_s^*}{\mu_F}\left(\frac{H_s^*}{\mu_H + \alpha_H}\right)}. \tag{19}$$

Host *J* cannot sustain the infection at all, and moreover can strongly dilute attacks.

All else being equal, the host attributes that contribute to initial pathogen establishment also help define the host that is expected to be superior in apparent competition. We conjecture that the host that is better able to spread the pathogen when that pathogen is initially rare (e.g., equal bite rates), is the one that in the long run will dominate,

when one pays attention to subsequent changes in host numbers post-establishment (see below). Further, if both hosts have equivalent mortalities, then the basic reproductive number (as is the case for a single host species with frequency-dependent attacks) is no longer affected solely by summed host densities but instead is determined by the ratio of vector numbers to summed host numbers. Increases in host density dilute transmission as infected hosts experience fewer attacks per unit time. In contrast, increased vector density will enhance pathogen establishment.

APPARENT COMPETITION AND EFFECTS OF BITING ON HOST FITNESS

So far, we have considered some effects that having two host species has on the initial dynamics of the pathogen, when it is rare. To analyze apparent competition, we now assume that one host species and the vector are at equilibrium, either without or with the pathogen, and ask whether or not the other host species can increase when rare. As noted from the examples given earlier, in some cases alternative hosts for vectors may exert apparent competition via the vector, even in the absence of a shared pathogen.

It is expected that there could be some duality in the apparent competitive effects between host species if bites by vectors on uninfected hosts have fitness consequences (reducing individual performance and consequently population growth rate), regardless of infection transmission. For simplicity, we assume that there is neither direct density dependence nor immigration in the host, but that there is immigration from an external source pool for the vector that sustains its numbers. In this case, in the absence of the disease, and assuming density dependence in vector attacks, the model can be collapsed to:

$$\frac{dJ_s}{dt} = \left(r_J - r_J g_J a F_s\right) J_s(t) - \mu_J J_s(t) \tag{20}$$

$$\frac{dH_s}{dt} = \left(r_H - r_H g_H a F_s\right) H_s(t) - \mu_H H_s(t) \tag{21}$$

$$\frac{dF_S}{dt} = \lambda + caF_S(J_S + H_S) - \mu_F F_S(t) \tag{22}$$

where c is a conversion efficiency of bites into new vectors and the total bites per host per unit time (aF_s) act to reduce host birth rate (r_J or r_H) at rates g_J and g_H, respectively (these convert bites per unit time to fractional reduction in birth rate), even in the absence of an infectious disease.

In the absence of either of the hosts in the local community, vectors persist; maintained at the ratio of immigration to the death rate:

$$F_S^* = \frac{\lambda}{\mu_F}. \tag{23}$$

In the presence of the vector, for each host to be able to increase when rare requires that the birth rate of the host exceed its intrinsic death rate, plus any attacks inflicted

upon it by the vector. This requires that for species J, $r_J > \frac{\mu_J}{1 - g_J a F_S *}$ and for species H, $r_H > \frac{\mu_H}{1 - g_H a F_S^*}$. However, once either species is present, a host (say species J) will reach an equilibrium with:

$$F_{S,J}^* = \frac{r_J - \mu_J}{r_J g_J a}. \tag{24}$$

At this vector equilibrium density ($F_{S,J}^*$), we can evaluate the condition under which the alternative host (H) invades by substituting this expression (24) into equation (21) and ascertaining if the *per capita* growth rate of the invading species (species H) is positive. After some manipulation, we find that:

$$\frac{r_J - \mu_J}{r_J g_J a} < \frac{r_H - \mu_H}{r_H g_H a}. \tag{25}$$

Similarly, in the absence of host type (J) the abundance of the vector at equilibrium (when only species H is present) is:

$$F_{S,H}^* = \frac{r_H - \mu_H}{r_H g_H a} \tag{26}$$

and invasion by the alternative host (J) occurs if:

$$\frac{r_H - \mu_H}{r_H g_H a} < \frac{r_J - \mu_J}{r_J g_J a}. \tag{27}$$

Clearly the two invasion expressions (Eqs. 25 and 27) are mutually exclusive. Only one host species or the other, but not both, can persist stably with the vector when the vector responds numerically to each host species and imposes a demographic cost on hosts as it attacks. Thus, it is expected that alterations in population growth rates through feeding by a vector will lead to apparent competitive effects. If bite rates differ between the two species, then factors that reduce the bite rate on a particular species will increase its apparent competitive effect on the alternative host. This result parallels those that are expected in general from apparent competition in other predator-prey models (e.g. Holt, 1977, 1997). In the absence of other limiting factors, if vectors impose fitness costs on their hosts, and can respond numerically to each host in a positive manner, then host coexistence may be precluded in these shared natural enemy interactions.

This simple model can obviously be generalized in many respects. In particular, one could introduce direct density dependence in the hosts. In general, we do not realistically expect direct vector impacts upon hosts to be sufficiently strong to lead to exclusion via apparent competition. Moreover, the dynamics might differ in important respects when vectors feed in a frequency-dependent fashion. However, the possibility of apparent competition is present at least as an element of interspecific interactions among host species, wherever multiple host species sustain a vector that imposes direct costs as it feeds.

APPARENT COMPETITION: INVASION OF ALTERNATIVE HOSTS

The invasion of an alternative host (*H*) into an established two-species system (host–vector) can also be explored with the above framework. Returning to the general model (Eqs. 1–4), if it is assumed that the invasion of the alternative host gives rise to infections then the dynamics of invasion of the alternative host (H_s, H_i), when this species (*H*) is rare and the other species are present at equilibrium, are governed by:

$$\frac{dH_s}{dt} = \lambda_H + r_H H_s(t) f_H(H) - \beta_{F_i H_s} F_i^* H_s(t) - \mu_H H_s(t) \tag{28}$$

$$\frac{dH_i}{dt} = \beta_{F_i H_s} F_i^* H_s(t) - (\alpha_H + \mu_H) H_i(t) \tag{29}$$

where all the parameters are as defined in Eqs. 1–2 and F_i^* is the equilibrial abundance of infected vectors determined from the resident host-vector interaction only. (We assume here that transmission is described either by Eq. 5 or Eq. 7, because with Eq. 6, as host density becomes very rare per capita transmission tends to infinity). Invasion conditions for this alternative host, assuming it does not immigrate ($\lambda_H = 0$), are obtained from the eigenvalues of:

$$\begin{pmatrix} r_H - \beta_{FiHs} F_i^* - \mu_H & 0 \\ \beta_{FiHs} F_i^* & -(\mu_H + \alpha_H) \end{pmatrix}. \tag{30}$$

Solving the resulting characteristic equation shows that invasion will be successful if:

$$r_H - \beta_{F_i H_s} F_i^* - \mu_H > 0\,. \tag{31}$$

Intuitively, this can seen by noting that, by assumption, infected individuals neither recover nor reproduce, so host increase is entirely determined by the susceptible fraction of the population (defined by Eq. 28). Invasion is less likely as 1) transmission of the disease (product of encounters and disease vectoring) increases, 2) the density of infected vectors increases, 3) the background death rate of the invading host increases and 4) the birth rate of the invading host decreases. Reciprocally, invasion by hosts of type *J* into an established vector-host interaction with host *H* present and at equilibrium with the vector occurs if:

$$r_J - \beta_{F_i J_s} F_i^* - \mu_J > 0\,. \tag{32}$$

Now the equilibrium abundance of infected vectors is determined from the resident host (*H*)-vector interaction. With density-dependent transmission (Eq. 5), hosts impact each other through the maintenance of infected vectors; as noted previously, with frequency dependence, this impact can be reduced by dilution of attacks. Density dependence in the resident host will tend to act indirectly to reduce the equilibrial abundance of infected vectors, and thus relax these conditions for invasion by each host, making their coexistence more feasible.

APPARENT COMPETITION DYNAMICS: EFFECTS OF TRANSMISSION AND BIRTH/IMMIGRATION

The above results clarify some of the essential implications of shared vectors and pathogens for limiting cases focused exclusively on invasion conditions and criteria. The multiplicity of feedbacks that are possible in this system make more complex dynamics possible when all species coexist. A fuller exploration of the population dynamics of shared vectors and pathogenic disease on multiple hosts requires numerical simulations making different assumptions about transmission and birth and/or immigration processes including density dependence. We show some representative examples in Fig. 3, and will elsewhere explore these effects in more detail.

Apparent competition leading to the reduction in density and/or exclusion of the inferior host occurs through a wide variety of different disease transmission and host birth processes. Given local births and focal host frequency-dependent disease transmission (Fig. 3), and weak or nonexistent direct density dependence in the host, it is expected that the inferior host will be excluded through the action of the shared vector and disease. Intriguingly, these different transmission routes also lead to different dynamical patterns. With local density-dependent births and mass action transmission, cyclic dynamics can occur in the vector and host populations (Fig. 3b), while under frequency-dependent transmission, the dynamics of the vector and the host populations in the examples we have examined are stable.

There are also different dynamical implications for the constant immigration of susceptible hosts and vectors, compared to local birth processes (Fig. 3a,c,e and Fig. 3b,d,f). Constant immigration leads to the potential for both disease amplification and dilution effects, depending on the particular form of transmission (Fig. 3a,c,e). Under mass action transmission, disease burdens are almost equivalent among hosts and vectors (Fig. 3a). Under frequency-dependent transmission (scaled by focal and total host abundance), the inferior host has low disease burdens. Disease effects are diluted in this inferior species and enhanced in the alternative host (and the vector) populations (Fig. 3 c,f). Immigration overall tends to stabilize local dynamics.

Different transmission processes also influence the dynamics of disease and the effects of disease dilution and amplification under local birth processes. Under mass action transmission (Fig. 3b), it is expected that the interaction between the hosts and vector will lead, at least transiently and sometimes persistently, to unstable dynamics in which disease transmission from vectors to hosts sweep through the population. This is expected to reduce host abundance. As the form of our equations (Eqs. 1–4) resemble classical predator-prey interactions, it may not be surprising that oscillations are observed. However, the form and persistence of the cycles are quite intriguing (see Figure 3b). It would appear that the time lag provided by the stage structure of the vectors can at times permit sustained limit cycle dynamics, under mass action transmission. Further, the interaction between the vector and its shared hosts can lead to apparent competition. Comparing Fig. 3d and Fig. 3f illustrates that under focal host frequency-dependent disease transmission (Eq. 6), the inferior host is driven to extinction. This is the host that suffers the higher levels of disease-induced mortality and it is not necessarily the

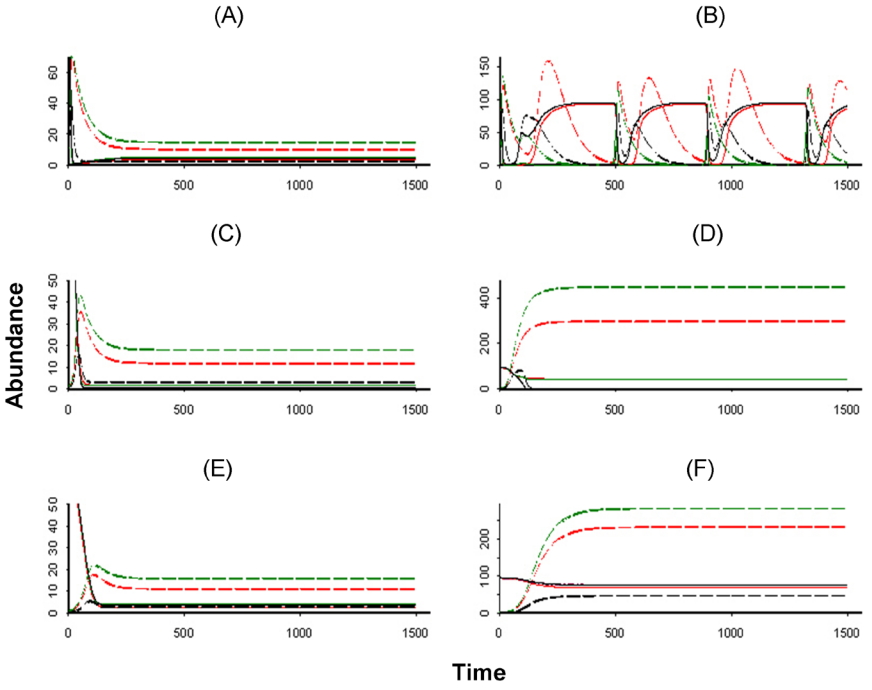

Fig. 3. Population dynamic patterns of the two host (red line, black line) –vector (green line) interaction under different transmission routes and processes of population growth (constant immigration, local births). (A) Constant host immigration, mass action disease transmission; (B) local births, mass action disease transmission; (C) constant host immigration, frequency-dependent transmission based on focal host abundance; (D) local births, frequency-dependent transmission based on focal host abundance; (E) constant host immigration, frequency-dependent transmission based on total host abundance; (F) local births, frequency-dependent transmission based on total host abundance. (Solid lines—susceptible host and vector densities, dashed line—infected host and vector densities). Parameter values, mass action transmission: $\lambda_F = 2.0$, $\lambda_J = 1.5$, $\lambda_H = 2.0$, $a = b = 0.15$, $r_H = 1.5$, $r_J = 2.0$, $r_F = 2.0$, $\alpha_H = 0.01$. $\alpha_J = 0.5$, $\mu_F = \mu_H = \mu_J = 0.1$. Parameter values, frequency-dependent transmission: $\lambda_F = 2.0$, $\lambda_J = 1.5$, $\lambda_H = 2.0$, $a = b = 0.75$, $r_H = 1.5$, $r_J = 2.0$, $r_F = 2.0$, $\alpha_H = 0.01$. $\alpha_J = 0.5$, $\mu_F = \mu_H = \mu_J = 0.1$. Numerical simulations were implemented in C using a standard 4th-order Runge–Kutta algorithm; code available on request.

host that has the lower birth rate. Under total frequency-dependent transmission, disease burden is distributed across multiple hosts and the effects of the disease on any particular species are diluted.

DISCUSSION

Here, we have explored how patterns of host species coexistence are affected by vector-borne diseases and apparent competition. Shared parasites are known to affect the structure of simple species assemblages (Bonsall and Hassell, 1997, 1998, 1999) and may be implicated in structuring entire food webs (Woolhouse et al., 2001; Lafferty et al., 2006). Patterns of species diversity and distributions can all be affected by vector-borne diseases (Keesing et al., 2006; Borer et al., 2009). Here, we have shown how different aspects of the ecology associated with multispecies interactions can affect community structure by governing species coexistence. For instance, the invasion of an alternative host is shown to be affected by vector density, the bite rate and probability of disease transmission during biting. The spread of vector-borne disease is shaped by host diversity: the presence of alternative hosts for the vector (and consequently the parasite) favors the spread of the disease as the effective transmission rate is amplified (both hosts are equally susceptible to infection and causing infection). Exclusion can occur even when there is pure frequency-dependent transmission due to the vector diluting its attacks across multiple hosts (as in the model represented by Eqs. 20–22). All these effects are likely to be sensitive to both the quantitative and qualitative aspects of the transmission function, and arguably this should be the focus of sustained and careful empirical work, as well as more theoretical exploration. Furthermore, it is expected that a richer understanding of the within-host (and within-vector) parasite dynamics will be crucial for understanding the outcome of shared pathogen interactions. For instance, Cable et al. (2007) have illustrated how epidemiological processes associated with disease morbidity and mortality scale with host metabolic rate; they show that this correlation is likely to be driven by the parasite cell cycle dynamics (speed of replication) and interaction with the host immune system. In different species, with different immune functions, these effects, manifest at the population and community level, will affect patterns of species coexistence and exclusion.

It is increasingly clear that the transmission of vector-borne diseases has important consequence for species diversity, disease ecology and, consequently, ecosystem function (Keesing et al., 2006). Similar to Rudolf and Antonovics (2005), we have shown that species diversity and coexistence are potentially affected by the form of the disease transmission process. In contrast to Rudolf and Antonovics (2005), we demonstrate that frequency-dependent transmission, mediated by the local abundance of both susceptible and infected hosts, can lead to species loss through apparent competition, because of the potential for the vector population itself to respond dynamically to host abundance. Models of vector-borne disease often assume that pathogen transmission is dependent on only the proportion of hosts infected (McCallum et al., 2001), and while this might be appropriate for some simple host–vector systems (e.g., Anderson and May, 1991), in more diverse communities where multiple species share vectors, simple frequency-dependent transmission expressions based solely on the abundance of infected individuals may not be appropriate. Vectors are consumers that depend upon their resources—hosts—and should tend to be more abundant, and thus more efficacious at sustaining

pathogens, when hosts are abundant and productive.

Vector distributions and abundance are likely to be affected by focal host density, the abundance of alternative hosts, the detailed spatial pattern of both hosts, and vector dispersal capacity. Here, we have illustrated how density-dependent, focal host frequency-dependent, and total host frequency-dependent transmission affect the dynamics and structure of simple food webs under apparent competition. We have not exhaustively explored any of these possibilities, but some salient points emerge from our analyses and numerical studies. If vector distribution is based on focal (local) host abundance, inferior species (suffering higher disease burdens, increased morbidity or mortality or lower birth rates) are likely to be excluded from the local community. Antonovics et al. (1995) considered a more general model of disease transmission, in which it was shown that density and frequency-dependent patterns of transmission represent extremes of the pathogen functional response curve. As vectors only visit a limited number of hosts, transmission may decline with increasing host density. Similarly, if vectors are affected by the presence of alternative hosts (and alternative host densities), then it is expected that the impact of the parasite on a focal host may be diluted (as alternative host abundance increases) or magnified (as alternative host abundance declines). Further understanding of the detailed dynamical underpinnings of processes of disease transmission within and between hosts is essential to interpreting the effects of infectious diseases on the patterns of community organization (Dobson, 2004; Keesing et al., 2006).

The process of local recruitment (constant immigration versus local births) has broad implications for community structure and diversity when species share parasites and experience apparent competition. At a local level, immigration precludes extinction even for species with negative growth rates. This suggests that one might expect differences in local community structure when comparing open communities dominated by immigration and emigration with closed communities governed by in situ density-dependent dynamics. Here we have shown how the local population dynamics alter as the patterns in recruitment change. Interestingly, in vector-borne disease systems, density-dependent local host births coupled with density-mediated transmission are predicted by our models to give rise to a range of complex population dynamics in which the vector and host densities oscillate, as well as disease levels. It can be relatively difficult to generate sustained oscillations in standard SIR-style models for directly transmitted disease. Our results suggest that the additional feedbacks that emerge when vector population dynamics are explicitly incorporated into the system (rather than merely swept under the rug of a transmission function) can have potentially important dynamical consequences.

In contrast, constant immigration of susceptible hosts and vectors is expected to lead to relatively stable dynamics either of two (host–vector) or three (host–host–vector) species interactions. This suggests that understanding the spatial scale of host regulation, versus that of disease transmission, will be crucially important in understanding the dynamical impacts of vectors and indirect interactions. While the role of metapopulations (populations linked through limited dispersal) have recently been shown to mitigate the effects of apparent competition mediated by natural enemies (Bonsall et al., 2005; Bull et al., 2006, 2007), the effects of regional spatial structures on the persistence and

dynamics of disease (and particular vector-borne disease) in multi-host systems still remains poorly explored. We expect this to be an important direction for future research.

Alternative hosts may play a particularly critical role in maintaining infectious diseases in spatially-extended systems (Hess, 1996; Gog et al., 2002). This is particularly clear for diseases such as plague in prairie dogs, which lead to rapid local extinctions, but the point applies much more broadly. Understanding the rate of movement of susceptible and infected hosts (and vectors) is important in judging the impact of infectious disease at the wider metacommunity scale. If disease and vectors are already widespread, then the additional movement of alternative hosts may have little impact on disease spread and persistence at the regional scale, but might have considerable impact at the local scale through apparent competition. Furthermore, both local and regional stochastic processes are likely to be of critical importance in determining both the epidemiological consequences and community assembly patterns of shared infectious diseases. Keeling and Gilligan (2000) demonstrate that historical patterns of bubonic plague are best understood though the (stochastic and spatial) interaction between rats, fleas, and humans. By developing a stochastic and spatial metapopulation model, small reservoir rodent populations were shown to be sufficient to give rise to the devastating epidemics of plague in humans observed through the 16th and 17th centuries (and noted in Graunt's Bills of Mortality). The distribution and abundance of alternative, secondary hosts may also act to influence both the persistence and spread of disease at different spatial scales. We suggest that alternative hosts might act to affect local heterogeneity in vector densities, connect isolated patches in metapopulations and buffer vector–disease interactions from extinction. All of these novel aspects of vector-borne disease ecology require consideration of both local and regional processes to understand the spread, maintenance, and effects of indirectly-transmitted parasites in multi-host communities. In conclusion, we have shown that pathogens transmitted by vectors have the potential to affect community structure and drive species locally extinct through the process of apparent competition. The broader implications of these effects for both local and regional community assembly warrant further theoretical and empirical attention.

ACKNOWLEDGMENTS

The work was supported by the Royal Society (MBB), and the University of Florida Foundation and the National Science Foundation, grant # EF 0525751 (RDH). We thank Lewi Stone, Mike Barfield, and two anonymous reviewers for critically reading the manuscript and providing detailed comments that improved the analysis and presentation.

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