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REVIEWS AND SYNTHESIS

Effects of species diversity on disease risk

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Abstract

The transmission of infectious diseases is an inherently ecological process involving interactions among at least two, and often many, species. Not surprisingly, then, the species diversity of ecological communities can potentially affect the prevalence of infectious diseases. Although a number of studies have now identified effects of diversity on disease prevalence, the mechanisms underlying these effects remain unclear in many cases. Starting with simple epidemiological models, we describe a suite of mechanisms through which diversity could increase or decrease disease risk, and illustrate the potential applicability of these mechanisms for both vector-borne and non-vector-borne diseases, and for both specialist and generalist pathogens. We review examples of how these mechanisms may operate in specific disease systems. Because the effects of diversity on multi-host disease systems have been the subject of much recent research and controversy, we describe several recent efforts to delineate under what general conditions host diversity should increase or decrease disease prevalence, and illustrate these with examples. Both models and literature reviews suggest that high host diversity is more likely to decrease than increase disease risk. Reduced disease risk with increasing host diversity is especially likely when pathogen transmission is frequency-dependent, and when pathogen transmission is greater within species than between species, particularly when the most competent hosts are also relatively abundant and widespread. We conclude by identifying focal areas for future research, including (1) describing patterns of change in disease risk with changing diversity; (2) identifying the mechanisms responsible for observed changes in risk; (3) clarifying additional mechanisms in a wider range of epidemiological models; and (4) experimentally manipulating disease systems to assess the impact of proposed mechanisms.

Keywords

Biodiversity, dilution effect, disease ecology, diversity, ecosystem function, host, pathogen.

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INTRODUCTION

Infectious diseases necessarily involve interactions between at least two species – the pathogen and the host. At least one more species is involved for pathogens that are transmitted from host to host by a vector, and for many diseases, multiple species of host can sustain the pathogen. In some vector-borne disease systems (e.g. West Nile virus), several to many species of vector can transmit the pathogen. Thus, infectious diseases are inherently ecological systems, involving interactions among small to large networks of species. It is perhaps not surprising, then, that a potential connection between species diversity and disease transmission has long been recognized.

Well over 100 years ago, medical entomologists suggested a connection between species diversity and transmission of vector-borne diseases of humans (reviewed in Service 1991). Researchers argued that malaria transmission might be reduced if alternative hosts for mosquito vectors (e.g. livestock) were placed around areas of human habitation, an idea termed 'zooprophylaxis'. Zooprophylaxis refers to the use of non-human animals to protect human health specifically by diverting vector meals away from humans. Later, a connection between diversity and disease was also

recognized by Elton (1958), who proposed that plant diseases could be ameliorated in 'complex' ecosystems if this complexity reduced the density of the host plant for a disease, an insight that was subsequently supported both by empirical research on plant diseases (Burdon & Chilvers 1982; Boudreau & Mundt 1997) and by epidemiological models demonstrating the sensitivity of disease transmission to host density (Anderson & May 1981).

Recently, there has been renewed interest in the potential effects of diversity on disease risk, in large part because of interest in identifying and evaluating utilitarian functions of biodiversity (Loreau *et al.* 2001). Despite the fact that effects of diversity on disease transmission have now been described for multiple diseases (e.g. Van Buskirk & Ostfeld 1995; Norman *et al.* 1999; Gilbert *et al.* 2001; Allan *et al.* 2003; LoGiudice *et al.* 2003; Ruedas *et al.* 2004; Telfer *et al.* 2005), the specific mechanisms underlying these effects are not well understood. Understanding these mechanisms is critically important, both for predicting net effects and for evaluating the generality of patterns found in specific disease systems. For example, various empirical and modelling investigations have suggested that increased species diversity could reduce disease risk by regulating the abundance of an important host species (Burdon & Chilvers 1982; Rudolf & Antonovics 2005), or by redistributing vector meals in the case of vector-borne diseases (Van Buskirk & Ostfeld 1995; Norman *et al.* 1999; LoGiudice *et al.* 2003). But other studies have suggested that increased diversity could *increase* disease risk if, for example, added species function as alternative sources of infection, or if they increase vector numbers or activity by providing additional sources of vector meals (Holt & Pickering 1985; Norman *et al.* 1999; Gilbert *et al.* 2001; Schmidt & Ostfeld 2001; Saul 2003; Dobson 2004).

Here, we describe a suite of mechanisms that could result in either a reduction or an increase in disease risk with increasing diversity. We focus principally on species richness as the measure of diversity, although variation in species evenness can have similar effects, as we illustrate in several examples. We begin with simple models of a single pathogen and host species, using these models to identify parameters that can be affected by increasing species diversity with potentially strong effects on pathogen dynamics. We then increase the complexity of the models to include a vector. We review recent research in a variety of disease systems, with an emphasis on pathogens with multiple hosts, and examine specific case studies that illustrate our underlying mechanisms. Finally, we discuss several recent efforts to generalize the conditions under which species diversity should dilute or amplify disease risk in disease systems with multiple hosts, and illustrate their applicability with specific examples. We conclude by suggesting important areas for future research.

A SIMPLE SYSTEM

To identify the specific mechanisms by which species diversity can decrease or increase disease risk, we begin with a simple susceptible-infected (SI) model of a disease system in which a microparasite is specialized on a single host species and transmission of the pathogen is non-vector-borne (e.g. by direct contact) (Ostfeld & Holt 2004, after Anderson & May 1981):

$$\mathrm{d}S/\mathrm{d}t = (b - m)S - \alpha\delta\mathrm{SI} + (\gamma + b')I, \quad (1)$$

$$\mathrm{d}I/\mathrm{d}t = \alpha\delta\mathrm{SI} - (\gamma + m')I. \quad (2)$$

Here S and I are, respectively, densities of healthy and infected hosts; b and b', and m and m', are, respectively, their birth and death rates. All newborns are uninfected. Recovery occurs at rate γ, and recovered individuals can be re-infected. We write the transmission rate of the infection as $\alpha\delta$, the product of the rate of encounter, α, between healthy and infected hosts, and the probability of transmission from an infected host to a susceptible host, per encounter, δ. This product is often collapsed into a single variable, β, the transmission rate; for our purposes, it is useful to distinguish two components of β (Anderson 1982). Equation 1 describes a host that is regulated solely by the pathogen. For a case, in which the total number of hosts is regulated by factors independent of pathogen dynamics (e.g. nest site availability), an alternative form of eqn 1, $S + I = K$, where K is carrying capacity, would be appropriate. The assumption that the host is at carrying capacity can be a reasonable approximation for systems where pathogens have weak or negligible demographic impacts on their hosts (e.g. the common cold in humans).

Even in this simple system with parasite specialization on a single host, the species diversity of a community can potentially influence disease dynamics in a number of different ways. One way to assess how diversity could influence disease dynamics is to assess how diversity could control the rate of change in the density of infected hosts, which is one measure of disease risk (eqn 2; Box 1). Adding species could reduce $\mathrm{d}I/\mathrm{d}t$ by any of the following routes:

Box 1 Metrics of disease risk.

Many different metrics of disease risk are used in epidemiological and ecological studies of disease systems, including those reviewed here. These metrics include the density of infected reservoir hosts, the prevalence of infection in reservoir hosts, the rate of change in the density of infected hosts, the density of infected vectors, and the infection prevalence in vectors, among many others. Which of these metrics are the most relevant for the epidemiology of particular types of disease systems remains a research frontier, as is whether different metrics vary in their sensitivity to species diversity.

- reducing the rate of encounter between susceptible and infectious individuals (α);
- reducing the probability of transmission given an encounter (δ);
- decreasing the density of susceptible individuals (S);
- increasing the recovery rate (γ);
- increasing the death rate of infected individuals (m');

Conversely, species diversity could have the opposite effects for each of these mechanisms, which we now consider in turn. Recall that we are considering a single host-single pathogen system, and therefore added species by definition cannot be hosts.

Encounter reduction

If non-host species reduce the probability of encounter between hosts, α, there would be fewer opportunities for conversions of susceptible to infected individuals (Fig. 1a). Added species could decrease α in several ways. Imagine, for example, that the added species is a predator which induces the host to move less, due to increased risk of predation. Susceptible hosts would then be less likely to encounter infected hosts, in the case of a directly transmitted disease, or less likely to encounter propagules of the pathogen in the case of an environmentally transmitted disease. Alternatively, imagine that individual hosts with a directly transmitted disease have a relatively fixed number of behavioural contacts, whether with conspecifics or heterospecifics (Ostfeld & Mills in press). The addition of a non-host species with which the host species made behavioural contact would reduce contacts between conspecifics, thus reducing the probability of encounters between susceptible and infected hosts.

Alternative scenarios could lead to an increase in encounters (*encounter augmentation*). For instance, if conspecifics were more clumped in the presence of heterospecifics, then encounters between infected and healthy hosts might be more frequent. Thus, a potentially important route through which diversity can influence disease dynamics, even in specialist host–pathogen systems, is through shifts in spatial organization and contact patterns among host individuals.

Transmission reduction

If added species reduce the probability that contact between individuals leads to transmission, δ, there would be fewer conversions of susceptible to infected individuals (Fig. 1b). In practice, transmission reduction would occur if adding species reduced the pathogen load or the pathogen's titre within the host. For example, the addition of a resource species (prey or mutualist) for the host species could decrease host stress, which could increase the efficacy of the immune response, thus resulting in a lower pathogen load and lower probability of transmission (for alternative

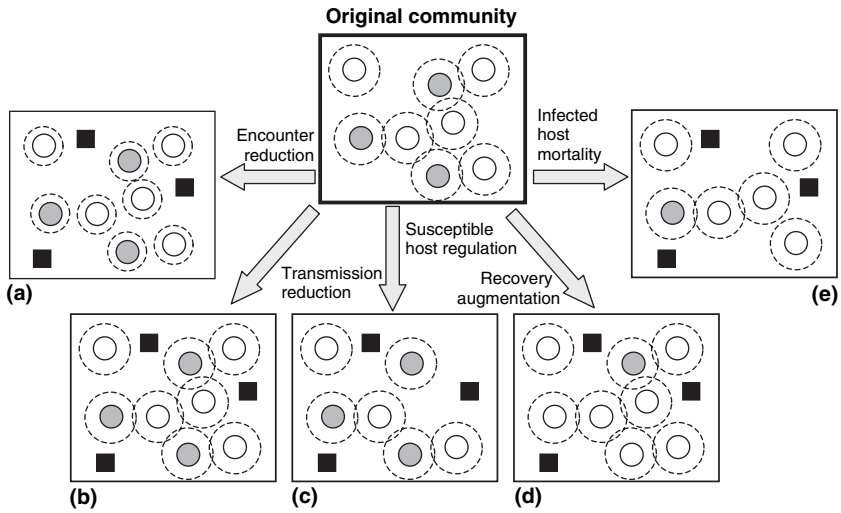

Figure 1 Conceptual model of the mechanisms by which diversity could reduce disease risk in a specialist host–pathogen system for a non-vector-borne disease. The original community consists of a single species with some individuals infected (filled circles) and some individuals uninfected and therefore susceptible (open circles). Each individual uses a particular home range (dashed lines). The addition of a second species (black squares) could (a) result in a reduction in space use by the host species, thus reducing encounters between susceptible and infected individuals (*encounter reduction*); (b) reduce the probability of transmission given encounters, as indicated here by no increase in the number of infected individuals despite contacts that could lead to transmission (*transmission reduction*); (c) reduce the number of susceptible hosts (*susceptible host regulation*); (d) increase the rate of recovery of infected individuals, as indicated by some infected individuals becoming uninfected (*recovery augmentation*); or (e) increase the mortality rate of infected individuals (*infected host mortality*).

scenarios, see Lafferty & Holt 2003). Added species might also reduce the duration of intraspecific contacts (e.g. via changes to time/activity budgets).

Susceptible host regulation

We use this term to describe situations in which added non-host species limit or regulate susceptible host numbers, S, via altered birth or death rates (Fig. 1c). For instance, interspecific competition for limiting resources could constrain the abundance of susceptible hosts. From eqn 2, this would then automatically reduce the maximal rate at which a pathogen could spread. Other interspecific interactions such as predation could also limit host numbers, and thus indirectly influence disease transmission.

Infected host mortality

Non-host species could influence disease dynamics via effects on the mortality of *infected* hosts (m'; Fig. 1e). For example, competing species could increase death rates of infected individuals via exploitative competition. Although competition could affect both susceptible and infected individuals, infection may make individuals less able to tolerate competitive interactions, and so suffer higher mortality rates. An increase in m' should dampen the spread of an infectious disease to surviving uninfected individuals. Alternatively, a predator could target infected individuals preferentially or exclusively, reducing risk (Packer *et al.* 2003; Ostfeld & Holt 2004).

Recovery augmentation

This arises if species added to the community facilitate the recovery of infected individuals (γ; Fig. 1d); for instance, if added species provide resources (e.g. are mutualists or prey), hosts may maintain bodily states that enhance their response to infection. Alternatively, the addition of resource species could increase the longevity of infected individuals.

We have emphasized how non-host species can alter the rate of change in the density of infected hosts, dI/dt. Alternative measures of disease risk can at times be more useful (Box 1). For instance, risk to humans might be measured in terms of total contact rates with infected hosts, or some subset of hosts (e.g. a particular life stage), or by the proportion of encountered hosts that are infected. For the above model with a non-regulatory pathogen (the alternative form of eqn 1 with host numbers fixed at K), increased species diversity acting through the above suite of mechanisms affects both the total equilibrial density of infected hosts, and the prevalence of the infection (the proportion of individuals infected) in the same direction as for dI/dt (details not shown). For a host that is regulated solely by the disease (eqn 1), most effects of these parameter shifts are in the same direction, with the notable exception of the transmission parameters; a decrease in transmission rate due to the addition of non-host species actually leads to an *increase* in the equilibrial density of infected hosts (with lower transmission rates, more infected hosts are required to keep the host species in equilibrium).

These mechanisms of reduction or augmentation for host-specific diseases could operate in concert in real systems. For example, a predator could both reduce host movement, diminishing contacts with pathogen propagules (*encounter reduction*), and preferentially consume infected host individuals, driving disease risk down through a second pathway (*infected host mortality*). The net result would be a 'dilution effect' – a reduction of disease risk due to increased species diversity (see Box 2). Alternatively, mechanisms for reduction and augmentation could operate simultaneously; for instance, an added predator species could reduce the abundance of infected individuals while also increasing per capita contact rates between infected and susceptible hosts, resulting in an unpredictable net effect.

Examples of effects of diversity in simple disease systems

Virtually all research on the effects of diversity on specialist, non-vector-borne pathogens has focused on plants, particularly in agricultural settings, where the potential for reducing disease prevalence through crop diversity has long been recognized (e.g. Elton 1958; van der Plank 1963; Browning & Frey 1969; Barrett 1978). Several authors have recognized that plant diversity could affect disease prevalence in multiple ways (e.g. Burdon & Chilvers 1982; Finckh *et al.* 2000), though most have emphasized the fundamental importance of host density in affecting the transmission of plant diseases, and the potential role that diversity could play in influencing host density (reviews in Burdon & Chilvers 1982; Boudreau & Mundt 1997, but see Pfleeger & Mundt 1998). Recent studies in natural plant systems have reinforced these ideas. For example, Knops *et al.* (1999) and Mitchell *et al.* (2002, 2003) investigated the effects of plant species richness on the prevalence of foliar fungal diseases, each specific to a particular host plant species. In these studies, plots with high species richness had significantly lower disease severity compared with plots with low species richness. Plots with high species richness had lower host density, and statistical analysis revealed that it was low host density that reduced disease severity rather than species diversity *per se* (Fig. 2). High species richness thus appears to have reduced host density through competition, an example of *susceptible host regulation*, described above. One potential

Box 2 The dilution effect.

The term 'dilution effect' has come to have a somewhat confused meaning in the literature on disease ecology. Part of this is due to confusion about the historical origins of this concept. For example, the term 'dilution effect' appeared several times in papers on disease ecology (e.g. Power 1987; Hochberg 1991) before its first formal characterizations in the past decade (Van Buskirk & Ostfeld 1995; Norman *et al.* 1999; Ostfeld & Keesing 2000a,b), though early uses of the term do not coincide with its more recent meaning. More importantly, some of the ideas central to the dilution effect – but not the term itself – also appeared much earlier in papers on vector-borne diseases of humans (e.g. zooprophylaxis, reviewed in Service 1991) and on diseases of plants (e.g. Barrett 1978; Burdon & Chilvers 1982).

A second source of confusion about the 'dilution effect' arises from the ways it has been applied more recently. In an earlier review, Ostfeld & Keesing (2000b) described the dilution effect as occurring when the presence of a diverse assemblage of relatively inefficient reservoir hosts reduces disease risk. They then characterized four conditions necessary for this effect to occur for zoonotic diseases (diseases for which the pathogen resides primarily in non-human animal hosts) that are vector-borne. Critically, the conditions they outlined focused on a particular measure of disease risk – the proportion of vectors infected with the pathogen. This usage corresponds with that of both Van Buskirk & Ostfeld (1995) and Norman *et al.* (1999). More recently, however, the term 'dilution effect' has been applied to situations in which species diversity reduces (a) other measures of disease risk (e.g. the density of infected vectors: Allan *et al.* 2003), or (b) the prevalence of non-vector-borne diseases (e.g. Yahnke *et al.* 2001; Mitchell *et al.* 2002; Ruedas *et al.* 2004). In general, the mechanisms underlying these other applications of the term have not been clearly delineated. Furthermore, even restrictive definitions of the 'dilution effect' can require multiple underlying mechanisms (e.g. Begon in press). The term has more recently been used phenomenologically (Allan *et al.* 2003; Rudolf & Antonovics 2005).

In sum, then, we are left with two alternatives for the use of the term 'dilution effect'. In the first case, the term could be used restrictively, to apply only to situations in which a pathogen can be acquired by a variety of hosts but is transmitted efficiently by only one or a few (*sensu* Van Buskirk & Ostfeld 1995; Norman *et al.* 1999; Ostfeld & Keesing 2000a,b). This restrictive definition, however, leaves many mechanisms by which species diversity reduces disease risk undefined and with no associated lexicon. It also fails to capture situations in which diversity operates via multiple pathways, e.g. when it deflects transmission events from highly competent to less competent hosts and simultaneously regulates density of highly competent hosts. Alternatively, the term 'dilution effect' could be used inclusively, to describe the net effect of species diversity reducing disease risk by any of a variety of mechanisms (as described in the main text), and for both vector-borne and non-vector-borne diseases. Because choosing the restrictive option would require the introduction of a new term for the net effect, and because we recommend using mechanistically specific and relevant terms for specific underlying mechanisms, we suggest here that the term 'dilution effect' be used inclusively to refer to the phenomenon – the net effect – when increased species diversity reduces disease risk, as we have done throughout this paper. We further suggest that its opposite, when increased species diversity *increases* disease risk, be called an 'amplification effect'.

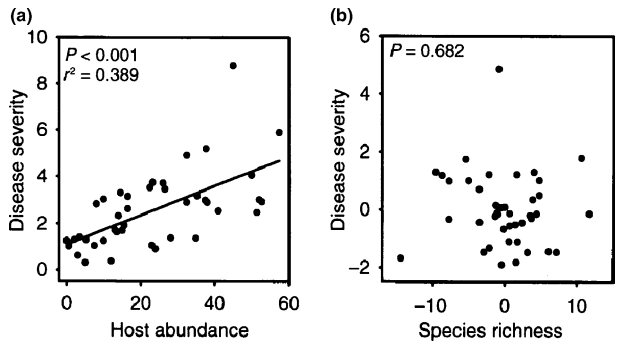

Figure 2 The effects on disease severity of (a) host abundance and (b) species richness after controlling for the effects of host abundance. The host is *Bouteloa*, an abundant C4 grass, and its foliar fungal pathogen is *Bipolaris* in experimental plots in Minnesota. Overall, species richness decreased disease severity by reducing the abundance of the specialist host; there were no significant effects of diversity beyond these effects on host abundance The mechanism of this effect is *susceptible host regulation*. Reprinted with permission from Mitchell *et al.* (2002).

effect that might also be involved is that an increase in the density of non-hosts (with no change in host density) may directly hamper disease transmission, for instance because of physical interference with the aerial movement of fungal spores – leading to *encounter reduction*, an idea previously raised by others (e.g. Burdon & Chilvers 1982).

A VECTOR-BORNE DISEASE SYSTEM

We next consider a system in which a pathogen is transmitted from host to host by a vector species, such as a mosquito, tick, flea, aphid, or mite. We will limit our analysis for now to a case where the pathogen can be harboured by a single species of host and transmitted by a single species of vector. There is no transovarial transmission of the pathogen among vector generations, so infected vectors give birth to uninfected offspring. Vectors that are exposed to the pathogen are assumed to become immediately infectious.

The dynamics of this system can be divided into two components: the loading of the pathogen onto the vector, and the loading of the pathogen from vector to host. We begin with pathogen transmission to the vector, and describe the rates of increase in uninfected (W) and infected (Z) vectors respectively, following Antonovics *et al.* (1995):

$$dW/dt = \lambda - \mu W - \alpha\delta IW, \quad (3)$$

$$dZ/dt = \alpha\delta IW - \mu Z. \quad (4)$$

For simplicity, the vector is assumed to have a constant recruitment rate (λ), and to be unaffected by the presence of the pathogen. The quantity μ is a vector death rate; as before, overall transmission from infected hosts to uninfected vectors is the product of two components, the rate of encounter of an uninfected vector with an infected host, α, and the probability that contact leads to transmission of the infection to the vector, δ.

For illustrative purposes, we assume that the host population is composed of either healthy or infected individuals, but that it is regulated by factors other than the pathogen (i.e. $S + I = K$). The dynamics of pathogen transfer from the vector to the host population are given by

$$dI/dt = \alpha\delta' SZ - (\gamma + m')I. \quad (5)$$

Compared with eqn 2, the transmission term now reflects the rate of encounters between vectors and healthy hosts, and the probability of transmission per encounter.

Non-host species can indirectly influence disease dynamics in this system by altering components of dZ/dt. The mechanisms identified in the non-vector-borne disease system described above still pertain in this case. For instance, a non-host species could reduce disease risk by decreasing density of infected hosts (I), for example by increasing the mortality rate of infected hosts, m' (*infected host mortality*); this reduces the rate of increase of infected vectors, which would then feed back to reduce the rate of increase of infected hosts. A non-host species could decrease the probability of transmission between infected hosts and vectors (*transmission reduction*). Or a non-host species could decrease risk by reducing the rate of contact between vectors and hosts, α, a form of *encounter reduction*; new dimensions of encounter reduction that can occur in a vector-borne disease system are explored below. One entirely new mechanism arises for vector-borne diseases: a non-host could regulate the size of the vector population, an effect we call *vector regulation* (see below).

Encounter reduction

Encounter reduction occurs when another species reduces the rate of contact that could lead to pathogen transmission. In the case of a vector-borne disease system, encounter reduction could occur because either (a) the presence of the added species affects *host* behaviour, reducing the probability of contact between vector and host or (b) it affects *vector* behaviour. In the latter case, the addition of a competitor or a predator of the vector could reduce movements by the vector, creating a situation analogous to that explored above for hosts with direct transmission. Moreover, if the added species is an alternative host for the vector, but not the pathogen, its presence may lure vectors away from the focal host, thus decreasing transmission. For example, a livestock species near a human dwelling might deflect mosquito meals that would otherwise be drawn from humans, resulting in less frequent contact between malarial mosquitoes and susceptible human hosts. This zooprophylaxis, described as early as 1903 (reviewed in Service 1991) and popularized by Macdonald (1957) and the World Health Organization (1982), has also been explored for diseases other than malaria (e.g. Cecere *et al.* 1997).

Vector regulation

This occurs when an added species reduces vector density, for instance because of mortality imposed on the vector. For instance, grooming by the host is an important cause of mortality for many vector species (e.g. Shaw *et al.* 2003); many hosts for these ectoparasite species can also mount immune responses to salivary antigens of the vector, which can attack the vector and reduce its survival (Randolph 1979). Alternatively, an added species could increase vector abundance by providing additional sources of vector meals (e.g. Cecere *et al.* 1997; Norman *et al.* 1999; Gilbert *et al.* 2001; Schmidt & Ostfeld 2001; Saul 2003; Fig. 3).

We have focused on how increased species diversity could reduce the rate of change in the density of infected vectors, dZ/dt. An alternative measure of risk might be the equilibrial abundance of infected vectors. For the above model, where the pathogen regulates neither the vector nor the host population, it can be shown that the equilibrial abundance of

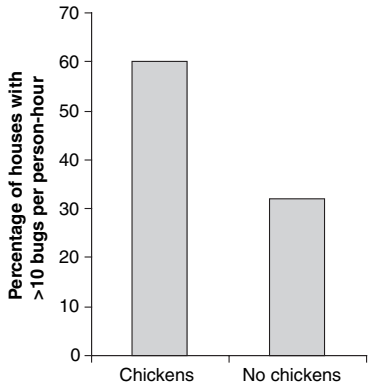

Figure 3 Effects of the presence of chickens in houses on the density of *Triatoma infestans*, the vector of Chagas disease in Argentina. Houses with chickens present had significantly higher densities of bugs than did houses without chickens, an example of species richness increasing vector density (*vector augmentation*). Figure drawn from data in Cecere *et al.* 1997.

fected vectors changes in parallel with dZ/dt, decreasing if there is a decrease in vector recruitment rates, host carrying capacity, contact rates or transmission probability per contact, or an increase in vector mortality rates (details not shown). Other species in the community can shift the abundance of infected vectors via changes in any of these quantites.

Examples from vector-borne disease systems

Relatively, few studies have documented the effects of diversity on vector-borne diseases with specialist pathogens, and those that have have focused on plants. Pitre & Boyd (1970) found that the presence of weeds in corn (*Zea mays*) fields in Mississippi deflected aphid (*Graminella nigrifons*) vectors of corn smut virus away from corn plants (which could be called 'phytoprophylaxis'), a form of *encounter reduction*. Because the weeds were preferred breeding sites for the aphids; however, the weedy fields had higher abundances of aphids (*vector augmentation*). The net effect of these two mechanisms operating together was a reduction in disease prevalence in the weedy (more diverse) fields – a dilution effect. In a similar system in Nicaragua, Power (1987) found that crop diversity reduced the prevalence of corn stunt on maize (*Z. mays*) with a leafhopper vector (*Dalbulus maidis*), but the mechanism behind these effects was not clear. Farrell (1976) also found a dilution effect for rosette virus disease of groundnuts (*Arachis hypogaea*) in Malawi. This virus, which is transmitted by an aphid vector (*Aphis craccivora*), was more prevalent in groundnut monocultures than in fields with both groundnuts and field beans (*Phaseolus vulgaris*), when treatments controlled for host plant density. Apparently, many aphids got trapped on the bean plants (*vector regulation*) and thus could not subsequently infect the groundnut hosts for the virus (*encounter reduction*).

MULTI-HOST SYSTEMS

More broadly, both vector-borne and non-vector-borne pathogens often are generalists that can infect multiple species of hosts (Woolhouse *et al.* 2001; Power & Flecker in press). Both theoretical and empirical investigations have demonstrated that the presence of a pathogen shared by multiple host species can influence host species diversity (Holt & Pickering 1985; Dobson & Hudson 1986; Bowers & Begon 1991; Begon *et al.* 1992; Grosholz 1992; Begon & Bowers 1994; Hudson & Greenman 1998; Power & Mitchell 2004; Rudolf & Antonovics 2005), but only more recently have the effects of host species diversity on the dynamics of the pathogen been explicitly considered (Holt *et al.* 2003; Dobson 2004; Rudolf & Antonovics 2005). This latter interest arose in part from modelling studies that suggested that under some conditions, the presence of a second host species could increase infection in a focal host species (Bowers & Begon 1991; Begon *et al.* 1992, but see Begon & Bowers 1994). The effect on pathogen prevalence of adding host species has remained a subject of debate and both theoretical and empirical investigation.

In general, the net effect of diversity upon disease dynamics in a focal host is likely to depend on the properties of that species relative to the entire community. For example, imagine a focal host species infected by a pathogen that can also infect other host species. If the focal host is a poor reservoir (i.e. does not transmit the pathogen effectively), adding other host species to the community might increase the prevalence of the pathogen in the focal host because the added hosts will be better reservoirs ('spillover' *sensu* Daszak *et al.* 2000; Power & Mitchell 2004). To illustrate this, humans alone cannot sustain rabies because human-to-human transmission is negligible. The presence of additional species that can infect humans (e.g. raccoons); however, can sustain infection in humans because of animal-to-human transmission (*encounter augmentation*). In an experimental plant community, Power & Mitchell (2004) found that host communities containing grass species that were poor reservoirs had low rates of infection with barley yellow dwarf virus. More diverse systems had higher rates of infection because they contained a highly competent reservoir for the virus – the wild oat, *Avena fatua* (Fig. 4). Examples like these demonstrate how species diversity can sometimes amplify disease prevalence.

On the other hand, imagine a focal host species that is a highly competent reservoir. Adding host species that are less competent reservoirs might decrease disease risk if those added species decrease the probability of encounter between the pathogen and the focal host species (*encounter reduction*). This could occur, for example, for an environmentally transmitted disease, if the added species removed

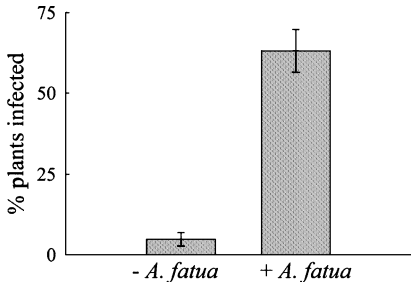

Figure 4 Effects of the presence of a particular host species, the wild oat *Avena fatua*, on the prevalence of barley yellow dwarf virus, a generalist plant pathogen. Experimental communities containing *A. fatua* had higher disease prevalence regardless of overall host species richness, demonstrating that the presence of one host that transmits the pathogen at a particularly high rate can be more important than species diversity *per se*. Reprinted with permission from Power & Mitchell (2004).

pathogens in a free-living, depletable pool of propagules. Intestinal parasites/pathogens that accumulate in latrines (e.g. raccoon roundworm, *Baylisascaris procyonis*; LoGiudice 2003) or decomposing tissues from infected carcasses (e.g. chronic wasting disease; T. Hobbs, personal communication) both serve as sites of infection, but both are at least potentially depletable by repeated visits from species that can harbour, but do not readily transmit, the propagules. These examples illustrate how host diversity could decrease disease prevalence.

Frequency-dependent vs. density-dependent transmission

Because of contradictory examples such as these, several recent theoretical studies have attempted to delineate under what general conditions host diversity should increase or decrease disease prevalence (Holt *et al.* 2003; Dobson 2004; Rudolf & Antonovics 2005). One key factor appears to be whether transmission of the pathogen is a function of the absolute density of infected hosts (density-dependent), or whether it is a function of the proportion of the total population that is infected with the pathogen (frequency-dependent). Density-dependent models of transmission are generally used to characterize diseases that are spread through environmental propagules or through random contact among individuals. Frequency-dependent transmission models are typically used to characterize sexually transmitted diseases (Getz & Pickering 1983; Thrall *et al.* 1993), because the number of sexual contacts is likely to be fixed, regardless of population density. Vector-borne diseases are also frequently considered to conform broadly to frequency-dependent models of transmission (e.g. Thrall *et al.* 1993), a situation that would apply if the number of contacts between vectors and hosts is fixed, e.g. because vectors actively search for their hosts and compensate for decreased density of hosts by increasing searching distances (Power 1987; Antonovics *et al.* 1995; Rudolf & Antonovics 2005).

Dobson (2004) and Rudolf & Antonovics (2005) argued that the effect on disease prevalence of adding host species will differ depending on whether the disease is characterized by density-dependent or frequency-dependent transmission. If pathogen transmission is density-dependent, adding hosts will typically decrease disease risk only if the added hosts reduce the abundance of the focal host (*susceptible host regulation*), assuming that transmission between species is lower than transmission within species. On the other hand, if the pathogen is transmitted in a frequency-dependent manner, adding hosts will decrease disease risk whether or not the added hosts reduce the abundance of the focal host. This is because adding a host species decreases the proportion of all infected individuals in the host community, resulting in a reduction in the number of contacts between susceptible and infected individuals (*encounter reduction*), again assuming that transmission between species is lower than transmission within species.

Between- vs. within-species transmission

The assumption that transmission is higher within species than between species is common to virtually all models of pathogen transmission among multiple host species and can even be required for host coexistence (e.g. Holt & Pickering 1985; Bowers & Begon 1991; Begon *et al.* 1992; Begon & Bowers 1994; Dobson 2004; Rudolf & Antonovics 2005). This assumption appears to be appropriate in many cases (Begon *et al.* 1999; Woolhouse *et al.* 2001). It also appears to be a necessary condition for host diversity to decrease disease risk. In cases with higher between- than within-species transmission, host diversity may increase disease prevalence. For example, Rhodes *et al.* (1998) found that side-striped jackal (*Canis adustus*) populations in Zimbabwe could not support rabies virus unless they were frequently reinoculated through contact with infected domestic dogs (*encounter augmentation*). Similarly, Caley & Hone (2004) used field and modelling efforts to establish that bovine tuberculosis (pathogen *Mycobacterium bovis*) in New Zealand was being maintained in low-density feral ferrets (*Mustela furo*) only through their contact with brushtail possums (*Trichosurus vulpecula*).

Holt *et al.* (2003) explored the consequences of relative rates of between- and within-species transmission for pathogen establishment in communities composed of two hosts. With only one host species and density-dependent transmission, there is a threshold density of that host above which the pathogen can become established; for pairs of hosts, there are various combined densities that permit establishment, depending on the amount of interspecific pathogen transmission. For example, at one extreme, if there is no interspecific transmission, at least one of the hosts must occur at or above its threshold density for the pathogen to become established. In contrast, if between-species transmission is greater than within-species transmission, the combination of host species more readily permits pathogen establishment than does either species alone – an example of disease amplification with increasing diversity. They also considered the possibility that one host cannot sustain the infection and, moreover, decreases the rate at which the other host becomes infected – an example of diversity diluting disease prevalence. In this case, as the density of the second host increases, the density of the first host required for pathogen establishment also increases. According to Holt *et al.* (2003), this latter situation is most plausible if transmission is via vectors or a depletable pool of environmental propagules.

Modelling of multi-host systems

While there is great current interest in the effects of host diversity on pathogen dynamics, most models of such systems are restricted to three species – a pathogen and two hosts. More comprehensive models quickly become analytically challenging (Begon & Bowers 1995; Hudson & Greenman 1998; Grenfell *et al.* 2002). Although some models have included more species (e.g. Bowers & Begon 1991; Gilbert *et al.* 2001; LoGiudice *et al.* 2003), the development of new statistical and analytical approaches may be necessary before significant progress can be made (Hudson & Greenman 1998; Grenfell *et al.* 2002). However, because parameters from single-host models also arise in multi-host models, the component processes we have explored above in a single host model should also pertain to systems involving a multiplicity of species, while additional mechanisms may also arise. In the next section, we consider how specific mechanisms can operate in concert in real disease systems to either amplify or dilute disease risk.

Examples from multi-host systems

Most empirical investigations of the effects of diversity on disease risk have focused on vector-borne pathogens, despite the potential for host diversity to also influence the prevalence of pathogens that are directly or environmentally transmitted. Two recent studies of non-vector-borne diseases suggest that host diversity can reduce disease risk, though the mechanisms underlying these effects are not clear. In a study of the ecology of Laguna Negra virus (the aetiological agent for hantavirus pulmonary syndrome in Paraguay), Yahnke *et al.* (2001) found that host communities that had high proportions of the most competent reservoir – the vesper mouse, *Calomys laucha* – also had the highest antibody prevalence in this reservoir. Virus transmission appears to be primarily through direct contact (Yahnke *et al.* 2001). Thus, the probability of conspecific encounters between *C. laucha* individuals, and hence of potential transmission events, may have decreased as the relative abundance of this species declined with increasing diversity (*encounter reduction*). If there is a relatively fixed number of contacts per individual host, one expects frequency dependent transmission, and these results would then conform to the expectations of Dobson (2004) and Rudolf & Antonovics (2005). In a study of another hantavirus, Choclo virus, in Panamá, Ruedas *et al.* (2004) found that at sites where the virus was present (either in humans or wildlife), the host community was less diverse than at comparable sites where no virus was found, suggesting that diversity reduced disease prevalence. The mechanisms for this effect were not clear.

In a recent study of a vector-borne disease of wildlife, Telfer *et al.* (2005) found that the presence of bank voles (*Clethrionomys glareolus*) reduced the infection prevalence in wood mice (*Apodemus sylvaticus*) of species of *Bartonella*, bacteria vectored by fleas. Bank voles appear to be poor reservoirs for the pathogen, but good hosts for the flea vector. Flea prevalence did not increase with overall rodent density, suggesting that *vector augmentation* did not occur. Importantly, fleas were less abundant on wood mice when bank voles were present and wood mice were at high densities, strongly suggesting that *encounter reduction* between fleas and hosts may have taken place in this system.

Lyme disease, a vector-borne zoonosis in which a spirochete bacterium, *Borrelia burgdorferi*, is passed from host to host by the bite of an ixodid tick, provides one of the best-studied examples of the effects of host diversity on disease risk. The tick vectors in this system feed on a wide variety of vertebrate hosts, but the white-footed mouse (*Peromyscus leucopus*) is the most competent reservoir for the pathogen in eastern North America. Mice appear to be particularly abundant in small forest fragments because their predators and competitors are absent or scarce (Nupp & Swihart 1996; Krohne & Hoch 1999; Rosenblatt *et al.* 1999), providing examples of *susceptible host regulation* and *infected host mortality*. *Encounter reduction* also appears to operate in this system. When the density of chipmunks, an alternative host for ticks, is high, the number of ticks on mice is lower (Fig. 5; Schmidt *et al.* 1999; Ostfeld et al in press). This

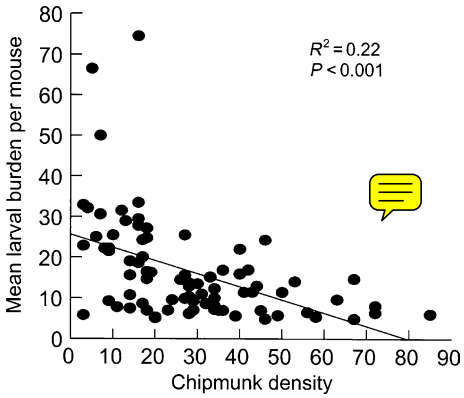

Figure 5 The effect of eastern chipmunk (*Tamias striatus*) density, varying across years and sites, on the average number of larval blacklegged ticks (*Ixodes scapularis*) infesting white-footed mice (*Peromyscus leucopus*) in eastern New York state. In years of low chipmunk density, tick burdens on mice were variable. In years of high chipmunk density, however, tick burdens on mice were always low, suggesting that an abundance of an alternative host for the ticks reduced rates of encounter between ticks and white-footed mice, the most competent reservoir for the Lyme bacterium (*Borrelia burgdorferi*). These data provide an example of *encounter reduction* – a decline in encounters that could lead to infection as a result of increasing species diversity (see main text). Reprinted with permission from Ostfeld *et al.* (in press).

suggests that the presence of another species at high density (an increase in species evenness rather than species richness) reduces encounters between the vector and the most competent reservoir for the pathogen, though the evidence for this mechanism comes from correlative rather than experimental data. The presence of alternative (non-mouse) hosts in diverse host communities can lead to *vector regulation*, because ticks that feed on mice are more likely to survive to moult than are ticks that feed on some other hosts (Randolph 1979; Craig *et al.* 1996; LoGiudice *et al.* 2003). Thus, in a diverse community, ticks feed on a greater number of hosts, and these alternative hosts decrease their survival. Schauber & Ostfeld (2002) suggested that *transmission reduction* might also operate in the Lyme disease system.

Allan *et al.* (2003) tested for a net effect of all of these mechanisms by evaluating disease risk in forest fragments in upstate New York. They predicted that the smallest fragments would have high densities of infected ticks, and thus high disease risk, because the small fragments had high densities of white-footed mouse due to lower predation and/or competition; and (b) more tick meals being taken on mice because of loss of both *encounter reduction* and *vector regulation*. Consistent with these predictions, they found that densities of infected ticks were more than four times higher in small fragments than in larger fragments.

For louping ill, a similar tick-borne disease system, several studies (Norman *et al.* 1999; Gilbert *et al.* 2001; Laurenson *et al.* 2003) describe the results of modelling and empirical investigations in which the louping ill virus is transmitted among hosts by the bite of another ixodid tick (*Ixodes ricinus*). The roles of hosts in this system are complex: only sheep (*Ovis aries*) and red grouse (*Lagopus lagopus*) produce sufficient viraemia to pass the viral infection to ticks, but mountain hares (*Lepus timidus*) can both transmit the infection through co-feeding ticks and also sustain the vector population (Gilbert *et al.* 2001). Red deer (*Cervus elephus*) do not transmit the virus, but are the primary host for the tick vector and thus can sustain the tick population. Norman *et al.* (1999) and Gilbert *et al.* (2001) found that intermediate abundances of a non-viraemic host for the tick vector (e.g. red deer) permit viral persistence in a viraemic host (e.g. grouse), whereas high or low abundances lead to viral fadeout. At low deer abundance, there are too few ticks to sustain the pathogen (*vector regulation*); at high deer abundance, tick bites get 'wasted' on the non-viraemic deer (*encounter reduction*), and the pathogen cannot persist.

Whether the *Bartonella*, louping ill and Lyme disease systems conform to the predictions of Dobson (2004) and Rudolf & Antonovics (2005) is not clear because the relationship between host and vector abundances is not known. As Dobson (2004) pointed out, the net effect of host diversity for vector-borne diseases will be in part a consequence of whether vector abundance is a function of host abundance. In some cases, e.g. mosquitoes, vector abundance may be independent of host abundance (and limited instead by, for example, availability of breeding sites; Dobson 2004). Recent studies of the use of zooprophylaxis – the addition of non-human hosts to siphon vector meals away from humans – for malaria mitigation suggest that even for mosquitoes, this conclusion might not be straightforward, given that in some situations, adding hosts increases mosquito density (e.g. Saul 2003). Whether the abundance of tick vectors is a function of host abundance remains controversial (Van Buskirk & Ostfeld 1995; Norman *et al.* 1999; Gilbert *et al.* 2001; Schmidt & Ostfeld 2001, R.S. Ostfeld, personal communication). For example, white-tailed deer (*Odocoileus virginianus*) are the primary hosts for adult ticks in eastern North America, and in areas (e.g. islands) where they have been extirpated, tick abundance is essentially zero. However, an empirically based model developed by Van Buskirk & Ostfeld (1995) found that even very small numbers of deer sustained substantial tick populations, suggesting that tick abundance is not a linear function of deer abundance. A crucial area requiring attention is the determination of what factors limit and regulate vector populations.

In cases where vector abundance is independent of host density, frequency-dependent rather than density-dependent transmission may best describe transmission dynamics (Dobson 2004); these diseases would be predicted to show reduced disease prevalence with increasing diversity (Dobson 2004; Rudolf & Antonovics 2005). Cases in which vector abundance is dependent on host abundance are more complex, and outcomes are much less easy to predict (Van Buskirk & Ostfeld 1995; Norman *et al.* 1999; Gilbert *et al.* 2001; Schmidt & Ostfeld 2001; Dobson 2004), especially when there are nonlinearities in the relationship between host abundance and vector abundance (Van Buskirk & Ostfeld 1995; Norman *et al.* 1999). In these cases, a simple tally of species presence/absence may be insufficient to gauge the importance of diversity for disease dynamics in a focal host species, because different processes dominate at different population sizes. Going from zero to low densities, an alternative host that is critical to vector dynamics might boost vector numbers, leading to an increase in disease in the focal host species. However, if the alternative host is ineffective at sustaining the pathogen, further increases may lead to a reduction in disease prevalence (Van Buskirk & Ostfeld 1995; Norman *et al.* 1999; Gilbert *et al.* 2001). Similar non-monotonic effects arise broadly in trophic interactions due to the interplay of nonlinear functional and numerical responses (Holt 1997).

DISCUSSION

We have described a suite of general mechanisms through which increases in species diversity in a disease system could decrease overall disease risk (dilution), or at times increase risk (amplification). We have argued that these mechanisms could occur with pathogens transmitted through direct host–host contact, through environmental contact with free-living pathogen propagules, or through vector–host interactions. An additional mechanism arises in vector-borne disease systems, and the interplay of these mechanisms can lead to complex patterns of disease risk shifting along gradients in species diversity.

Clearly, the existence of disease requires at least two species (pathogen and host), and vector-borne diseases require a minimum of three species. Our discussions of the interactions between diversity and disease risk assume that the minimal level of diversity necessary to support a particular disease exists, and we have assessed the consequences of changes in diversity beyond this minimal level.

One key question in multi-host disease systems is whether the most competent reservoir is present in species-poor communities. If so, species added to these communities have, by definition, lower (if any) reservoir competence and thus have the potential to decrease disease risk. If the most competent reservoir is not present in species-poor communities, by contrast, then an increase in diversity could include the addition of the most competent reservoir itself, which is likely to result in an amplification of disease risk. Ostfeld & Keesing (2000b) considered evidence that the most competent reservoir for a variety of vector-borne zoonoses was typically present in species-poor communities. They found support for this assumption for two diseases (Lyme disease and zoonotic cutaneous leishmaniasis), but insufficient evidence to evaluate it for others. To our knowledge, the generality of this pattern for non-vector-borne diseases has not been established, though there is suggestive evidence for both Sin Nombre and Laguna Negra viruses, the aetiological agents of Hantavirus pulmonary syndrome in the south-western USA and South America, respectively (Mills *et al.* 1998; Yahnke *et al.* 2001). We suspect that this phenomenon might be widespread, and result from evolutionary bias by pathogens towards widespread, abundant hosts (Ostfeld & Keesing 2000b).

Disease risk could in theory be reduced by manipulating abundances of specific host species (Fig. 4), or via habitat modifications that alter parameters such as encounter rates. For instance, risk could be most strongly reduced by the addition of the most effective 'dilution host' (LoGiudice *et al.* 2003) rather than by the addition of many host species chosen at random (i.e. by increasing diversity *per se*). Thus, in principle, management of single species (e.g. dilution hosts or predators on the most competent reservoir) could create a dilution effect without strongly increasing diversity. In agricultural settings, manipulation of disease prevalence through the addition of particular species can be both tractable and effective (but not always; see Boudreau & Mundt 1997), and there are also opportunities for disease mitigation through targeted manipulation of diversity in livestock production (e.g. foot and mouth disease in the UK; Keeling *et al.* 2003). But in natural systems, insufficient information will usually be available to craft such a highly targeted strategy, and broader measures to foster the maintenance of species richness will be needed (Ostfeld & LoGiudice 2003). For instance, large blocks of continuous habitat often support higher species diversity than do small patches in a fragmented landscape (Rosenblatt *et al.* 1999). Rich communities are more likely to maintain species that can impact the abundance of the most competent reservoir for zoonoses (e.g. predators that can limit the numbers of their rodent prey, Ostfeld & Holt 2004). The efficiency of this broad-brush strategy to controlling disease can only be assessed by a larger number of systematic studies of how disease risk in focal disease systems varies along gradients in species richness and composition.

As is clear from the examples we have provided above, most studies of the effects of species diversity on disease risk have focused on just a few of the mechanisms we have described: *encounter reduction*, *susceptible host regulation*, and *vector regulation*. In contrast, to our knowledge, there are no examples in the literature of *recovery augmentation* (an increase in rate of recovery from infection as a result of increased species diversity), though such an effect seems plausible. Similarly, there are virtually no examples of *transmission reduction* (but see Schauber & Ostfeld 2002). We suggest that the exploration of the existence of these mechanisms in particular disease systems is one important area for future research.

Many disease systems are much more complex than our simple illustrative models, and their complexities may introduce additional mechanisms through which diversity could affect disease risk. For example, models with nonlinearities in transmission dynamics (Hochberg 1991; Rosà *et al.* 2003), and latent periods following infection of vectors (Anderson & May 1981) include parameters that could be influenced by the addition of species; moreover, many disease systems involve multiple species of vectors or pathogens (e.g. Hochberg & Holt 1990). Additional mechanisms might also arise for diseases caused by macroparasites that require multiple hosts to complete their life cycles. Because the parameters from our basic model are relevant in a variety of other types of disease systems, we argue that the mechanisms presented here are likely to be observed across a diversity of systems, while additional mechanisms might arise as well.

By deconstructing the terms of simple host–pathogen models, we have attempted to characterize the rich diversity of mechanisms by which increased diversity in host communities might decrease or increase disease risk, even in host-specific disease systems. Systems involving multiple host species, or vectors, have an even richer array of such mechanisms. The mechanisms suggested by these models appear plausible; some have now been demonstrated to operate in specific systems. We suggest that future research on mechanisms should focus on: (1) describing patterns of change in disease risk with changing diversity; (2) identifying the mechanisms responsible for observed changes in risk; (3) clarifying additional mechanisms in a wider range of epidemiological models; and (4) experimentally manipulating disease systems to assess the impact of proposed mechanisms. An additional frontier is to explore the degree to which pathogen diversity is a function of host diversity, and how the existence of multiple parasites influences both underlying mechanisms and net effects of diversity on the dynamics of specific diseases.

Recent syntheses (Holt *et al.* 2003; Dobson 2004; Rudolf & Antonovics 2005) have made some progress in identifying key factors that will determine the net effect of increased host diversity in multi-host disease systems, particularly whether pathogen transmission is density-dependent or frequency-dependent, and whether pathogen transmission within species exceeds that between species. Key directions for future research in multi-host disease systems include determining the applicability of these generalizations to specific disease systems, and developing analytical techniques for tackling complex models of multi-host systems.

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REFERENCES

Allan, B.F., Keesing, F. & Ostfeld, R.S. (2003). Effects of habitat fragmentation on Lyme disease risk. *Cons. Biol.*, 17, 267–272.

Anderson, R.M. (1982). Epidemiology. In: *Modern Parasitology* (ed. Cox, F.E.G.). Blackwell Scientific Publications, Oxford, pp. 204–225.

Anderson, R.M. & May, R.M. (1981). The population dynamics of microparasites and their invertebrate hosts. *Philos. Trans. R. Soc. Lond. Ser. B*, 291, 451–524.

Antonovics, J., Iwasa, Y. & Hassell, M.P. (1995). A generalized model of parasitoid, venereal, and vector-based transmission processes. *Am. Nat.*, 145, 661–675.

Barrett, J.A. (1978). A model of epidemic development in variety mixtures. In: *Plant Disease Epidemiology* (eds. Scott, P.R. & Bainbridge, A.). Blackwell, Oxford, pp. 129–137.

Begon, M. (in press). Effects of host diversity on disease dynamics. In: *Infectious Disease Ecology: Effects of Ecosystems on Disease and of Disease on Ecosystems* (eds Ostfeld, R.S., Keesing, F. & Eviner, V.). Princeton University Press, Princeton, NJ.

Begon, M. & Bowers, R.G. (1994). Host–host–pathogen models and microbial pest control: the effect of host self-regulation. *J. Theor. Biol.*, 169, 275–287.

Begon, M. & Bowers, R.G. (1995). Beyond host–pathogen dynamics. In: *Ecology of Infectious Diseases in Natural Populations* (eds. Grenfell, B.T. & Dobson, A.P.). Cambridge University Press, Cambridge, pp. 478–509.

Begon, M., Bowers, R.G., Kadianakis, N. & Hodgkinson, D.E. (1992). Disease and community structure: the importance of host self-regulation in a host–host–pathogen model. *Am. Nat.*, 139, 1131–1150.

Begon, M., Hazel, S.M., Baxby, D., Bown, K., Cavanagh, R., Chantrey, J. *et al.* (1999). Transmission dynamics of a zoonotic pathogen within and between wildlife host species. *Proc. R. Soc. Lond. B*, 266, 1939–1945.

Boudreau, M.A. & Mundt, C.C. (1997). Ecological approaches to disease control. In: *Environmentally Safe Approaches to Crop Disease Control* (ed. Rechcigl, N.A. & J.E. Rechcigl). CRC Publications, Boca Raton, FL, USA, pp. 33–62.

Bowers, R.G. & Begon, M. (1991). A host–host–pathogen model with free-living infective stages, applicable to microbial pest control. *J. Theor. Biol.*, 148, 305–329.

Browning, J.A. & Frey, K.J. (1969). Multiline cultivars as a means of disease control. *Ann. Rev. Phytopath.*, 59, 355–382.

Burdon, J.J. & Chilvers, G.A. (1982). Host density as a factor in plant disease ecology. *Ann. Rev. Phytopath.*, 20, 143–166.

Caley, P.J. & Hone, J. (2004). Disease transmission between and within species, and the implications for disease control. *J. Appl. Ecol.*, 41, 94–104.

Cecere, M.C., Gürtler, R.E., Chuit, R. & Cohen, J. (1997). Effects of chickens on the prevalence of infestation and population density of *Triatoma infestans* in rural houses of northwest Argentina. *Med. Vet. Entomol.*, 11, 383–388.

Craig, L.E., Norris, D.E., Sanders, M.L., Glass, G.E. & Schwartz, B.S. (1996). Acquired resistance and antibody response of raccoons (*Procyon lotor*) to sequential feedings of *Ixodes scapularis* (Acari: Ixodidae). *Vet. Parasitol.*, 63, 291–301.

Daszak, P., Cunningham, A.A. & Hyatt, A.D. (2000). Emerging infectious diseases of wildlife: threats to biodiversity and human health. *Science*, 287, 443–449.

Dobson, A.P. (2004). Population dynamics of pathogens with multiple host species. *Am. Nat.*, 164, S64–S78.

Dobson, A.P. & Hudson P.J. (1986). Parasites disease and the structure of ecological communities. *TREE*, 1, 11–15.

Elton, C.S. (1958). *The Ecology of Invasions by Animals and Plants*. Methuen & Co., London.

Farrell, J.A.K. (1976). Effects of intersowing with beans on the spread of groundnut rosette virus by *Aphis craccivora* Koch (Hemiptera, Ahpididae) in Malawi. *Bull. Entom. Res.*, 66, 331–333.

Finckh, M.R., Gacek, E.S., Goyeau, H., Lannou, C., Merz, U., Mundt, C.C. *et al.* (2000). Cereal variety and species mixtures in practice, with emphasis on disease resistance. *Agronomie*, 20, 813–837.

Getz, W.M. & Pickering, J. (1983). Epidemic models: thresholds and population regulation. *Am. Nat.*, 121, 892–898.

Gilbert, L., Norman, R., Laurenson, K., Reid, H.W. & Hudson, P.J. (2001). Disease persistence and apparent competition in a three-host community: an empirical and analytical study of large-scale,wild populations. *J. Anim. Ecol.*, 70, 1053–1061.

Grenfell, B.T., Amos, W., Arneberg, P., Bjornstad, O.N., Greenman, J.V., Harwood, J. *et al.* (2002). Visions for future research in wildlife epidemiology. In: *Ecology of Wildlife Diseases* (eds Hudson, P., Rizzoli, A., Grenfell, B., Heesterbeek, H., Dobson, A.P.). Oxford University Press, Oxford, pp. 151–164.

Grosholz, E.D. (1992). Interactions of intraspecific, interspecific, and apparent competition with host–pathogen population dynamics. *Ecology*, 73, 507–514.

Hochberg, M.E. (1991). Non-linear transmission rates and the dynamics of infectious disease. *J. Theor. Biol.*, 153, 301–321.

Hochberg, M.E. & Holt, R.D. (1990). The coexistence of competing parasites. 1. The role of cross-species infection. *Am. Nat.*, 136, 517–541.

Holt, R.D. (1997). Community modules. In: *Multitrophic Interactions in Terrestrial Ecosystems, 36th Symposium of the British Ecological Society* (eds Gange, A.C. & Brown, V.K.). Blackwell, Oxford, UK, pp. 333–349.

Holt, R.D. & Pickering, J. (1985). Infectious disease and species coexistence: a model of Lotka–Volterra form. *Am. Nat.*, 126, 196–211.

Holt, R.D., Dobson, A.P., Begon, M., Bowers, R.G. & Schauber, E.M. (2003). Parasite establishment in host communities. *Ecol. Lett.*, 6, 837–842.

Hudson, P. & Greenman, J. (1998). Competition mediated by parasites: biological and theoretical progress. *TREE*, 13, 387–390.

Keeling, M.J., Woolhouse, M.E.J., May, R.M., Davies, G. & Grenfell, B. (2003). Modelling vaccination strategies against foot-and-mouth disease. *Nature*, 421, 136–142.

Knops, J.M.H., Tilman, D., Haddad, N.M., Naeem, S., Mitchell, C.E., Haarstad, J. *et al.* (1999). Effects of plant species richness on invasion dynamics, disease outbreaks, insect abundances and diversity. *Ecol. Lett.*, 2, 286–293.

Krohne, D.T. & Hoch, G.A. (1999). Demography of *Peromyscus leucopus* populations on habitat patches: the role of dispersal. *Can. J. Zool.*, 77, 1247–1253.

Lafferty, K. & Holt, R.D. (2003). How should environmental stress affect the population dynamics of disease?. *Ecol. Lett.*, 6, 654–664.

Laurenson, M.K., Norman, R.A., Gilbert, L., Reid, H.W. & Hudson, P.J. (2003). Identifying disease reservoirs in complex systems: mountain hares as reservoirs of ticks and louping-ill virus, pathogens of red grouse. *J. Anim. Ecol.*, 72, 177–185.

LoGiudice, K. (2003). Trophically transmitted parasites and the conservation of small populations: Raccoon roundworm and the imperiled Allegheny woodrat. *Conserv. Biol.*, 17, 258–266.

LoGiudice, K., Ostfeld, R.S., Schmidt, K.A. & Keesing, F. (2003). The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. *Proc. Natl. Acad. Sci.*, 100, 567–571.

Loreau, M., Naeem, S., Inchausti, P., Bengtsson, J., Grime, J.P., Hector, A. *et al.* (2001). Biodiversity and ecosystem functioning: current knowledge and future challenges. *Science*, 294, 804–808.

Macdonald, G. (1957). *The Epidemiology and Control of Malaria*. Oxford University Press, London.

Mills, J.N., Johnson, J.M., Ksiazek, T.G. *et al.* (1998). A survey of hantavirus antibody in small-mammal populations in selected United States National Parks. *Am. J. Trop. Med. Hyg.*, 58, 525–532.

Mitchell, C.A., Tilman, D. & Groth, J.V. (2002). Effects of grassland plant species diversity, abundance, and composition on foliar fungal disease. *Ecology*, 83, 1713–1726.

Mitchell, C.A., Reich, P.B., Tilman, D. & Groth, J.V. (2003). Effects of elevated CO_2, nitrogen deposition, and decreased species diversity on foliar fungal plant disease. *Global Change Biol.*, 9, 438–451.

Norman, R., Bowers, R.G., Begon, M. & Hudson, P.J. (1999). Persistence of tick-borne virus in the presence of multiple host species: tick reservoirs and parasite-mediated competition. *J. Theor. Biol.*, 200, 111–118.

Nupp, T.E. & Swihart, R.K. (1996). Effect of forest patch area on population attributes of white-footed mice (*Peromyscus leucopus*) in fragmented landscapes. *Can. J. Zool.*, 74, 467–472.

Ostfeld, R.S. & Holt, R.D. (2004). Are predators good for your health? Evaluating evidence for top-down regulation of zoonotic disease reservoirs. *Front. Ecol. Evol.*, 2, 13–20.

Ostfeld, R.S. & Keesing, F. (2000a). Biodiversity and disease risk: the case of Lyme disease. *Conserv. Biol.*, 14, 722–728.

Ostfeld, R.S. & Keesing, F. (2000b). The function of biodiversity in the ecology of vector-borne zoonotic diseases. *Can. J. Zool.*, 78, 2061–2078.

Ostfeld, R.S. & LoGiudice, K. (2003). Community disassembly, biodiversity loss, and the erosion of an ecosystem service. *Ecology*, 84, 1421–1427.

Ostfeld, R.S. & Mills, J.N. (in press). Social behavior, demography, and rodent-borne pathogens. In: *Rodent Societies* (eds Wolff, J.O. & Sherman, P.W.). University of Chicago Press, Chicago, IL.

Ostfeld, R.S., Keesing, F. & LoGiudice, K. (in press). Community ecology meets epidemiology: the case of Lyme disease. In: *Disease Ecology: Community Structure and Pathogen Dynamics* (eds Collinge, S. & Ray, C.). Oxford University Press, Oxford.

Packer, C., Holt, R.D., Hudson, P.J., Lafferty, K.D. & Dobson, A.P. (2003). Keeping the herds healthy and alert: implications of predator control for infectious disease. *Ecol. Lett.*, 6, 797–802.

Pfleeger, T.G. & Mundt, C.C. (1998). Wheat leaf rust severity as affected by plant density and species proportion in simple communities of wheat and wild oats. *Phytopathology*, 88, 708–714.

Pitre, H.N. & Boyd, F.J. (1970). A study of the role of weeds in corn fields in the epidemiology of corn stunt disease. *J. Econ. Entom.*, 63, 195–197.

van der Plank, J.E. (1963). *Plant Diseases: Epidemics and Control*. Academic Press, New York.

Power, A.G. (1987). Plant community diversity, herbivore movement, and an insect-transmitted disease of maize. *Ecology*, 68, 1658–1669.

Power, A.G. & Flecker, A.S. (in press). Effects of vector diversity on disease dynamics. In: *Infectious Disease Ecology: Effects of Ecosystems on Disease and of Disease on Ecosystems* (eds Ostfeld, R.S., Keesing, F. & Eviner, V.). Princeton University Press, Princeton, NY.

Power, A.G. & Mitchell, C.E. (2004). Pathogen spillover in disease epidemics. *Am. Nat.*, 164, S79–S89.

Randolph, S.E. (1979). Population regulation in ticks: the role of acquired resistance in natural and unnatural hosts. *Parasitology*, 79, 141–156.

Rhodes, C.J., Atkinson, R.P.D., Anderson, R.M. & Macdonald, D.W. (1998). *Rabies in Zimbabwe: Reservoir Dogs and the Implications for Disease Control*. 353, 999–1010.

Rosà, R., Pugliese, A., Norman, R. & Hudson, P.J. (2003). Thresholds for disease persistence in models for tick-borne infections including non-viraemic transmission, extended feeding and tick aggregation. *J. Theor. Biol.*, 224, 359–376.

Rosenblatt, D.L., Heske, E.J., Nelson, S.L., Barber, D.M., Miller, M.A. & MacAllister, B. (1999). Forest fragments in East-central Illinois: islands or habitat patches for mammals? *Am. Midl. Nat.*, 141, 115–123.

Rudolf, V.H. & Antonovics, J. (2005). Species coexistence and pathogens with frequency-dependent transmission. *Am. Nat.*, 166, 112–118.

Ruedas, L.A., Salazar–Bravo, J., Tinnin, D.S., Armién, B., Cáceres, L., García, A. *et al.* (2004). Community ecology of small mammal populations in Panamá following an outbreak of Hantavirus pulmonary syndrome. *J. Vector Ecol.*, 29, 177–191.

Saul, A. (2003). Zooprophylaxis or zoopotentiation: the outcome of introducing animals on vector transmission is highly dependent on the mosquito mortality while searching. *Malar. J.*, 2, 32–50.

Schauber, E.M. & Ostfeld, R.S. (2002). Modeling the effects of reservoir competence decay and demographic turnover in Lyme-disease ecology. *Ecol. Appl.*, 12, 1142–1162.

Schmidt, K.A. & Ostfeld, R.S. (2001). Biodiversity and the dilution effect in disease ecology. *Ecology*, 82, 609–619.

Schmidt, K.A., Ostfeld, R.S. & Schauber, E.M. (1999). Infestation of *Peromyscus leucopus* and *Tamias striatus* by *Ixodes scapularis* (Acari: Ixodidae) in relation to the abundance of hosts and parasites. *J. Med. Entomol.*, 36, 749–757.

Service, M.W. (1991). Agricultural development and arthropod-borne diseases: a review. *Rev. Saude Pública*, 25, 167–178.

Shaw, M.T., Keesing, F., McGrail, R. & Ostfeld, R.S. (2003). Factors influencing the distribution of larval blacklegged ticks on rodent hosts. *Am. J. Trop. Med. Hyg.*, 68, 447–452.

Telfer, S., Bown, K.J., Sekules, R., Begon, M., Hayden, T. & Birtles, R. (2005). Disruption of a host-parasite system following the introduction of an exotic host species. *Parasitology*, 130, 661–668.

Thrall, P.H., Antonovics, J. & Hall, D.W. (1993). Host and pathogen coexistence in vector-borne and venereal diseases characterized by frequency-dependent disease transmission. *Am. Nat.*, 142, 543–552.

Van Buskirk, J. & Ostfeld, R.S. (1995). Controlling Lyme disease risk by modifying the density and species composition of tick hosts. *Ecol. Appl.*, 5, 1133–1140.

Woolhouse, M.E.J., Taylor, L.H. & Haydon, D.T. (2001). Population biology of multihost pathogens. *Science*, 292, 1109–1112.

World Health Organization (1982). *Manual on Environmental Management for Mosquito Control with Special Emphasis on Mosquito Vectors*. WHO offset publication no. 66, Geneva, Switzerland.

Yahnke, C.J., Meserve, P.L., Ksiazek, T.G. & Mills, J.N. (2001). Patterns of infection with Laguna Negra virus in wild populations of *Calomys laucha* in the central Paraguayan chaco. *Am. J. Trop. Med. Hyg.*, 65, 768–776.

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