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The function of biodiversity in the ecology of vector-borne zoonotic diseases

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Abstract: This is a critical evaluation of the influence of species diversity within communities of vertebrates on the risk of human exposure to vector-borne zoonoses. Vertebrates serve as natural reservoirs of many disease agents (viral, bacterial, protozoal) that are transmitted to humans by blood-feeding arthropod vectors. We describe the natural history of the Lyme disease zoonosis to illustrate interactions among pathogens, vectors, vertebrate hosts, and risk to humans. We then describe how the presence of a diverse assemblage of vertebrates can dilute the impact of the principal reservoir (the white-footed mouse, *Peromyscus leucopus*) of Lyme disease spirochetes (*Borrelia burgdorferi*), thereby reducing the disease risk to humans. Exploring the logic of what we call the dilution effect reveals four conditions that are necessary for it to apply generally to vector-borne zoonoses: (1) the feeding habits of the vector are generalized; (2) the pathogen is acquired by the vector from hosts (as opposed to exclusively transovarial transmission); (3) reservoir competence (the ability of a particular host species to infect a vector) varies among host species; and (4) the most competent reservoir host tends to be a community dominant, as defined by the proportion of the tick population fed by that species. When these conditions are met, vertebrate communities with high species diversity will contain a greater proportion of incompetent reservoir hosts that deflect vector meals away from the most competent reservoirs, thereby reducing infection prevalence and disease risk. Incorporating the likelihood that the abundance of competent reservoirs is reduced in more diverse communities, owing to the presence of predators and competitors, reinforces the impact of the dilution effect on the density of infected vectors. A review of the literature reveals the generality, though not the universality, of these conditions, which suggests that the effects of diversity on disease risk may be widespread. Issues in need of further exploration include (*i*) the relative importance of diversity per se versus fluctuating numbers of particular species; (*ii*) the relevance of species richness versus evenness to the dilution effect; (*iii*) whether the dilution effect operates at both local and regional scales; and (*iv*) the shape of empirically determined curves relating diversity to measures of disease risk. Further studies linking community ecology with epidemiology are warranted.

Résumé : On trouvera ici une évaluation critique de l'influence de la diversité des espèces des communautés de vertébrés sur les risques, pour les humains, d'une exposition à des zoonoses transmises par des vecteurs. Les vertébrés sont les réservoirs naturels de plusieurs vecteurs de maladie (virus, bactéries, protozoaires) transmis aux humains par des arthropodes hématophages. Nous décrivons ici l'histoire naturelle de la zoonose de la maladie de Lyme pour illustrer les interactions entre les pathogènes, les vecteurs, les vertébrés hôtes et les risques pour les huma ins. Nous poursuivons en expliquant comment la présence d'un groupe diversifié de vertébrés peut diluer l'impact du réservoir principal (la Souris à pattes blanches, *Peromyscus leucopus*) des spirochètes associés à la maladie de Lyme (*Borrelia burgdorferi)*, diminuant par le fait même les risques de transmission de la maladie aux humains. La logique qui sous-tend l'effet de dilution a permis de reconnaître quatre conditions nécessaires pour que cet effet s'applique de façon générale aux zoonoses transmises par des vecteurs : (1) les vecteurs ont des habitudes alimentaires généralistes, (2) le pathogène passe de l'hôte au vecteur par transmission active (par opposition à la transmission exclusivement trans-ovarienne), (3) l'aptitude à servir de réservoir varie d'une espèce d'hôte à l'autre et (4) l'espèce hôte la plus apte à servir de réservoir a tendance à être l'espèce dominante de la communauté, telle que définie par la proportion de la population de tiques nourrie à même cette espèce. Lorsque ces conditions sont remplies, les communautés de vertébrés très diversifiées contiennent une plus grande proportion d'hôtes réservoirs inaptes qui font dévier les vecteurs loin des espèces réservoirs plus appropriées, ce qui limite la prévalence des infections et les risques de maladie. En tenant compte de la probabilité que l'abondance des réservoirs appropriés soit réduite dans les communautés plus diversifiées à cause de la présence de prédateurs et de compétiteurs, on augmente l'impact de l'effet de dilution sur la densité des vecteurs infectés. Une revue de la littérature met en lumière la généralité, sinon l'universalité, de ces conditions, ce qui semble

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indiquer que les effets de la diversité sur les risques de maladie peuvent être répandus. Il faudra explorer davantage les sujets suivants : (*i*) l'importance relative de la diversité per se par opposition à la densité fluctuante d'espèces particulières; (*ii*) l'influence sur l'effet de dilution de la richesse en espèces par opposition à la régularité; (*iii*) les limites du champ d'opération de l'effet de dilution (à l'échelle locale ou à des échelles régionales et (*iv*) la forme des courbes déterminées empiriquement et reliant la diversité aux mesures des risques de maladie. D'autres études sur la relation entre l'écologie des communautés et l'épidémiologie s'imposent.

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Introduction

The importance of species diversity (the number and relative abundance of different species within an ecological community) in the performance of ecosystem functions, such as primary production and resource extraction, is widely debated. The primary experimental method used for addressing this issue is to assemble communities of, for instance, herbaceous plants or protists that differ in the number of species which are drawn from the pool of possible species (Tilman et al. 1996; McGrady-Steed et al. 1997; Naeem and Li 1997). A key issue in this controversy is whether any measured variation in ecosystem processes among the assembled communities is due to differences in species richness per se, or to differences in the specific identity or functional roles of the species added (Schwartz et al. 2000; Wardle et al. 1999). Resolving this issue is important for policy and management actions that would impact biodiversity. If species diversity per se consistently has an impact on ecosystem processes, then the loss of species, irrespective of their particular functional roles, would be expected to affect ecosystem services such as efficient capture of energy from sunlight or filtering of pollutants. On the other hand, if particular functional groups or individual species have a disproportionately large effect on ecosystem functions, the loss of other components of the ecological community would likely have little impact on ecosystem services.

Consensus seems to be emerging that greater diversity in the functional roles of species in experimental communities leads to greater efficiency in the use of scarce resources and increases the resilience of the community in the face of environmental variability (e.g., drought) (Tilman et al. 1996; Naeem and Li 1997). Similarly, high diversity within an ecological community may interfere with the ability of competitively superior species to become numerically dominant (Pimm 1991). Under such conditions, diversity begets diversity (Mayer and Pimm 1997).

Recent studies suggest that species diversity may also be important in the ecology of infectious diseases, particularly vector-borne zoonoses, diseases of humans in which the disease agent resides predominantly in a non-human animal host and is transmitted among hosts (including humans) via the bite of a vector, typically an arthropod. On one hand, high species diversity in vertebrate hosts of vectors may play a beneficial role by impeding dominance by particular species that act as key reservoirs of the pathogen. On the other hand, high diversity of vertebrate hosts may provide generalist vectors or pathogens with a hedge against local extinction that would accompany the extirpation of a primary host, and therefore may play a role in increasing the disease risk to humans.

In this paper we address the role of species diversity in the ecology and epidemiology of vector-borne zoonoses. We begin by describing the natural history of one of the best known such diseases, Lyme disease, in order to lay the foundation for a conceptual model of the role of vertebrate diversity in disease risk. We then briefly describe conceptual and mathematical models of the *dilution effect* in the Lyme disease epidemic. Four crucial assumptions of the dilution effect are then critically analyzed in light of recent studies of a variety of vector-borne zoonoses. We end with a general discussion of the generality of the dilution effect.

Lyme disease and the dilution effect

Natural history of Lyme disease

Lyme disease was first described in the 1970s, when a cluster of cases of childhood arthritis in Lyme, Connecticut, U.S.A., was linked to tick bites and a spirochetal pathogen subsequently named *Borrelia burgdorferi*. The discovery of this spirochetal pathogen and the link to disease symptoms associated with it led to a flurry of research designed to uncover the enzootic cycle both in North America and in Europe, where similar symptoms had been linked to tick bites. Within 10 years of the initial description, a basic understanding of the natural history of Lyme disease had developed for foci in both eastern North America and northern Europe.

Lyme disease is transmitted via the bite of a member of the *Ixodes ricinus* complex (Acari: Ixodidae), which includes *Ixodes scapularis* in eastern and central North America, *Ixodes pacificus* in western North America, *Ixodes ricinus* in Europe, and *Ixodes persulcatus* in Asia (Lane et al. 1991; Barbour and Fish 1993; Piesman and Gray 1994). Each of these tick species has a life cycle that includes three active stages, larva, nymph, and adult, each of which takes a single blood meal from a single individual before dropping off the host and either molting to the next stage (in the case of larvae and nymphs) or reproducing and dying (in the case of adults). Because transovarial transmission of *B. burgdorferi* is highly inefficient, the vast majority of larval ticks hatch uninfected with the spirochete and therefore unable to infect their host during feeding (Piesman et al. 1986; Patrican 1997*a*). The larval meal represents an opportunity for the tick to acquire an infection, which is maintained through all subsequent molts. Larval ticks in the *I. ricinus* complex tend to be highly generalized in their host selection, feeding from a wide variety of mammalian, avian, and reptilian hosts. The specific identity of the host of larval ticks is important in the enzootiology of Lyme disease, because host species vary dramatically in their probability of infecting a feeding larval tick. In eastern North America, the white-footed mouse (*Peromyscus leucopus*) is highly efficient at infecting feeding ticks, and is considered the principal natural reservoir of Lyme disease (reviewed by Giardina et al. 2000). Eastern chipmunks (*Tamias striatus*) also appear to be competent reservoirs in North

America (Slajchert et al. 1997; Schmidt and Ostfeld 2000). In Europe and Asia, voles (*Clethrionomys glareolus*), mice (e.g., *Apodemus* spp.), introduced gray squirrels (*Sciurus carolinensis*), red squirrels (*Sciurus vulgaris*), and blackbirds (*Turdus merula*) are competent reservoirs (Matuschka et al. 1992*b*, 1994; Humair and Gern 1998; Humair et al. 1998). Other hosts, such as deer (*Odocoileus virginianus* and *Capreolus capreolus*), lizards (*Sceloporus occidentalis* and *Lacerta vivipara*), and ovenbirds (*Seiurus aurocapillus*) do not transmit *B. burgdorferi* to feeding ticks, and are considered incompetent reservoirs (Telford et al. 1988; Jaenson and Tälleklint 1992; Magnarelli et al. 1992; Lane and Quistad 1998).

Ticks infected during their larval meal become active about 1 year later, after they molt into infected nymphs capable of transmitting the pathogen to their hosts. Those not infected during their larval meal have a second opportunity to acquire the Lyme disease pathogen during their nymphal meal. Ticks that become infected during either their larval or their nymphal meal will molt into an infected adult, which becomes active between several months and >1 year later. Thus, both nymphs and adults are capable of transmitting Lyme disease, as well as perpetuating the enzootic cycle, when they bite a reservoir host. The synchrony between annual peaks in activity of nymphs and human cases of Lyme disease suggests that most cases of Lyme disease result from transmission of the pathogen by nymphs rather than by adults (Barbour and Fish 1993). Given the small size of nymphs of *Ixodes* spp. and their tendency to reach a seasonal activity peak in summer, when humans are most likely to enter tick habitat, it is not surprising that the nymphal stage is most dangerous to people.

Two parameters describing the tick population are crucial in determining the probability of human exposure to Lyme disease within specific localities that people use domestically or recreationally. The first is nymphal infection prevalence (NIP) within the local population, defined as the proportion of nymphal ticks that are infected with *B. burgdorferi*. NIP will determine the probability that a given bite from a nymphal tick will transmit Lyme disease to the human host. NIP will be a function of the distribution of larval ticks among vertebrate hosts. The larger the proportion of larvae that feed from highly competent reservoirs, the higher will be the infection prevalence in the nymphal generation. The second parameter is the local density of infected nymphs (DIN), which will strongly influence the probability that a person will encounter a tick capable of transmitting Lyme disease. DIN will be a function of both NIP and the local population density of ticks. The density of nymphs, in turn, will be influenced by both the distribution of ticks among vertebrate hosts, which vary in their ability to support successful feeding by ticks, and by biotic and abiotic conditions affecting tick survival and reproduction.

The dilution effect

Recent research has suggested that variation in the diversity of vertebrate hosts of ticks might influence the risk of human exposure to Lyme disease as measured by either NIP or DIN (Ostfeld and Keesing 2000; Schmidt and Ostfeld 2000). This assertion is based on the following observations about the ecology of Lyme disease in eastern and central North America. First, the main vector, *I. scapularis*, shows little or no transovarial transmission of *B. burgdorferi*, therefore larval ticks typically hatch free of Lyme disease bacteria. Second, *I. scapularis* is a generalist vector: larvae and nymphs feed from dozens of species of vertebrate hosts, including mammals, birds, and reptiles. Third, *I. scapularis* larvae acquire Lyme disease bacteria more efficiently from white-footed mice than from other hosts. A few other hosts, such as chipmunks and American robins (*Turdus migratorius*), are competent reservoirs, but most hosts show a low probability of infecting feeding ticks. Fourth, the white-footed mouse is one of the most abundant and widespread of all possible hosts for ticks, being present in both species-rich and species-poor vertebrate communities. From these observations, Ostfeld and Keesing (2000) argued that species-poor vertebrate communities should be characterized by a high relative abundance of white-footed mice, and that species-rich communities should include a higher relative abundance of non-mouse species that are poorer disease reservoirs. As a result, high vertebrate diversity should *dilute* the impact of white-footed mice on tick infection prevalence and consequently reduce the risk of human exposure to Lyme disease.

Schmidt and Ostfeld (2000) further analyzed the dilution effect using both empirical and modeling approaches. First, they found that at sites in southeastern New York State, eastern chipmunks are highly competent reservoirs of Lyme disease, infecting almost 70% of larval ticks that fed to repletion from them. Chipmunks were only moderately less competent than white-footed mice, which infected >90% of larval ticks that fed to repletion. Second, they demonstrated that the infection prevalence of questing nymphal ticks was dramatically lower than would be expected if all larval meals were taken from either mice or chipmunks. Third, using a simple mathematical model, they postulated that at their study sites, about 60% of larval meals must be taken from non-mouse, non-chipmunk hosts, in order to account for the observed infection prevalence of questing nymphs (38%). They interpreted this result as demonstrating the dilution effect at the scale of local vertebrate assemblages. The presence of a diverse assemblage of relatively inefficient reservoir hosts reduces NIP.

However, Schmidt and Ostfeld (2000) noted that species diversity in a community can be measured as the number of species (species richness), the relative abundance of the various species (species evenness), or a combination of richness and evenness, as represented by diversity indices such as the Shannon Index (Magurran 1988). These metrics of diversity have different implications for disease risk. Adding species to a species-poor community without changing the absolute abundance of the dominant species in the community may provide the tick population with more feeding opportunities than it would have in the absence of the added hosts. If the added species are incompetent reservoirs, the additional diversity would decrease NIP, but the added feeding opportunities might simultaneously increase the total density of nymphs. To investigate the net effects of variation in species richness and evenness on disease risk, Schmidt and Ostfeld (2000) used computer simulation modeling to assess the effects of both host species richness and evenness on the density of infected nymphs.

Schmidt and Ostfeld (2000) assembled simulated vertebrate communities by adding between 6 and 12 species drawn randomly from a pool of potential community members to an

initial community consisting of white-footed mice and eastern chipmunks. These species were assumed to have several attributes. First, each species was assumed to have a low to moderate fixed reservoir competence (also termed infectivity or reservoir capacity), which is the ability of a particular host species to infect a vector; this was assigned randomly from a uniform distribution between 0 and 0.20. This assumption was based on characteristics of host communities in eastern North America (reviewed by Giardina et al. 2000) in which most hosts are weakly capable of infecting feeding ticks. Holding reservoir competence at a fixed level for each species was a simplifying factor that we explore further below. Second, each species was assumed to have relative dominance in the community, which is the product of its characteristic population size and average tick burden. This assumption allowed us to represent species that exhibit covariation in population density and average tick burden. For example, for some larger-bodied species (e.g., raccoons, *Procyon lotor*), population densities are likely to be relatively low but the tick burden per individual is likely to be high (Fish and Dowler 1989). Third, each species was assigned an interaction coefficient with white-footed mice (mean = 0.90, SD = 0.15), so each added species modified the relative dominance of mice. Although any given species could have either a net antagonistic or net mutualistic effect on mice, we assumed that an antagonistic effect is more likely, therefore the mean interaction coefficient was <1.0. The effect of these simulated species can be either a direct reduction in mouse abundance (e.g., predation, competition) or a siphoning of tick meals away from mice. Finally, to reflect the observation (see below) that numerically dominant members of host communities are often, but perhaps not always, the most competent reservoirs, we allowed the correlation between host dominance and reservoir competence to vary. Running the simulations with both high and low correlations between host dominance and reservoir competence allowed us to assess outcomes when dominance and competence are not linked.

The model showed that the effect of species richness on disease risk is contingent on the community composition of hosts. When neither interaction coefficients among hosts nor correlations between community dominance and reservoir competence were incorporated, increasing species richness had a positive effect on DIN simply by providing more feeding opportunities for ticks. However, when we allowed additional hosts to have a net negative impact on the abundance of, or tick burdens on, highly competent reservoirs, increasing species richness dramatically decreased DIN and therefore disease risk (Schmidt and Ostfeld 2000).

Unlocking reservoir competence

Often, reservoir competence is determined using xenodiagnosis, a process in which uninfected vectors are fed on hosts that have been either artificially (via injection) or naturally (via vector) infected with the pathogen. The reservoir competence of field-caught hosts can be measured by determining the infection prevalence of previously uninfected vectors that are known to have parasitized the host. When the infection status of the host is known, field determinations of reservoir competence can provide an ecologically realistic estimation of the probability that a particular host species will infect a vector in nature. Laboratory measurements of reservoir competence, on the other hand, typically assess the maximum value for hosts that have recently been infected. Because the actual reservoir competence of an individual host tends to decline with time following inoculation with the pathogen (Levin et al. 1995; Shih et al. 1995; Lindsay et al. 1997; Markowski et al. 1998), these maximum values may not accurately represent reservoir competence under natural conditions. The reservoir competence of particular species can be characterized by three different parameters: (1) a maximum value achieved shortly after inoculation; (2) a characteristic rate of decay with time since inoculation; and (3) a characteristic probability that repeated inoculations will return competence values to the maximum (E.M. Schauber and R.S. Ostfeld, in preparation). The wide variation in estimates of the reservoir competence of particular host species (see below) may be due to failure to incorporate these three parameters when determining competence. By specifying values for these parameters, the reservoir competence of a host over the course of a season of varying vector activity, or its *effective reservoir competence*, can be estimated (E.M. Schauber and R.S. Ostfeld, in preparation). Effective reservoir competence is useful in assessing the net effects of particular species on the infection prevalence of vectors.

Models of E.M. Schauber and R.S. Ostfeld (in preparation) suggest that species diversity in host communities can influence disease risk via an additional pathway: reducing the effective reservoir competence of each host. Imagine a host community that is dominated by a species with a high maximum reservoir competence, such as one with many white-footed mice and few alternative hosts. Because the population of larval ticks will feed predominantly on this competent reservoir, many will become infected and molt the next year into infected nymphs. As a result of high NIP, inoculation rates of both the highly competent and the less competent reservoirs will tend to be rapid, pushing the effective reservoir competence of all hosts toward their maxima. High effective reservoir competence will reinforce high NIP, resulting in a positive feedback loop. Now, imagine another host-species-rich community with low maximum reservoir competence. The same initial population of larval ticks will have a lower average probability of feeding on a competent reservoir, and fewer will molt the next year into infected nymphs. As a result of the lower NIP, inoculation rates of all hosts will be low, and larvae will be more likely to feed after reservoir competence has decayed, resulting in low effective reservoir competence and a positive feedback loop toward low NIP. The model suggests that high diversity in the vertebrate community may reduce disease risk not only by diluting the effects of the most competent reservoirs, but also by reducing the reservoir competence of hosts. As we indicate below, evaluating the veracity of this model requires further research into the various parameters that influence effective reservoir competence.

Generality of the dilution effect in vector-borne zoonoses

Although Lyme disease is the most common vector-borne disease in parts of North America and Europe (Centers for Disease Control and Prevention 2000; Smith et al. 1998), many other vector-borne zoonoses plague humans through-

out the world. Often these diseases are debilitating and even lethal. It is therefore useful to assess the degree to which the dilution effect may apply to diseases other than Lyme disease. Four attributes are necessary for the dilution effect to operate in a disease system: a generalist vector, a significant role for oral (as opposed to transovarial) acquisition of the pathogen by vectors, variation in reservoir competence among hosts, and a positive correlation between reservoir competence and numerical dominance of hosts in the community. We describe these four attributes below and then explore their likely occurrence for vector-borne zoonoses in general.

A generalist vector

Arthropod parasites vary widely in their degree of specialization on hosts. For instance, for a vector-borne zoonosis to exist, the vector must at least parasitize both a non-human animal and a human. For zoonoses in which vectors are extreme specialists, i.e., parasitize one or a few host species, diversity in the host community will be relatively unimportant in determining vector abundance and infection prevalence because most hosts will be irrelevant to the enzootic cycle. Only in those cases in which variation in host diversity represents a change in feeding opportunities for the vector will the dilution effect apply.

Oral acquisition of the pathogen

Some pathogens are transmitted across vector generations via transovarial passage from mother to offspring. For these zoonoses, vectors are often infected when they hatch, which liberates them from immediate reliance on a blood meal from a host for pathogen acquisition. Only in those cases in which a significant proportion of pathogen acquisition by vectors is from host meals will the dilution effect apply.

Variation in reservoir competence among hosts

If the probability of transmitting the disease agent to a feeding vector is similar for all hosts, then variation in host diversity is unlikely to influence the infection prevalence of vectors because a meal from each host species will have a similar probability of transmitting the pathogen. Only when different host species vary in their reservoir competence will changes in the composition of the host community influence the disease risk.

A positive correlation between reservoir competence and numerical dominance in the community

If the most competent reservoirs of a pathogen tend to be rare members of their communities, they will probably be absent from species-poor communities and present only in species-rich communities (Davies et al. 2000). In such cases, the disease risk will be higher in more diverse communities, which is the opposite of the dilution effect. Only in situations in which high species richness is accompanied by the inclusion of species with low average reservoir competence will the dilution effect apply.

Exploration of the necessary attributes

A generalist vector

Although comprehensive assessments of the degree to which zoonotic vector species are selective of hosts are rare, it appears that extreme specialists are few. For instance, some species of mosquitoes feed largely on birds and others largely on mammals, but most or all of these mosquitoes feed on at least several species within each vertebrate class (Tempelis 1975). The widespread use of domestic chickens and livestock as sentinels to detect mosquito-borne diseases such as malaria, dengue fever, and various mosquito-borne encephalitis viruses capitalizes on the tendency for vector mosquitoes to feed on both human and non-human hosts. Indeed, zooprophylaxis, which is related to the dilution effect, consists of using domestic animals as alternative hosts that may deflect host-seeking mosquitoes from human hosts (Hess and Hayes 1970). However, whereas the dilution effect is concerned with the impact of diversity of native host communities on both abundance and infection prevalence of vectors, zooprophylaxis is concerned with the role of domestic animals in vector use of human hosts.

Ixodid ticks appear to vary greatly in their degree of host generalization, from species such as *Ixodes neotomae*, which attacks relatively few mammalian hosts, and not humans, to *I. scapularis* and *I. ricinus*, which parasitize at least several dozen host species in three vertebrate classes (Lane et al. 1991; Matuschka et al. 1992*b*; Kurtenbach et al. 1995; Kollars et al. 1999). Tick vectors of the Crimean–Congo hemorrhagic fever (CCHF) virus (genera *Hyalomma*, *Rhipicephalus*, and *Amblyomma*) infest numerous species of wild and domestic ungulates, rodents, and ground-dwelling birds (Camicas et al. 1990; Zeller et al. 1994). *Rhipicephalus sanguineus*, a tick vector of tick typhus, Rocky Mountain spotted fever, Q-fever, Lyme disease, tularemia, and CCHF is known to parasitize domestic dogs, livestock, wild carnivores, ungulates, and other mammals (Walker et al. 2000). Similarly, in the Neotropics, several syntopic species of sand fly (genus *Lutzonyia*) vectors of zoonotic cutaneous leishmaniasis (ZCL) vary dramatically in their host specificity, but few take more than 50% of their blood meals from any single host species (Christensen and de Vasquez 1982). Old World sand fly (genus *Phlebotomus*) vectors of ZCL are known to be attracted to and feed on several species of native rodents and rabbits, as well as domestic stock and pets (Schlein et al. 1984; Johnson et al. 1993; Githure et al. 1996). Triatomine bug vectors of Chaga's disease feed on a wide variety of mammalian and avian hosts. Zeledón et al. (1973) reported a single adult *Triatoma dimidiata* as having fed on six species of hosts belonging to five mammalian orders. A population of mosquitoes (*Aedes albopictus*) from Missouri, U.S.A., fed from at least nine species of mammals and members of four orders of birds (Savage et al. 1993). Tsetse fly (*Glossina* spp.) vectors of African trypanosomiasis are known to feed on dozens of species of both wild and domestic mammals (reviewed by Milligan and Baker 1988). We conclude that host generalization by disease vectors appears to be widespread. However, we hasten to add that, with few exceptions, the absolute degree of host generalization by arthropod vectors, as represented by the distribution of blood meals among hosts, is poorly understood.

Oral versus transovarial transmission of pathogens

Many pathogens have multiple routes of transmission to vectors, including transovarial, venereal, and oral. For some vector-borne zoonoses, especially mosquito-borne viral diseases (Table 1), transovarial transmission is relatively effi-

cient, leading to moderate or high infection prevalences in newly hatched larvae produced by infected mothers. For other vector-borne zoonoses, including many viral, bacterial, and protozoal diseases, transovarial transmission is highly inefficient or nonexistent, leading to low to zero infection prevalences in F_1 generations produced by infected mothers (Table 1).

When transovarial or venereal transmission is the main route of pathogen transmission, infection prevalence of vectors may be largely independent of the source of blood meals, therefore the dilution effect is less likely to be a strong determinant of disease risk. Nevertheless, even for zoonoses with efficient transovarial transmission, efficiency (defined as the percentage of infected offspring produced by infected mothers, sometimes called filial transmission) rarely exceeds 80%, and is often <50%. Infection prevalence in the F_2 generation often declines dramatically from that in the F_1 generation. If transovarial transmission were the only means of pathogen acquisition, infection prevalence of vectors would decline exponentially with succeeding generations, leading to the virtual loss of the pathogen in relatively few generations. Therefore, even for zoonoses with significant vertical transmission, the distribution of blood meals among hosts is likely to influence the infection prevalence of vectors, and therefore disease risk (e.g., Turell and LeDuc 1983). Further studies are warranted to compare the infection prevalence expected if transmission is exclusively transovarial with the actual infection prevalence of vectors. This difference, the effective degree of oral transmission, should be correlated with the potential for the dilution effect to influence disease risk.

Variation in reservoir competence among hosts

The reservoir competence (or infectivity) of hosts is typically assessed by means of one of three methods. First, the proportion of vectors acquiring an infection from a blood meal is determined using xenodiagnosis, as described above. Second, hosts are sampled to determine whether they possess circulating antibodies to specific pathogens, under the assumption that demonstrated recent or current exposure to a pathogen reflects a host's state of infectivity to vectors. Third, hosts are sampled for the presence of the pathogen in the bloodstream or other tissues, under the same assumption as for antibody tests. The latter two methods by themselves are often unreliable because the presence of either particular antibodies or pathogens may not accurately reflect the probability of transmission from host to vector. For example, circulating levels of *B. burgdorferi*-specific immunoglobulins in three species of wild rodents were found to be negatively correlated with spirochete infectivity to ticks (Kurtenbach et al. 1994). Similarly, in the case of visceral leishmaniasis, both culture methods and polymerase chain reaction techniques demonstrated the presence of *Leishmania chagasi* in individual opossums (*Didelphis marsupialis*) that were not infective, as determined by xenodiagnosis (Travi et al. 1998). Whether the presence of pathogens or antibodies specific to those pathogens in hosts is correlated with infectivity to vectors is rarely examined for vector-borne zoonoses (cf. Reithinger and Davies 1999).

For nearly all vector-borne zoonoses of which we are aware, reservoir competence appears to vary substantially among hosts (Table 2). However, accurate determination of reservoir competence in specific disease foci is problematic for several reasons. First, intraspecific variation in reservoir competence appears to be commonplace (Table 2) but the causes of this variation are typically unknown. Second, the burden of vectors on hosts can influence reservoir competence, but the presence of heavy vector burdens in some cases increases, but in other cases decreases, reservoir competence (Kurtenbach et al. 1995). Third, reservoir competence is likely to be determined by a combination of characteristics that are intrinsic to the host (e.g., immune response to the pathogen) and extrinsic to the host (e.g., the relative abundance of other hosts in the community and the abundance of vectors). Fourth, some hosts that are incompetent reservoirs may nevertheless infect vectors when they are simultaneously parasitized by infected and uninfected vectors (Ogden et al. 1997; Patrican 1997*b*). In such cases, pathogens are transmitted from vector to vector without being maintained or amplified in the host (Stafford et al. 1995; Randolph 1998). Further studies to assess the mechanisms behind both inter- and intra-specific variation in infectivity are warranted. Such studies would facilitate accurate determination of whether host species have modal values of reservoir competence, and if so, whether modal values vary substantially among species.

A positive correlation between reservoir competence and numerical dominance in the community

Few data exist to allow this condition to be explored. Although many studies of vector-borne zoonoses involve assessment of the reservoir competence of various hosts, few simultaneously assess the relative abundance of each host species, or average parasite burdens on those hosts. Searches for reservoir hosts of zoonotic pathogens typically focus exclusively on the most abundant host species. In fact, if a host species is not both abundant and highly infective to vectors, it is usually ignored, under the assumption that only species having both characteristics can be important epidemiologically. In marked contrast, the dilution effect argues that, because of their potential to divert meals away from highly infective hosts, both less common species and those with low infectivity are likely to be more important than was previously recognized.

In many endemic foci of Lyme disease in North America, the white-footed mouse is both the most competent reservoir and the numerically dominant member of the community of tick hosts (Mather et al. 1989; Ostfeld 1997; Lindsay et al. 1999; Ostfeld and Keesing 2000). In Lyme disease foci in Europe and Asia, the bank vole (*Clethrionomys glareolus*) tends to be the numerically dominant tick host; in some studies it has been found to be also the most competent reservoir (Tälleklint et al. 1993; Matuschka et al. 1994), whereas others have found higher reservoir competence in syntopic mice (genus *Apodemus*) (Matuschka et al. 1992*a*; Humair et al. 1993). It is noteworthy that some European studies of Lyme borreliosis have shown positive correlations between average tick burden on hosts and reservoir competence of those hosts (Matuschka et al. 1992*a*, 1992*b*, 1994; Ogden et al. 1997). This finding is consistent with the model of Schmidt and Ostfeld (2000), which defines host community dominance in terms of the number of tick meals supplied by a given host species, rather than host density per se.

Table 1. Selected zoonotic diseases for which vertical (i.e., transovarial or filial) transmission of the pathogen is highly inefficient (<5%) or more efficient (>5%).

Disease	Vector	References
Vertical transmission <5%		
California group encephalitis virus (LaCrosse virus)	*Aedes albopictus*	Tesh and Gubler 1975
	Culex fatigans	Tesh and Gubler 1975
Chikungunya virus	*Aedes aegypti*	Jupp et al. 1981; Mourya 1987
	Aedes albopictus	Zytoon et al. 1993; Mourya 1987
	Aedes furcifer	Jupp et al. 1981
Crimean–Congo hemorrhagic fever virus	*Amblyomma hebraeum*	Sheperd et al. 1991
	Boophilus decoloratus	Sheperd et al. 1991
	Hyalomma dromedarii	Logan et al. 1990
	Hyalomma impeltatum	Logan et al. 1990
	Hyalomma marginatum	Sheperd et al. 1991
	Hyalomma truncatum	Logan et al. 1989, 1990; Sheperd et al. 1991
	Rhipicephalus appendiculatis	Logan et al. 1989, 1990; Sheperd et al. 1991
	Rhipicephalus evertsi	Sheperd et al. 1991
Dengue fever virus	*Aedes aegypti*	Lee et al. 1997
	Aedes albopictus	Lee et al. 1997
Eastern equine encephalitis virus	*Culiseta melanura*	Morris and Srihongse 1978
Granulocytic ehrlichiosis	*Ixodes pacificus*	Ogden et al. 1998*b*
	Ixodes ricinus	Ogden et al. 1998*b*
	Ixodes scapularis	Ogden et al. 1998*b*
Human granulocytic ehrlichiosis	*Ixodes scapularis*	Levin et al. 1999
Lyme disease	*Ixodes pacificus*	Schoeler and Lane 1993
	Ixodes ricinus	Zhioua et al. 1994
	Ixodes scapularis	Patrican 1997*a*, 1997*b*; Levin et al. 1999
Murine typhus	*Xenopsylla cheopis*	Farhang-Azad et al. 1985
Murray Valley encephalitis	*Aedes aegypti*	Kay and Carley 1980
Rift Valley fever	*Aedes juppi*	Gargan et al. 1988
St. Louis encephalitis	*Culex pipiens*	Francy et al. 1981
Vesicular stomatitis	*Aedes albopictus*	Tesh and Gubler 1975
	Culex fatigans	Tesh and Gubler 1975
	Lutzonyia longipalpus	Tesh et al. 1987
Vertical transmission >5%		
California group encephalitis virus	*Aedes dorsalis*	Turell et al. 1982
	Aedes melanimon	Turell et al. 1982
Keystone strain	*Aedes atlanticus*	LeDuc et al. 1975
LaCrosse virus	*Aedes atropalpus*	Freier and Beier 1984
	Aedes triseriatus	Woodring et al. 1998; Schopen et al. 1990; Patrican and DeFoliart 1987
	Culiseta inornata	Schopen et al. 1990
Snowshoe hare virus	*Aedes triseriatus*	Schopen et al. 1990
	Culiseta inornata	Schopen et al. 1990
Crimean–Congo hemorrhagic fever virus	*Hyalomma marginatum*	Zeller et al. 1994
	Hyalomma truncatum	Wilson et al. 1991; Gonzalez et al. 1992
Jamestown Canyon virus	*Aedes provocans*	Boromisa and Grayson 1990
	Aedes stimulans	Boromisa and Grimstad 1986
Japanese encephalitis virus	*Aedes alcasidi*	Rosen et al. 1989
	Aedes vexans	Rosen et al. 1989
	Armigeres flavus	Rosen et al. 1989
	Armigeres subalbatus	Rosen et al. 1989
	Culex pipiens	Rosen et al. 1989
	Culex quinquefasciatus	Rosen et al. 1989
	Culex tritaeniorhynchus	Rosen et al. 1989
Powassan virus	*Ixodes scapularis*	Costero and Grayson 1996
Rift Valley fever	*Aedes lineatopennis*	Linthicum et al. 1985
Rio Grande virus	*Lutzonyia anthophora*	Endris et al. 1983

Note: Vertical transmission includes the percentages of progeny infected from known infected females and from all females.

Table 2. Variation both within and among host species in reservoir competence (RC) for selected diseases.

Host					
Common name	Scientific name	Method	RC	Source	Location
Lyme disease					
Old World field mouse	*Apodemus agrarius*	XW	58	Matuschka et al. 1992*a*	Germany
Yellow-necked mouse	*Apodemus flavicollis*	XW	8	Humair et al. 1993	Switzerland
		XW	27	Matuschka et al. 1994	Germany
		XW	25	Matuschka et al. 1992*a*	Germany
Wood mouse	*Apodemus sylvaticus*	XW	21	Humair et al. 1993	Switzerland
		XW	0	Matuschka et al. 1994	Germany
		XW	46	Gern et al. 1993	Switzerland
Short-tailed shrew	*Blarina brevicauda*	XW	37	Telford et al. 1990	Mass., U.S.A.
		XW	37	Mather 1993	Mass., U.S.A.
Veery	*Catharus fuscescens*	XW	21	Magnarelli et al. 1992	Conn., U.S.A.
		XW	43	Rand et al. 1998	Maine, U.S.A.
Bank vole	*Clethrionomys glareolus*	XW	15	Humair et al. 1993	Switzerland
		XW	72	Matuschka et al. 1994	Germany
		XW	39	Matuschka et al. 1992*a*	Germany
Gray catbird	*Dumetella carolinensis*	XW	7	Anderson and Magnarelli 1984	Conn., U.S.A.
		XW	3	Magnarelli et al. 1992	Conn., U.S.A.
		XW	0	Mather 1993	Mass., U.S.A.
		XW	10	Anderson et al. 1985	Conn., U.S.A.
Common yellowthroat	*Geothlypis trichas*	XW	17	Anderson and Magnarelli 1984	Conn., U.S.A.
		XW	2	Magnarelli et al. 1992	Conn., U.S.A.
		XW	24	Rand et al. 1998	Maine, U.S.A.
Worm-eating warbler	*Helmitheros vermivorus*	XW	7	Magnarelli et al. 1992	Conn., U.S.A.
Wood thrush	*Hylocichla mustelina*	XW	0	Magnarelli et al. 1992	Conn., U.S.A.
Edible dormouse	*Glis glis*	XW	95	Matuschka et al. 1994	Germany
Sand lizard	*Lacerta agilis*	XW	0	Matuschka et al. 1992*a*	Germany
Swamp sparrow	*Melospiza georgiana*	XW	19	Anderson and Magnarelli 1984	Conn., U.S.A.
Song sparrow	*Melospiza melodia*	XW	12	Rand et al. 1998	Maine, U.S.A.
Field vole	*Microtus agrestis*	XW	0*	Matuschka et al. 1994	Germany
Meadow vole	*Microtus pennsylvanicus*	XW	62	Markowski et al. 1998	R.I., U.S.A.
		XW	6	Mather 1993	Mass., U.S.A.
		XW	1	Mather 1993	Mass., U.S.A.
White-tailed deer	*Odocoileus virginianus*	XW	0	Mather 1993	Mass., U.S.A.
		XW	1	Mather 1993	Mass., U.S.A.
		XW	1	Telford et al. 1988	Mass., U.S.A.
Rice rat	*Oryzomys palustris*	XIV	76	Levin et al. 1995	Ga., U.S.A
White-footed mouse	*Peromyscus leucopus*	XIS	22	Lindsay et al. 1997	Canada
		XW	25	Anderson and Magnarelli 1984	Conn., U.S.A.
		XW	40	Fish and Daniels 1990	N.Y., U.S.A.
		XW	46	Mather et al. 1989	Mass., U.S.A.
		XW	46	Mather 1993	Mass., U.S.A.
		XW	81	Mather 1993	Mass., U.S.A.
		XW	76	Mather 1993	Mass., U.S.A.
		XW	77	Mather 1993	R.I., U.S.A.
		XW	88	Mather et al. 1989	Mass., U.S.A.
		XW	83	Mather et al. 1989	Mass., U.S.A.
		XW	35	Rand et al. 1998	Maine, U.S.A.
		XW	91	Giardina et al. 2000	N.Y., U.S.A.
Deer mouse	*Peromyscus maniculatus*	XIV	33*	Peavey and Lane 1995	Calif., U.S.A.
Pheasant	*Phasianus colchicus*	XIV	23	Kurtenbach et al. 1998	U.K.
		XIS	5	Kurtenbach et al. 1998	U.K.
Eastern towhee	*Pipilo erythrophthalmus*	XW	0	Rand et al. 1998	Maine, U.S.A.
Raccoon	*Procyon lotor*	XW	0	Mather 1993	Mass., U.S.A.
Norway rat	*Rattus norvegicus*	XIV	78*	Matuschka et al. 1997	Germany
Black rat	*Rattus rattus*	XIV	90*	Matuschka et al. 1997	Germany
Ovenbird	*Seiurus aurocapillus*	XW	1	Magnarelli et al. 1992	Conn., U.S.A.

Table 2 (*continued*).

Host					
Common name	Scientific name	Method	RC	Source	Location
Common shrew	*Sorex araneus*	XW	10	Humair et al. 1993	Switzerland
Eastern chipmunk	*Tamias striatus*	XW	20	Mather 1993	Mass., U.S.A.
		XW	20	Mather et al. 1989	Mass., U.S.A.
		XW	61	Giardina et al. 2000	N.Y., U.S.A.
House wren	*Troglodytes aedon*	XW	9	Magnarelli et al. 1992	Conn., U.S.A.
Carolina wren	*Troglodytes carolinensis*	XW	16	Magnarelli et al. 1992	Conn., U.S.A.
Robin	*Turdus migratorius*	XW	0	Magnarelli et al. 1992	Conn., U.S.A.
		XW	17	Rand et al. 1998	Maine, U.S.A.
Red fox	*Vulpes vulpes*	XW	13*	Mather 1993	Mass., U.S.A.
Hooded warbler	*Wilsonia citrina*	XW	9	Magnarelli et al. 1992	Conn., U.S.A.
Visceral leishmaniasis					
Dog	*Canis familiaris*	XWI	29	Sherlock 1996	Brazil
Opossum	*Didelphis marsupialis*	XWI	14*	Sherlock 1996	Brazil
		XIS	2*	Travi et al. 1998	Colombia?
Human	*Homo sapiens*	XWI	15	Sherlock 1996	Brazil
Cutaneous leishmaniasis					
Spiny mouse	*Acomys russatus*	PD	0	Schlein et al. 1984	Israel
Spiny mouse	*Acomys subspinosus*	PD	0	Githure et al. 1996	Kenya
Four-toed jerboa	*Allactaga euphratica*	PD	0	Saliba et al. 1994	Jordan
Grass rat	*Arvicanthis niloticus*	PD	1	Githure et al. 1996	Kenya
Cheesman's gerbil	*Gerbillus cheesmani*	PD	0	El Sibae et al. 1993	Saudi Arabia
Wagner's gerbil	*Gerbillus dasyurus*	PD	0	Schlein et al. 1984	Israel
Baluchistan gerbil	*Gerbillus nanus*	PD	0	Schlein et al. 1984	Israel
Greater gerbil	*Gerbillus pyramidum*	PD	0	Schlein et al. 1984	Israel
Long-eared desert hedgehog	*Hemiechinus auritus*	PD	0*	Yaghoobi-Ershadi et al. 1996	Iran
Asiatic porcupine	*Hystrix indica*	PD	0*	Schlein et al. 1984	Israel
Lacertid lizard	*Latastia longicauda*	PD	40	Githure et al. 1996	Kenya
Multimammate mouse	*Mastomys natalensis*	PD	1	Githure et al. 1996	Kenya
Jird	*Meriones crassus*	PD	0	Saliba et al. 1994	Jordan
		PD	7	El Sibae et al. 1993	Saudi Arabia
		PD	10	Schlein et al. 1984	Israel
Libyan jird	*Meriones libycus*	PD	18	Yaghoobi-Ershadi and Javidian 1996	Iran
		PD	25	Yaghoobi-Ershadi et al. 1996	Iran
		PD	0	Saliba et al. 1994	Jordan
		PD	5	El Sibae et al. 1993	Saudi Arabia
House mouse	*Mus musculus*	PD	0*	Schlein et al. 1984	Israel
Rock hyrax	*Procavia capensis*	PD	0*	Schlein et al. 1984	Israel
Fat sand jird	*Psammomys obesus*	PD	23	Saliba et al. 1994	Jordan
Norway rat	*Rattus norvegicus*	PD	0*	El Sibae et al. 1993	Saudi Arabia
Black rat	*Rattus rattus*	PD	0*	El Sibae et al. 1993	Saudi Arabia
		PD	0*	Schlein et al. 1984	Israel
Great gerbil	*Rhombomys opimus*	PD	54	Yaghoobi-Ershadi and Javidian 1996	Iran
		PD	32	Yaghoobi-Ershadi et al. 1996	Iran
Large naked-soled gerbil	*Tatera robusta*	PD	13	Githure et al. 1996	Kenya
Monitor lizard	*Varanus* sp.	PD	67*	Githure et al. 1996	Kenya
Sand fox	*Vulpes ruppelli*	PD	0*	Schlein et al. 1984	Israel
Chaga's disease					
Three-toed sloth	*Bradypus variegatus*	PD	0	Travi et al. 1994	Colombia
Dog	*Canis familiaris*	XW	41	Gürtler et al. 1993	Argentina
Two-toed sloth	*Choloepus hoffmani*	PD	0*	Travi et al. 1994	Colombia
Silky anteater	*Cyclopes didactylus*	PD	0*	Travi et al. 1994	Colombia
Nine-banded armadillo	*Dasypus novemcinctus*	PD	100*	Travi et al. 1994	Colombia
Opossum	*Didelphis marsupialis*	PD	72	Travi et al. 1994	Colombia
Cat	*Felis cattus*	XW	28	Gürtler et al. 1993	Argentina

Table 2 (*concluded*).

Host					
Common name	Scientific name	Method	RC	Source	Location
Brown "four-eyed" opossum	*Philander opossum*	PD	0	Travi et al. 1994	Colombia
Kinkajou	*Potos flavus*	PD	50*	Travi et al. 1994	Colombia
Spiny rat	*Proechimys semispinosus*	PD	1	Travi et al. 1994	Columbia
Black rat	*Rattus rattus*	PD	6	Travi et al. 1994	Colombia
Climbing rat	*Tylomys mirae*	PD	13	Travi et al. 1994	Colombia

Note: Reservoir competence is defined as the percentage of vectors that acquire an infection during a blood meal taken from a host. It is estimated using a variety of techniques, including xenodiagnosis of wild-caught hosts of unknown infection status (XW), wild-caught hosts known to be infected (XWI), captive hosts infected by syringe (XIS), and captive hosts infected by vector (XIV) and by direct detection of pathogens (PD). An asterisk denotes a study in which <10 hosts of a particular species were tested.

For ZCL in Jordan, Saliba et al. (1994) found that the fat sand rat or jird, *Psammomys obesus*, was both the most abundant rodent and the most competent reservoir for *Leishmania* spp. Analyzing ZCL in Iran, Yaghoobi-Ershadi et al. (1996) found that the numerically dominant species and most competent reservoir at one site was the Libyan jird (*Meriones libycus*), but at another site was the great gerbil (*Rhombomys opimus*). At a Saudi Arabian site where *Meriones crassus* was captured more frequently than were *Meriones libycus* or three other co-occurring rodents, *M. crassus* was the most competent reservoir for ZCL (El Sibae at al. 1993).

We conclude that although the evidence for a link between abundance and reservoir competence of hosts is suggestive for a few zoonoses, data are insufficient for a thorough evaluation of this condition more generally. We expect pathogens that are transmitted by generalist vectors to have had opportunities to interact, and possibly to coevolve, with multiple host species across many ecological communities. Under such conditions, we expect that selection might favor pathogen genotypes that are able to exploit the dominant members of ecological communities, which would provide them with the most stable "habitats" promoting persistence. We also urge that more attention be paid to host species that are poor or incompetent reservoirs for zoonotic agents, and to rarer members of vertebrate communities. Such species may provide vector populations with abundant opportunities to feed but little opportunity for infection.

General discussion

According to the dilution-effect model, increasing the diversity of the community of hosts of vectors will lead to a greater proportion of blood meals being taken from rarer, less competent reservoirs, resulting in lower infection prevalence in the vector population (Ostfeld and Keesing 2000; Schmidt and Ostfeld 2000). The suppressive effects of high diversity on disease risk will be reinforced when species added to the host community either diminish the population density of the primary reservoir host, e.g., via predation or competition, or reduce the absolute vector burden on the reservoir host, e.g., by diverting vector meals away from the reservoir host to incompetent reservoirs (Schmidt and Ostfeld 2000). Moreover, the presence of alternative host species with low reservoir competence may, by reducing encounter rates between infected vectors and hosts, reduce the effective reservoir competence of other host species (E.M. Schauber and R.S. Ostfeld, in preparation). Although the dilution-effect model was developed for Lyme disease, it may apply, in principle, to many other vector-borne zoonoses. The four attributes necessary for the dilution effect to operate seem to apply broadly (Table 3), but data are insufficient for a rigorous analysis of its general applicability.

Case studies?

Two recent studies of non-Lyme vector-borne zoonoses suggest the operation of the dilution effect. In a study of Chaga's disease in Colombia, Travi et al. (1994) determined that the opossum *Didelphis marsupialis* is the principal reservoir host for the protozoal agent *Trypanosoma cruzi*. They found that Chaga's disease occurs in human settlements in tropical dry forest but not in tropical wet forest, despite the presence of both opossums and triatomine vectors in both areas. Travi et al. (1994) attributed the lower disease risk in wet forest to the higher abundance of alternative, eutherian hosts for triatomines, which may have reduced the infection prevalence of vectors and therefore contact between infected insects and humans. Similarly, Ogden et al. (1998*a*) compared the infection prevalence of granulocytic *Ehrlichia* spp. in ticks (*I. ricinus*) at two U.K. study sites that differed dramatically in the diversity of vertebrate hosts. At an upland site with abundant reservoir-competent sheep but a low diversity of alternative hosts, tick infection prevalence, and consequently disease risk for humans, was high. In contrast, at a woodland site with abundant populations of rodents, pheasants (*Phasianus colchicus*), and roe deer (*Capreolus capreolus*), which are inefficient reservoirs of granulocytic *Ehrlichia* spp., the infection prevalence of nymphal and adult ticks was significantly lower.

Prospects for the future

Several key questions require resolution if the importance and generality of the dilution effect are to be determined.

(1) To what extent is the infection prevalence of vectors determined by diversity within the entire community of vertebrate hosts, by diversity of particular components of the host community, or simply by population fluctuations of the most competent host(s), e.g., mice in the case of Lyme disease?

If disease risk were related solely to the population abundance of a single reservoir host, then the dilution effect would not operate. On the other hand, overall diversity (e.g., the Shannon index) within a community of hosts will decrease whenever the most abundant species increases in absolute

abundance, if the remaining species remain at constant density. Therefore, we expect the dynamics of particular, common hosts to covary with community diversity. Experimental studies that separately manipulate absolute or relative abundance of particular species and community diversity per se will facilitate obtaining an answer to this question. In addition, multiple regression models in which abundances of single host species and diversity metrics for the entire community are used as independent variables should prove useful.

(2) Which metrics of species diversity will perform best as predictors of vector infection prevalence?

Species diversity can be estimated using metrics such as species richness (number of species) or diversity indices, which incorporate species richness and evenness (proportional representation of each species). Which metric seems most appropriate will vary according to specific features of models linking diversity to disease risk. In a model in which ticks passively encounter hosts in proportion to host abundance, diversity indices that incorporate evenness would seem most appropriate, because evenness, not richness, would capture the probability of encounter between a passive, nonselective vector and each species of host. In a model in which vectors are selective of hosts, or in which host species interact (via predation or competition), species richness would seem more appropriate for capturing the impacts of the addition or deletion of particular species.

(3) Does the dilution effect occur only at the scale of local ecological communities, or is it important in explaining patterns of vector infection prevalence at large geographic scales?

Schmidt and Ostfeld (2000) demonstrated that the presence of inefficient vertebrate reservoirs reduces tick infection prevalence within local ecological communities, supporting the dilution effect model locally for Lyme disease. Ostfeld and Keesing (2000) provided evidence that the dilution effect may operate for Lyme disease at the scale of the eastern United States. Further studies of both local-scale and regional-scale interactions between host diversity and disease risk are warranted.

(4) At broad spatial scales, can vertebrate diversity be predicted from the degree of habitat destruction or alteration in the region?

Claims that habitat destruction and alteration affect disease transmission are frequent but rarely demonstrated. For some diseases, such as yellow fever, dengue fever, and African trypanosomiasis, disturbance of natural habitats appears to alter the behavior of vectors so that encounter rates between vectors and humans are elevated (Walsh et al. 1993; Molyneux 1997). The dilution effect suggests an alternative mechanism by which habitat alteration can influence disease risk. Habitat destruction and the fragmentation of landscapes into small isolated units are known to cause reduction or elimination of some vertebrate species (Nupp and Swihart 1996, 1998; Rosenblatt et al. 1999) and therefore of diversity. Often, the species most sensitive to such habitat destruction are large species that occupy high trophic levels, such as raptors and carnivorous mammals. Loss of these species, which are rarely found to be competent reservoirs for vector-borne zoonoses (for an exception see Reithinger and Davies 1999), may increase disease risk both via reduction in feeding opportunities from these incompetent hosts and via the loss of a regulatory effect of such predators on typically more reservoir-competent rodents.

(5) Are the curves relating diversity to disease risk linear or curvilinear?

If disease risk, as measured by vector infection prevalence or density of infected vectors, decreases more or less linearly with increasing vertebrate diversity, a critical implication would be that any loss of diversity will result in a measurable increase in disease risk. On the other hand, analyses of Lyme disease by Ostfeld and Keesing (2000) and Schmidt and Ostfeld (2000) suggest that the association between diversity and disease risk is better described by a saturating function, so changes in diversity have a strong effect only on species-poor communities. Similar, saturating curves describing the relationship between diversity and ecosystem function appear to be commonplace (McGrady-Steed et al. 1997; Schwartz et al. 2000). In such cases, the system may be more resilient to the loss of diversity; highly diverse vertebrate communities should be affected only modestly, if at all, by modest losses in diversity, whereas already depauperate communities should be affected dramatically.

Disease systems inconsistent with the dilution effect should be sought. One likely exception to the beneficial impact of host diversity on disease risk is the plague epizootic, in which the pathogen (*Yersinia pestis*) is transmitted from non-human mammal reservoirs to humans by several species of fleas. Unlike most zoonotic pathogens, *Y. pestis* is highly pathogenic to virtually all hosts, often causing mortality in ca. 99% of infected individuals across a wide range of species (Gage et al. 1995; Anderson and Williams 1997; Cully et al. 1997). As a result of high mortality rates, plague tends to occur as a rapidly developing epizootic that decimates host populations and then goes locally extinct (Barnes 1993; Anderson and Williams 1997). In this case, low diversity in the host community appears to facilitate rapid extinction of the plague epizootic, whereas more diverse mammal communities provide the pathogen with refugia from rapid local extinction, maintaining its potential to infect people (R. Parmenter, personal communication).

It is important to note that our arguments concerning the role of diversity in disease risk are limited to diversity within communities of vertebrate hosts and reservoirs. The effects of variation in diversity within other components of zoonotic disease systems are worthy of exploration in their own right. For example, for both cutaneous and visceral leishmaniases, a diverse assemblage of sand fly species and parasites (*Leishmania* spp.) are involved, but the effects of vector and pathogen diversity are poorly understood. Clearly, disease risk to humans will typically be higher in geographic areas that contain a greater diversity of pathogens and vectors, such as the tropics, than in regions with fewer infectious agents, which include the boreal zone.

Finally, it is worth exploring whether directly transmitted zoonoses, in addition to vector-borne diseases, also are subject to the dilution effect. Rodents are the reservoirs for numerous bacterial and viral diseases of humans that are transmitted by direct contact or inhalation of aerosols containing

Table 3. Pathogens, vectors, and reservoir species for key vector-borne diseases of humans.

Disease	Pathogen	Vector
Viral diseases		
LaCrosse virus	Bunyavirus	*Aedes* sp., *Culex* sp. (mosquitoes)
Rift Valley fever	Bunyavirus	*Aedes* sp., *Culex* sp. (mosquitoes)
Oropouche	Bunyavirus	*Culex quinquifasciatus* (mosquito), *Culicoides* sp. (midge)
Crimean–Congo hemorrhagic fever	Bunyavirus	*Hyalomma* sp., *Rhipicephalus* sp., *Amblyomma* sp. (ticks)
St. Louis encephalitis	Flavivirus	*Culex* sp. (mosquito)
Yellow fever	Flavivirus	*Aedes* sp., *Haemogogus* sp. (mosquitoes)
West Nile virus	Flavivirus	*Culex* sp. (mosquito)
Japanese encephalitis	Flavivirus	*Culex* sp., *Aedes* sp. (mosquitoes)
Murray Valley encephalitis	Flavivirus	*Culex annulirostris*, *Aedes aegypti* (mosquitoes)
Kyasanur forest disease	Flavivirus	*Haemaphysalis* sp. (tick)
Tick-borne encephalitis[c]	Flavivirus	*Ixodes* spp. and other ticks
Omsk hemorrhagic fever	Flavivirus	*Dermacentor pictus* (tick)
Equine encephalitis		
Western	Togavirus	*Culex tarsalis* (mosquitoes)
Eastern	Togavirus	*Coquillettidiea perturbans*, *Aedes* sp., *Culiseta* sp. (mosquitoes)
Venezuelan	Togavirus	*Psorophora ferox*, *Culex* sp., *Aedes* sp. (mosquitoes)
Ross River virus	Togavirus	*Aedes* sp., *Cules* sp. (mosquitoes)
Chikungunya virus	Togavirus	*Aedes* sp. (mosquitoes)
Sinbis virus	Togavirus	*Culex* sp. (mosquito)
Colorado tick fever	Reovirus	*Dermacentor andersoni* (tick)
Bacterial diseases		
Murine typhus	*Rickettsia typhi*	*Xenopsylla cheopis*, *Leptopsylla segnis* (flies)
Rickettsial pox	*Rickettsia akari*	*Liponyssoides sanguineus* (mite)
Human epidemic typhus	*Rickettsia prowazeki*	*Pediculus humanus* (louse)
Scrub typhus	*Rickettsia tsutsugamushi*	*Leptotrombidium* mite
Rocky Mountain spotted fever	*Rickettsia rickettsia*	*Dermacentor* sp., *Amblyomma* sp., *Rhipicephalus* sp. (ticks)
Tick typhus	*Rickettsia sibirica*	Various ticks
Boutonneuse fever	*Rickettsia conorii*	Various ticks
Human ehrlichiosis	*Ehrlichia* sp.	*Ixodes scapularis*, *Amblyomma* sp. (tick)
Plague	*Yersinia pestis*	*Xenopsylla* sp. (flea), *Amblyomma* sp. (tick)
Q-fever	*Coxiella burnetii*	Various ticks
Tularemia	*Francisella tularensis*	Various ticks
Relapsing fever	*Borrelia hermsii*	*Ornithodoros hermsi* (tick)
Lyme disease	*Borrelia burgdorferi*	*Ixodes* sp. (tick)
Protozoal diseases		
Human babesiosis	*Babesia microti*	*Ixodes scapularis* (tick)
Leishmaniasis		
Visceral	*Leishmania* spp.	*Phlebotomus* spp. (sand flies) *Lutzonyia* spp. (sand flies)
Cutaneous	*Leishmania* spp.	*Phlebotomus* spp. (sand flies)
Chaga's disease	*Trypanosoma cruzi*	*Rhodnius prolixus*, *Triatoma* sp. (hemipterans)
Sleeping sickness	*Trypanosoma brucei*	*Glossina* spp. (flies)

Note: Pathogens and vectors are identified to genus when multiple species of a genus are involved as pathogen or vector.
[a]The main vertebrate taxa from which the vector acquires the pathogen during blood meals. Because data are often incomplete or lacking, the list of taxa is not exhaustive.
[b]The main vertebrate taxa that are parasitized by the vector but that play at most a minor role as a disease reservoir.
[c]Includes Louping III, Powassan virus, Russian spring–summer encephalitis.

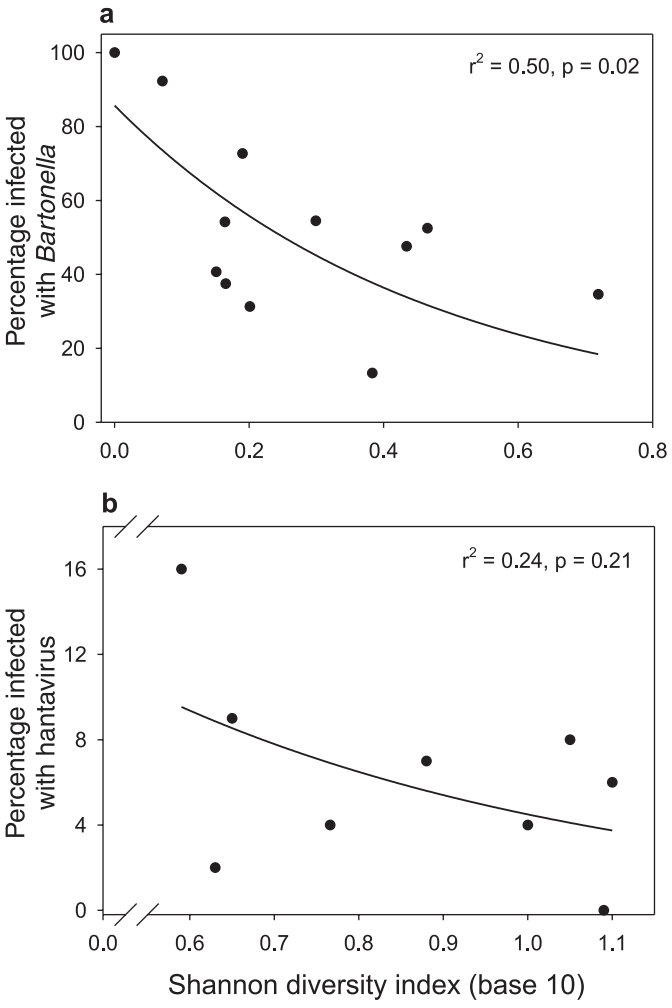
pathogens from urine and feces. Horizontal transmission of these pathogens within rodent populations is associated with intraspecific contact (Mills and Childs 1998). High diversity within vertebrate communities may reduce rates of intraspecific contact either by direct reduction of population density of primary reservoirs or by facilitating interspecific contact at the expense of intraspecific contact.

To explore whether the dilution effect holds promise for understanding geographic patterns in directly transmitted rodent-borne zoonoses, we analyzed data from Kosoy et al.

Principal reservoir hosts(s)[a]	Tangential host(s), including humans[b]	References
Chipmunks, rabbits, tree squirrels	Mammals (?)	Johnson 1990; Woodring et al. 1996
Cattle and domestic stock	Small mammals	Monath 1988; Holbrook 1996
Monkeys, sloths	Primates, birds, rodents	Monath 1988; Holbrook 1996; Pinheiro et al. 1981
Lagomorphs, rodents	Mammals, birds	Monath 1988; Hoogstraal 1979; Piesman and Gage 1996
Passerines	Mammals	Johnson 1990; Woodring et al. 1996; Monath 1988
Monkeys	Humans	Woodring et al. 1996; Monath 1988, 1990; Nasci and Miller 1996
Pigeons, crows	Mammals, birds	Monath 1988; Nasci and Miller 1996; Tesh 1990
Aquatic birds, pigs	Water buffalo	Nasci and Miller 1996; Kettle 1995; Igarashi 1994
Horses, dogs, chickens	Mammals (?)	Monath 1988; Kettle 1995; Doherty 1977
Rodents, shrews	Monkeys, cattle	Hoogstraal 1981; Kettle 1995
Insectivores, rodents, deer, grouse, songbirds	Mammals, birds	Hoogstraal 1981; Hayes and Wallis 1977
Muskrat, water voles	Other rodents (?)	Hoogstraal 1981; Kettle 1995
Passerines	Mammals, horses	Monath 1988; Hayes and Wallis 1977
Passerines	Primates, rodents, horses, pheasants	Monath 1988; Kettle 1995; Scott and Weaver 1989
Rodents, horses	Mammals (?)	Kettle 1995
Cattles, horses, kangaroos, wallabies	Mammals (?)	Kettle 1995; Monath 1991
Monkeys	Primates (?)	Nasci and Miller 1996; Kettle 1995; Monath 1991
Songbirds	Mammals (?)	Monath 1988, 1991; Kettle 1995
Sciurid rodents	Rodents	Piesman and Gage 1996; Kettle 1995
Rats	Rodents	Azad 1990
Mice, voles	Rodents	Kettle 1995; Rehacik 1979
Flying squirrels, humans	Rodents (?)	Piesman and Gage 1996
Rats	Rodents, birds, insectivores	Piesman and Gage 1996; Kettle 1995
Rodents	Mammals, birds	Piesman and Gage 1996; Hoogstraal 1967
Rodents	Mammals, birds	Hoogstraal 1967
Rodents, lagomorphs	Mammals, birds	Kettle 1995; Hoogstraal 1967
Rodents	Mammals, birds, reptiles	Desvignes and Fish 1997; Telford et al. 1996
Rodents, lagomorphs	Mammals	Thomas 1996; Twigg 1978
Wild and domestic ungulates, marsupials	Mammals (?)	Kettle 1995
Rodents, lagomorphs	Mammals, birds	Kettle 1995
Sciurid rodents	Mammals, birds (?)	Piesman and Gage 1996; Kettle 1995
Rodents	Mammals, birds, reptiles	Piesman and Gage 1996; Ostfeld 1997
Rodents	Mammals, birds	Piesman and Gage 1996
Rats, canids, badger	Mammals (?)	Kettle 1995; Tesh and Guzman 1996; Arias and Naiff 1981
Edentates, opossums		
Rodents, hyraxes	Rodents, ungulates	Kettle 1995; Tesh and Guzman 1996
Rodents, armadillos, dogs	Mammals, birds, reptiles	Kettle 1995; Marquardt 1996
Bovids	Mammals, reptiles	Kettle 1995; Marquardt 1996; Molyneux 1980

(1997) and Mills et al. (1998) on *Bartonella* spp., a bacterial zoonosis, and the hantavirus pulmonary syndrome (HPS), respectively. In both cases we regressed the infection prevalence within entire rodent communities, as a surrogate for disease risk to humans, against the species diversity (Shannon index (base 10)) within those communities. Consistent with expectations from the dilution effect, the overall prevalence of infection with *Bartonella* spp. was significantly lower in more diverse rodent assemblages (Fig. 1*a*). We observed a similar pattern for HPS, although the regression

Fig. 1. Relationship between diversity of the community of rodent reservoirs for directly transmitted diseases and the percentage of individual rodents infected with the bacterial pathogen *Bartonella* sp. (data from Kosoy et al. 1997) (A) and viral agents of the hantavirus pulmonary syndrome (data from Mills et al. 1998) (B).



was not statistically significant (Fig. 1*b*). For *Bartonella* spp., an exponential decay curve fit the data better (r^2 = 0.50) than did a linear regression (r^2 = 0.32), which suggests a saturating effect of increasing reservoir diversity on disease risk.

Conclusions

A crucial task for scientists is to determine the ecological consequences of the erosion of biodiversity. Some of these consequences involve changes in the ways in which ecological systems capture and use resources (including pollutants), respond to disturbances, such as drought, and resist invasions by exotic species (Schwartz et al. 2000; Levine 2000). Some experimental and comparative studies reveal utilitarian functions of biodiversity that are important in attaining such desirable goals as pest or weed control, filtration of pollutants, and sustained resource extraction (e.g., Tilman et al. 1996; Daily et al. 1997). We have highlighted an additional utilitarian benefit of biodiversity: protection of human health. High diversity within communities of vertebrates that serve as hosts for vectors or reservoirs for zoonotic diseases may dilute the power of disease transmission to humans. Using both empirical and theoretical evidence we have demonstrated the existence of the dilution effect for Lyme disease in North America (Ostfeld and Keesing 2000; Schmidt and Ostfeld 2000; E.M. Schauber and R.S. Ostfeld, in preparation). However, many crucial aspects of the interactions between biodiversity and zoonotic diseases remain to be explored. Our approach in this paper has been to dissect the logic of the dilution-effect argument and determine the features of disease systems that are required for the dilution effect to operate. Our exploration of the degree to which these features exist across the various vector-borne zoonoses suggests that the dilution effect may apply broadly. However, such a conclusion is not definitive. Although many features of these diseases are well understood by biomedical researchers, in most cases knowledge of the key ecological variables is incomplete or lacking. In part, the absence of relevant information seems to result from a schism between the ways in which epidemiologists, vector biologists, and ecologists view infectious diseases (Rogers 1988). For instance, little effort has been devoted to determining comprehensively the distribution of vector meals on all members of the host community, or assessing for these hosts the probability, under natural conditions, that they will infect the vector. We conclude that the perspectives of, and expertise gained from, the science of ecology have much to contribute to the epidemiology of infectious diseases.

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