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BIODIVERSITY AND THE DILUTION EFFECT IN DISEASE ECOLOGY

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Abstract. Many infectious diseases of humans are caused by pathogens that reside in nonhuman animal reservoirs and are transmitted to humans via the bite of an arthropod vector. Most vectors feed from a variety of host species that differ dramatically in their reservoir competence; that is, their probability of transmitting the infection from host to vector. We explore a conceptual model of what we termed the "dilution effect," whereby the presence of vertebrate hosts with a low capacity to infect feeding vectors (incompetent reservoirs) dilute the effect of highly competent reservoirs, thus reducing disease risk. Using Lyme disease as an example, we demonstrate the presence and estimate the magnitude of the dilution effect for local sites in eastern New York State. We found that the prevalence of Lyme disease spirochetes, *Borrelia burgdorferi,* in field-collected *Ixodes* ticks (37.6% and 70.5% for nymphal and adult stages, respectively) was dramatically lower than expected (~90% and >95% for nymphal and adult stages, respectively) if ticks fed predominantly on highly competent reservoirs, white-footed mice (*Peromyscus leucopus*) and eastern chipmunks (*Tamias striatus*). We inferred the role of additional host species using an empirically based model that incorporated data on tick burdens per host, relative population densities of hosts, and reservoir competence of each host. Assuming an empirically realistic reservoir competence of 5% for non-mouse and non-chipmunk hosts, we determined that alternative hosts must provide 61% and 72% of larval and nymphal meals, respectively. Using computer simulations, we assembled simulated host communities that differed in species richness, evenness, and net interactions between alternative hosts and mice. We found that increasing species richness (but not evenness) reduced disease risk. Effects were most pronounced when the most competent disease reservoirs were community dominants and when alternative hosts had a net negative influence on the dominance of mice as a host for ticks. Our results highlight a critical role of biodiversity and host community ecology in the transmission of vector-borne zoonotic diseases that in turn has important consequences for human health.

Key words: biodiversity and human health; Borrelia burgdorferi; *dilution effect; disease ecology; disease reservoirs; infectious disease;* Ixodes scapularis; *Lyme disease;* Peromyscus leucopus; Tamias striatus; *vector-borne disease; zoonosis.*

INTRODUCTION

Many infectious diseases of humans, termed zoonoses, are caused by pathogens that reside predominantly in nonhuman animal hosts. These nonhuman hosts, which are often rodents (Mills and Childs 1998), are considered disease reservoirs when their bodies are the principal sites of maintenance or growth of the pathogen population. When the primary mode of transmission of the pathogen between reservoir and the human victim is via the bite of an arthropod, the disease is considered a vector-borne zoonosis. For vector-borne zoonoses to exist, the arthropod must be sufficiently generalized in its choice of hosts that individuals have a reasonable probability of biting both a nonhuman reservoir host and a human within their lifetimes. Examples of zoonoses that are transmitted to humans by moderately to highly generalized arthropod vectors include Leishmaniasis, Chagas' disease, West Nile virus, plague, and Lyme disease.

Epidemics of vector-borne zoonoses tend to elicit a reactionary response involving attempts to eradicate the vector, the reservoir host, or both. Eradication programs appear to be based on the assumption, rooted in classical disease ecology, that disease transmission is determined by a single pathogen that resides predominantly within single intermediate and definitive hosts. This classical framework promotes the logic that knocking out the system's dominant players is both

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necessary and sufficient to reduce disease transmission. Such efforts often entail application of nonspecific pesticides or destruction of vector or host habitat, both of which may foster unintended and undesirable ecological consequences. In addition, vector and host eradication programs rarely succeed in eliminating disease.

In reality, pathogens often reside in many species of vectors and hosts, which vary in their ability to support survival and reproduction of the pathogen population. An exclusive focus on the dominant players may fail to take advantage of natural ecological processes that influence disease transmission. Both vectors and reservoirs exist within ecological communities characterized by cryptic and indirect interactions. In this paper, we argue that a more contemporary perspective from community ecology may help us understand and prevent transmission of infectious diseases.

Various species of hosts for vectors differ considerably in their ability to transmit a specific pathogen to a feeding vector. This ability is termed the host's reservoir competence. Weakly competent or incompetent intermediate hosts may play a crucial role in determining the average infection prevalence of the vector. Communities of hosts characterized by high species richness or evenness are likely to contain a high proportion of hosts that are inefficient in transmitting the disease agent to a feeding vector, a phenomenon called the "dilution effect" (Ostfeld and Keesing 2000; see also Matuschka et al. 1991, Matuschka and Spielman 1992). The greater the abundance of weakly competent reservoir species, the stronger the dilution effect and the lower the probability of disease transmission for any given bite from a vector. However, host communities that contain many species of incompetent reservoirs could increase the density of vectors by providing the vector population with more feeding opportunities than they would have in species-poor communities. Thus, more diverse communities could simultaneously decrease infection prevalence and increase the population density of vectors, with unpredictable net effects on disease risk in humans.

In this paper we explore how the diversity and composition of host communities may influence the prevalence of vector-borne zoonoses. We use Lyme disease as our model system because this disease is by far the most common vector-borne disease in the United States (Lane et al. 1991, Barbour and Fish 1993, CDC 1999), and because the natural history of the pathogen (a spirochete, *Borrelia burgdorferi*), the primary tick vector (the black-legged tick, *Ixodes scapularis*), and its vertebrate hosts, are relatively well understood (Lane et al. 1991, Piesman and Gray 1994, Ostfeld 1997). Because it is difficult or impossible to experimentally manipulate diversity and composition of entire vertebrate communities, we use a combination of empirical analysis and modeling. We proceed by: (1) summarizing the natural history of the Lyme disease system; (2) constructing a model of infection prevalence of tick vectors that considers multiple host species; (3) parameterizing the model based on our empirical studies; (4) quantifying the magnitude of the dilution effect; (5) assembling simulated host communities in order to explore the influences of key properties of the host community, including species richness, community diversity, and community interactions; and (6) ending with a general discussion of the dilution effect and the role of host biodiversity as it influences the prevalence of vector-borne diseases.

Natural History

The black-legged tick occurs in forested and shrubby habitats throughout the eastern half of the United States and southern Canada (Ginsberg and Ewing 1989, Lane et al. 1991). This species and its Lyme disease-bearing relatives from western North America and Eurasia (*Ixodes pacificus, I. ricinus,* and *I. persulcatus*) require three blood meals to complete their life cycle. The initial stage, called the larva, hatches uninfected with Lyme disease spirochetes despite adult infection due to highly inefficient vertical transmission (Piesman et al. 1986, Patrican 1997). Larval ticks may take their single blood meal from any one of a variety of vertebrate hosts, particularly rodents, medium-sized mammals (e.g., raccoons, *Procyon lotor*), ground-dwelling birds, and lizards (Anderson and Magnarelli 1984, Battaly et al. 1987, Magnarelli et al. 1992, Battaly and Fish 1993, Mannelli et al. 1993, Oliver et al. 1993, Brillhart et al. 1994, Levine et al. 1997). Those that feed successfully drop off the host and molt the following year into the second juvenile stage, called the nymph. Nymphs similarly take their single blood meal from a member of the same group of vertebrates, and after dropping off the host, molt into the adult stage. Adult black-legged ticks are more specialized than are the larval and nymphal stages, feeding predominantly on large mammals, particularly white-tailed deer (*Odocoileus virginianus*).

Ticks may become infected with the Lyme disease spirochete if they take a blood meal from an infected host, and once acquired by the tick, infections are maintained for life (Mather 1993). Thus, nymphal ticks, that have obtained a single blood meal (as a larva), have had one chance to become infected, whereas adults, that have obtained two blood meals (one each as a larva and nymph), have had two opportunities to become infected. Accordingly, adult ticks have higher infection prevalence than do nymphs (Lane et al. 1991).

Ixodes scapularis is a host generalist and many of its hosts are poor reservoirs or incapable of transmitting Lyme spirochetes. In theory, the relative importance of each species of host in infecting ticks could be determined by a complete accounting of the distribution of juvenile ticks on all potential host species, the relative densities of hosts, and their respective reservoir competence. In practice, logistical difficulties have prevented the performance of such studies. Instead, the

focus of ecological research on Lyme disease has been directed at the interaction between ticks and the host species with the highest competence, i.e., white-footed mouse (*Peromyscus leucopus*) and, to a lesser extent, eastern chipmunks (*Tamias striatus*). These hosts provide the greatest contribution to the infection prevalence of the vector. In the next section, we model infection prevalence based solely on the presence of mice and chipmunks. We then use its parameterized form (based on our empirical studies) to show how the distribution of ticks on alternative hosts can be inferred and the magnitude of the dilution effect estimated.

Modeling Infection Prevalence

Based on epidemiological analyses, Barbour and Fish (1993) determined that the primary factor determining the risk of human exposure to Lyme disease is the local density of nymphal ticks infected with *B. burgdorferi*. Therefore, we focus on two indicators of Lyme disease risk; nymphal infection prevalence (NIP), and the density of infected nymphs (DIN). Nymphal infection prevalence represents the probability that any given tick bite will, on average, transmit a *Borrelia* infection to the host. Density of infected nymphs represents the local probability of human exposure to an infection event. In this section, we focus on NIP and later turn to DIN. To illustrate the model's structure, we begin by considering only two host species, white-footed mice and eastern chipmunks, before proceeding to an analysis of a more complete model. We emphasize that we do not consider a dynamical model of Lyme disease transmission, but rather focus on parameters (e.g., reservoir competence, community interactions) that deal with the mechanisms of disease transmission and are themselves important components of a larger dynamic system.

Assume that densities of mice and chipmunks at time t (= 1 yr) are given as M_t and C_t, respectively. Let α and β be the probability that a tick becomes infected with *B. burgdorferi* from either a mouse or chipmunk, respectively. Note that these probabilities represent the product of the probability that a host individual is infected with *Borrelia* (host infection prevalence), and the probability that a host individual transmits the spirochetes to a feeding tick (reservoir competence in the strict sense). We combine these two probabilities into a single term because we are interested in the total probability that an individual tick feeding from an average host individual (mouse or chipmunk) will acquire *Borrelia*.

Let a_L and a_N be the feeding bias of larval and nymphal ticks, respectively, towards mice relative to chipmunks (standardized to one). That is, the relative number of larval ticks that feed from the average mouse is a_L times the number that feed from the average chipmunk, and similarly for nymphal ticks in the case of a_N. A greater number of larvae per mouse than per chipmunk is implied when $a_L > 1$, while $a_L < 1$ implies the opposite. The biases encapsulated by a_L and a_N may include habitat preferences of the rodents that influence the encounter probability with ticks, host preferences of the questing ticks, and/or biases in removal of ticks by grooming. Given these parameters, the probability that a questing nymph is infected as a larva is

$$(P_N)_t = \alpha[a_L M_t/(a_L M_t + C_t)] + \beta[C_t/(a_L M_t + C_t)]. \quad (1)$$

Likewise, the probability that a questing adult is infected as either a larva or a nymph is

$$(P_A)_{t+1} = [1 - (P_N)_t]\{\alpha[a_N M_{t+1}/(a_N M_{t+1} + C_{t+1})] + \beta[C_{t+1}/(a_N M_{t+1} + C_{t+1})]\} + (P_N)_t \quad (2)$$

where $(P_N)_t$ and $(P_A)_{t+1}$ denote nymphal and adult tick infection prevalence, respectively. The subscripts t and $t + 1$ for nymphal and adult infection prevalence, respectively, reflect that *Ixodes* has a 2-yr life cycle with larval ticks feeding in the summer of year t, and nymphal ticks emerging and feeding the following summer (year $t + 1$). Also notice that infection prevalence is determined by relative host densities and not their absolute densities.

To simplify our model, we assume that the relative densities of the two rodent species are spatially and temporally consistent. This assumption is supported by recent field studies in which densities of mice and chipmunks were monitored over time. Summer densities of mice and chipmunks were positively correlated with a slope of 0.519 ($n = 26$, $r = 0.560$, $P < 0.001$) and an intercept of 4.87, which was not significantly different from zero ($P > 0.40$) (see Schmidt et al. 1999 for details of our trapping protocol and enumeration of rodent densities). Year ($n = 3$) and plot ($n = 6$) effects, included as independent variables, were not significant ($F < 0.60$, $P > 0.50$). From these results, we concluded that at the scale of ~2.5-ha plots, mouse density is, on average, 1.92× the density of chipmunks, and that relative density does not vary in space or time, at least within the same habitat type (also see Slajchert et al. 1997). Because of the lack of year effects on the relative densities of the two rodent species, we make the assumption in all analyses below that the relative density of mice and chipmunks, on which $(P_N)_t$ and $(P_A)_{t+1}$ depend, is constant among years. Thus, we drop the subscripts denoting years from the terms in Eqs. 1 and 2. Furthermore, we let C and M refer to relative densities; we set chipmunk density to unity (C = 1) and change the relative densities of rodents by varying M.

Eq. 1 and 2 assume ticks infest their rodent hosts in proportion to host availability provided there is no feeding bias (when a_L and $a_N = 1$). However, given a feeding bias, the relative density of mice is adjusted downward (a_L or $a_N < 1$) or upward (a_L or $a_N > 1$) by the product of relative density and feeding bias, $a_L M$. This product combines both the relative numerical dif-

ferences between host populations and feeding biases into a parameter combination we call relative host dominance. This is defined as the number of ticks, specified by subscript as larvae (L) or nymphs (N), that feed from mice relative to chipmunks. Because density and feeding bias are standardized to chipmunks, the relative dominance of chipmunks is always equal to unity, whereas a_LM can have any positive, nonzero value.

Proportional host dominance, $D_{i,j}$ is similarly defined as the proportion of ticks (j = L or N) that feed from host i (in the current model, i = M and C for mice and chipmunks, respectively). For mice, proportional host dominance is given by: $D_{M,L} = a_L M/(a_L M + C)$; and $D_{M,N} = a_N M/(a_N M + C)$. Proportional chipmunk dominance can be similarly expressed in longhand; or, because the sum of mouse and chipmunk dominance must equal one, $D_{C,L} = (1 - D_{M,L})$ and $D_{C,N} = (1 - D_{M,N})$.

Adding host species to the model

We know that larval and nymphal black-legged ticks parasitize other species of hosts, and that these hosts tend to have much lower reservoir competence than do white-footed mice and eastern chipmunks. However, little information exists for predicting how the presence and abundance of alternative hosts affects Lyme disease risk (Ostfeld and Keesing 2000). To incorporate a third category of hosts, which we call Host X, we modify Eqs. 1 and 2 for three host species; i.e., mice, chipmunks, and Host X:

$$P_N = \alpha[a_L M/(a_L M + C + a_X X)] + \beta[C/(a_L M + C + a_X X)] + \chi[a_X X/(a_L M + C + a_X X)]; \quad (3)$$

$$P_A = (1 - P_N)\{\alpha[a_L M/(a_L M + C + a_X X)] + \beta[C/(a_L M + C + a_X X)] + \chi[a_X X/(a_L M + C + a_X X)]\} + P_N. \quad (4)$$

Host X has population density X, feeding bias a_X (that may or may not differ for larvae and nymphs), and a probability of transmitting infection, χ, analogous to α and β for rodent hosts. We cannot separate a_X from X without empirical data, but we can infer the role of Host X through its relative dominance, $a_X X$. The term $a_X X$ represents the dilution factor necessary to give congruent results between the expected tick infection prevalence from our model and the empirically measured tick infection prevalence. This dilution factor is due to a combination of the density of Host X, more host individuals on which to attach, and the feeding bias of Host X, the relative number of ticks per host individual. The relative magnitude of each is unknown. For example, raccoons occur at low densities (about one individual per 10 ha), but each may harbor hundreds of ticks (Mannelli et al. 1993), and thus raccoons may effectively dilute disease prevalence despite their low densities.

Solving Eqs. 3 and 4 for $a_X X$ in terms of χ yields

$$a_X X_L = [\alpha(a_L M) + \beta(C) - P_N(a_L M + C)] \div (P_N - \chi); \quad (5)$$

$$a_X X_N = \{(1 - P_N)[\alpha(a_N M) + \beta(C)] - (P_A - P_N)(a_N M + C)\} \div (P_A - P_N - \chi + \chi P_N). \quad (6)$$

Parameterizing the Model

In this section, we use our empirical studies of Lyme disease from New York State to estimate the model's parameters in order to evaluate the magnitude of the dilution effect. Our field studies take place on six 2.25-ha plots in areas dominated by red oak (*Quercus rubra*) and chestnut oak (*Q. prinus*) (see Ostfeld et al. 1996*a* and Jones et al. 1998 for thorough descriptions of the study sites). We employ a battery of field and laboratory techniques in order to estimate the following parameters: (1) absolute and relative densities of the two numerically dominant species of rodent, the white-footed mouse and the eastern chipmunk; (2) densities of host-seeking larval, nymphal, and adult ticks; (3) numbers of larval and nymphal ticks attached to mice and chipmunks (adult ticks are never found on these hosts); (4) the probability that larval and nymphal ticks feeding from free-ranging mice and chipmunks acquire the Lyme disease infection; and (5) the proportion of host-seeking nymphal and adult ticks that are infected with the Lyme disease spirochete (tick infection prevalence).

Host use by juvenile ticks

Using regression analyses, Schmidt et al. (1999) estimated the relative host use from larval and nymphal ticks feeding on chipmunks and mice captured at the same trap station during the same week. This criterion controlled as much as possible for variation in the local densities of ticks in both space and time. We observed distinct differences in tick burdens between the two tick life stages, with more larvae on mice and more nymphs on chipmunks. Specifically, larval tick burdens on mice were, on average, 2.24× higher than larval burdens on chipmunks (a_L = 2.24), whereas nymphal tick burdens were, on average, 0.30× the levels found on chipmunks (a_N = 0.30).

Reservoir competence of the most common hosts

In 1998 and 1999 we captured mice and chipmunks (the two most common hosts at our sites and representing >95% of small mammal captures) in the field, and held them in the laboratory for two days of observations. Hosts brought into the laboratory were a random sample of the host population, and were col-

lected during the seasonal peak in larval activity, which is the appropriate time to measure their ability to infect feeding larvae. Ticks dropping off these animals were collected twice daily from pans of water held underneath caged animals. After ticks molted into nymphs or adults, they were examined for the presence of *B. burgdorferi* using direct immunofluorescence microscopy (Lane and Burgdorfer 1987). We used only newly molted nymphs, which had fed only once as larvae, to estimate reservoir competence. Thirty-five mice and 30 chipmunks provided 161 and 141 molted nymphs, respectively. Reservoir competence, calculated as the mean proportion of newly molted nymphs infected by an individual rodent, was $\alpha = 0.935$ ($\pm$ 0.032 SE) and $\beta = 0.687$ ($\pm$ 0.071 SE) for mice and chipmunks, respectively.

This "wild caught" calculation of reservoir competence is appropriate provided that all individuals are infected with Lyme disease. All 35 mice yielded at least one infected nymph, indicating that all mice were infected with *Borrelia.* Twenty-five of 30 chipmunks (83.3%) yielded at least one infected nymph. The five chipmunks that did not yield any infected nymphs fed a mean of 1.2 larvae per host, and were among the 16 chipmunks that produced two or fewer fed larvae. Given that the mean proportion of nymphs infected by chipmunks was 0.687, we would expect four or five of the 16 chipmunks to yield no infected nymphs even if all 16 hosts were infected. Thus, we can safely assume that all individual mice and chipmunks were infected. The high competence reported here is consistent with other studies of mice (Levine et al. 1985, Mather et al. 1989), but is higher than typically found in chipmunk populations (Godsey et al. 1987, Slajchert et al. 1997).

Infection prevalence of wild-caught ticks

At monthly intervals between April and November of each year, we collected all life stages of questing black-legged ticks by dragging 1-m^2 pieces of white corduroy cloth (see Falco and Fish 1992) along 400-m transects within each of the forest plots. Ticks were identified and brought to the lab where nymphs and adults were tested for the presence of *B. burgdorferi* using direct immunofluorescence. Between 1995 and 1998, 478 of 1271 nymphal ($P_N = 37.6\%$) and 572 of 811 adult ($P_A = 70.5\%$) ticks tested positive for *Borrelia.*

Quantifying the dilution effect

In the absence of alternative host species, the expected infection prevalence is between 0.687 and 0.935 for nymphs and >0.96 for adults (the minimum probability of escaping infection from two meals assuming mice and chipmunks are equally likely hosts = $[1 - (0.687 + 0.935)/2]^2 = 0.036$). We suggest that the dramatically lower empirical infection prevalence of wild-caught nymphs (37.6%) and adults (70.5%) is caused by the presence of alternative, poorly competent hosts that dilute the infection prevalence of the tick population. This difference is the estimate of the dilution effect due to non-mouse, non-chipmunk hosts.

Given that we have established the existence of a dilution effect, we can now use the model to explore its magnitude. That is, we ask how many tick meals must be taken from the collection of non-mouse, non-chipmunk hosts to give the infection prevalence values we observe in host-seeking ticks. We use Eqs. 5 and 6 to calculate the relative dominance for Host X at any specified value of χ for each life stage, and from that, the proportional dominance of each host species. If we assume $\chi = 0.05$, an empirically realistic competence value (birds, Magnarelli et al. 1992; Nichols and Callister 1996; voles, Mather et al. 1989, but see Markowski et al. 1998; raccoons, Norris et al. 1996, Ouellette et al. 1997, Slajchert et al. 1997; deer, Telford et al. 1988; lizards, Manweiler et al. 1990, Lane and Quistad 1998), the proportional dominance values are $D_{X,L} = 0.611$, $D_{M,L} = 0.316$, and $D_{C,L} = 0.073$. In other words, Host X is responsible for 61.1% of all larval tick meals, mice for 31.6%, and chipmunks for the remaining 7.3%. Similarly, for nymphal meals we obtain as $D_{X,N} = 0.716$, $D_{M,N} = 0.104$, and $D_{C,N} = 0.179$. Host X accounts for 71.6% of nymphal tick meals. Host X, in fact, is the major contributor of blood meals at each juvenile life stage of the tick, and as a consequence of its low competence, strongly dilutes the impact of both mice and chipmunks.

The relationship between the relative dominance of Host X and nymphal infection prevalence (NIP) is hyperbolic with accelerating NIP as Host X declines (Fig. 1). For the parameterized model, this relationship begins to accelerate rapidly as the relative dominance of Host X is reduced below 10. Further decreases result in rapidly increasing NIP. On the other hand, further increases in Host X's relative dominance past ~20 yield much smaller decreases in NIP per unit increase in X's dominance. While a relative dominance of 20 may seem large, medium-sized mammals, while they have lower population densities than chipmunks, may be capable of supporting hundreds of ticks (e.g., Mannelli et al. 1993 found, on average, 420 larvae per raccoon). The absolute limit of Host X's relative dominance is an important issue that will have to be assessed empirically. In contrast, increasing the relative dominance of the highly competent rodent species increases NIP. This is particularly true of mice. Decreasing absolute mouse dominance is also a potentially effective method of reducing NIP.

Caveats

We have assumed that infection prevalence can be entirely explained by patterns of host use (i.e., tick burdens), host abundance, and reservoir competence. In other words, the difference between empirically measured infection prevalence and that expected when assuming mice and chipmunks are the only hosts is due

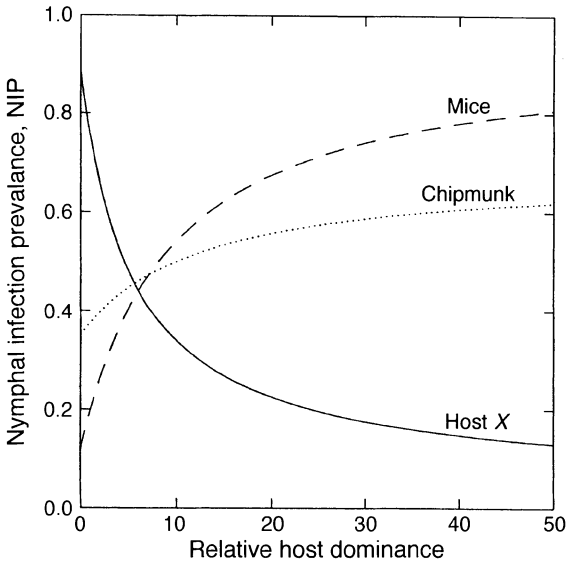

FIG. 1. Nymphal infection prevalence (NIP; proportion of nymphal ticks infected) as a function of the relative dominance of each host species. Each curve assumes that the relative dominance of the alternative hosts remains constant at our empirically determined values (mice = 4.33, chipmunks = 1.0, Host X = 8.37). Increasing the dominance of highly competent hosts (mice or chipmunks) results in greater NIP; whereas, increasing the dominance of Host X (incompetent species) decreases NIP.

entirely to the presence of Host X. This assumption may potentially overestimate or underestimate the impact of Host X. Specifically, we recognize at least two potential errors. First, survival rates of ticks may be influenced by the presence of *Borrelia*. For example, if ticks with *Borrelia* have lower survivorship, the dilution attributed to Host X will be overestimated. Second, temporal variation may exist in the probability of hosts passing on infection within a year. We can dismiss the first source of error because infection prevalence of questing adult ticks does not differ between individuals collected in the fall or in the spring of the following year (R. S. Ostfeld, *unpublished data*). This suggests that overwinter survival of adult ticks is not biased by whether they carry *Borrelia*. The second source of error can also be dismissed. We based our infection prevalence data on ticks removed from rodents that were collected between 19 June and 2 September and observed no trend toward either decreasing or increasing infection prevalence over time (R. S. Ostfeld, *unpublished data*). While the density of hosts, tick burdens, and infection prevalence have been noted to vary between years, these fluctuations are not significantly correlated to one another or to host density of the previous year in any consistent manner (Schmidt et al. 1999). Finally, other biases may be due to clumping of either tick or host populations, but we lack the data to evaluate these possibilities.

SIMULATIONS: HOST COMMUNITIES AND LYME DISEASE RISK

The dilution effect as defined (see Ostfeld and Keesing 2000) refers to infection prevalence; any increase in the abundance of poorly competent hosts will decrease the infection prevalence in the vector population. However, such an increase in host abundance and diversity may also increase the number of feeding opportunities for larval ticks, and thus the absolute number of ticks. Therefore, we now address the effects of changes in host community composition on the density of infected nymphal ticks, DIN, as a measure of human risk of contracting Lyme disease (Barbour and Fish 1993). Because vertebrate communities (Host X) are composed of many hosts that vary in both dominance and reservoir competence, a large number of unknowns hinders our ability to provide a meaningful assessment of Lyme disease risk in relation to community composition using analytical modeling, and we turn to simulation modeling.

We used computer simulations to explore relationships between DIN and several potentially important and quantifiable community parameters: (1) host species richness, SPP; (2) host community diversity, H'; (3) the correlation between host dominance and reservoir competence, CORR; and (4) the mean (i.e., community) interaction coefficient between the species that make up Host X and mice, CI. We included CORR to reflect the observed pattern in zoonoses that the most competent disease reservoir often is a numerically dominant member of the host community (Ostfeld and Keesing 2000). We included CI to reflect the likelihood that species added to the vertebrate community will interact with mice and chipmunks, particularly as predators (e.g., carnivores) or competitors (e.g., other rodents, insectivores, birds, deer). We calculated community diversity, by adapting the Shannon index, $H' = -\Sigma\,[p_i \times \log(p_i)]$, where p_i gives the proportion of tick meals obtained from the ith (i = 1, 2, . . . , 20, C, M) species. Note that we examine the diversity of tick meals across species and not species densities, because species density can be misleading if large host species occur at low densities but harbor large numbers of feeding ticks (e.g., raccoons).

Our initial host community was composed solely of mice and chipmunks, and was parameterized to closely match our empirical results (α = 0.935, β = 0.687, $D_{M,L}$ = 5, $D_{C,L}$ = 1). Reservoir competence of each host was constant throughout the simulations. Relative dominance of mice varied with the number of species added to the host community. However, we held the relative dominance of chipmunks fixed throughout (although proportional dominance declined as species were added). Thus we concentrated on interactions between additional host species and mice, which have by far the greater impact than do chipmunks on DIN (see *Parameterizing the model: Quantifying the dilution effect*).

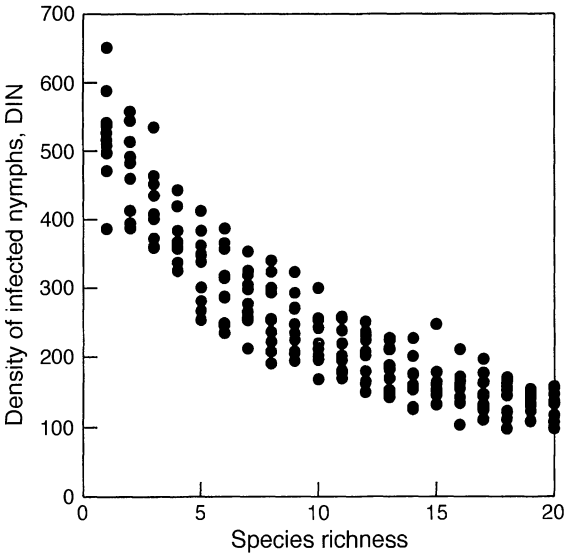

FIG. 2. The density of infected nymphs (DIN) declines with species richness (number of non-mouse, non-chipmunk hosts). Data were generated from 10 simulated host communities at each level of species richness (plus mice and chipmunks). DIN as used in Figs. 2–6 is based on the relative dominance and reservoir competence values assigned to host species in the model simulations and is for comparative purposes.

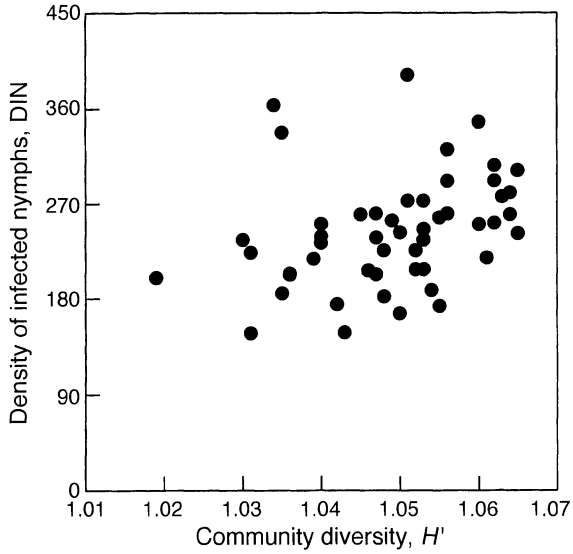

FIG. 3. The density of infected nymphs (DIN) is unrelated to community diversity (Shannon diversity index, H'). Data were generated from 50 simulated host communities, each containing 10 hosts species (plus mice and chipmunks).

We sequentially added up to 20 additional host species (X_i = 1, 2, . . . , 20) to the basic mouse and chipmunk community. Each component species, X_i, was given: (1) a relative dominance drawn from a normal distribution with $\bar{X} = 2$ and $\sigma = 1.0$; (2) a reservoir competence drawn from a uniform distribution between 0.0 and 0.20; and (3) an interaction coefficient (CI) drawn from a normal distribution ($\bar{X} = 0.90$, $\sigma = 0.15$). Mouse dominance was adjusted with each added species by the product of CI and mouse dominance in the community lacking X_i. The interaction coefficient determines the effect of species X_i on the dominance level of mice, where CI > 1.0 indicates a net positive effect on dominance level of mice, and CI < 1.0 indicates a net negative impact on mouse dominance. On average, the dominance of mice was reduced by 10% with each species added, while any individual species X_i may either magnify or reduce the dominance of mice. We chose a mean CI < 1.0 because we hypothesized that the species pool of forest floor vertebrates from which Host X can be drawn consists largely of carnivorous and omnivorous mammals, and granivorous and insectivorous birds. Thus, it is likely that Host X will have a net antagonistic effect on mice through a combination of predation and competition. We expect net mutualistic effects of Host X on mice to be uncommon.

We calculated the nymphal infection prevalence for each simulated host community using equations of the form given by Eq. 3 extended for additional hosts species. We calculated the density of ticks using the sum of relative dominance values for each community. There is no direct correspondence between this sum and actual tick densities, but we can use the dominance sum to draw comparisons between simulated communities. Therefore, the results presented in Figs. 2–6 should be used for comparative purposes only; e.g., to

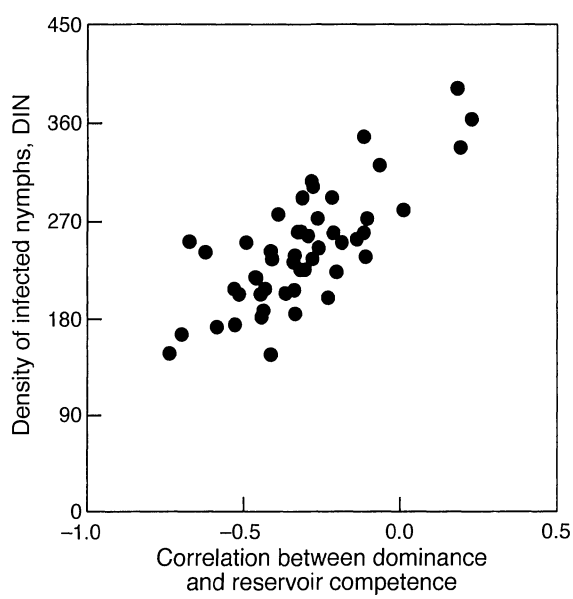

FIG. 4. The density of infected nymphs (DIN) increases when highly competent hosts are also dominant species in the community (as indicated by the correlation between host dominance and reservoir competence). Data were generated from 50 simulated host communities each containing 10 hosts species (plus mice and chipmunks).

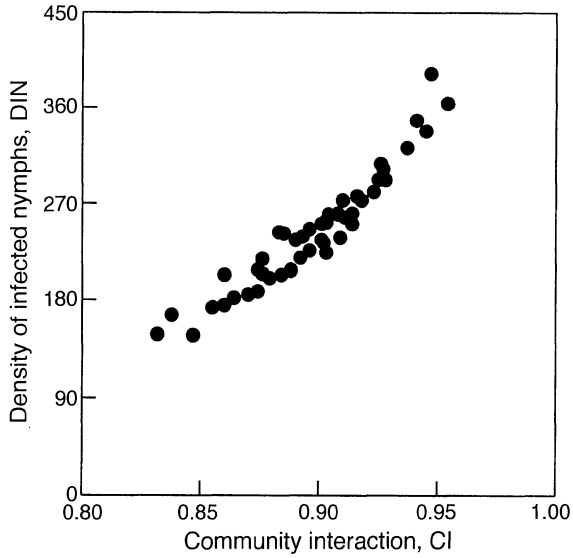

FIG. 5. Density of infected nymphs (DIN) as a function of the mean community interaction (CI). CI = 1.00 indicates no net interaction between host species and mice. The more negative the mean effect of Host X on mice (i.e., the lower is the CI), the lower the DIN. Data were generated from 50 simulated host communities, each containing 10 hosts species (plus mice and chipmunks).

determine the magnitude of reductions or increases in disease risk as influenced by community properties.

Our simulations show that an increase in species richness causes a dramatic reduction in DIN (Fig. 2). The shape of the relationship is nonlinear, with a diminishing effect of species addition on DIN in already species-rich communities. The shape of the relationship between SPP and DIN matches the empirical nonlinear relationships seen in natural large-scale community assemblages (Ostfeld and Keesing 2000).

Because of the relationship between species richness and DIN, we examined the remaining variables under constant species richness. We held species richness constant at 10 species (plus mice and chipmunks for a total of 12 species) and determined DIN, H', CORR, and CI for 50 simulated host communities. The density of infected nymphs shows no relationship to community diversity (H') when species richness is held constant (Fig. 3). However, DIN increases strongly as the correlation between host species dominance and reservoir competence increases (CORR; Fig. 4). In host communities in which the numerically dominant species is/are the most competent reservoir(s), we expect disease risk to be particularly high. DIN also increases strongly as the mean community interaction decreases (i.e., CI = 1.0 indicates no interaction between host species and mice; Fig. 5). The more negative the effect of Host X on mice (CI $<$ 1.0) the lower is the risk of Lyme disease (Fig. 5).

Finally, to explore the simultaneous influences of SPP, H', CORR, and CI, we assembled 120 simulated communities, 20 each of six to 12 species added to a community containing mice and chipmunks. We analyzed the output using ANCOVA, with DIN as the dependent variable, SPP as a covariate, and H', CORR, and CI as independent variables. This analysis showed that the effect of species richness (in our model) is contingent on the community composition of hosts. Factoring out H' by including it as a main effect in the ANCOVA model, but without incorporating CORR or CI into the ANCOVA, species richness (adjusted least square means) had a positive effect on DIN (Fig. 6a). More hosts simply provide more feeding opportunities for ticks. It is only when additional hosts have negative impacts on the dominance of highly competent hosts (either via changes in host abundance or feeding preferences of ticks) that increasing species richness is effective at reducing Lyme disease risk (Fig. 6b).

To summarize, our simulations indicate that the strength of community interactions and the relationships between host dominance and reservoir compe-

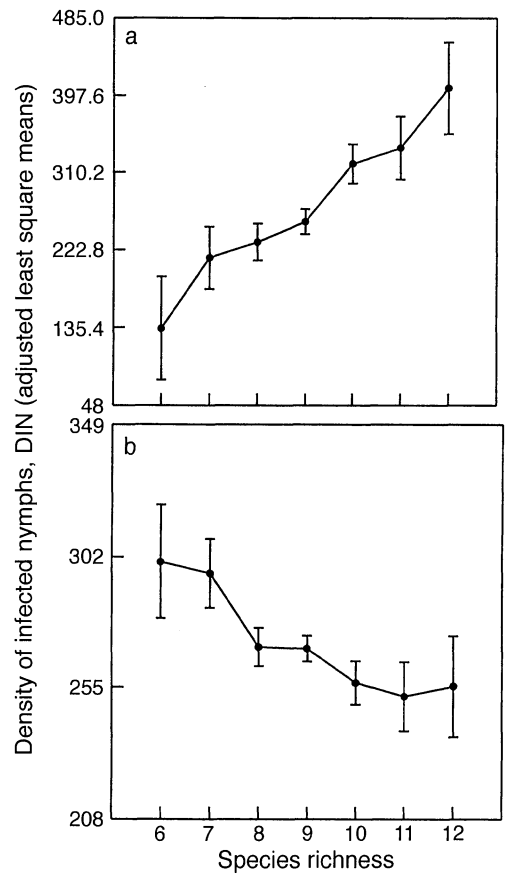

FIG. 6. Density of infected nymphs (DIN) as a function of the number of species added to a community of mice and chipmunks. DIN is given as the adjusted least square means (± 1 SE) calculated (a) using H' as an independent variable, but without inclusion of CORR and CI into the ANCOVA model, or (b) by factoring out the effects of CORR, CI, and H'.

tence strongly influence the density of infected nymphs and thus Lyme disease risk in humans. The addition of host species without accounting for their effects on the population size of highly competent hosts or on feeding preferences of ticks resulted in an increase in DIN and, therefore, in disease risk. However, when our model accounted for the negative interactions between the species added to the community and both the population sizes and tick burdens on highly competent hosts, increasing species richness dramatically reduced DIN. Such an effect, of course, would not be observed in situations in which additional species exhibit mutualisms with highly competent hosts. In either case, consideration of the effects of changes in species richness on disease risk should incorporate interactions among the various hosts, in addition to the direct interactions between hosts and ticks.

General Discussion

We have used a combination of empirical and modeling approaches to ask how changes in the community of vertebrate hosts will influence the infection prevalence of Lyme disease vectors and the density of infected nymphal ticks, and hence the risk of human exposure to disease. Previously, we have described a conceptual model of the "dilution effect" (Ostfeld and Keesing 2000) whereby the presence of vertebrate hosts that have a low capacity to infect feeding vectors dilutes the effect of highly competent reservoirs, thus reducing disease risk (also see Matuschka and Spielman 1992, Matuschka et al. 1992). Zooprophylaxis, the diversion of bites by disease vectors from humans to alternative hosts (e.g., Hess and Hayes 1970), may similarly reduce disease risk to humans, but fails to consider the density or infection prevalence of the vector. In this paper, we focused on Lyme disease as a model system and generalized the results through the creation of simulated host communities.

Our empirical observations at study sites in southeastern New York State indicate that the average reservoir competence of white-footed mice and eastern chipmunks, the two species we capture most frequently, is extremely high. In the absence of alternative hosts, >70% of nymphal and >95% of adult ticks that fed from these hosts during earlier instars can be expected to acquire the Lyme spirochete. However, the actual nymphal infection prevalence (NIP) is dramatically reduced (NIP ~30%) by the presence of a group of nonmouse, non-chipmunk hosts of low reservoir competence that is highly effective in diluting the effects of the primary rodent hosts. We term this conglomerate of hosts "Host *X*," and estimate that given an empirically realistic competence of 0.05 for Host *X*, ~60% of larvae and 70% of nymphs feed on Host *X* at our study sites.

Our modeling approach indicates that the greater the abundance of Host *X* relative to mice and chipmunks, the lower the infection prevalence of ticks (Fig. 1); and, therefore, the lower the probability that any given tick bite will transmit Lyme disease to a human host. White-footed mice and eastern chipmunks are among the most abundant vertebrates in many forest ecosystems throughout the eastern United States (Kaufman and Kaufman 1989, Hamilton and Whitaker 1998), and they are among the hosts most commonly parasitized by black-legged ticks (Schmidt et al. 1999 and references therein). The positive effect of these two hosts on the infection prevalence of ticks can be reduced by decreasing their abundance relative to that of Host *X*. Our simulation results suggest that such a reduction may be accomplished by increasing the species richness of the community of vertebrate hosts for ticks (also see Van Buskirk and Ostfeld 1995, 1998). If so, this would constitute a tangible benefit to humans of preserving biodiversity.

However, if species richness in the vertebrate community increases without changing the absolute abundance of the most competent reservoirs, then the added species may simply increase the number of feeding opportunities for ticks and, therefore, tick density. Such an effect may increase disease risk if increasing tick density is not offset by decreasing infection prevalence. Indeed, analysis of our simulation model indicated that when species additions are assumed to have no effect on either density of or tick burdens on the most competent reservoirs, disease risk will increase with species richness (Fig. 6a). When we made the more realistic assumption that additional species are likely to compete with or prey on mice, and that their presence reduces the absolute number of ticks feeding on mice by siphoning away tick meals, then high species richness will dramatically reduce the density of infected nymphs (DIN) and thus disease risk (Figs. 2 and 6b).

The structure of our model host community is admittedly simplistic. For instance, we limited interactions within host communities to those between host species and mice, and did not include explicit changes in host preference as communities are assembled (e.g., Hess and Hayes 1970). However, in the absence of empirical details on the structure of complete vertebrate assemblages, we avoided adding components that require making specific and detailed assumptions regarding host community structure. Furthermore, our analysis looks for statistical trends among general community descriptors such as species richness and community diversity. While detailed and complex simulated communities might, if realistic and correctly parameterized, describe relationships more accurately, at this initial stage of analysis we hope to suggest which community components may be critical for effective operation of the dilution effect.

Generality of the dilution effect

We propose that the presence of the following features of a disease system are necessary to result in decreasing disease risk with increasing species richness

in the host community. First, the pathogen must exhibit a substantial degree of horizontal transmission, from vector to host and host to vector, rather than relying exclusively or primarily on vertical transmission. Vertical transmission of some pathogens, e.g., some viruses and rickettsiae, via transovarial passage across generations, liberates the pathogen from reliance on vertebrate reservoirs. Most zoonotic pathogens appear to require a substantial degree of horizontal transmission, being acquired by the vector from a pool of vertebrate reservoirs rather than residing exclusively within the population of vectors. Second, the vector must exhibit generalized feeding habits, parasitizing more than one host species. Virtually all arthropod vectors of human diseases are known to parasitize at least several and sometimes dozens of host species. Finally, substantial differences among hosts must exist in their reservoir competence, that is, in the probability that they will infect a feeding vector. Although this condition appears likely to be met for many or most disease systems, insufficient evidence exists to evaluate its generality. Surprisingly little research has been performed to evaluate reservoir competence of vertebrate hosts for disease vectors. Comparative studies of reservoir competence are warranted. To the extent that these conditions are met in other disease systems, we contend that the dilution effect will be a general phenomenon.

Conclusion

Using Lyme disease as a model system, we have shown that the community composition of hosts for vector-borne zoonoses has important consequences for infection prevalence among vectors and, in turn, for human health. A community of abundant alternative hosts with reduced competence mitigates Lyme disease risk in this system by decreasing the average infection prevalence of ticks. When alternative hosts have net negative impacts on the most competent reservoirs, via competition and predation, the dilution effect is compounded. We believe this dilution effect to be a general phenomenon of disease ecology because the majority of vector-borne zoonoses are characterized by the features necessary for it to occur. Unfortunately, little is known regarding how vectors of many zoonoses interact with their host communities to determine disease risk. We hope that by highlighting a critical role of host community ecology through an illustration of the dilution effect, we will stimulate further research in a field that has great consequences for human health.

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