CHAPTER 3

Community ecology meets epidemiology: the case of Lyme disease

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3.1 Background

Any vector-borne zoonotic disease involves *at least* four species: the human victim, the pathogen, the vector, and the wildlife reservoir. Typically, though, the pathogen (usually viral or bacterial) can infect multiple species of wildlife hosts, and the vector (usually an arthropod) can feed from many wildlife species in addition to the human and wildlife reservoir. In addition, population density of the reservoir (usually a mammal or bird) and vector might be controlled or regulated by natural enemies or resources. To fully understand disease risk, scientists therefore might need to study interactions among a formidable number of species, and potentially between each species and its abiotic environment. Thus, although epidemiology appears to have primacy as the science devoted to analysis of variation in human disease risk and incidence, a key role for community ecology also seems fundamental.

The crucial importance of community ecology in understanding disease risk is best explored by considering a particular disease system whose ecological components are relatively well understood. Lyme disease (LD) is a tick-borne zoonosis caused by the bacterium *Borrelia burgdorferi*. LD was first described in the mid-1970s in the US state of Connecticut, but it is now known from Eurasia, Africa, and Australia in addition to North America (Barbour and Fish 1993). In some temperate regions, LD is the most commonly reported vector-borne disease (CDC 2003). Ticks in the *Ixodes ricinus* complex are the primary vectors; in the United States, the most important vector is the blacklegged tick, *I. scapularis*. In the enzootic cycle, *B. burgdorferi* is reciprocally transmitted between ticks and wildlife reservoir hosts; LD exists because humans can serve as hosts for both the tick and the pathogen and thus can become an accidental part of this cycle. The *Ixodes* tick life cycle includes four stages: egg, larva, nymph, and adult. Larvae, nymphs, and adults each take a single blood meal, lasting from several days to a week, from a vertebrate host before dropping off and molting into the next stage (in the case of larvae and nymphs) or before reproducing and dying (in the case of adults). Larvae and nymphs are highly generalized in their choice of hosts and are known to parasitize dozens of species of mammals, birds, and lizards. Adults tend to be restricted to larger mammals, particularly white-tailed deer, *Odocoileus virginianus*. Two years are required for the life cycle to be completed (Barbour and Fish 1993; Ostfeld 1997).

3.2 Vertebrate communities and Lyme-disease risk

Larval ticks, which hatch from eggs uninfected, can acquire a *B. burgdorferi* infection if they feed on an infected vertebrate host. Those that acquire infection during the larval meal then molt into infected nymphs capable of transmitting the disease agent during the nymphal blood meal. The probability that a larval tick will become infected during its larval blood meal is largely a function of the species of host on which it feeds. The white-footed mouse, *Peromyscus leucopus*, is considered the principal natural reservoir for *B. burgdorferi* infection in North America; recent research shows that 92% of larvae feeding from free-ranging white-footed mice

acquire infection and molt into infected nymphs (LoGiudice *et al*. 2003). A few other competent reservoirs exist, principally eastern chipmunks (*Tamias striatus*) and short-tailed shrews (*Blarina brevicauda*), but most vertebrate species have low capacity to infect feeding ticks.

Most cases of LD are transmitted by nymphs. Nymphs are tiny and therefore hard to detect and they have a potentially high probability of being infected with *B. burgdorferi*. Nymphs are also active in summer, which increases their contact rates with humans. Adult ticks have an even higher probability of being infected, but are much more conspicuous and are most active in mid-to-late autumn when encounters with humans are less likely. Nymphal infection prevalence (NIP), the proportion of host-seeking nymphs infected with the disease agent, is considered an important measure of human risk of exposure to LD (Ostfeld *et al*. 2001; LoGiudice *et al*. 2003), because it reflects the probability that a tick bite will result in a case of LD. In the United States, LD incidence is high in the northeastern and upper midwestern regions. In this "hyperendemic" zone, NIP tends to be above 15%. In contrast, LD incidence is quite low despite abundant populations of blacklegged ticks in southeastern and lower midwestern states, where NIP tends to be <5% (Ostfeld and LoGiudice 2003). Therefore, NIP would appear to be an epidemiologically relevant measure of disease risk at both small and large scales.

Nymphal infection prevalence is determined by the distribution of larval meals among members of the vertebrate community, which differ strongly in reservoir competence—the probability that they will infect ticks that feed on them. A vertebrate community dominated by highly competent reservoirs should produce a nymphal cohort having high NIP, whereas a community with many incompetent reservoirs should produce lower NIP. Natural variation in species composition within the community of hosts from which ticks feed, which is essentially the entire community of ground-dwelling mammals, birds, and lizards, should be accompanied by variation in NIP. As vertebrate species composition changes due to anthropogenic or natural causes, it might be possible to predict the consequent changes in disease risk.

3.3 Modeling community disassembly

Habitat destruction, fragmentation, and conversion are responsible for changing both diversity and species composition of vertebrate communities worldwide (Hilton-Taylor 2000). Frequently, species diversity declines and species composition shifts towards domination by species most resistant to, or even favored by, these habitat changes. Therefore, a strong potential exists for habitat fragmentation to influence risk of human exposure to LD via its impacts on both diversity and species composition within the community of ground-dwelling terrestrial vertebrates. To predict how habitat loss and fragmentation will change LD risk, precise knowledge of three factors is essential: (1) what functional role is played by each potentially important host species in contributing to overall NIP; (2) which species are most likely to be lost from (or severely reduced within) remaining tick habitat as the landscape is fragmented; and (3) how the loss of species changes the abundance or functional role of the remaining species. We address each of these fundamental issues next.

3.3.1 Species-specific contributions to NIP

Nymphal infection prevalence in any given year is equal to the proportion of the prior year's larval cohort that acquired infection, adjusted by any difference in survival (from fed larva to questing nymph) between infected and uninfected ticks. Because no evidence suggests that infection with *B. burgdorferi* reduces survival probability of larval *I. scapularis*, NIP can be considered equivalent to the proportion of larvae acquiring infection in the previous year. Within endemic zones for LD, virtually every terrestrial mammal and ground-dwelling bird species acts as a host for larval *I. scapularis*. These species vary strongly, however, in the fraction of the larval cohort they feed and in the fraction of feeding larvae they infect. To account for species-specific contributions to overall NIP, LoGiudice *et al*. (2003; see also Ostfeld and LoGiudice 2003) constructed a simple model with the following parameters: Density (number ha^{-1}) of host species $i = N_i$; species-specific body burdens (mean larval ticks $host^{-1}$) = B_i; and species-specific reservoir

competence (proportion of larvae acquiring infection) = C_i. Therefore, $m_i = N_i B_i$, where m_i is the number of larval meals taken from species i, and $I_i = m_i C_i$, where I_i is the number of nymphs infected from their larval meal on species i; and the total number of nymphs infected from their larval meal is $I_T = \sum m_i C_i$. The number of nymphs not infected in their larval meal on species i is given by $U_i = m_i \times (1 - C_i)$, and the total number of nymphs not infected is $U_T = \sum m_i (1 - C_i)$. Thus, the total nymphal infection prevalence is $NIP_T = I_T / (I_T + U_T)$.

To validate this model, LoGiudice *et al*. (2003) exhaustively live-captured mammals and birds within a hyperendemic zone for LD in Dutchess County, New York State (US), and directly estimated N_i, B_i, and C_i. For some species, estimates of N_i had to be drawn from published studies conducted in similar oak-mixed hardwood habitats. Because long-term studies of white-footed mice and eastern chipmunks reveal dramatic interannual variation in population density (Ostfeld *et al*. 1996; 2001; Schmidt and Ostfeld 2001), LoGiudice *et al*. (2003) allowed N_i values for these species to vary within the bounds of observed variation. The model provided predicted values of NIP for a fully intact host community with varying densities of mice and chipmunks (Fig. 3.1). Empirical data on NIP as a function of the previous year's rodent density closely matches the model's predictions (Fig. 3.1), which suggests that: (1) important hosts for ticks were not missed in the field sampling; (2) estimates of parameter values for each host species are reasonably accurate; and (3) a combination of high species richness and low mouse density reduces NIP and therefore LD risk.

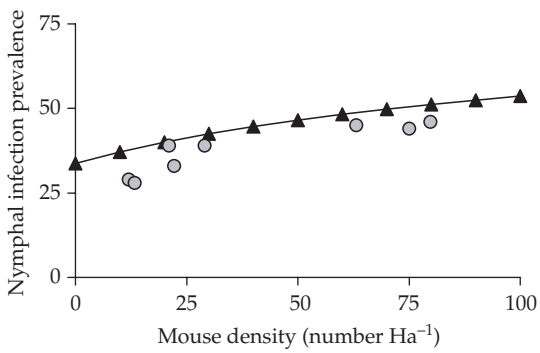

Figure 3.1 Change in LD risk, as measured by nymphal infection prevalence (NIP=percentage of nymphs infected with *B. burgdorferi*) with changing density of white-footed mice. Dark triangles and line represent model predictions based on a complete host community consisting of ca. 12 species of mammals and 4 species of birds, with each species except mice fixed at their long-term average densities. Less diverse communities show higher values of NIP at all mouse densities (LoGiudice *et al*. 2003). Gray circles represent empirical values of NIP in year t and mouse density in year $t-1$ averaged over three 2.25-ha forest plots on which ticks and small mammals have been monitored for the past 10 years (updated from LoGiudice *et al*. 2003). The good fit between predicted and observed NIP as mouse density varies suggests relatively complete accounting of host species, an appropriate model, and accurate parameter values (empirically determined) of host abundance, tick burdens, and reservoir competence.

3.3.2 Species loss with habitat fragmentation

Habitat fragmentation and destruction are considered the primary causes of declines in abundance and local extirpation of many species (e.g. Hilton-Taylor 2000). However, some species appear relatively insensitive to habitat fragmentation and others clearly benefit. Intrinsic traits of species that influence their sensitivity to habitat fragmentation are known in only a few cases (e.g. Kirkland and Ostfeld 1999; Davies *et al*. 2000; Pereira *et al*. 2004), and the importance of these traits appears to vary taxonomically and geographically. At present, for mammals and birds in the northeastern United States (within the LD hyperendemic zone), little is known concerning either which species are most vulnerable to habitat fragmentation or which intrinsic traits most strongly influence vulnerability.

In contrast, intensive study of the mammalian fauna in agricultural landscapes of the midwestern United States, suggests that: (1) species richness declines log-linearly with decreasing forest fragment size (i.e. there is a strong species–area relationship; Rosenblatt *et al*. 1999); (2) carnivorous and some granivorous species occur only in relatively large fragments (Rosenblatt *et al.* 1999); and (3) white-footed mice occur in fragments of all sizes, but their density is highest in the smallest forest patches (Nupp and Swihart 1996, 2000; Krohne and Hoch 1998).

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Whether responses to habitat fragmentation are idiosyncratic or predictable on the basis of

species-specific traits is unknown. Assessing the importance of species-specific traits, such as body size, trophic level, degree of trophic, or habitat specialization, dispersal ability, intrinsic rate of increase, variability in population size, etc., is fraught with difficulties. A key obstacle is the covariation in many of these traits within and among species (Davies *et al*. 2000). For instance, large-bodied animals typically have low intrinsic rates of increase and need extensive habitat areas (which would increase their vulnerability to extirpation with habitat fragmentation), but also tend to be highly mobile and have relatively constant population densities (which would decrease vulnerability to fragmentation-caused extirpation). Consequently, no strong empirical basis exists for posing "disassembly rules" (Ostfeld and LoGiudice 2003) for the mammalian and avian faunas in fragmented forest landscapes of the northeastern United States.

Our strategy, therefore, is to pose a plausible set of disassembly rules, and ask whether different rules result in different predictions concerning the way that LD risk changes with changing species richness and composition (Ostfeld and LoGiudice 2003). Our rules allow us to construct virtual communities with different richness levels and species composition, and our model (see Section 3.3.1) allows us to predict the value of NIP that arises from each community. We next describe a set of plausible rules for community disassembly and assess their consequences for NIP by applying each rule to determine the sequence of species lost from a fully intact community.

Nihilism

For the nihilism "rule" (actually, an anti-rule), species respond entirely idiosyncratically to habitat fragmentation, such that no intrinsic trait is a good predictor. This rule is simulated by selecting species for removal entirely at random. Note that this is the rule often used for assessing the impact of species diversity on ecosystem functions in experimental field or microcosm approaches (Loreau *et al*. 2001; Tilman *et al*. 1997).

Quasi-nihilism

For this rule, we rely on strong empirical evidence that white-footed mice do not decline or disappear from small forest fragments (reviewed above). We assume that mice are present in all communities, but randomly select all other species for removal.

Body size/home-range size

Here we assume that larger-bodied (higher mass) species are most sensitive to habitat fragmentation, owing to the scaling of habitat size to body size. Because home-range size scales with body size for the mammals of northeastern United States (Ostfeld and LoGiudice 2003), rules involving body size and home range are equivalent. For this and the remaining disassembly rules, we assume that white-footed mice and white-tailed deer are necessary for the tick life cycle to be completed, and therefore are present in all communities.

Trophic level

For this rule, we assume that the most carnivorous species are most sensitive to habitat fragmentation, owing to their need for greater habitat area to meet dietary requirements (Pimm and Lawton 1977; Holt 1996). Herbivores and omnivores are expected to be least sensitive, and insectivores of intermediate sensitivity.

Midwestern United States

For this "rule" we simply apply empirical observations by Rosenblatt *et al.* (1999) and Nupp and Swihart (2000) regarding the sequence of mammalian species loss with decreasing habitat area in the midwestern United States. A few species found at our sites are not included in the communities studied in the Midwest; for those species, we apply the trophic level rule.

The consequences of applying different disassembly rules to changes in NIP are dramatic (Figs 3.2 and 3.3). Under the Nihilism "rule," declining species richness results in declining NIP because the probability of mice being included in the community is reduced as richness becomes lower. In contrast, including mice in all communities under the Quasi-nihilism "rule" causes NIP to soar with declining richness as larger and larger proportions of the larval cohort feed on mice (Fig. 3.2). NIP is constrained between ca. 40% at

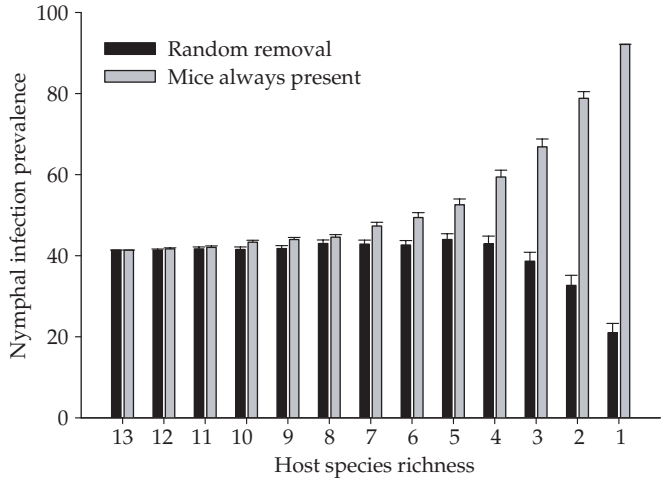

Figure 3.2 Results of a simulation model in which nymphal infection prevalence (defined in Fig. 3.1) changes as species richness in the host community varies. Two community disassembly rules are represented: the nihilism "rule," in which species are removed in random sequence; and the quasi-nihilism rule, in which mice are assumed to be present in all communities and all other species are removed in random sequence. Bars represent means with standard errors from 100 simulations at each level of species richness. See Ostfeld and LoGiudice (2003) for more details. Redrawn and updated from Ostfeld and LoGiudice (2003).

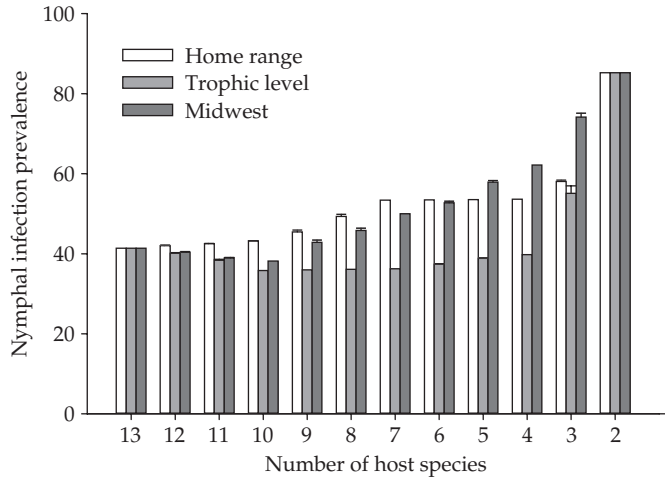

Figure 3.3 Results of a simulation model in which nymphal infection prevalence (defined in Fig. 3.1) changes under three nonrandom disassembly rules: the body size/home-range size rule, in which species with larger bodies/home ranges are lost first; the trophic level rule, in which the most carnivorous species are lost first; and the Midwest "rule," in which species are lost in roughly the order observed in fragmented landscapes in midwestern United States (see text).

maximal species richness and ca. 85% when only white-footed mice and white-tailed deer remain (Fig. 3.3). Nevertheless, at intermediate levels of species richness, different disassembly rules create different patterns of change in NIP. For example, under the home-range rule, species loss from 13 to 7 elicits a gradual increase in NIP, followed by a plateau until the final two species are lost at which point NIP soars (Fig. 3.3). The Midwest "rule" gives rise to a more gradual increase in NIP throughout the entire range of species loss. In marked contrast, the trophic level rule results in a *decrease* in NIP from high to intermediate levels of species richness. Only when the final two species are lost does NIP skyrocket to the maximal level.

Differences among the disassembly rules in the relationship between species richness and NIP are affected both by the species lost and by the composition of the remaining community. In other words, whether the loss of a particular species results in an increase or decrease in NIP, and the magnitude of that effect, depends on the aggregate effects of the remaining species on NIP. To exemplify this concept, first consider a high-diversity community that consequently has a low NIP. If shrews (*Sorex* and *Blarina* spp.) are lost from this community, the result will be a decrease in NIP, because shrews have a moderately high reservoir competence and feed a large number of larval ticks (Fig. 3.4). This early loss of shrews under the

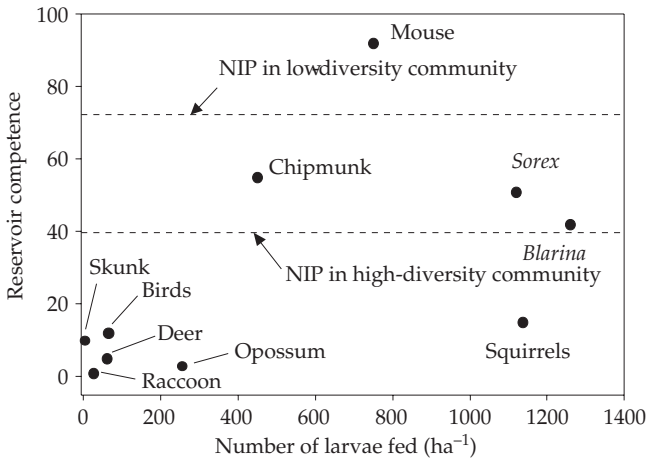

Figure 3.4 Position of host species in phase space, defined as the combination of realized reservoir competence (measured proportion of larvae that acquire infection through feeding from a particular host species; Schauber and Ostfeld 2002) and number of larvae fed by that host species per hectare. The "Reservoir competence" axis can be viewed as a measure of the direction of species-specific effects on total NIP, and the "number of larvae fed" axis can be viewed to represent the magnitude of species-specific effects on total NIP. In a fully intact community at our field sites, NIP values range between 35% and 40% (lower dashed line), whereas in species-poor communities, NIP is much higher (upper dashed line, see Allan *et al*. 2003). In a species-poor community, with high NIP, loss of species such as *Sorex* and *Blarina* shrews and chipmunks would have the effect of increasing NIP even further. But in a species-rich community, with lower NIP, loss of these same species would reduce NIP even further. This situation illustrates the contingent nature of the net effects of species loss on this particular ecosystem function.

trophic level rule appears responsible for the general reduction in NIP with species loss at high and intermediate levels of species richness (Fig. 3.3). However, if shrews are lost from a low-diversity community that has a high NIP, the effect is to strongly elevate NIP even further. This effect of shrews is responsible for the patterns arising from the home-range and Midwest rules.

Host species can be arrayed in a phase diagram with the *x*-axis representing the total number of larvae fed by that species, and the *y*-axis representing reservoir competence (Fig. 3.4). Whether NIP increases or decreases following loss of species *X* depends on whether the reservoir competence of species *X* is below or above the NIP value for the current community (Fig. 3.4). This example illustrates a concept increasingly recognized by community ecologists—that species-specific functional roles can depend on the composition of the remaining community. It also reinforces the importance of knowing the sequence with which species are lost as habitat is degraded, in order to predict the consequences for ecosystem functioning (Ostfeld and LoGiudice 2003).

3.3.3 Impact of species loss on abundance or functional role of remaining species

So far, we have assumed that all host species interact only with ticks directly and not with one another, an assumption that is quite likely to be false. Many of the species in these host communities are likely to interact directly via predator–prey or competitive relationships. Moreover, indirect effects of each species on other members of the community could occur via trophic webs or "competition" for ticks.

Recall that species-specific effects on NIP are determined by population density, average tick burden per individual, and reservoir competence. Despite the convenient assumption that each of these parameters can be treated as a species-specific constant, we have every reason to expect that each parameter is influenced by the composition of the remaining community. Unfortunately, for lack of data, we cannot incorporate the net effect of each species on parameter values for all other species into our models. Nevertheless, it seems useful to describe qualitatively how the population density, mean tick burdens, and reservoir competence of a

focal species are likely to be influenced by the presence of other species in the community.

Population density

Community ecologists have long recognized that species influence one another's abundance both through direct effects (predator–prey, competitive, and mutualistic interactions) and indirect effects mediated by third parties (reviewed by Pimm 1991). For instance, the loss of a carnivorous or omnivorous mammalian species (e.g. skunk, raccoon) that preys on omnivorous/granivorous rodents (e.g. mice, chipmunks, squirrels) could result in enhanced population growth of all rodent species. Or, if the carnivore preferentially fed on one of the species (e.g. mice), then the loss of the carnivore species may elicit disproportionate increases in population growth of mice, which may then suppress populations of the sciurid rodents with which they compete. The observed correlation between carnivore loss and increased abundance of white-footed mice in fragmented landscapes in Indiana and Illinois (Nupp and Swihart 1996, 2000; Krohne and Hoch 1998; Rosenblatt *et al*. 1999) suggests a direct trophic interaction, but the evidence is weak. To our knowledge, the existence of indirect interactions, such as apparent competition, between mice and sciurid rodents in these or similar landscapes has not been assessed. It might be necessary to construct interaction webs in which all pairwise interactions are quantified in order to predict net effect of species loss on abundance of the remaining species.

Average tick burdens

Ticks are weakly mobile and move at most a few meters by crawling, relying on vertebrate hosts for longer-distance movements. As such, their ability to actively pursue and select hosts is quite limited, and their host-finding strategy is largely sit-and-wait. The active host-seeking season for each tick stadium (larval, nymph, or adult stage) extends for only a few months (Fish 1993; Ostfeld *et al.* 1996a). The number of available, host-seeking ticks should be "depleted" rapidly when encounter rates with hosts are high, but might decline slowly when encounter rates are low. It follows that average tick burdens on any given species of host should be reduced when the total abundance of hosts (of all species) is high, and increased when total host abundance is low.

Nevertheless, effects of the abundance of alternate hosts (or all hosts) on average tick burdens on focal hosts have been neglected until recently. A study of impacts of alternate host abundance on focal-host tick burdens was undertaken by Schmidt *et al*. (1999), who assessed how varying density of eastern chipmunks influenced average tick burdens on white-footed mice. When chipmunk density was low, larval tick burdens per mouse were variable and often extremely high, whereas when chipmunk density was high, larval burdens on mice were lower and less variable (Fig. 3.5). This relationship supports the contention that host-seeking ticks can be depleted by increasing availability of hosts, and that this can reduce tick burdens on individual host species. The data in Fig. 3.5 also support the assertion that average larval burdens on host species might be determined as much by host community composition as by species-specific characteristics.

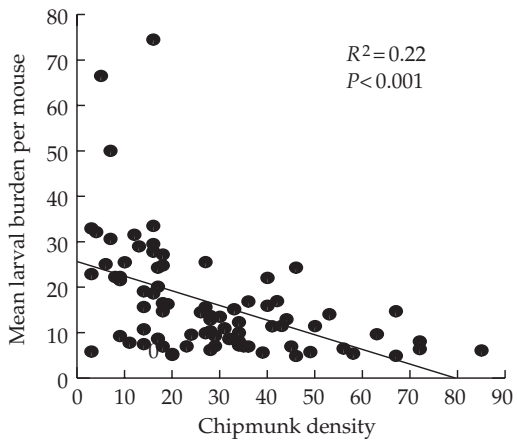

Figure 3.5 Effects of variation in the population density of chipmunks (number per 2.25-ha plot) and average larval tick burdens on the white-footed mice on that plot. Each data point represents a different plot-year covering our sites of long-term monitoring of rodents and ticks at the Institute for Ecosystem Studies in Millbrook, New York. The relationship demonstrates the strong potential for changes in the abundance of one host (in this case, chipmunks) to affect encounter rates between ticks and a focal host (white-footed mice).

Reservoir competence

Reservoir competence for any vector-borne pathogen is typically measured by determining the probability that an infected host will transmit an infection to a feeding vector. Generally, reservoir competence is high when the immune response by the host to the pathogen is insufficient to prevent high concentrations of pathogens in blood or other tissues, resulting in the acquisition of pathogens by the vector during its blood meal. For many host species, however, reservoir competence initially increases following the infection event (during the pathogen dissemination phase) and then declines as the immune system removes pathogens. Schauber and Ostfeld (2002) termed the latter "reservoir competence decay." In the case of LD, and probably for many other zoonoses, hosts become repeatedly inoculated during vector meals, and each new inoculation results in a temporary increase in reservoir competence as pathogen levels circulating in the host increase (reviewed in Schauber and Ostfeld 2002). Therefore, true reservoir competence values for a given host change dynamically with the rate of inoculation, and published values for particular host species almost certainly represent averages that hide these dynamics.

Schauber and Ostfeld (2002) found that, even if the shape of reservoir competence decay is a species-specific trait, the "realized" reservoir competence—which is the probability that a blood meal taken by a vector from a host under field conditions will result in infection of the vector—depends strongly on the composition of the host community. If the community is dominated by highly competent reservoirs (e.g. mice), then the infection prevalence in the tick population should be high. As a result, the inoculation rate of all hosts should be high, and hosts' realized reservoir competence should remain near the maximum of the competence decay curve. On the other hand, if the host community is dominated by incompetent reservoirs, tick infection prevalence should be lower, resulting in lower inoculation rates and realized reservoir competence values that are closer to their minimum under decay.

In conclusion, all three key parameters that determine any individual species' contribution to NIP are quite likely to be influenced by the presence and abundance of other species in the host community. Therefore, considering them to be species-specific traits, as we have done in our modeling, probably oversimplifies the LD system considerably. Determining the net effect of changes in species composition on population density, average tick burdens, and reservoir competence of all host species, however, is a major logistical and conceptual challenge. We suggest that a combination of experimental, modeling, and comparative studies will allow investigators to approach this important issue (Box 3.1).

Box 3.1 Approaches for assessing the role of indirect interactions in disease dynamics

Most studies of disease dynamics have focused on a relatively small set of species directly involved in transmission. Most studies of malaria, for example, have emphasized monitoring abundances of reservoir hosts and mosquito vectors, and determining the prevalence of the *Plasmodium* parasite in these animals. Once the appropriate values are obtained from these observations, they are used to model transmission dynamics.

If disease systems are seen as embedded within ecological communities, however, then studying them also requires investigating species and interactions only *indirectly* involved in disease transmission. For example, investigators interested in the abundance of different malaria vectors might consider bottom-up controls of vegetation on mosquito recruitment (for an example, see Chapter 7, this volume). In the context of LD, we might consider potential top-down and bottom-up controls on the abundance of competent hosts. In eastern North America, white-footed mice (*P. leucopus*) were known to be the most competent reservoir for the bacterial agent of LD (*B. burgdorferi*), as early as 1984 (Anderson and Magnarelli 1984; Levine *et al*. 1985). But what regulated mouse populations was not understood until long-term monitoring showed correlations between mouse abundance and pulses of acorn production (mast) by oak trees (Elkinton *et al*. 1996; Ostfeld *et al*. 1996b; Wolff 1996). To determine if acorn production *caused* high mouse abundance, Jones *et al*. (1998) conducted a large-scale field experiment in which they added more than a million acorns to experimental plots. This experimental pulse of acorn production caused significant increases in mouse densities the following year (Jones *et al*. 1998).

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Box 3.1 *continued*

Sometimes, though, field experiments of the dynamics of complex communities are not possible. For example, LoGiudice *et al*. (2003) were interested in the individual roles played by each host species in infecting ticks with the Lyme bacterium. Removing each host species from large plots of forest was infeasible. However, through extensive fieldwork and literature reviews, they estimated the densities of each host species, and then estimated their tick burdens and reservoir competencies directly. (Reservoir competence was measured by bringing field-caught animals into the laboratory and placing them in cages suspended over pans of water. Embedded ticks dropped off after feeding and were collected from pans. Ticks were allowed to molt and were tested for *B. burgdorferi* using a direct immunofluoresence assay. For details, see Ostfeld *et al*. 2001). With these data, they built a simple model that accounted for each species' contribution to the proportion of ticks infected with the Lyme bacterium. They tested the model by comparing the proportion of ticks the model predicted would be infected with the proportion of ticks that actually were infected in their forest plots. With close agreement between these values, Ostfeld and LoGiudice (2003) used the model to simulate the removal of individual host species and the effect of these removals on tick infection.

Laboratory experiments can also be useful for understanding complex ecological interactions that are impractical or impossible to study in the field. Shaw *et al*. (2003) were interested in why field-caught white-footed mice were infested with on average twice as many larval ticks as were eastern chipmunks (*T. striatus)*. In a carefully controlled laboratory experiment, they tested two alternative explanations for this effect. They found that ticks oriented towards white-footed mice twice as often as they oriented towards eastern chipmunks (*T. striatus*), but that mice partially counterbalanced this attraction by being more efficient groomers than chipmunks were.

This careful integration of monitoring, experimentation, and modeling has its limitations for elucidating the epidemiological consequences of species interactions in complex ecological communities. LoGiudice *et al*. (2003) sought to make their model for estimating tick infection more realistic by allowing the removal of a particular host species to affect parameter values for other species. For example, what would be the net effect of removing eastern chipmunks from a forest community if this removal increased tick burdens on white-footed mice, or increased mouse density by reducing competition, or reduced mouse density by increasing predation pressure on mice? Unfortunately, LoGiudice *et al*. (2003) were unable to adapt their model in this way because they found that there were simply no field data to use to parameterize such a model, nor could they develop a realistic field protocol to gather these data themselves. Developing mathematical and experimental or comparative techniques for grappling with complex ecological systems is the greatest challenge for the future of disease ecology; it is also the greatest challenge for other disciplines that investigate the behavior of intricate networks of interactions.

3.4 Discussion

Epidemiology is the scientific discipline devoted to understanding the incidence, distribution, and control of disease in a population. For infectious diseases, the simplest disease system is one with a single species each of pathogen and host, and a highly specialized relationship between the two. Under these conditions, the importance of community ecology to epidemiology might be limited to: (1) the reciprocal roles of the host and pathogen in regulating each other; and (2) the roles played by various other (non-host, non-pathogen) species in controlling abundance of the host species (Keesing *et al*., in preparation, Box 3.2; see also Holt and Dobson, Chapter 2, this volume). However, when the pathogen can infect more than one species of host (the situation for all zoonoses), community ecology becomes even more fundamental, because species composition and interspecific interactions can influence abundance of all host species and contact rates between each host and the pathogen. Finally, vector-borne diseases increase the importance of community ecology even further by adding vector-host and vector-pathogen interactions to the mix.

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We have focused on the LD system because of its epidemiological importance and relevance as a model of other vector-borne zoonoses. The LD system involves several key features that are

Box 3.2 What causes the "dilution effect" in disease systems?

In several recent studies, vertebrate communities composed of a diversity of host species have been found to pose lower LD risk than communities composed of few host species. This phenomenon, termed the "dilution effect" (Ostfeld and Keesing 2000a), may occur in a number of vector-borne disease systems (Ostfeld and Keesing 2000b). Despite the potential generality of this effect, however, there is confusion about the mechanisms underlying its occurrence. Keesing *et al*. (in review) argue that dilution is in fact the net effect of a suite of potential mechanisms, and that these mechanisms may operate in both vector-borne and non-vector-borne disease systems.

The mechanisms through which dilution could occur can be elucidated by examination of a model of a simple disease system. Imagine a system in which a microparasite is specialized on a single host species, and is passed from host to host through direct contact. The dynamics of the host population can be described by a susceptible-infected (SI) model (after Anderson and May 1978):

$$\frac{dS}{dt} = (b-m)S - \alpha\delta SI + (\gamma + b')I \quad (3.1)$$

$$\frac{dI}{dt} = \alpha\delta SI - (\gamma + m')I \quad (3.2)$$

where b and b' are the birth rates of susceptible and infected individuals, respectively, and m and m' are their respective death rates. Susceptible individuals become infected based on a rate of encounter with infected individuals, α, and a probability of transmission given an encounter, δ. Infected individuals recover at a rate γ to become susceptible again.

One measure of disease risk in this system is the rate of change in the density of infected individuals, dI/dt (Equation (3.2)). If the host species is already present in the focal community, the addition of a non-host species could result in declining disease risk if the presence of that species:

- decreased encounter rates between susceptible and infected individuals (α)—"encounter dilution";
- decreased rates of transmission given an encounter (δ)—"transmission dilution";
- decreased the abundance of susceptible individuals (S)—"susceptible host regulation",
- increased the rate of recovery of infected individuals (γ)—"recovery dilution"; or
- increased the rate of mortality of infected individuals (m')—"infected host mortality".

The first of these modes of dilution, encounter dilution, could occur if an added species (e.g. a predator) affected space use of the host in a way that reduced its rate of contact with other hosts. The third mode, susceptible host regulation, could occur if a non-host species added to this system were a predator or competitor of the host. Examples of this type of regulation abound.

Now we consider more complex systems in which multiple host species can be infected with the pathogen. We identify the host species with the highest probability of transmission (δ) as the most competent reservoir (MCR). In this system, disease risk is a function of the total density of infected hosts, including the MCR and other host species. We begin with a focal community initially containing the MCR. Adding a *non-host* species to this community could lead to dilution through any of our original five modes; these would decrease the density of infected MCR individuals. Because of our measure of risk, however, adding a *host* species would only *increase* disease risk, unless its addition resulted in a concomitant reduction in the density of infected MCR individuals through one of our five original modes. With a vector-borne disease system, such as LD, we introduce one additional mode of dilution, vector regulation, which occurs when the addition of a species regulates the abundance of the vector.

In the LD system, many of these modes appear to operate simultaneously. In diverse communities in the northeastern United States, predators and competitors of white-footed mice (*P. leucopus*), the MCR, might regulate its abundance, decreasing disease risk through "susceptible host regulation" and "infected host mortality" (Rosenblatt *et al*. 1999; Nupp and Swihart 1996, 2000; Allan *et al*. 2003). These same species serve as hosts for ticks, and thus may also decrease contact rates between infected ticks and uninfected mice (see Fig. 3.5), a form of "encounter dilution." Immature ticks that attempt to feed on non-mouse hosts tend to be less likely to survive than are ticks that feed on mice (Wilson *et al.* 1990; Craig *et al*. 1996; Ostfeld and Lewis 1999), a type of "vector regulation." And "transmission dilution" occurs when the presence of alternate species increases the average reservoir competence decay (*sensu* Schauber and Ostfeld 2002; see main text), resulting in a lower average rate of transmission.

Whether the dilution effect occurs in a given disease system is determined by the net effect of this suite of mechanisms operating simultaneously. Of course, these modes can in principle operate in reverse, such that adding species to a disease system could increase disease risk through an "amplification effect." Only further studies of the community ecology of a diversity of disease systems will reveal the frequency of these patterns.

characteristic of many other vector-borne disease systems: reliance by the pathogen on acquisition by vectors from hosts (rather than purely vertical transmission), a generalist vector that parasitizes many host species, and strong variation among host species in average reservoir competence (Ostfeld and Keesing 2000a). These characteristics suggest that disease risk to humans, which is affected by both the proportion of vectors infected with the pathogen and the abundance of infected vectors, will be influenced by host community composition. In the LD system, the most competent reservoir (white-footed mouse) is also ubiquitous, abundant, and insensitive to human disturbance of natural habitats. Consequently, as host diversity is eroded, the impact of white-footed mice increases and so does risk of human exposure to the pathogen. Conversely, high diversity in the vertebrate host community will provide a strong "dilution effect" (Ostfeld and Keesing 2000a), which protects humans from disease risk.

However, prior empirical and modeling approaches to studying the effect of species diversity and composition on LD risk have largely been limited to determining the individual roles played by each species in feeding and infecting ticks, and then assessing how species presence or absence affects tick infection prevalence or abundance. We have argued that such an approach provides a good beginning, but is limited by the likely possibility that species-specific effects are not fixed, but rather are strongly influenced by the composition of the remaining host community. Our empirically based simulation models suggest that knowing the relative sensitivities of various host species to habitat loss and degradation can be important in predicting consequences for human health. But the results also suggest that basic knowledge of the community ecology of familiar host species is insufficient for making quantitative predictions. The next key step will be to determine how the presence/absence of species influences the abundance, tick burden, and reservoir competence of the remaining species.

Zoonotic disease systems involve a network of interacting species directly involved in disease transmission, and understanding the complexity of interactions among these species is crucial for understanding disease risk. But disease systems also involve a larger network of species that play *indirect* but potentially critical roles in determining disease risk (see Box 3.1). In the LD system in eastern North America, oak trees (*Quercus* species) determine much of the interannual variation in LD risk, despite the fact that they are not directly involved as hosts for either the pathogen or the vector of this disease. Instead, because they produce occasional hyperabundant crops of nutritious acorns (mast), oaks indirectly affect LD risk by influencing the abundance of key hosts for the pathogen, including the white-footed mouse (Ostfeld *et al*. 1996b; Jones *et al*. 1998). Clearly, the concepts, approaches, and tools of community ecology are fundamental to the epidemiology of zoonotic disease.

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