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Quantifying dilution and amplification in a community of hosts for tick-borne pathogens

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Abstract. Recent controversy over whether biodiversity reduces disease risk (dilution effect) has focused on the ecology of Lyme disease, a tick-borne zoonosis. A criticism of the dilution effect is that increasing host species richness might amplify disease risk, assuming that total host abundance, and therefore feeding opportunities for ticks, increase with species richness. In contrast, a dilution effect is expected when poor quality hosts for ticks and pathogens (dilution hosts) divert tick blood meals away from competent hosts. Even if host densities are additive, the relationship between host density and tick encounters can be nonlinear if the number of ticks that encounter a host is a saturating function of host density, which occurs if ticks aggregate on the remaining hosts rather than failing to find a host before death. Both dilution and amplification are theoretical possibilities, and assessing which is more prevalent required detailed analyses of empirical systems. We used field data to explore the degree of tick redistribution onto fewer individuals with variation in intraspecific host density and novel data-driven models for tick dynamics to determine how changes in vertebrate community composition influence the density of nymphs infected with the Lyme bacterium. To be conservative, we allowed total host density to increase additively with species richness. Our long-term field studies found that larval and nymphal ticks redistribute onto fewer individuals as host densities decline, that a large proportion of nymphs and adults find hosts, and that mice and chipmunks feed a large proportion of nymphs. White-footed mice, eastern chipmunks, short-tailed shrews, and masked shrews were important amplification hosts that greatly increased the density of infected nymphs. Gray squirrels and Virginia opossums were important dilution hosts. Removing these two species increased the maximum number of larvae attached to amplification hosts by 57%. Raccoons and birds were minor dilution hosts under some conditions. Even under the assumption of additive community assembly, some species are likely to reduce the density of infected nymphs as diversity increases. If the assumption of additivity is relaxed, then species that reduce the density of small mammals through predation or competition might substantially reduce disease risk.

Key words: biodiversity; community ecology; ecosystem services; Ixodes scapularis*; Lyme disease.*

Introduction

In the past few years, vigorous debate among disease ecologists and conservation scientists has explored how frequently and under what conditions the loss of biodiversity increases the risk of contracting zoonotic diseases (Keesing et al. 2010, Ostfeld and Keesing 2012, 2013, Randolph and Dobson 2012, Wood and Lafferty 2012, Ostfeld 2013). The argument that biodiversity loss potentially increases disease risk begins with the observation that many multi-host pathogens are harbored by abundant and widespread species with fast life histories that are present in both species-rich and species-poor communities. West Nile virus, for example, can be transmitted by many species of birds, but among its most competent hosts in North America are American Robins, Blue Jays, and Common Grackles (Kilpatrick et al. 2006), which are abundant in both species-rich and species-poor habitats (Allan et al. 2009). Rodents provide another example, because many species are abundant, resilient to anthropogenic impacts on the environment, and function as particularly efficient hosts for zoonotic pathogens, including those that cause Lyme disease, hantavirus diseases, plague, leishmaniasis, various hemorrhagic fevers, human anaplasmosis, and babesiosis (Daszak et al. 2000, Ostfeld and Holt 2004). The underlying causes of the observed relationship between host persistence across gradients in anthropogenic disturbance and host quality as producers of pathogens are not yet clear, but recent research suggests that one mechanism may be that ecologically resilient species with fast life histories may invest relatively less in adaptive immunity, compared to species that are more

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sensitive to biodiversity loss, for instance because of long generation times (Previtali et al. 2012, Ostfeld et al. 2014). An alternative mechanism could be that pathogens have adapted to specialize on abundant hosts as a form of evolutionary host habitat selection. Regardless of the underlying mechanism, the ecological consequences are clear. When amplification hosts (i.e., hosts that infect many vectors) become abundant in ecological communities, pathogen transmission is likely to rise. In contrast, communities with higher levels of biodiversity might include hosts that reduce disease risk by transmitting pathogens to vectors at lower rates, by reducing vector survival, or by reducing the abundance of amplification hosts. This decrease in disease risk with addition of less efficient hosts is sometimes called the dilution effect and these hosts are called dilution hosts.

Assessing the relative magnitudes of dilution and amplification of hosts within a community requires considerable data on host abundance and quality, as well as the development and analysis of mathematical models. These models must capture key aspects of host–vector–pathogen dynamics and be grounded in empirical data. Our principal goal in this paper is to develop such a model, focusing on the within-season dynamics of the agent of Lyme disease circulating in vertebrate populations in the northeastern USA.

Lyme disease is by far the most common vector-borne disease of humans in the USA, and both its annual incidence and geographic range are still increasing (Bacon et al. 2008). Lyme disease is caused by the bacterium *Borrelia burgdorferi*, which is passed from host to host through the bite of *Ixodes* tick vectors. In the U.S. northeast and upper midwest, where over 96% of U.S. Lyme disease cases occur, the primary Lyme disease vector is the blacklegged tick (*Ixodes scapularis*), which feeds just once during each life stage (larva, nymph, and adult). Larval blacklegged ticks are extreme host generalists and will feed from any of dozens of vertebrate taxa including birds, reptiles, and mammals (Fig. 1A). The infected nymphs that arise from those larval blood meals are responsible for almost all human cases of Lyme disease (Barbour and Fish 1993). Although *I. scapularis* ticks can acquire *B. burgdorferi* from many vertebrate hosts, these host species vary greatly in the probability that they will transmit the pathogen to a feeding tick attached to a given host. This probability, which is the product of the probability that the host is infected and the probability that it transmits the pathogen to a feeding tick, is called the realized reservoir competence (Table 1). The most competent host species for transmitting *B. burgdorferi* are common and widespread small mammals: white-footed mice (*Peromyscus leucopus*), eastern chipmunks (*Tamias striatus*), short-tailed shrews (*Blarina brevicauda*), and masked shrews (*Sorex cinereus*; Table 1; LoGiudice et al. 2003).

Vertebrate hosts differ not only in their ability to transmit pathogens, but also in the probability that larval ticks successfully feed having encountered each host (i.e., their permissiveness). For example, almost 50% of larval ticks that attempt to feed from white-footed mice are successful

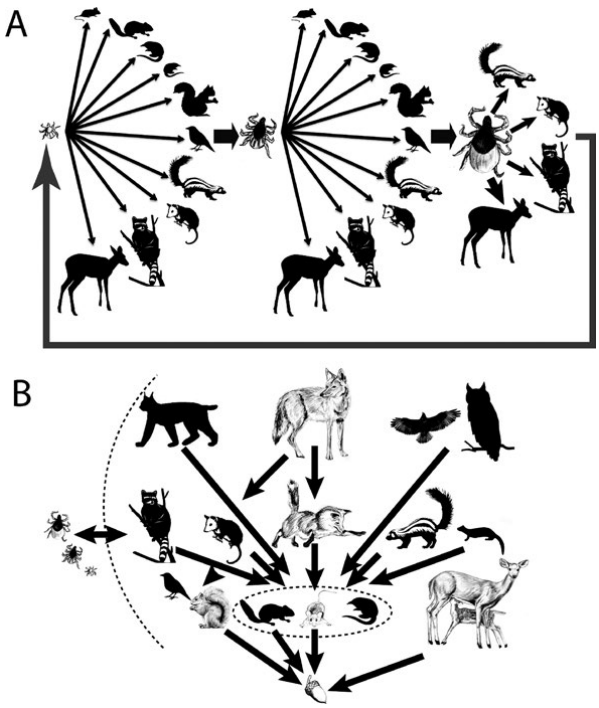

FIG. 1. A complete population model for the community ecology of Lyme disease would (A) include all tick–host interactions in the larval, nymph, and adult life stages and then feed back to influence larval abundance, and (B) incorporate predatory and competitive interactions within the vertebrate host community. Such a model would likely be intractable, but empirical data on the interactions between larval ticks and hosts allow us to parameterize a detailed multi-host model of the critical larvae–nymph transition, which determines the density of infected nymphs from a given larval cohort. We conservatively assume that host densities are additive (no interaction among hosts), although less competent, larger bodied hosts often prey upon and compete with pathogen-amplifying small mammals, which could reduce the probability that a larval tick successfully transitions into an infected nymph.

in feeding to repletion, a level of success significantly higher than any other host tested (Keesing et al. 2009, Table 1). In marked contrast, only 3.5% of larval ticks attempting to feed on a Virginia opossum (*Didelphis virginianus*) feed to repletion, with the remaining 96.5% being killed by the host during grooming (Keesing et al. 2009, Table 1). Because mice, and to a lesser extent chipmunks, are both competent and permissive hosts, as well as being widespread and typically abundant, their population dynamics are thought to be important factors influencing tick-borne disease risk. Shrews are also competent reservoirs that host large numbers of larvae (Table 1), but their permissiveness has not been tested.

A criticism of the dilution effect in this system is that although increasing host diversity may reduce tick infection prevalence, adding host species could increase tick abundance (vector amplification) simply by providing more feeding opportunities to ticks (Randolph and Dobson 2012, Wood and Lafferty 2012). This argument rests on an implicit assumption of additivity: each species has its own density that is set by factors other than the

TABLE 1. Data used to parameterize the model and estimates of the proportion of fed larvae produced by each species.

Species	Scientific name	Individuals per hectare (D_i)	Post-grooming body burden (B_i)	Feeding survival (S_i)	*B. burgdorferi* reservoir competence (R_i)
White-footed mouse	*Peromyscus leucopus*	40	27.8	0.49	0.921
Eastern chipmunk	*Tamias striatus*	20	36	0.24	0.550
Masked shrew	*Sorex cinereus*	25	55.5	**0.49, 0.24**	0.512
Short-tailed shrew	*Blarina brevicauda*	25	62.9	**0.49, 0.24**	0.418
Eastern gray squirrel	*Sciurus carolinensis*	8.1	142	0.17	0.147
Ground foraging birds	Various	31.6	**1.7, 11.4**	0.27	0.117
Striped skunk	*Mephitis mephitis*	0.05	66.8	**0.24, 0.04**	0.097
Raccoon	*Procyon lotor*	0.2	127	**0.24, 0.04**	0.013
Virginia opossum	*Didelphis virginiana*	**0.2, 1**	254	0.035	0.026
White-tailed deer	*Odocoileus virginianus*	0.25	239	0.49	0.046

Notes: Parameters for body burden taken from LoGiudice et al. (2003), but we additionally ran the model with the higher body burden found on birds in Keesing et al. (2009). Feeding survival parameters were taken from Keesing et al. (2009) where available. Where these data were unavailable (cells with numbers in bold), we used a range of parameter values from other species. We assumed that larval survival on shrews would be bracketed by the higher survival on mice and the lower survival on chipmunks. We assumed that larval survival on raccoons and skunks would be similar to survival on chipmunks, but we also ran the model with the exceptionally low survival found on opossums. We assumed that deer would be poor groomers with high survival similar to mice. Density estimates are taken from LoGiudice et al. (2003) and Keesing et al. (2009) with the exception of opossum where we use their value of 1/ha and also use a lower population density estimate. Reservoir competence for *B. burgdorferi* was taken from LoGiudice et al. (2003). In the manuscript, our base parameters refer to the first value the cells with two values and alternative parameterizations refer to the second value.

densities of the other co-occuring species. In other words, more species-rich communities by necessity will contain more total host individuals. When assuming additivity, additional species could lead to more tick feeding opportunities, which would increase tick abundance if those ticks would otherwise have failed to find a host. However, there is no a priori reason to assume that total host density and/or biomass increase additively with increasing species richness because alternative hosts may actually be predators or competitors of reservoir hosts. Depending on the strength of predatory and competitive interactions, increased diversity can lead to roughly constant community density or even far lower community density if additional hosts in a community reduce the abundance of particular focal hosts (Fig. 1B). For example, disappearance of top predators (Estes et al. 2011, Ripple et al. 2013), a loss of diversity, releases their prey from top-down regulation, leading to greatly elevated total host abundance in predator-free communities. Similarly, the loss of competitors can result in ecological release with consequences for disease. For example, the loss of large herbivores in an African savanna permitted surges in the abundance of small-bodied rodents, which in turn sustain pathogen-carrying fleas at higher abundance (McCauley et al. 2008, Young et al. 2014), and overhunting of granivorous white-lipped peccaries (*Tayassu pecari*) resulted in elevated abundance of the rodent hosts for hantavirus (Galetti et al. 2015). Within the hotspots of Lyme disease, the restoration of top predators to northeastern forests would almost certainly very substantially reduce the densities of deer, which is clearly a violation of the assumption of additive community density. Additivity is also likely violated among small mammals in many systems due to strong interspecific competition, with competitive exclusion as a limiting case (reviewed in Grant 1972, Kelt et al. 1995, Nupp and Swihart 2001). However, experimental research in our system was unable to successfully manipulate rodent densities because translocated individuals rarely remained at sites where they were introduced, and removals resulted in excess immigration that counterbalanced density reductions (Brunner et al. 2013).

Even in a hypothetical situation when host densities are additive as species diversity changes, the distribution of ticks may change in important ways. For instance, ticks may aggregate on the remaining hosts as host density or diversity declines. We call this shift in host use by ticks when some hosts are scarce or missing "redistribution" (Keesing et al. 2009). If there is no redistribution and host densities are additive, then all hosts are amplification hosts because ticks that encounter them would otherwise simply have failed to feed. Some critiques of the dilution effect have merely assumed this de facto (Randolph and Dobson 2012, Wood and Lafferty 2012).

By contrast, if there is complete redistribution, or if only the infection prevalence rather than density of nymphs is considered, then removing incompetent hosts causes larvae to instead feed on competent small mammals, and in this case, the incompetent hosts would be deemed dilution hosts (LoGiudice et al. 2003, Keesing et al. 2009). Whether one assumes that ticks completely redistribute, or do not redistribute at all, leads to opposite conclusions about whether there is a dilution or amplification effect (if species densities are additive). The degree of redistribution is thus crucial to gauging whether a given host species is a dilution or amplification host.

To analyze this question in the context of a concrete empirical system, here we first analyze 19 years of field data on both questing and attached larva and nymph populations. To supplement our long-term data on immature ticks, we also analyze data on adult feeding success from two classic papers in Lyme disease ecology (Deblinger et al. 1993, Ginsberg and Zhioua 1999), to address the relationship between host density and the number of ticks attached to hosts. Specifically, we determine whether the number of ticks fed by hosts increases linearly with host density or saturates at low, moderate, or high host densities. We then expand on current models by explicitly considering both vector and pathogen amplification using a data-driven model of the larva–nymph transition. We were able to include many of the complexities associated with host–vector–pathogen interactions in the larva-to-nymph transition because our system is so data-rich with respect to these interactions. However, the nymph-to-adult and adult-to-larva transitions are admittedly data-poor (as is arguably true for most tick population studies in the literature). We concentrate our efforts on the within-year seasonal dynamics of the larva-to-nymph transition, which we believe is critical to Lyme disease ecology because (1) this transition produces the nymphs that are responsible for infecting people, and (2) tick populations appear most limited by hosts for larvae (discussed below, Figs. 2-3). To build upon previous work (e.g., Keesing et al. 2009), we incorporate the process of questing, attachment, and feeding by larval ticks as dynamic outcomes of a model, which allows tick redistribution to occur as hosts are added or removed.

Our model examines in detail the within-season dynamics of the larval tick population exposed to a multi-host prey community, a key dimension of understanding this system that has been neglected by most earlier theoretical studies motivated by this system (e.g., VanBuskirk and Ostfeld 1995, Porco 1999, Schauber and Ostfeld 2002, Levi et al. 2012). We parameterize the model with empirical data on the quality of vertebrate hosts, using a plausible range of parameter values for missing data, to evaluate the disease amplification and dilution potential of individual host species under the assumption that host densities are additive. Our approach in this paper fits with broader currents in epidemiological modeling, moving beyond single host-pathogen systems (reviewed by, e.g., Lloyd-Smith et al. 2009), first to theoretical or conceptual models of dilution and amplification in multi-host systems (e.g., Dobson 2004, Keesing et al. 2006, Joseph et al. 2013), and then to the complexities of an empirical, multispecies community.

Field data

We used 19 years of data from a tick and small-mammal trapping program at the Cary Institute of Ecosystem Studies in Dutchess County, southeastern New York, USA (Ostfeld et al. 2006, and *unpublished data*) to assess the level of empirical support for larval redistribution as host abundance varies (full methods in the Appendix S1). Rather than remove an entire host species (which is experimentally difficult), we used natural variation in mouse and chipmunk abundance to determine whether a decline in rodent density leads to (1) increased tick loads (i.e., ticks concentrate on fewer individuals) and (2) increased densities of questing ticks (i.e., ticks fail to find hosts at low rodent densities but are removed from the pool of questing ticks as rodent densities increase). Because mice are much more abundant than chipmunks, and are highly correlated with chipmunks (Pearson's test $P < 0.0001$, $r = 0.52$), mouse density is nearly perfectly correlated with total rodent (mouse + chipmunk) density ($r = 0.98$, $P < 10^{-16}$; Appendix S1: Fig. S1). Thus inferences based on mouse density cannot be distinguished from those based on total rodent density.

We quantified the degree of larval and nymphal redistribution onto fewer hosts using the relationship between mouse density and the average number of larvae or nymphs per mouse during the trapping session coinciding with the larval and nymphal peaks. Redistribution would be evident by each mouse hosting more larvae or nymphs at lower mouse densities, or, equivalently, by the total number of larvae or nymphs feeding on mice increasing more slowly as mouse densities increase. We used maximum-likelihood to fit the saturating (Michaelis-Menten) function, $y = \alpha x/(\beta + x)$, where x is the density of mice, and y is the total number of larvae or nymphs counted on mice per hectare (i.e., the product of mouse density and larvae or nymphs per mouse). The parameter α determines the saturation point and β is the half-saturation parameter. We also present results of larvae or nymphs per mouse vs. mouse density using the per-capita formulation $y = \alpha/(\beta + x)$, where α and β are identical to above. In addition, this equation is used to relate peak questing (host-seeking ticks/100 m^2) larval or nymphal density (y) to mouse density (x) to determine if abundant mouse populations can potentially reduce the density of questing ticks by encountering them and removing them from the pool of host-seeking ticks. Noting that white-tailed deer are an important host for adult blacklegged ticks, we also utilized data from two classic papers in Lyme disease ecology (Deblinger et al. 1993, Ginsberg and Zhioua 1999) to address the degree to which adult ticks redistribute onto remaining hosts (deer) when host density declines.

Data results

Larvae counted per mouse declined nonlinearly as mouse density increased ($R^2 = 0.56$), indicating that larvae can potentially concentrate on fewer hosts as host density declines (Fig. 2A). Total number of larvae feeding on mice per hectare saturated at $a = 3914$ larvae/ha, with half saturation parameter $b = 94.4$ mice/ha (Fig. 2B; Michaelis-Menten functional form $y = ax/(b + x)$). The peak density of questing larvae per hectare was unrelated to mouse density (Fig. 2C). Note that this density refers to larvae collected on drag cloths, which is much lower than the estimated actual

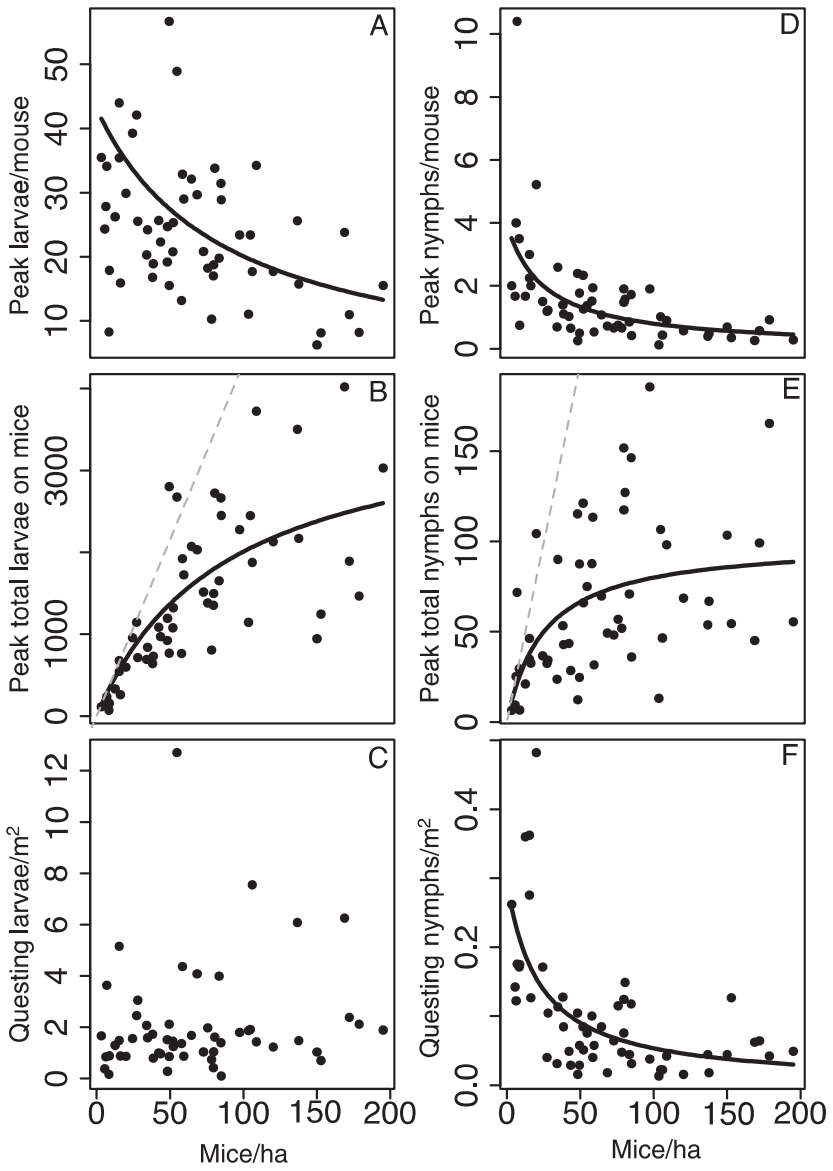

FIG. 2. Quantifying the relationships between hosts and ticks of each life stage. Nonlinear model fits were in all cases supported by at least four AIC units. (A and B) Larvae counted per mouse declined nonlinearly as mouse density increased ($R^2 = 0.56$), indicating that larvae can concentrate on fewer hosts as host density declines. Gray dashed line follows the initial slope, which is the expected relationship if total larvae fed increases additively with mouse density. (C) The peak density of questing larvae per hectare was unrelated to mouse density. (D and E) Nymphs counted per mouse also declined nonlinearly ($R^2 = 0.23$), and the total number of nymphs feeding on mice saturated at moderate mouse densities, indicating that most nymphs find hosts. Gray dashed line follows the initial slope, which is the expected relationship if total nymphs fed increases additively with mouse density. (F) The peak density of questing nymphs declined sharply with increasing mouse density ($R^2 = 0.40$). The density of questing nymphs reached high densities when mice become very rare, providing evidence that mice and chipmunks feed a large proportion of the nymph population, and that nymphs redistribute onto fewer hosts until a low threshold vertebrate density is reached.

density of questing larvae that was used to parameterize the model (drag cloth efficiency for larval sampling is estimated to be less than 10%; Daniels et al. 2000). Nymphs counted per mouse also declined nonlinearly (Fig. 2D; $R^2 = 0.23$), and the total number of nymphs feeding on mice saturated at $a = 104$ nymphs/ha, with half saturation parameter $b = 28.3$ mice/ha (Fig. 2E). The total number of nymphs on mice saturated at much lower mouse densities than did the number larvae on mice, allowing it to be closer to complete saturation at the highest mouse densities. In contrast to questing larvae, the peak density of questing nymphs did decline nonlinearly with increasing mouse density (Fig. 2F; $R^2 = 0.40$) and reached high densities when mice became very rare. The negative relationship between mouse density and the density of questing nymphs suggests that a high proportion of nymphs find a host and that mice and chipmunks together feed a large proportion of nymphs. One could view the patterns of Fig. 2A, D, and F as a kind of intraspecific dilution effect; the more rodents, the lower the tick burden per rodent.

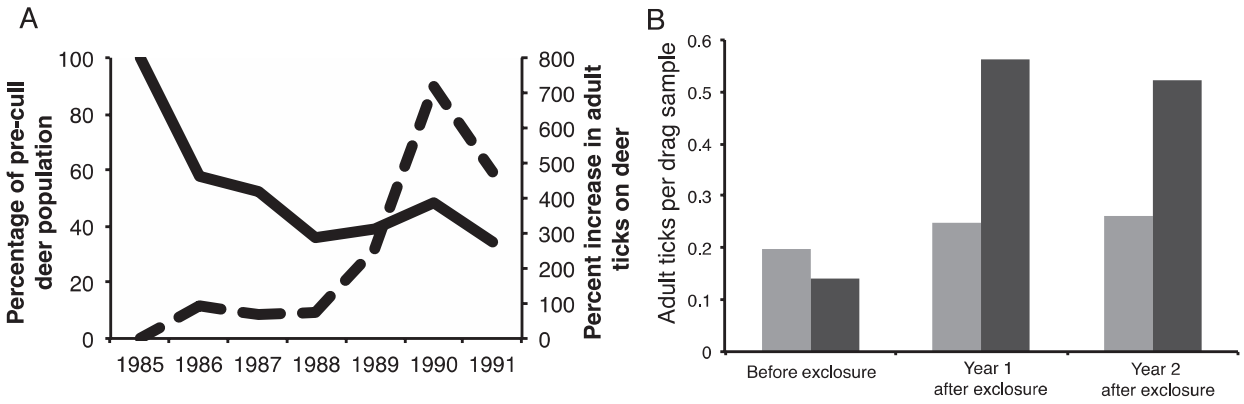

FIG. 3. The relationship between deer density and tick feeding success. (A) Data from experimental deer cull published in Deblinger et al. (1993) showing the decline in the deer density index (solid line) accompanied by an increase in the number of adult ticks counted on deer (dashed line). (B) Data from Ginsberg and Zhioua (1999) on the encounter rate (per minute of cloth dragging) of questing adult ticks in control (light gray) and deer exclosure treatment (dark gray) before the exclosure was erected, 1 yr after exclosure, and 2 yr after exclosure.

The data from an experimental deer cull published in Deblinger et al. (1993) showed that the decline in the deer density index was accompanied by an increase in the number of adult ticks counted on deer (Fig. 3A), suggesting that within this range of deer densities, deer density is not limiting adult tick feeding opportunities because the number of adult ticks feeding on the deer population is invariant to deer density. Data from Ginsberg and Zhioua (1999) on the encounter rate of questing adult ticks before a deer exclosure was erected, 1 yr after exclosure, and 2 yr after exclosure indicated that >50% of adult ticks had already attached to deer by mid-November (Fig. 3B). This is early in the phenology of the adult life stage, which overwinters and is active again through the following spring (Levi et al. 2015), suggesting that a larger portion of the adult life stage eventually finds a host.

TICK–HOST MODEL

The field data demonstrating tick redistribution across all life stages, strong redistribution of nymphs at moderate mouse densities, and heavy dependence of nymphs on rodents (mice and/or chipmunks), motivates us to develop a dynamic model to describe the transition from questing tick larvae to infected or uninfected nymphs (parameter list in Appendix S1: Table S1). The purpose of the model is to determine the effect of the presence of each host on the density of infected nymphs produced from emerging larvae in a single year. The model consists of differential equations for densities of questing larvae, Q, and for each larval tick host i of N species, the larvae that have attached to host i, A_i, and larvae that have successfully fed to repletion and detached from host i, F_i (Fig. 4A). Questing larvae emerge with a time-dependent phenology at rate $E(t)$, and they are removed from the questing class if they attach to any of the N hosts, or if they die, leading to the differential equation:

$$\frac{dQ}{dt} = E(t) - \sum_{i-1}^{N} a_i D_i Q - \mu_Q Q \qquad (1)$$

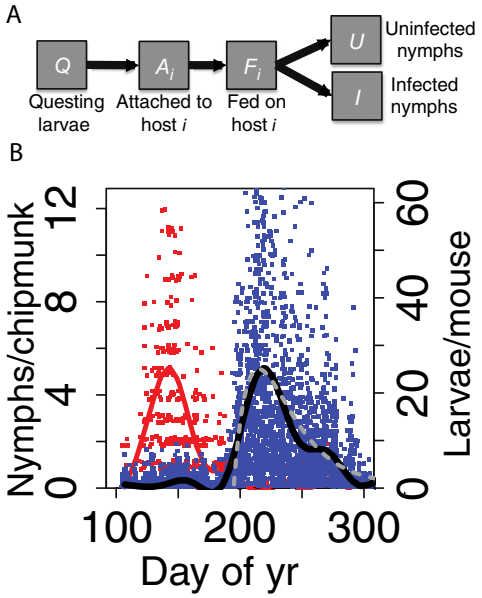

FIG. 4. (A) Structure of our differential equation model for the flow of questing larvae, Q, to attached larvae on each host species, A_i, to larvae that have successfully fed on each host species, F_i. The larvae that have successfully fed transition into infected or uninfected nymphs depending on the reservoir competence of the host. (B) Phenology of nymphs counted on the heads of chipmunks (red) followed by larvae counted on the heads and ears of mice (blue) on six 2.25-ha trapping grids at the Cary Institute of Ecosystem Studies in Dutchess County, New York, USA, in 2012. Our larval emergence function (Eq. 2) with a peak timing of 20 d ($b = 0.05$) is plotted with a gray dashed line and closely matches the timing of larval body burdens fit with a generalized additive model (black line). We ignore the small early larval peak associated with overwintering larvae from the previous cohort.

where μ_Q is the per-capita death rate of questing larvae, and a_i is the per-capita rate at which questing larvae encounter host i, which has density D_i. We are interested in differences between communities differing in which host species are present; D_i is set to 0 for hosts that are absent. Since additivity is assumed, the value of D_i for host i does

not depend on the composition of the rest of the community (i.e., no competition or predation). The emergence function can be approximated by:

$$E(t) = Hte^{-bt} \tag{2}$$

which describes the increase in larval abundance in late summer, when the vast majority of larvae emerge, followed by a peak and subsequent decline found in field data (Fig. 4B; $t = 0$ is assumed to be the emergence start time, before which $Q = 0$). The parameter H is the initial slope of Eq. 2, which also influences the height of the peak emergence rate, $H/(be)$, which occurs at $t = 1/b$. The total number of larvae in the cohort is H/b^2.

Attached larvae can either die on the host, with per-capita death rate μ_i, or successfully feed and drop off. The time required for larvae to feed to repletion is assumed to be constant ($\tau = 4$ d), so the drop-off rate is the product of the rate at which larvae attached to the host τ days ago and the probability that they successfully survived those τ days, which is $e^{-\mu_i\tau}$. This leads to a delay differential equation for the density of attached larvae on host species i with the form:

$$\frac{dA_i}{dt} = a_i D_i Q - \mu_i A_i - a_i D_i e^{-\mu_i\tau} Q(t-\tau). \tag{3}$$

The replete larvae that drop off their host move into a fed class. The density F_i of fed larvae produced in the current season from host i is described by:

$$\frac{dF_i}{dt} = a_i D_i e^{-\mu_i\tau} Q(t-\tau). \tag{4}$$

No death rate is included in Eq. 4 since we assume the mortality of fed larvae is dominated by failure to molt or overwintering deaths, accounted for below, Eqs. 6-7. The cumulative number of fed larvae produced from each host at time t can be found by integrating Eq. 4 with respect to time, giving:

$$F_i(t) = \int_0^t \frac{dF_i}{dt}\,dt = a_i D_i e^{-\mu_i\tau} \int_\tau^t Q(t-\tau)dt. \tag{5}$$

The total number of fed larvae produced from host i can be found by setting t to the time of the end of the season, which is often approximately $F_i(\infty)$ (see Fig. 5), which we assume.

To become nymphs the following year, larvae that fed on species i must successfully molt, which has probability M_i, and successfully overwinter, which has probability O_i (we assume that both can depend on the host, but not the time at which feeding ended). The density of nymphs the next year, DON, is then:

$$DON = \sum_{i=1}^{N} F_i(\infty) M_i O_i. \tag{6}$$

The density of nymphs infected with *B. burgdorferi* is:

$$DIN = \sum_{i=1}^{N} F_i(\infty) M_i O_i R_i \tag{7}$$

where R_i is the realized reservoir competence of vertebrate host i for *B. burgdorferi* (the probability that it will infect a tick that feeds on it). Because there is considerably less variation among species in the probability that larvae successfully molt, and because there are limited data on the probability of successful overwintering, we assume that all species produce fed larvae with similar molting success and overwinter survival, $M_i O_i = 0.4$ (Brunner et al. 2011).

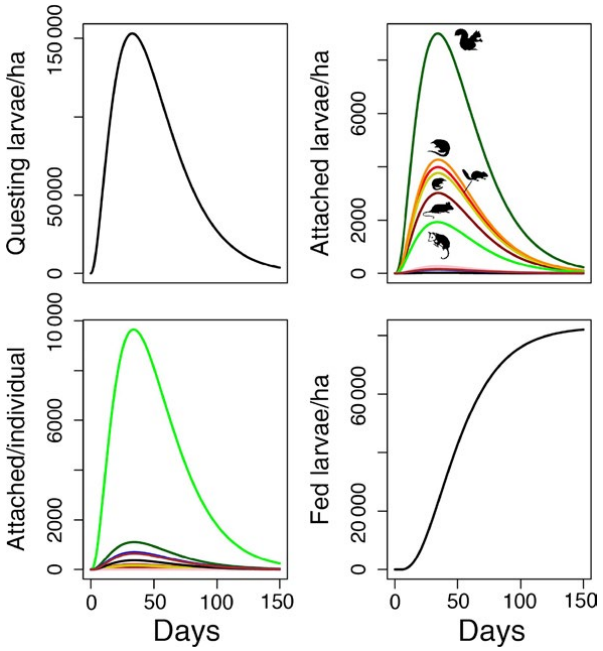

FIG. 5. Results from numerical solution (using base parameter set in Table 1, and $\mu_Q = 0.01$/d) of the differential equations for the densities of questing larvae, attached larvae on each host species, and fed larvae over a season. Attached larvae per host individual is also shown, indicating that individual opossums encounter many more larvae than do other hosts when assuming base parameters. Individual skunks and raccoons are also predicted to encounter thousands of larvae per individual if we assume that larval survival on these species is similar to survival on opossums. Per unit area, mice (dark red), short-tailed shrews (orange), chipmunks (red), masked shrews (yellow), squirrels (dark green), and opossums (light green) hold significant quantities of larvae. Raccoons (blue), skunks (black), and deer (brown) can individually hold significant quantities, but relatively few larvae attach to birds (pink).

Even when assuming additivity, increasing host diversity results in lower disease risk as long as the decline in tick survival and infection probabilities outweighs the increase in the probability of encountering a host. For example, if adding opossums, (excellent groomers and incompetent reservoirs) to a vertebrate community does not reduce the density of any other species, then larval ticks will be more likely to encounter a vertebrate host and potentially become infected. Whether this results in more infected nymphs depends in large part on the probability that these larvae would otherwise (in the absence of opossums) have found a more competent and permissive host such as a small mammal, which would have a higher probability of producing an infected nymph. This can be formalized by comparing the fractional change in the density of infected nymphs, ΔDIN_j, going from a community with all N host

species to a community of $N-1$ species, in which one focal species (j) has been removed. The change in DIN when host j is removed can be written as:

$$\Delta DIN_j = \frac{\sum_{i\neq j} F_{i,j}(\infty) M_i O_i R_i}{\sum_i F_i(\infty) M_i O_i R_i} - 1 \quad (8)$$

where $F_{i,j}(\infty)$ is the total number of larvae that fed on host i when host j is removed from the community. Host j is a dilution host if $\Delta DIN_j > 0$ (DIN increases in j's absence) and an amplification host if $\Delta DIN_j < 0$, with the magnitude of ΔDIN_j determining the strength of dilution or amplification. (The summation with index i is over all hosts, and with index $i \neq j$ is over all hosts except j.)

We solved the differential Eqs. 1–4 numerically using the deSolve package in R statistics. The delay differential equation was implemented with the DDE function. We were also able solve the equations analytically (see Appendix S1), but most analytical solutions are unwieldy. However, we were able to obtain an intuitive and simple solution for $F_i(\infty)$:

$$F_i(\infty) = \frac{H}{b^2} \frac{a_i D_i e^{-\mu_i \tau}}{\sum_{i=1}^{N} a_i D_i + \mu_Q} \quad (9)$$

where H/b^2 is the total abundance of larvae (from integrating Eq. 2):

$$\frac{a_i D_i}{\sum_{i=1}^{N} a_i D_i + \mu_Q}$$

is the probability that a tick encounters host i rather than encountering another host or dying, and $e^{-\mu_i \tau}$ is the probability of surviving on host i to reach the fed class. This equation can also be used for $F_{i,j}(\infty)$ by excluding host j from the sum.

We can now derive the conditions for a focal species, j, to be a dilution host, which is $\Delta DIN_j > 0$, or, equivalently:

$$\sum_{i\neq j} F_{i,j}(\infty) M_i O_i R_i > \sum_i F_i(\infty) M_i O_i R_i. \quad (10)$$

M_i and O_i can be canceled because we assume that molting success and overwinter survival do not vary across species, and using $F_i(\infty)$ from Eq. 9 gives:

$$\sum_{i\neq j} \frac{a_i D_i e^{-\mu_i \tau} R_i}{\sum_{i\neq j} a_i D_i + \mu_Q} > \sum_i \frac{a_i D_i e^{-\mu_i \tau} R_i}{\sum_i a_i D_i + \mu_Q}. \quad (11)$$

Consider the conditions for species 2 to be a dilution host in a two-species host community:

$$\frac{a_1 D_1 e^{-\mu_1 \tau} R_1}{a_1 D_1 + \mu_Q} > \frac{a_1 D_1 e^{-\mu_1 \tau} R_1 + a_2 D_2 e^{-\mu_2 \tau} R_2}{a_1 D_1 + \mu_Q + a_2 D_2 + \mu_Q} \quad (12)$$

which can be simplified to:

$$R_2 e^{-\mu_2 \tau} < \frac{a_1 D_1}{a_1 D_1 + \mu_Q} R_1 e^{-\mu_1 \tau}. \quad (13)$$

The result in Eq. 13 is relevant to the debate over the dilution effect. For example, if we assume that $\mu_Q = 0$, such that questing larvae never die and so always eventually encounter a host, then host 2 is a dilution host if the product of the probability of surviving and becoming infected is lower on host 2 than on host 1. In this scenario, hosts that seem to be poor hosts really do reduce DIN. In contrast, as μ_Q gets large, the quantity on the right of Eq. 13 gets small and host 2 can be an amplification host even if it has low reservoir competence and is not permissive to tick feeding.

Equation 13 can be generalized to N species where species j is a dilution host if:

$$R_j e^{-\mu_j \tau} < \frac{\sum_{i\neq j} a_i D_i R_i e^{-\mu_i \tau}}{\sum_{i\neq j} a_i D_i + \mu_Q}. \quad (14)$$

Notably, the density of the potential dilution host does not appear in Eqs. 13 and 14, although the amount of dilution does depend on host density. H and b also do not affect Eq. 8 or inequalities 10–14.

Parameterization

Host specific encounter and mortality rates

We determined host-specific encounter and mortality rates using field data, experiments, and the literature. B_i is the number of larvae successfully feeding on an individual of host species i during the larval peak, which we call post-grooming body burden. S_i is the proportion of attached larvae that survive to successfully feed, and D_i is the population density (1/ha) of species i (Table 1). B_i has been determined for most hosts by bringing wild-caught animals into the lab during the larval peak and counting engorged larvae that fall off the animal (see LoGiudice et al. 2003). S_i has been determined experimentally by placing 100 larvae on each host and counting the number that successfully feed to repletion, and D_i is taken from the literature and field data (see Keesing et al. 2009, Table 1). The probability of larval survival while feeding has not been determined for all hosts. For hosts lacking data, we used both higher and lower values of larval survival. We assumed that short-tailed shrews (*Blarina brevicauda*) and masked shrews (*Sorex cinereus*) might groom similarly to mice (high survival) or chipmunks (moderate survival), that raccoons and skunks would groom similarly to chipmunks (moderate) or opossums (very low survival), and that larvae would have high survival on deer similar to that on mice. Larval survival on deer may be higher if deer are especially poor groomers, but increasing the survival probability to $S_i = 0.8$ did not change our results. We used two values of questing larvae mortality ($\mu_Q = 0.01, 0.1$).

We convert the survival probability, S_i, into the mortality rate μ_i, using:

$$e^{-\mu_i \tau} = S_i \rightarrow \mu_i = \frac{-\ln(S_i)}{\tau}. \quad (15)$$

In our model, the body burden of each host species (the number of attached ticks per host individual) is a dynamic outcome of the model. We solved for the encounter rate a_i by considering the steady state peak body burden A_i^*/D_i (total density of larvae attached to host species i divided by

the population density of species *i*) when the peak density of questing larvae is assumed to be constant, $Q(t) = Q^*$. Setting Eq. 3 to zero and solving for a_i yields:

$$a_i = \frac{A_i^*}{D_i}\left(\frac{\mu_i}{Q^*(1-e^{-\mu_i\tau})}\right). \quad (16)$$

The peak body burden A_i^*/D_i is equivalent to the post-grooming body burden divided by probability of surviving, B_i/S_i, assuming that Q^* is near the peak questing larval density (since B_i was measured on animals captured near this peak). Substituting our observed parameters into Eq. 10 yields the formula for a_i:

$$a_i = \frac{-B_i \ln(S_i)}{Q^* S_i(1-S_i)\tau} \quad (17)$$

Previous research using removal sampling found mean density of questing larvae during the peak, Q^*, of 115 000/ha (Daniels et al. 2000). Q^* is an important parameter because it influences the calculated value of the encounter rate a_i, which is a key parameter determining whether ticks that would have fed on host A instead redistribute to feed on host B when host A is removed (Eq. 14). We therefore ran our model for values of Q^* from 10 000 to 200 000 (equivalent to a range of densities of 1–20 larvae/m^2) in intervals of 5000.

Other parameters

Complete methods of estimating all other parameters are presented in Appendix S1. Briefly, we assumed that on average larvae survive a season ~100 d long, from mid-July to late October, leading to a mortality rate of $\mu_Q = 0.01$/d. We also ran the model at the low value of $\mu_Q = 0.1$/d, corresponding to an average life span of 10 d, to illustrate how the results change if larvae are much less likely to encounter a host before death. We fit the parameters of the emergence function to field data and using model output. These parameters do not influence the relative density of infected nymphs when a host species is removed (Eq. 11), but they do influence our numerical output and the predicted absolute density of nymphs.

Model Results

Numerical solutions

The temporal pattern of questing and attached larvae generated from the model (Fig. 5) had a form qualitatively similar to the temporal pattern of larval emergence given by Eq. 2, and the empirical temporal pattern of attachment observed on mice (Fig. 4B). The total number of fed larvae saturated by about 150 d, and few questing larvae remained alive, which is consistent with the small cohort of overwintering larvae that we observe in late spring (Fig. 4B). Using our base parameter values (Table 1), far more larvae attached to squirrels than to other hosts, a result driven by the very large pre-grooming body burdens (*B*/*S*) of squirrels, and their moderately high population density. After squirrels, shrews and chipmunks had the most larvae attached, followed by mice. Substantially fewer larvae attached to opossums and low numbers of larvae attached to any other hosts (Fig. 5). Per animal, opossums had by far the most larvae attached, followed by squirrels, raccoons, and deer (Fig. 5). The opossum result is driven by their high post-grooming body burden and very low survival of attached larvae, which when put together (*B*/*S*) leads to a high attachment rate. If we assume that larval survival on raccoons and skunks is similarly low to survival on opossums, then these other carnivores also host large quantities of larvae. We explore the variation of results dependent on parameter values as follows.

Larval redistribution after removing opossums and squirrels

Removing opossums and squirrels from the host community using our base parameter values (which used the lower opossum density) resulted in a 57% increase in the number of larvae attached to each amplification host (mice, chipmunks, and shrews; Fig. 6). The timing of peak attachment also occurred later without squirrels and opossums because larvae remain in the questing pool longer before finding a host.

Testing hosts for dilution and amplification

With our base parameter values, squirrels were the most important dilution hosts followed by opossums (Figs. 7A and 8A). Removing these species substantially increased the density of infected nymphs over the range of Q^* that we assessed (Fig. 7A). Mice, chipmunks, short-tailed shrews, and masked shrews were the most important amplification hosts (Figs. 7A and 8A). Varying the parameter Q^* did not qualitatively impact our inferences, but the fractional change in *DIN* declined as Q^* increased so that squirrels and opossums became be less effective dilution hosts and small mammals became slightly more potent amplification hosts (Fig. 7A). If the death rate of questing larvae were very high (life span of 10 d), which reduces the possibility of finding an alternative host before death, opossums and squirrels become substantially less potent dilution hosts (Fig. 7B), and the predicted change in *DIN* became more sensitive to Q^*.

Our results changed substantially with alternative parameter values. If the actual opossum density were 1/ha as has previously been assumed (LoGiudice et al. 2003, Keesing et al. 2009), then opossums became by far the most important dilution host (Fig. 8C). If the body burden of birds were substantially higher, as in Keesing et al. (2009), then birds became minor dilution hosts (Fig. 8D). Using the smaller parameter values for the survival of larvae on shrews and mesocarnivores (i.e., survival on raccoons and skunks is similar to opossums and survival on shrews is similar to chipmunks; Table 1) caused raccoons

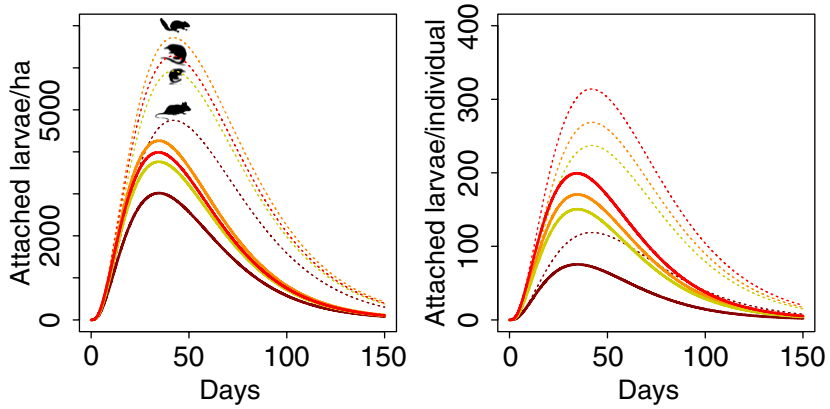

FIG. 6. The redistribution of larvae onto amplification hosts, including mice (dark red), short-tailed shrews (orange), chipmunks (red), and masked shrews (yellow), when opossums and squirrels were removed resulted in a 57% increase in maximum body burdens. Larvae attached to amplification hosts per hectare and per individual with an intact host community (solid lines), and when squirrels and opossums are removed (thin dotted lines). The right-shift in the curve with squirrels and opossums removed occurs because larvae remain in the questing pool longer before finding a host.

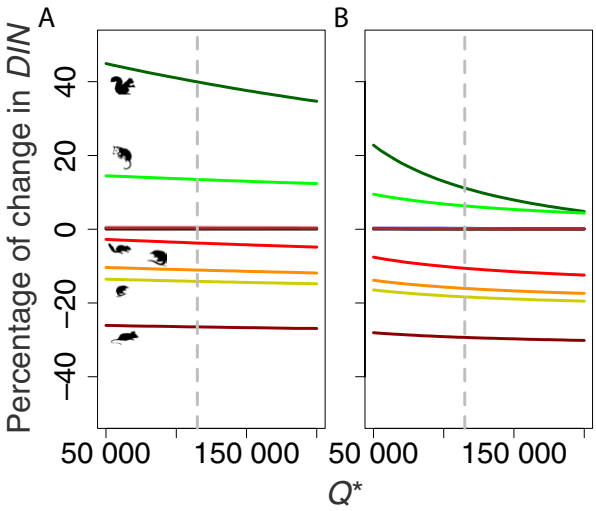

FIG. 7. The percent change in the density of infected nymphs (*DIN*) when removing each host species as a function of the peak larval density parameter Q^*, which influences the encounter rate between questing larvae and hosts. Hosts whose removal has a visible influence on *DIN* are labeled with silhouettes. Raccoons, skunks, deer, and birds all overlap with a near zero percent change in *DIN* when these species are not available to larvae. (A) Removing mice (dark red), chipmunks (red), masked shrews (yellow), and short-tailed shrews (orange) leads to a substantial decrease in *DIN*; they are amplification hosts. Removing squirrels (dark green) and opossums (light green) leads to a substantial increase in *DIN*. (B) Even if the mortality rate of questing larvae increases from $\mu_Q = 0.01$ to $\mu_Q = 0.1$/d, which corresponds to an implausible decreasing in the average lifespan from 100 d to 10 d, removing squirrels and opossums still increases the density of nymphs infected with *B. burgdorferi*.

to become moderately effective dilution hosts and shrews to become less important amplification hosts, as expected, but this also reduced the number of larvae that encounter squirrels and opossums (because more have encountered raccoons and shrews first), which somewhat reduced their effectiveness at reducing *DIN* (Fig. 8E). Using the same parameters as in Keesing et al. (2009), which includes the higher values of opossum density and bird body burden in Table 1, then opossums encountered so many questing larvae that birds became relatively inconsequential hosts (i.e., neither strong dilution or amplification hosts; Fig. 8F).

DISCUSSION

The initial description of the dilution effect in this system focused on the reduction in nymphal infection prevalence as hosts other than the white-footed mouse, the most competent reservoir species, were introduced (LoGiudice et al. 2003, Ostfeld and LoGiudice 2003). Later work integrated vector amplification/reduction caused by differential larval survival on each host species and different scenarios for the degree of tick redistribution as host communities change (Keesing et al. 2009). In contrast, we have attempted to (1) use field data to explore the degree of tick redistribution due to variation in intraspecific host density and (2) to use models to explore the degree and consequence of tick redistribution to interspecific variation in host densities.

Tick redistribution

Our field data indicated that both larvae and nymphs concentrate on fewer animals as mouse density declines. The total number of nymphs feeding on mice saturated at moderate mouse densities, suggesting that the availability of hosts for nymphs is not limiting to tick populations in most years. We also found that the peak density of questing nymphs is several times higher when mouse densities are very low. These results have several important implications: (1) the density of infected nymphs (Lyme disease risk) is much higher in years when the rodent population crashes because questing nymphs that would have fed on

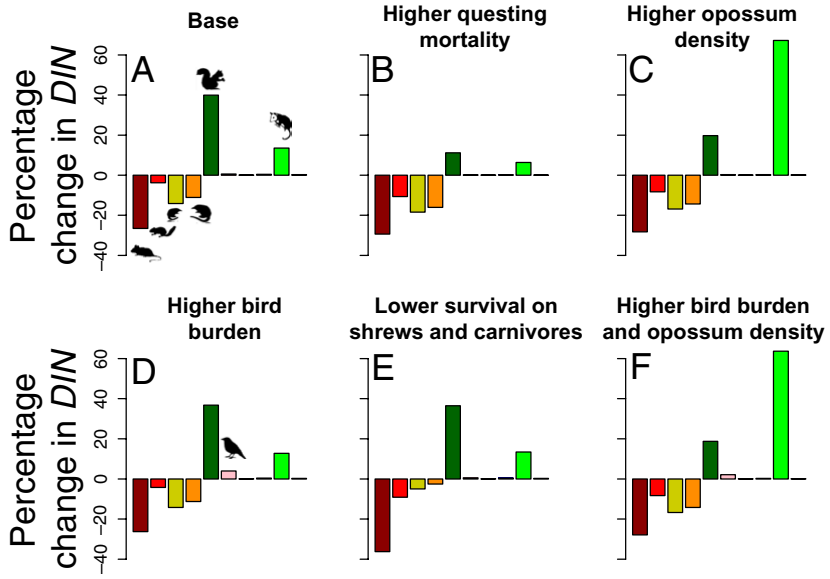

FIG. 8. Percent change in *DIN* at our point estimate $Q^* = 115\,000$ taken from Daniels et al. (2000) and using different parameter values. The model outputs correspond to (A) our base parameter values including the first value in the split cells of Table 1 (as in Fig. 5A) and $\mu_Q = 0.01$/d, (B) increasing the questing mortality rate to $\mu_Q = 0.1$ (as in Fig. 7B), (C) increasing opossum density to 1/h as previously assumed in LoGiudice et al. (2003) and Keesing et al. (2009) and, (D) increasing body burden of birds to 11.4 as found in Keesing et al. (2009), (E) lower survival rates of attached larvae on shrews and mesocarnivores (Table 1), and (F) increasing both opossum density and the body burden of birds to match the parameterization in Keesing et al. (2009).

rodents now remain questing, (2) a high proportion of nymphs find hosts, (3) the combination of mice and/or chipmunks feed a large proportion of nymphs, (4) larvae feed on many hosts, including more larvae feeding on shrews, chipmunks, and squirrels than on mice, which (5) may be responsible for the density of questing larvae being unrelated to mouse density, but (6) mice were nevertheless the most important amplification host (Figs. 5–8).

The data from Deblinger et al. (1993) and Ginsberg and Zhioua (1999) demonstrated that adult ticks are also able to concentrate on fewer deer as deer density declines. Ginsberg and Zhioua (1999) demonstrated that over half of adult ticks had already attached to deer by mid-November, while some unknown additional fraction of ticks would have attached after this date, including the following spring after adults overwinter, and onto other medium-bodied hosts. The large proportion of nymphs and adults that find hosts depletes the pool of questing nymphs and adults, suggesting that nymphs and adults can redistribute onto fewer individuals more readily than larvae, but even larval feeding begins to saturate as host density increases. However, this may be due to differences in host behavior at high densities rather than depletion of the questing larval pool (Fig. 2).

Modeling results

Understanding host-vector-pathogen interactions requires a detailed understanding of the annual within-season dynamics of vectors interacting with hosts. In this paper, we have utilized long-term data to parameterize a model for larval feeding and transition to nymphs in a multi-host community. By dynamically modeling the process of host questing, attachment, and feeding, we allowed the degree of tick redistribution as host community composition changed to be a dynamic outcome of the model. We used field-derived parameter estimates, and a range of plausible parameter values, to assess the robustness of our results. Our results differ considerably from previous work on the Lyme disease biodiversity-dilution effect. For example, LoGiudice et al. (2003) found both species of shrews to be among the most important dilution hosts when considering their impact on reducing nymphal infection prevalence. However, consistent with Brisson et al. (2007), we found that shrews feed enough larvae that would otherwise fail to feed that these hosts can increase the density of infected nymphs. With or without host additivity, mice, chipmunks, and the two species of shrews are likely to be amplification hosts.

Our results are consistent with the findings of Keesing et al. (2009), in that we demonstrate that some hosts can reduce both the abundance and infection prevalence of nymphs. In our model, squirrels and opossums were clear dilution hosts, diverting larvae away from small mammals, killing many of them, and infecting a small proportion of the rest. However, raccoons, birds, and skunks were either inconsequential hosts or mild dilution hosts, depending on parameter values, because they did not encounter enough ticks to divert a large number of blood meals away from amplification hosts. It is noteworthy that birds, which are sometimes considered to be important hosts (Brinkerhoff et al. 2011), were deemed to be dilution rather than amplification hosts, especially when using the higher parameter value for their body burden (Fig. 8D, F).

This occurred because birds are only somewhat permissive to feeding and only somewhat competent as reservoirs (Table 1).

Although we tested the sensitivity of the model using a range of parameter values, substantially different parameter values would change our conclusions. For instance, opossums, but not raccoons, greatly reduced the density of infected nymphs. This difference is due to our field data showing twice as many larvae on opossums than raccoons. If our opossum body burden data greatly overestimates the rate at which opossums encounter larvae, then opossums would be less efficient dilution hosts (i.e., more similar to raccoons). Similarly, we found that, as expected, much lower survival of questing larvae would reduce the magnitude of the dilution effect (Figs. 7 and 8). This is consistent with previous research in the more environment of California, where questing tick survival is low, which found that larvae did not find an alternate host after the removal of their primary lizard host (Swei et al. 2011).

We emphasize that our specific model and conclusion focus on phenomena that emerge within a single growing season. Like previous research, we model the larvae–nymph transition because of limitations in data availability for the nymph-adult and adult-larvae transitions (Fig. 1). However, our field data suggest that hosts for larvae are most limiting (i.e., total number of larvae on mice does not fully saturate as mouse density increases and there is no evidence that hosts are depleting the pool of questing larvae; Fig. 2) and that mice and chipmunks feed a large proportion of the nymph population, suggesting that the dilution hosts from the larvae to nymph stage are unlikely to feed so many nymphs that they increase overall tick abundance. Additionally, in our long-term data on all questing life stages at Cary (Ostfeld et al. 2006), we find little evidence of any demographic momentum between subsequent generations.

Our results indicate (1) that mice, chipmunks, and both species of shrews are important amplification hosts, (2) that the density of infected nymphs is expected to increase when squirrels and opossums are removed, and (3) that most other species are relatively inconsequential hosts with respect to the larval to nymph transition. Result (1) is likely to be strengthened in a model including nymph-adult transitions because nymphs would be less likely to find a host without these common species (e.g., questing nymph density greatly increases at the lowest mouse densities in Fig. 2F), which would be expected to feed back to fewer adults and perhaps fewer larvae in the subsequent generation. Result (2) would not hold if squirrels and opossums feed enough nymphs that would otherwise fail to find a host that their removal reduces the number of adults and consequently the number of larvae. This is unlikely given that the number of nymphs on mice saturated at moderate mouse densities (Fig. 2D, E), and the density of questing nymphs increased substantially when mice were rare, which indicated that mice (and/or chipmunks by correlation) feed a large fraction of the nymph population (Fig. 2F). Result (3) is unlikely to hold in all circumstances because reducing deer populations to very low levels can cause hosts for adult ticks to become limiting resulting in lower densities of larvae (Daniels et al. 1993, Daniels and Fish 1995), although this is not always the case (Perkins et al. 2006). The deer density where saturation occurs is unknown and may depend on the density of other medium-bodied hosts that can support adult ticks. However, adult ticks do readily concentrate on fewer deer as densities decline, and the density of questing adults can appear very low when deer densities are high because most of the population has already attached to hosts (Fig. 3).

Future research

An important task for future work would be to extend this model to multiple annual cycles. This would require accounting for density dependence at some stage in the tick life cycle, which has been found in the probability of survival on hosts (e.g., Randolph 1993, Levin and Fish 1998) but see contradictory results in Hazler and Ostfeld (1995), and also considering consequences of temporal variation in host numbers and community composition. A host species that within a typical season might seem unimportant for tick population dynamics could be quite important in less typical years, due to the absence of other hosts to sustain tick numbers. For example, our results suggest that a large fraction of nymphs feed on mice and chipmunks, so that squirrels and shrews are not typically important hosts, but these hosts may help sustain the tick population during mouse and chipmunk population crashes. Finally, some hosts that are seemingly unimportant in terms of local host and pathogen dynamics may nevertheless be important as modulators of dispersal among habitat (i.e., birds).

Future research should consider the impact of host community composition throughout the complete life cycle of ticks as well as the impact of species interactions in a diverse, temporally varying, and spatially structured host community. This will require substantial new empirical data to improve our understanding of (1) the number and survival of nymphs on each host, (2) the functional form of density dependence for each life stage of the tick population, and (3) how the presence of predators and competitors influences the density of important amplification hosts.

Relevance to the dilution effect

We have adhered to the assumption that biodiversity loss leads to fewer blood-meal opportunities because fewer species reduces overall host density (Randolph and Dobson 2012). The assumption of an additive relationship between species richness and host density pervades both epidemiological models and much discussion of the dilution effect (Dobson 2004, Randolph and Dobson 2012). If one assumes that host densities are additive (i.e., no species interactions so that adding species increases

total host density) and that all hosts are otherwise identical, then host diversity is expected to increase pathogen emergence (R_0) if transmission is density dependent and reduce pathogen emergence if transmission is frequency dependent (Dobson 2004, Rudolf and Antonovics 2005). However, the community ecology of species interactions and patterns of biodiversity loss suggest that the assumption of additivity is unlikely to be met when considering competitive and trophic interactions introduced with increasing host diversity. Some species are more sensitive to the anthropogenic or natural forces that reduce biodiversity and others are less sensitive or more resilient. Resilient species tend to be habitat and dietary generalists, to be smaller, to have a faster pace of life, and to occupy lower trophic levels as compared to more sensitive species (Julliard et al. 2004, Cardillo et al. 2005, Jiguet et al. 2007, Lavergne et al. 2013, Ostfeld et al. 2014). Small-bodied species with a fast life history, such as many rodents, not only persist as habitats are fragmented and biodiversity declines, but also typically become more abundant, which may be due to reduced predation and competition (Nupp and Swihart 1998, 2000). While small mammals (mice, chipmunks, and shrews) are primary reservoir hosts, much of the remaining host community includes a diversity of mesocarnivore predators (including raccoon, skunk, and opossum, as well as weasel, fox, bobcat, coyote, and fisher) that may reduce the density of small mammals (Fig. 1B). Similarly, eastern gray squirrels, raccoons, and opossums compete with mice and chipmunks for acorns and other seeds, which are an important overwinter food resource (Ostfeld et al. 1996).

Diverse host communities thus appear to provide two types of dilution hosts: ones that are sufficiently abundant and heavily parasitized that they deflect a large number of tick blood meals away from the most competent species (e.g., squirrels, opossums), and ones that can reduce the abundance of the most competent hosts through species interactions such as competition and predation. Because abundance is a prerequisite for being a dilution host of the first type, these dilution hosts are likely to be less sensitive to anthropogenic disturbance. Consequently, disease risk is likely to increase due to dominance by amplification hosts predominantly at the lowest levels of species richness observed in these landscapes (Ostfeld and LoGiudice 2003, Keesing et al. 2009).

Dilution hosts of the second type occur when higher levels of biodiversity shift vertebrate biomass from small mammals to competitors and predators that empirical data suggest are better groomers and incompetent hosts for *B. burgdorferi* (Table 1), with the opposite occurring with lower diversity. This amplification-host reduction mechanism has been previously proposed as a driver of increasing Lyme disease cases due to loss of predation service (Levi et al. 2012). Many possible dilution hosts of the second type are more sensitive to anthropogenic disturbance. Ecological communities are now highly altered in eastern deciduous forests of the northeast and upper midwest USA that are hotspots for tick-borne disease. The now extinct passenger pigeon was the most abundant seed predator in these forests and would have competed with mice and chipmunks for acorns, which are a crucial resource for overwinter survival (Ostfeld et al. 2001). Historic predator communities featured top predators, such as puma (*Puma concolor*) and wolves (*Canis lupus*), had larger and more widespread populations of bobcats (*Lynx rufus*), fisher (*Martes pennanti*), and marten (*Martes americana*), which are still extirpated from much of their former range, and contained few or no coyotes (*Canis latrans*). An unresolved question is whether historic predation levels in Lyme disease hotspots previously exceeded the thresholds that would have suppressed disease. What is known is that predators in other systems are able to maintain rodent prey at low abundance across a wide range of resource availability (Korpimäki et al. 2004) and that deer populations irrupted in the absence of top predators (perhaps first noted in Leopold et al. 1947). The degradation of top-down forces as predators are extirpated has unknown consequences that could have widespread ripple effects for disease dynamics in many anthropogenic landscapes.

Acknowledgments

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Literature Cited

Allan, B. F., et al. 2009. Ecological correlates of risk and incidence of West Nile Virus in the United States. Oecologia 158:699–708.

Bacon, R. M., K. J. Kugeler, and P. S. Mead. 2008. Surveillance for Lyme disease: United States, 1992–2006. Morbidity and Mortality Weekly Report 57(SS10):1–9.

Barbour, A. G., and D. Fish. 1993. The biological and social phenomenon of Lyme disease. Science 260:1610–1616.

Brinkerhoff, J. R., C. M. Folsom-O'Keefe, K. Tsao, and M. A. Diuk-Wasser. 2011. Do birds affect Lyme disease risk? Range expansion of the vector-borne pathogen *Borrelia burgdorferi*. Frontiers in Ecology and the Environment 9:103–110.

Brisson, D., D. E. Dykhuizen, and R. S. Ostfeld. 2007. Conspicuous impacts of inconspicuous hosts on the Lyme disease epidemic. Proceedings of the Royal Society B 275:227–235.

Brunner, J. L., L. Cheney, F. Keesing, M. Killilea, K. Logiudice, A. Previtali, and R. S. Ostfeld. 2011. Molting success of *Ixodes scapularis* varies among individual blood meal hosts and species. Journal of Medical Entomology 48:860–866.

Brunner, J. L., S. Duerr, F. Keesing, M. Killilea, H. Vuong, and R. S. Ostfeld. 2013. An experimental test of competition among mice, chipmunks, and squirrels in deciduous forest fragments. PLoS ONE 8:e66798.

Cardillo, M., G. M. Mace, K. E. Jones, J. Bielby, O. R. P. Bininda-Emonds, W. Sechrest, C. D. L. Orme, and A. Purvis. 2005. Multiple causes of high extinction risk in large mammal species. Science 309:1239–1241.

Daniels, T. J., and D. Fish. 1995. Effect of deer exclusion on the abundance of immature *Ixodes scapularis* (Acari: Ixodidae) parasitizing small and medium-sized mammals. Journal of Medical Entomology 32:5–11.

Daniels, T. J., D. Fish, and I. Schwartz. 1993. Reduced abundance of *Ixodes scapularis* (Acari: Ixodidae) and Lyme disease risk by deer exclusion. Journal of Medical Entomology 30:1043–1049.

Daniels, T. J., R. C. Falco, and D. Fish. 2000. Estimating population size and drag sampling efficiency for the blacklegged tick (Acari: Ixodidae). Journal of Medical Entomology 37:357–363.

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

Deblinger, R. D., M. W. Wilson, D. W. Rimmer, and A. Spielman. 1993. Reduced abundance of immature *Ixodes dammini* (Acari: Ixodidae) following incremental removal of deer. Journal of Medical Entomology 30:144–150.

Dobson, A. 2004. Population dynamics of pathogens with multiple host species. American Naturalist 164:S64–S78.

Estes, J. A., et al. 2011. Trophic downgrading of planet earth. Science 333:301–306.

Galetti, M., R. Guevara, C. L. Neves, R. R. Rodarte, R. S. Bovendorp, M. Moreira, J. B. Hopkins, and J. D. Yeakel. 2015. Defaunation affects the populations and diets of rodents in Neotropical rainforests. Biological Conservation 190:2–7.

Ginsberg, H. S., and E. Zhioua. 1999. Influence of deer abundance on the abundance of questing adult *Ixodes scapularis* (Acari: Ixodidae). Journal of Medical Entomology 36:376–381.

Grant, P. 1972. Interspecific competition among rodents. Annual Review of Ecology and Systematics 3:79–106.

Hazler, K. R., and R. S. Ostfeld. 1995. Larval density and feeding success of *Ixodes scapularis* on two species of *Peromyscus*. Journal of Parasitology 81:870–875.

Jiguet, F., A. S. Gadot, R. Julliard, S. E. Newson, and D. Couvet. 2007. Climate envelope, life history traits and the resilience of birds facing global change. Global Change Biology 13:1672–1684.

Joseph, M. B., J. R. Mihaljevic, S. A. Orlofske, and S. H. Paull. 2013. Does life history mediate changing disease risk when communities disassemble?. Ecology letters 16:1405–1412.

Julliard, R., F. Jiguet, and D. Couvet. 2004. Common birds facing global changes: what makes a species at risk? Global Change Biology 10:148–154.

Keesing, F., R. D. Holt, and R. S. Ostfeld. 2006. Effects of species diversity on disease risk. Ecology Letters Ecology Letters 9:485–498.

Keesing, F., J. Brunner, S. Duerr, M. Killilea, K. LoGiudice, K. Schmidt, H. Vuong, and R. S. Ostfeld. 2009. Hosts as ecological traps for the vector of Lyme disease. Proceedings of the Royal Society B 276:3911–3919.

Keesing, F., et al. 2010. Impacts of biodiversity on the emergence and transmission of infectious diseases. Nature 468:647–652.

Kelt, D. A., M. L. Taper, and P. L. Meserve. 1995. Assessing the impact of competition on community assembly: a case study using small mammals. Ecology 76:1283–1296.

Kilpatrick, A. M., P. Daszak, M. J. Jones, P. P. Marra, and L. D. Kramer. 2006. Host heterogeneity dominates West Nile virus transmission. Proceedings of the Royal Society B 273:2327–2333.

Korpimäki, E., P. R. Brown, J. Jacob, and R. P. Pech. 2004. The puzzles of population cycles and outbreaks of small mammals solved? BioScience 54:1071–1079.

Lavergne, S., M. E. K. Evans, I. J. Burfield, F. Jiguet, and W. Thuiller. 2013. Are species' responses to global change predicted by past niche evolution? Philosophical Transactions of the Royal Society B 368:20120091.

Leopold, A., L. K. Sowls, and D. L. Spencer. 1947. A survey of over-populated deer ranges in the United States. Journal of Wildlife Management 11:162–177.

Levi, T., A. M. Kilpatrick, M. Mangel, and C. C. Wilmers. 2012. Deer, predators, and the emergence of Lyme disease. Proceedings of the National Academy of Sciences USA 109:10942–10947.

Levi, T., F. Keesing, K. Oggenfuss, and R. S. Ostfeld. 2015. Accelerated phenology of blacklegged ticks under climate warming. Philosophical Transactions of the Royal Society of London B 370:20130556.

Levin, M. L., and D. Fish. 1998. Density-dependent factors regulating feeding success of *Ixodes scapularis* larvae (Acari: Ixodidae). Journal of Parasitology 84:36–43.

Lloyd-Smith, J. O., D. George, K. M. Pepin, V. E. Pitzer, J. R. Pulliam, A. P. Dobson, P. J. Hudson, and B. T. Grenfell. 2009. Epidemic dynamics at the human-animal interface. Science. 326:1362–1367.

LoGiudice, K., R. S. Ostfeld, K. A. Schmidt, and F. Keesing. 2003. The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. Proceedings of the National Academy of Sciences USA 100:567–571.

McCauley, D. J., F. Keesing, T. Young, and K. Dittmar. 2008. Effects of the removal of large herbivores on fleas of small mammals. Journal of Vector Ecology 33:263–268.

Nupp, T. E., and R. K. Swihart. 1998. Influence of forest fragmentation on population attributes of white-footed mice and eastern chipmunks. Journal of Mammalogy 79:1234–1243.

Nupp, T. E., and R. K. Swihart. 2000. Landscape-level correlates of small-mammal assemblages in forest fragments of farmland. Journal of Mammalogy 81:512–526.

Nupp, T., and R. Swihart. 2001. Assessing competition between forest rodents in a fragmented landscape of midwestern USA. Mammalian Biology 66:345–356.

Ostfeld, R. S. 2013. A Candide response to Panglossian accusations by Randolph and Dobson: biodiversity buffers disease. Parasitology 140:1196–1198.

Ostfeld, R. S., and R. D. Holt. 2004. Are predators good for your health? Evaluating evidence for top-down regulation of zoonotic disease reservoirs. Frontiers in Ecology and the Environment 2:13–20.

Ostfeld, R. S., and F. Keesing. 2012. Effects of host diversity on infectious disease. Annual Review of Ecology, Evolution, and Systematics 43:157–182.

Ostfeld, R. S., and F. Keesing. 2013. Straw men don't get Lyme disease: response to Wood and Lafferty. Trends in Ecology and Evolution 28:502–503.

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

Ostfeld, R. S., C. G. Jones, and J. O. Wolff. 1996. Of mice and mast: ecological connections in eastern deciduous forests. BioScience 46:323–330.

Ostfeld, R. S., E. M. Schauber, C. D. Canham, F. Keesing, C. G. Jones, and J. O. Wolff. 2001. Effects of acorn production and mouse abundance on abundance and *Borrelia burgdorferi* infection prevalence of nymphal *Ixodes*

scapularis ticks. Vector Borne and Zoonotic Diseases 1:55–63.

Ostfeld, R. S., C. D. Canham, K. Oggenfuss, R. J. Winchcombe, and F. Keesing. 2006. Climate, deer, rodents, and acorns as determinants of variation in Lyme-disease risk. PLoS Biology 4:1058–1068.

Ostfeld, R. S., T. Levi, A. Jolles, L. B. Martin, P. R. Hosseini, and F. Keesing. 2014. Life history and demographic drivers of reservoir competence for three tick-borne zoonotic pathogens. PLoS ONE 9:e107387.

Perkins, S. E., I. M. Cattadori, V. Tagliapietra, A. P. Rizzoli, and P. J. Hudson. 2006. Localized deer absence leads to tick amplification. Ecology 87:1981–1986.

Porco, T. C. 1999. A mathematical model of the ecology of Lyme disease. Journal of Mathematics Applied in Medicine and Biology 16:261–296.

Previtali, M. A., R. S. Ostfeld, F. Keesing, A. E. Jolles, R. Hanselmann, and L. B. Martin. 2012. Relationship between pace of life and immune response in wild rodents. Oikos 121:1483–1492.

Randolph, S. E. 1993. Density-dependent acquired resistance to ticks in natural hosts, independent of concurrent infection with *Babsia microti*. Parasitology 108:413–419.

Randolph, S. E., and A. D. M. Dobson. 2012. Pangloss revisited: a critique of the dilution effect and the biodiversity-buffers-disease paradigm. Parasitology 139:847–863.

Ripple, W. J., et al. 2013. Status and ecological effects of the world's largest carnivores. Science 343:1241484.

Rudolf, V. H. W., and J. Antonovics. 2005. Species coexistence and pathogens with frequency-dependent transmission. American Naturalist 166:112–118.

Schauber, E. M., and R. S. Ostfeld. 2002. Modeling the effects of reservoir competence decay and demographic turnover in Lyme disease ecology. Ecological Applications 12:1142–1162.

Swei, A., R. S. Ostfeld, R. S. Lane, and C. J. Briggs. 2011. Impact of the experimental removal of lizards on Lyme disease risk. Proceedings of the Royal Society B 278:2970–2978.

VanBuskirk, J., and R. S. Ostfeld. 1995. Controlling Lyme disease by modifying the density and species composition of tick hosts. Ecological Applications 5:1133–1140.

Wood, C. L., and K. D. Lafferty. 2012. Biodiversity and disease: a synthesis of ecological perspectives on Lyme disease transmission. Trends in Ecology and Evolution 28:239–247.

Young, H. S., et al. 2014. Declines in large wildlife increase landscape-level prevalence of rodent-borne disease in Africa. Proceedings of the National Academy of Sciences USA 111:7036–7041.

Supporting Information

Additional supporting information may be found in the online version of this article at http://onlinelibrary.wiley.com/doi/10.1890/15-0122.1/suppinfo

Data Availability

Data associated with this paper have been deposited in the KNB Data Repository: https://knb.ecoinformatics.org/#view/knb.779.2