Deer, predators, and the emergence of Lyme disease

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Lyme disease is the most prevalent vector-borne disease in North America, and both the annual incidence and geographic range are increasing. The emergence of Lyme disease has been attributed to a century-long recovery of deer, an important reproductive host for adult ticks. However, a growing body of evidence suggests that Lyme disease risk may now be more dynamically linked to fluctuations in the abundance of small-mammal hosts that are thought to infect the majority of ticks. The continuing and rapid increase in Lyme disease over the past two decades, long after the recolonization of deer, suggests that other factors, including changes in the ecology of small-mammal hosts may be responsible for the continuing emergence of Lyme disease. We present a theoretical model that illustrates how reductions in small-mammal predators can sharply increase Lyme disease risk. We then show that increases in Lyme disease in the northeastern and midwestern United States over the past three decades are frequently uncorrelated with deer abundance and instead coincide with a range-wide decline of a key small-mammal predator, the red fox, likely due to expansion of coyote populations. Further, across four states we find poor spatial correlation between deer abundance and Lyme disease incidence, but coyote abundance and fox rarity effectively predict the spatial distribution of Lyme disease in New York. These results suggest that changes in predator communities may have cascading impacts that facilitate the emergence of zoonotic diseases, the vast majority of which rely on hosts that occupy low trophic levels.

coyote range expansion | *Ixodes* | mesopredator release | trophic cascade | zoonosis

There is growing recognition that changes in host community ecology and trophic interactions can contribute to the emergence of infectious diseases (1–3). In particular, the transmission of vector-borne zoonotic diseases to humans depends on multiple species interactions that influence host and vector abundance and infection prevalence. Most zoonotic pathogens are harbored by wildlife that occupy low trophic levels (1). The extirpation of top predators and the consequent restructuring of predator communities (4, 5) may thus increase the risk of zoonotic diseases if predation of reservoir hosts plays a key role in disease suppression. A paradigmatic case of disease emergence that is thought to be driven by changes in the host community is Lyme disease (Fig. 1).

Lyme disease is the most prevalent vector-borne disease in North America, and both the annual incidence and geographic range are still increasing (6). The disease is caused by the bacteria *Borrelia burgdorferi*, which is transmitted to humans in the eastern United States primarily by the nymphal stage of *Ixodes scapularis* ticks (7). The emergence of Lyme disease has been attributed to the century-long population recovery of deer, which are not competent hosts for transmitting *B. burgdorferi* to ticks but are nonetheless important reproductive hosts for adult ticks (7, 8). Support for this hypothesis comes partly from studies of experimental removal or exclusion of deer, which has often led to reduced tick densities (9). However, substantial research indicates that experimental or natural increases of deer density above a low threshold often have little effect on nymphal tick abundance (reviewed in ref. 10; see also refs. 11–13; Table S1). This research suggests that when deer are sufficiently abundant, other factors, such as hosts for immature ticks, may become limiting. Decades after the recolonization of deer, and despite a shift in management objectives from increasing deer populations to stabilizing or reducing them (14), Lyme disease cases have increased enormously (380% increase in Minnesota, 280% in Wisconsin, and 1,300% in Virginia from 1997 to 2007; Fig. S1), which suggests that other previously unidentified ecological changes may now be facilitating the emergence of Lyme disease.

A growing body of evidence implicates small-mammal abundance as a key determinant of the density of infected nymphs, the primary measure of entomological risk for Lyme disease (12, 15, 16). Molecular evidence suggests that four species of small mammals (the white-footed mouse *Peromyscus leucopus*, Eastern chipmunk *Tamias striatus*, short-tailed shrew *Sorex brevicauda*, and masked shrew *Sorex cinereus*) are responsible for infecting 80–90% of ticks (17). Thus, it is possible that changes in the ecology of small mammals play a role in the continuing increase of Lyme disease. Small-mammal populations are influenced both by resource availability, which has been correlated with the subsequent density of infected nymphs (12, 15) and by predation (18). The latter finding has led to the suggestion that predation may play a key role in suppressing Lyme disease (1).

A major change in predator–prey interactions in North America over the last half-century has resulted from the range expansion and population growth of a new top predator—the coyote, *Canis latrans*, which has spread across the continent following the extirpation of gray wolves, *Canis lupus* (19). The expansion of coyotes likely suppressed the abundance of several small-mammal predators, with the reduction of foxes by interference competition with coyotes being the best documented (20–22). The replacement of foxes by coyotes would likely reduce predation rates on small-mammal prey (i.e., the reverse of mesopredator release) because red fox (*Vulpes vulpes*) densities are typically an order of magnitude higher than coyote densities (23–25), and small mammals make up a larger fraction of their diets, particularly in the eastern United States, where coyotes have hybridized with wolves (26) and rely far more on deer (27, 28). Further, red fox cache prey for later consumption and are thus capable of killing large quantities of prey when prey are abundant (e.g., after an acorn mast). The high abundance of foxes (29), their ability to kill large quantities of small mammals due to both dietary preference and prey-caching behavior, and their adaptability to human-dominated landscapes makes them potentially highly important to suppressing Lyme disease hosts in areas around human habitation. Thus, somewhat paradoxically, the expansion of coyotes likely decreased predation rates on small mammals by suppressing more-efficient predators (foxes).

Here we test the hypothesis that changes in predation have contributed to the continuing emergence of Lyme disease by analyzing disease models that explicitly incorporate predation intensity, and by examining spatial and temporal correlations at multiple scales between Lyme disease, coyote, fox, and deer abundance.

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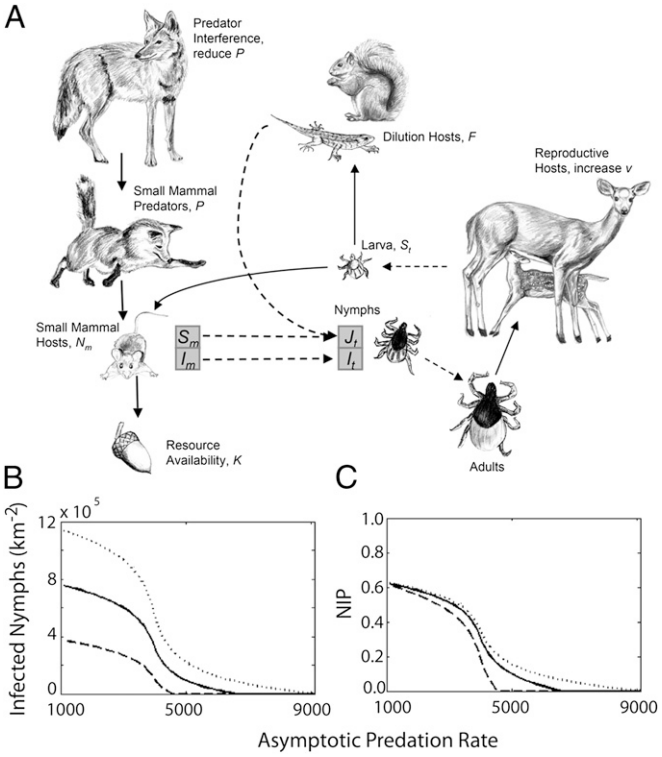


Fig. 1. (*A*) A simplified web of interactions involved in the ecology of Lyme disease. Solid lines indicate negative interactions, such as predation or parasitism. Dotted lines indicate resulting state transitions of ticks. Susceptible larva, S_t, infected nymphs, I_t, uninfected nymphs, J_t, and small-mammal hosts, N_m, broken into susceptible, S_m, and infected, I_m, classes are dynamically modeled. The density of dilution hosts, *F*, and predators, *P*, are incorporated into the model with parameters. Reproductive hosts are included with a parameter for the birth rate of ticks, ν. Our model uses ecologically realistic assumptions, such as logistic population growth, a type II functional response for ticks, and a type III functional response for generalist predators. (*B*) The model reveals a sharp nonlinear increase in the density of infected nymphs (DIN) and (*C*) nymphal infection prevalence (NIP) as the maximum predation rate (predator density × their consumption rate as prey increase to infinity) declines. The dotted, solid, and dashed lines corresponds to ν = 1.5, 1, and 0.5 million larva born per km^2 per year, respectively.

Results

Host–Vector Dynamical Model. We built a host–vector model to determine how changes in predation might impact Lyme disease risk (Fig. 1, *Methods*, and Table S2), and found that predation can have a strong nonlinear influence on both the density and infection prevalence of nymphs (Fig. 1 and Fig. S2). At intermediate predator densities, small changes in predation can cause large changes in Lyme disease risk. For example, a 20% reduction in predation near the inflection point in Fig. 1*B* more than doubles the density of infected nymphs. This nonlinearity is due to the interaction of predation with the quadratic shape of logistic population growth. Host densities near carrying capacity are by definition unproductive. Increasing the predation rate reduces host density, which increases population growth rates. When the host population is maximally productive near intermediate host densities, further increases in predation cannot be compensated for with more reproduction, which allows small increases in predation to cause greater reductions in host density (Fig. S3). Additionally, at these intermediate densities the host turnover rate is highest (maximal steady-state birth and death rates), which reduces host infection prevalence because hosts are born uninfected.

In this model, increasing deer abundance can also increase the density of infected nymphs if it increases the tick birth rate (Fig. 1*B*). However, the relationship between deer abundance and the tick birth rate is highly uncertain because adult ticks may be able to increasingly concentrate bloodmeals on fewer deer or alternate hosts as deer abundance declines. To explore the hypothesis that the relationship between deer and Lyme disease risk (density of infected nymphs) saturates (i.e., further increases in already abundant deer have little impact on nymph abundance), we reanalyzed data from deer removal studies that recorded deer abundance and the response of nymphs (30). Deer abundance was a poor predictor of tick abundance (measured as nymphs per mouse) 2 y later (Fig. 2*A*), which did not decline despite great reduction in deer abundance. Similarly, reducing deer density from >90 km^{-2} to 10 km^{-2} at Bluff Point coastal reserve in Groton, CT, only reduced tick density below 20 deer per km^2 (Fig. 2*B*) (31).

The model suggests that nymphal infection prevalence is only weakly influenced by the tick birth rate (Fig. 1*C*), because the fraction of ticks that are infected depends primarily on the composition of the host community and only weakly on the abundance of ticks. This finding is consistent with observations that nymphal infection prevalence does not decline inside deer exclosures (32), but does increase with small-mammal abundance (15, 16). The density of infected nymphs is a more direct Lyme disease risk factor than the infection prevalence of nymphs. If adult tick feeding rates saturate, then the key drivers of both the density and infection prevalence of nymphs would be hosts for immature ticks. Thus, the impact of predators would be greater than suggested here if reducing the density of hosts for immature ticks significantly reduces the tick birth rate (see additional model results in Figs. S4–S6).

Temporal Correlations. Over the past 30 y, correlations between deer abundance and Lyme disease were not significant or mixed in direction (Fig. 3), regardless of whether we scaled antlered-deer harvest by hunting license sales or used raw antlered-deer harvest data (Tables S3 and S4). Thus, we examined the potential role of predators as drivers of Lyme incidence with data on proxies of coyote and fox abundance (i.e., harvest by hunters). Harvests varied up to 10-fold as coyotes increased and foxes declined during the emergence of Lyme disease (Fig. 3). In Minnesota, fox hunter harvest decreased 95% from a high of 78,000 in 1991 to a low of 4,000 in 2008, whereas coyote harvest increased 2,200% from a low of 2,000 in 1982 to 46,000 in recent years. In Wisconsin, coyote hunter harvests increased 660% from a low of 6,847 in 1984 to over 52,000 in 2009, whereas fox harvests decreased 80% from over 25,000 to under 5,000 over that time. In Pennsylvania, only 1,810 coyotes were harvested in 1990, but harvests increased nearly 1,600% to a high of over 30,000 in 2009. In Virginia, where Lyme disease cases have only recently increased (more than 300% increase from 2005 to 2007), coyotes have also increased only recently, averaging ~3,000 in the 1990s, reaching nearly 10,000 in 2004, and increasing to a recent high of nearly 25,000 (Fig. S1).

Lyme disease cases were positively correlated with coyote abundance and negatively correlated with fox abundance in all four states (Fig. 3). The best models, using a model selection approach based on an information theoretic criterion (33), included measures of predator abundance for all four states. In contrast, deer abundance was present in the best fitting model only in Virginia (Fig. 3*D*).

Spatial Correlations. To test whether the spatial distribution of Lyme disease is correlated with the spatial distribution of deer or small-mammal predators, we examined Lyme disease incidence in Wisconsin, Pennsylvania, Virginia, and New York. Across space, Lyme disease incidence did not consistently increase with deer abundance. Deer and Lyme incidence were negatively correlated in Wisconsin and Pennsylvania, positively correlated in Virginia, and uncorrelated in New York (Fig. 4 *C–F*). In contrast, the spatial distribution of Lyme disease incidence in New York (the only state for which we had spatial data on predator abundance),

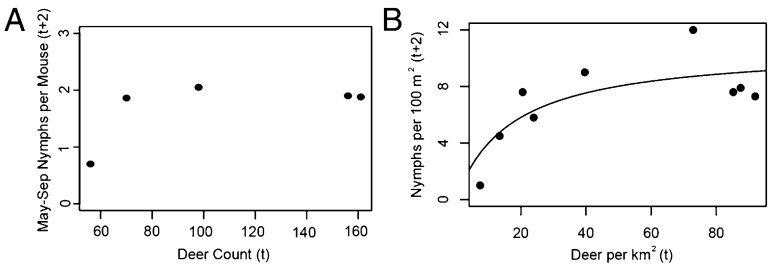


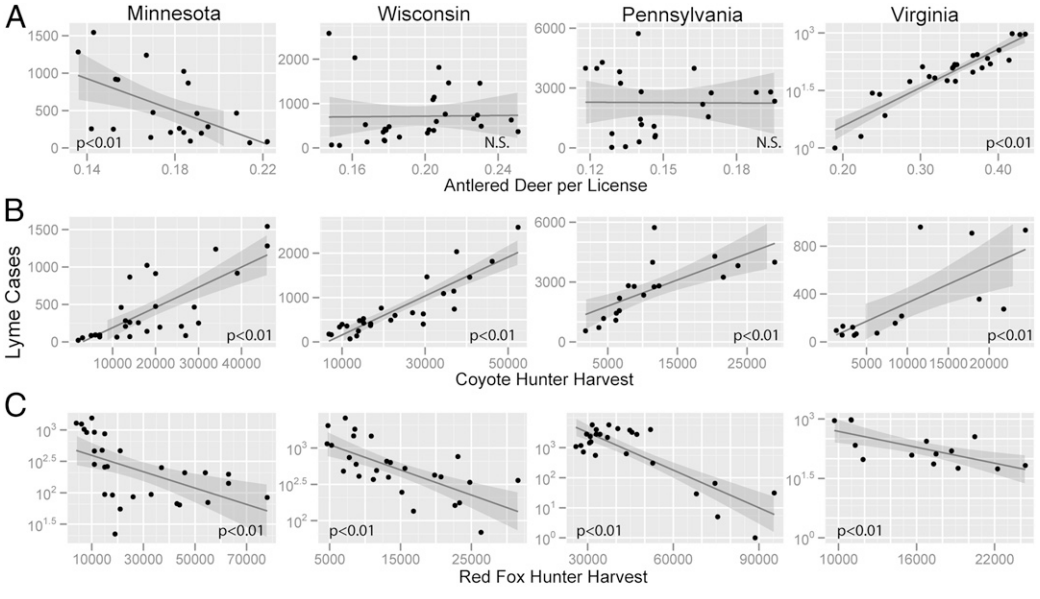
Fig. 2. Relationships between deer abundance and Lyme disease risk measured by the density of infected nymphs. (*A*) *I. scapularis* nymph abundance, measured as nymphs per mouse, in response to deer removal experiment in Deblinger et al. (30). (*B*) Nymph density (100 m^{-2}) as a function of deer density (per km^2) from Stafford et al. (31). When all data are included, there is a saturating relationship, and there is no significant relationship without the point with the lowest deer density despite nearly 10-fold variation in deer density.

is positively correlated with coyotes and negatively correlated with foxes (Fig. 4 *A* and *B*), which suggests a more important role for variation in the abundance of predators than deer. Lyme disease is notably rare in western New York, where fox are abundant, despite having among the highest deer abundance in the state. It is worth noting that the nonlinear relationship between foxes and Lyme in Fig. 4*B* closely resembles model predictions (Fig. 1). Previously compiled data on catch-per-unit effort of red fox by trappers and buck harvest density match the spatial distribution of carnivores and deer derived from harvest-independent data (34).

Temporal Correlations at Smaller Spatial Scales. Harvest-independent data from multiple regions of Wisconsin also suggest that Lyme incidence is more tightly linked to changes in predator abundance (coyote increase and fox decrease leading to lower overall predation rates) than deer abundance. In Wisconsin, where Lyme disease incidence has increased greatly over the past decade, landowner wildlife surveys indicate that a fox decline and coyote increase occurred throughout the state (Fig. 5), which corroborates the statewide trends from hunter harvest data (Fig. 3). Deer observations have been stable or declining over this period (Fig. 5), although due to high deer abundance, these surveys may be a less-sensitive index for deer. However, on a fine spatial scale, deer density in management units with the highest Lyme incidence did not change over the last decade, whereas Lyme disease cases increased 300% (Fig. S7). Deer densities increased at most sites from the early 1980s until the mid 1990s, which may have caused the initial emergence of Lyme disease in Wisconsin. However, in the past 15 y, deer abundance has slowed markedly, with one-fourth of units showing no increase and several others increasing only a small percentage (Fig. S7).

Discussion

The increase in deer during the early 20th century is thought to have allowed tick populations to grow and spread from small remnant populations, and this likely contributed significantly to the initial rise in Lyme disease cases (7). However, in recent decades, Lyme disease has continued to increase substantially in many places where deer populations have stabilized (Figs. 2 and 4). Further, we detected no relationship between the spatial distribution of Lyme disease and deer abundance in four states (Fig. 4). The weak correlations between changes in deer and Lyme disease incidence is consistent with a saturation in the probability that an adult tick finds a host (e.g., deer) with deer density (Fig. 2). Additionally, recent work from New York found no relationship between threefold variation in deer abundance and the density of infected nymphs over 13 y (12), and there was no response in nymph abundance to a recent deer culling program in New Jersey (13). Thus, though there is convincing evidence linking deer to high nymph densities from deer exclosure



D

Variable	R-2	ΔAICc	w
Coy+Fox	0.79	0.00	0.85
Deer +Coy+Fox	0.79	3.49	0.15
Fox	0.58	11.03	0.00
Deer+Fox	0.64	11.23	0.00
Deer+Coy	0.44	20.17	0.00
Deer	0.34	20.58	0.00
Coy	0.32	21.21	0.00

Variable	R-2	ΔAICc	w
Coy	0.73	0.00	0.37
Coy+Fox	0.75	0.11	0.35
Deer+Coy	0.74	1.55	0.17
Deer +Coy+Fox	0.76	2.60	0.10
Fox	0.47	17.94	0.00
Deer+Fox	0.47	20.59	0.00
Deer	0.07	33.19	0.00

Variable	R-2	ΔAICc	w
Deer+Coy	0.61	0.00	0.43
Coy	0.53	0.34	0.36
Deer+Coy+Fox	0.64	2.78	0.11
Coy+Fox	0.54	3.01	0.09
Fox	0.28	7.77	0.01
Deer+Fox	0.31	10.45	0.00
Deer	0.00	13.78	0.00

Variable	R-2	ΔAICc	w
Coy+Fox	0.83	0.00	0.39
Deer	0.76	1.14	0.22
Deer+Coy	0.81	1.77	0.16
Deer +Coy+Fox	0.87	2.00	0.14
Deer+Fox	0.79	3.22	0.08
Coy	0.66	6.01	0.02
Fox	0.47	12.07	0.00

Fig. 3. Temporal trends between Lyme cases and (*A*) deer harvest per license, the hunter harvest of (*B*) coyotes, and (*C*) foxes are consistent with the predation hypothesis. As predicted by the model, the relationship between foxes and Lyme is nonlinear (Lyme cases are on a log scale). (*D*) Statistical models were compared with AIC$_c$. All models with greater than 1% model weight did not have temporally autocorrelated residuals ($P > 0.05$ Box–Pierce test). Model selection in Pennsylvania underestimates the importance of foxes because we use only data since 1990, the first year that coyote data were collected (fox-only model is best if coyotes are excluded and the full fox and deer time series are analyzed).

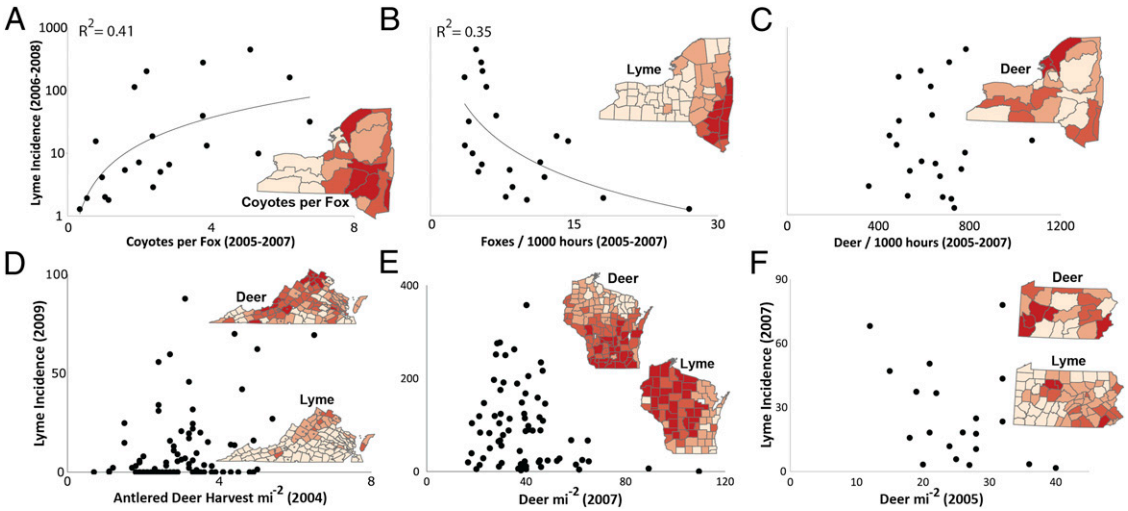


Fig. 4. Spatial relationships among deer, predators, and Lyme disease. (*A*) In New York, observation rates from the bow-hunter wildlife survey indicate that Lyme disease incidence (cases per 100,000) is positively correlated with coyotes, (*B*) negatively correlated with foxes, and (*C*) unrelated to deer. Coyote observations are scaled by foxes to highlight the transition in the predator community and its impact on Lyme disease. (*D*) Deer as estimated by the buck harvest density are positively (but weakly) correlated with Lyme disease incidence in Virginia counties (R^2 = 0.1, P = 0.001). (*E*) In contrast, deer density estimates (from sex-age-kill models) are negatively correlated with Lyme incidence in Wisconsin counties (R^2 = 0.06, P = 0.05, but driven by few data points—not significant when removed) and (*F*) negatively correlated in Pennsylvania deer management units (R^2 = 0.14, P = 0.09), where the unit with the lowest deer density has the second-highest Lyme incidence. (*Insets*) Darker red indicates more-abundant wildlife populations and higher Lyme incidence (in four classes: 0–10, 10–50, 50–100, and >100 cases per 100,000).

studies, and from the complete or near-complete deer removal on islands, linking deer abundance to ticks when deer are abundant has been less successful, particularly at mainland sites where there are many other potential reproductive hosts for *Ixodes* ticks and where most Lyme disease cases are contracted (reviewed in Table S1).

At the same time, over the past three decades there has been a regional red fox decline coincident with an expanding coyote population. Both spatial and temporal evidence across multiple states suggest that these changes in predator abundance are more closely linked with increases in Lyme disease than are changes in deer abundance. Our theoretical model suggested that changes in predation can in fact lead to the observed increases in Lyme risk, in that both the density and infection prevalence of nymphal ticks are sensitive to reduced predation (Fig. 1). Taken together with the empirical data on spatial and temporal patterns of Lyme incidence, deer, and predator abundance, these results suggest that the red fox declines may have resulted in increased Lyme disease risk due to the loss of predation as an ecosystem service. Detailed studies and experimental manipulation of predators could help elucidate whether controlling Lyme disease might be best accomplished by

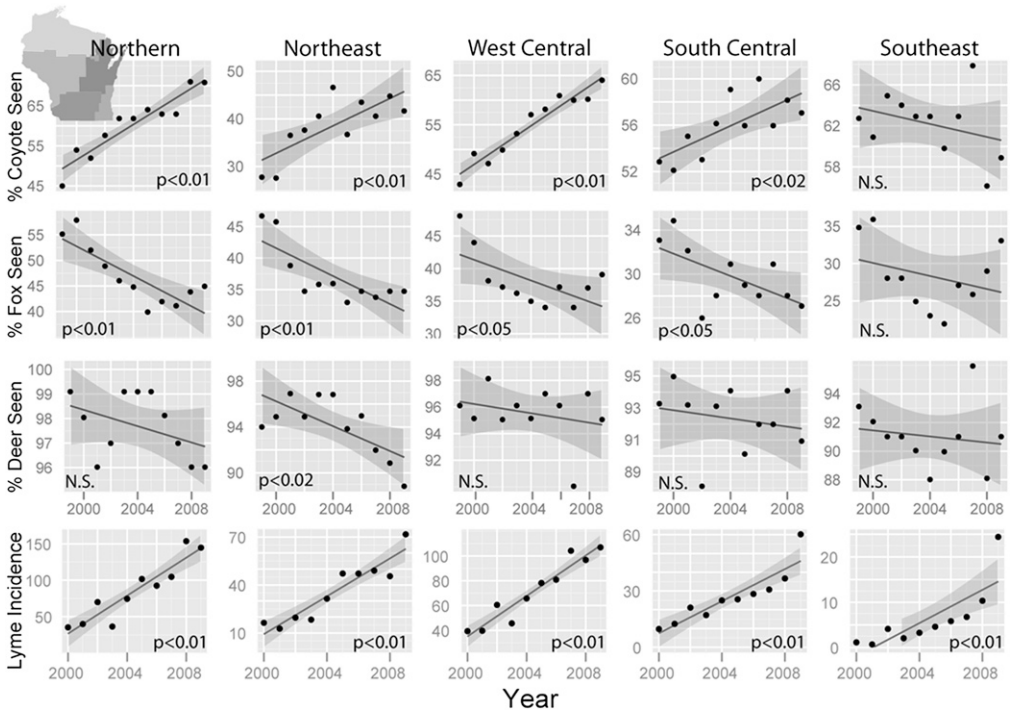


Fig. 5. The percent of surveyed rural landowners who saw coyotes, foxes, and deer in five geographic regions of Wisconsin from 1999 to 2009 according to the annual *Summer Wildlife Inquiry* run by the Department of Natural Resources. Lyme incidence in each region is the weighted average (by area) of county-level incidence.

a combination of predator manipulation and severe reductions in deer densities necessary to reduce tick abundance.

More broadly, these results suggest a need to explore the role of predation in the community ecology of other emerging zoonotic diseases, which overwhelmingly rely on hosts that occupy low trophic levels (1). Due to the widespread eradication of large carnivores (4), top predators in many terrestrial ecosystems are now medium-sized carnivores such as coyotes (5). These medium-sized carnivores can indirectly increase the abundance and diversity of low trophic-level species, such as rodents and songbirds, by suppressing populations of smaller carnivores such as foxes (20). Strong interactions among predators (35) that lead to cascading effects on prey have been documented for over 60 systems worldwide (21). As top predators are extirpated in some parts of the world, and recolonize in others, it will be important to understand the consequences for community composition and for the abundance of low trophic-level species in particular. Such restructuring of predator communities may have unintended consequences for human disease.

Methods

Host–Vector Disease Model. We use a vector-borne, susceptible-infected (36) modeling framework that describes the dynamics of ticks and small-mammal hosts, and includes parameters to account for the density of alternate hosts and deer. We group multiple species into a functional group of small-mammal hosts with density, N_m. The small-mammal host population growth rate, $G(N_m)$, is logistic with maximum intrinsic growth rate, r, and carrying capacity, K. The mortality rate, $M(N_m)$, follows a Holling type III functional response, which is characteristic of prey-switching generalist predation, with maximum predation rate, a, half-saturation parameter, c, and predator density, P (37–39). This functional response can exhibit alternative stable states in a small region of parameter space, but we stress that our results depend only on an S-shaped functional response, which is characteristic of switching or aggregating behavior in response to more-abundant prey (Fig. S3). An S-shaped functional response is also obtained with a type II functional response when predators respond numerically to increasing prey density (i.e., a combined numerical and functional response; *SI Text, Parameters and Derivations*).

The differential equation for the total host population is

$$\begin{aligned}\frac{dN_m}{dt} &= G(N_m) - M(N_m)\\ &= rN_m\left(1-\frac{N_m}{K}\right) - \frac{aPN_m{}^2}{c^2+N_m{}^2}\end{aligned} \quad [1]$$

The small-mammal host population consists of susceptible, S_m, and infected, I_m, classes. Susceptible hosts become infected with probability T_{mt} when bitten by an infected nymph, I_t. A fraction of tick bites occur on incompetent "dilution" hosts, F, so that these hosts divert blood meals away from small mammals but also increase total host abundance. The tick bite rate, $\beta(N_m + F)$, follows a type II functional response. Because each tick life stage requires a single blood meal, the functional response saturates at 1 as the abundance of hosts increases (i.e., all ticks can feed if there are infinite hosts). The half-saturation parameter, b_0, represents the density of small mammals where half of ticks would be expected to feed. Thus, the tick bite rate can be interpreted as the fraction of ticks that successfully feed given the total population of hosts, $N_m + F$.

The differential equations for susceptible and infected small-mammal hosts are

$$\begin{aligned}\frac{dS_m}{dt} &= G(N_m) - T_{mt}I_t\frac{S_m}{N_m+F}\beta(N_m+F) - \frac{S_m}{N_m}M(N_m)\\ &= rN_m\left(1-\frac{N_m}{K}\right) - \frac{T_{mt}I_tS_m}{b_0+N_m+F} - S_m\frac{aPN_m}{c^2+N_m{}^2}\end{aligned} \quad [2]$$

and

$$\begin{aligned}\frac{dI_m}{dt} &= T_{mt}I_t\frac{S_m}{N_m+F}\beta(N_m+F) - \frac{I_m}{N_m}M(N_m)\\ &= \frac{T_{mt}I_tS_m}{b_0+N_m+F} - I_m\frac{aPN_m}{c^2+N_m{}^2},\end{aligned} \quad [3]$$

where susceptible hosts are created by birth and lost by infection or predation, and infected hosts are created by infection and lost by predation. We assume no increase in predation risk associated with being infected. Therefore, the relative abundance of the susceptible and infected classes determines the relative predation rate of each class.

Larval ticks, S_t, which are all susceptible, have birth rate ν and per-capita death rate μ_l. We use a constant birth rate that can be varied independently, because it is unknown how vertebrate biomass and community composition influence the tick birth rate. Any larval tick that successfully feeds on either a small-mammal host or dilution host leaves this class so that the differential equation for larva is

$$\begin{aligned}\frac{dS_t}{dt} &= \nu - \beta(N_m+F)S_t - \mu_lS_t\\ &= \nu - \frac{N_m+F}{b_0+N_m+F}S_t - \mu_lS_t\end{aligned} \quad [4]$$

Nymphs die at rate, μ_n, and also leave their class by successfully feeding. Nymphs become infected when larva successfully contract *Borrelia* from an infected host (i.e., this depends on the frequency of infected hosts) with probability T_{tm}. Thus, the differential equation for infected nymphs, I_t, is

$$\begin{aligned}\frac{dI_t}{dt} &= \frac{I_m}{N_m+F}\beta(N_m+F)T_{tm}S_t - \beta(N_m+F)I_t - \mu_nI_t\\ &= \frac{T_{tm}I_mS_t}{b_0+N_m+F} - \frac{N_m+F}{b_0+N_m+F}I_t - \mu_nI_t\end{aligned} \quad [5]$$

Uninfected nymphs, J_t, can be uninfected because a larval tick fed on a susceptible or dilution host or because a larval tick fed on an infected host but did not contract *Borrelia*. The equation for uninfected nymphs thus has an additional term to account for the probability that feeding on an infected host did not cause infection, but can be simplified to

$$\begin{aligned}\frac{dJ_t}{dt} &= \frac{S_m+F}{N_m+F}\beta(N_m+F)S_t + (1-T_{tm})\frac{I_m}{N_m+F}\beta(N_m+F)S_t - \beta(N_m+F)J_t - \mu_nJ_t\\ &= \frac{S_m+F+I_m(1-T_{tm})}{b_0+N_m+F}S_t - \frac{N_m+F}{b_0+N_m+F}J_t - \mu_nJ_t\end{aligned} \quad [6]$$

We solved for the steady states as a function of the steady-state small-mammal density $\overline{N_m}$. The closed-form solutions, which are presented in *SI Text*, *Steady-State Solutions*, explicitly demonstrate the strength of the known multiple drivers of Lyme disease.

Data Analysis. ***Spatial Analysis.*** New York enlists bow hunters to survey wildlife from tree stands. We averaged the observation rates of each species from 2005 to 2007 in each management unit to compare with Lyme disease incidence from 2006 to 2008. Lyme disease incidence is recorded at a county scale, so we allocated incidence to management units as a weighted average based on the relative area of each county in each wildlife management unit groupings.

In Virginia we used buck harvest per square mile reported in the Virginia deer management plan (14) as a proxy for deer density. Both the harvest data and Lyme disease data are on the county spatial scale. Wisconsin and Pennsylvania produce deer density estimates using the sex-age-kill model (40), which estimates density in management units using data on harvest, age, and sex structure, and fawn-to-doe ratios. Lyme disease incidence is recorded at a county scale. In Pennsylvania, wildlife management units are larger than counties, so we allocated Lyme incidence to management units as above. In Wisconsin, wildlife management units are smaller than counties, so we allocated deer density to counties based on the relative area of each wildlife management unit in each county. For Wisconsin, we additionally analyze changes in deer densities since 1981 in 25 randomly chosen management units intersecting counties with the highest incidence (Fig. S7).

Time-Series Methods. We use harvest-based proxies for white-tailed deer, coyote, and red fox abundance. To compare the populations of coyotes and foxes with annual Lyme disease cases, we use hunter harvest as a proxy for abundance. Any longitudinal changes in hunting effort are unlikely to be biased in favor of one of these species over another, suggesting that a decline in fox harvests and an increase in coyote harvests represent real population changes. Data on trapper harvest is more widely available but is not reliable because it is influenced by exogenous factors such as pelt prices and changes in trapping regulations designed to prevent incidental catch of high-value or endangered species. Many states, including the four we consider, have liberal

coyote- and fox-hunting regulations, including very long or continuous seasons and no bag limits. We therefore conduct our analysis on the subset of large states from which we could obtain hunter harvest time-series data: Wisconsin, Minnesota, Pennsylvania, and Virginia (New York does not collect hunter harvest data); the exception is Pennsylvania, for which we have only total harvest (hunter + trapper) data, which are not as reliable an index for foxes but are likely representative of the population expansion of coyotes as they colonized the state.

As a proxy for deer abundance, we use antlered deer harvest, which is routinely used by wildlife management agencies to monitor trends in deer abundance. Antlered deer harvest is a robust estimate of the statewide deer population due to the large number of hunters that sample the deer population with success rates dependent on the abundance of deer. We scale antlered deer harvest by hunting license sales to capture changes in hunter participation (Fig. S8). Analysis of the hunter functional response from 10 datasets supports a type I functional response (41), which suggests that hunter success rates are expected to increase linearly, rather than simply monotonically, with deer density. Additionally, hunter success rates (Fig. S8; <25% in MN, PA, and WI, and <40% in VA) suggest increases in deer abundance would be represented by increased harvests, because hunters are not saturated with deer. Longitudinal hunter harvest data has been shown to correlate well with trends in deer density and has been used in the literature not only for crude population trends but also for more sophisticated time-series analysis (42–45).

Combining the available wildlife harvest time series, we evaluate the relative support of the predation and deer hypotheses. We additionally analyze antlered deer harvest data not corrected for license sales (Table S3) and harvests of deer, coyotes, and foxes all scaled by hunting license sales (Table S4) to ensure that our results are statistically robust to changes in hunter participation. We use deer (big game) license sales throughout because small-game hunters focus on a variety of species, and individuals may only report that they are coyote or fox hunters if they opportunistically kill one of these species incidental to other activities (46). The strength of each candidate model was evaluated using corrected Akaike Information Criterion (33, 47).

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Supporting Information

Levi et al. 10.1073/pnas.1204536109

SI Text

Parameters and Derivations. Although our analysis is qualitative and we produce closed-form solutions, we nevertheless find plausible parameter values to see if reasonable levels of predation can influence Lyme disease.

M(N_m). We model predation with a type III functional response, but our results can also be obtained by combining a type II functional response with a numerical response.

For example, if we instead model predation with a type II functional response

$$M(N_m) = \frac{aPN_m}{b + N_m},$$

but also note that predator density, P, should increase and eventually saturate with prey density, then we obtain

$$P = \frac{\alpha N_m}{\beta + N_m}.$$

Combining these two equations yields

$$M(N_m) = \frac{a\alpha P{N_m}^2}{\left(b\beta + 2b\beta N_m + {N_m}^2\right)},$$

which is simply a more general form of the type III functional response that has the same sigmoid shape and qualitative properties (this can be understood intuitively by recognizing that the squared-term dominates the expression in the denominator).

F. We estimate the density of noncompetent dilution hosts following LoGiudice et al. (1). We sum the density estimates of dilution hosts to obtain $F \sim 4{,}120$. We ignore the fact that dilution hosts are somewhat reservoir competent because of evidence that 80–90% of ticks are infected by a few small-mammal species (2). We thus consider a class of dilution hosts rather than considering the variability among hosts. The nonzero infectiousness of dilution hosts can prevent complete *Borrelia* extinction even when small mammals are rare, but this does not impact the qualitative relationship between predation and Lyme disease risk.

b_0. We use tick densities estimated with mark-recapture techniques (3) to estimate the half-saturation parameter of the tick functional response, b_0.

Daniels et al. (3) found larval densities of ~11.5 million km^{-2} and nymph densities of 1.2 million km^{-2}. The nymph population was ~10% of the larva population. We reason that at least 10% of larva successfully fed, allowing us to estimate b_0.

$$\beta(N_m + F) = \frac{N_m + F}{b_0 + N_m + F} = 0.10$$

Following LoGiudice et al. (1), the reservoir-competent small-mammal density (N_m) ranges from 5,000 to 200,000 km^{-2}. To estimate b_0, we use an intermediate (nonresource pulse) value of 10,000 km^{-2}. Substituting in F and solving for b_0, a reasonable estimate of b_0 is ~80,000, meaning that half of ticks are expected feed if the total host population ($N_m + F$) is 80,000 km^{-2}.

aP and c. One classic study (4) quantified the impact of generalist predators on two species of small mammals over 40 km^2 in southern Sweden. This study found that generalist predators were responsible for far more predation on voles and wood mice than specialist predators. We use predation rate data from this study to fit the parameters aP and c. A precise estimate of aP is not necessary because we explore the steady states of the differential equations as a function of a variable maximum predation rate, aP (Fig. 1 *B* and *C* and Fig. S2). We thus only need a reasonable half-saturation parameter. Although this study comes from Sweden, the predator community is similar to that of the northeastern United States, with red foxes being the dominant predator of small mammals.

We fit the per capita predation rate $\frac{aP \cdot N}{c^2 + N^2}$ (a type III functional response divided by N) to the data with and without two potential outliers. These data come from monthly predation rates that should show considerably more variation than annual predation rates because annual measures smooth over seasonal and stochastic variability. The best estimate of aP is 241,391 per 40 km^2, which is equivalent to 6,034 annual kills per km^2.

Steady-State Solutions. Eqs. **1–6** can be solved for steady-state solutions that depend only on the steady-state small-mammal density, $\overline{N_m}$. The steady states are given by

$$\overline{S_t} = \frac{\nu\left(b_0 + \overline{N_m} + F\right)}{\overline{N_m} + F + \mu_l\left(b_0 + \overline{N_m} + F\right)}, \quad \textbf{[S1]}$$

$$\overline{I_t} = \overline{N_m}\left(b_0 + \overline{N_m} + F\right)\left(\frac{T_{tm}\nu}{\left(\overline{N_m} + F + \mu_l\left(b_0 + \overline{N_m} + F\right)\right)\left(\overline{N_m} + F + \mu_n\left(b_0 + \overline{N_m} + F\right)\right)} - \frac{aP}{T_{mt}\left(c^2 + \overline{N_m}^2\right)}\right), \quad \textbf{[S2]}$$

$$\overline{J_t} = \overline{N_m}\left(b_0 + \overline{N_m} + F\right)\left(\frac{\nu\left(1 - T_{tm} + \frac{F}{\overline{N_m}}\right)}{\left(\overline{N_m} + F + \mu_l\left(b_0 + \overline{N_m} + F\right)\right)\left(\overline{N_m} + F + \mu_n\left(b_0 + \overline{N_m} + F\right)\right)} + \frac{aP}{T_{mt}\left(c^2 + \overline{N_m}^2\right)}\right), \quad \textbf{[S3]}$$

$$\overline{S_m} = \frac{\left(\overline{N_m} + F + \mu_l\left(b_0 + \overline{N_m} + F\right)\right)\left(\overline{N_m} + F + \mu_n\left(b_0 + \overline{N_m} + F\right)\right)aP\overline{N_m}}{\left(c^2 + \overline{N_m}^2\right)T_{mt}T_{tm}\nu}, \quad \textbf{[S4]}$$

and

$$\overline{I_m} = \overline{N_m}\left[1 - \frac{aP\left(\overline{N_m} + F + \mu_l\left(b_0 + \overline{N_m}\right)\right)\left(\overline{N_m} + F + \mu_n\left(b_0 + \overline{N_m}\right)\right)}{\left(c^2 + \overline{N_m}^2\right)T_{mt}T_{tm}\nu}\right]. \quad \textbf{[S5]}$$

All quantities are restricted to be nonnegative, and the abundance of any one class of either hosts or ticks is restricted to be less than the total abundance of hosts or ticks.

The infection prevalence of hosts (HIP) and nymphs (NIP) can be derived from the steady states

$$HIP = 1 - \frac{aP\left(\overline{N_m} + F + \mu_l\left(b_0 + \overline{N_m} + F\right)\right)\left(\overline{N_m} + F + \mu_n\left(b_0 + \overline{N_m} + F\right)\right)}{\left(c^2 + \overline{N_m}^2\right)T_{ml}T_{tm}\nu} \quad \textbf{[S6]}$$

and

$$NIP = \frac{\overline{I_t}}{\overline{I_t} + \overline{J_t}} = T_{tm}\frac{\overline{N_m}}{\overline{N_m} + F}\left(1 - \frac{aP\left(\overline{N_m} + F + \mu_l\left(b_0 + \overline{N_m} + F\right)\right)\left(\overline{N_m} + F + \mu_n\left(b_0 + \overline{N_m} + F\right)\right)}{\left(c^2 + \overline{N_m}^2\right)T_{ml}T_{tm}\nu}\right). \quad \textbf{[S7]}$$

Combining Eqs. **S6** and **S7**, we recover the intuitive result that relates the nymphal infection prevalence to the infection prevalence of hosts,

$$NIP = \frac{\overline{I_t}}{\overline{I_t} + \overline{J_t}} = T_{tm}\frac{\overline{N_m}}{\overline{N_m} + F}HIP. \quad \textbf{[S8]}$$

The fraction of hosts that are reservoir competent determines the relationship between host infection prevalence and nymphal infection prevalence.

The steady-state solutions provide a framework for understanding the role of the known multiple drivers of Lyme disease risk. For example, the steady-state densities of susceptible and infected hosts and ticks can be assessed as a function of predation, aP, relative to the density of the tick birth rate, ν (Fig. S4), or dilution hosts, F (Fig. S5). Increasing both predation and the density of dilution hosts reduces Lyme disease risk as long as the tick birth rate, ν, remains constant. However, by reducing the density or activity level of small mammals, predation likely reduces the tick birth rate if a larger fraction of immature ticks cannot find the hosts necessary to transition into reproductively mature adult ticks. In contrast, increased density of dilution hosts takes blood meals away from disease-amplifying small mammals, but by supplying blood meals, dilution hosts can increase the tick birth rate if hosts for immature ticks are limiting.

Thus, predation is always expected to reduce the density of infected nymphs, but the magnitude of this reduction in Lyme disease risk depends on how much predation of small mammals reduces the tick birth rate (Fig. S4, black arrows). In contrast, increasing the density of dilution hosts is expected to lower nymph infection prevalence but may have minimal impact on the density of infected nymphs (Fig. S6, black arrows) (5).

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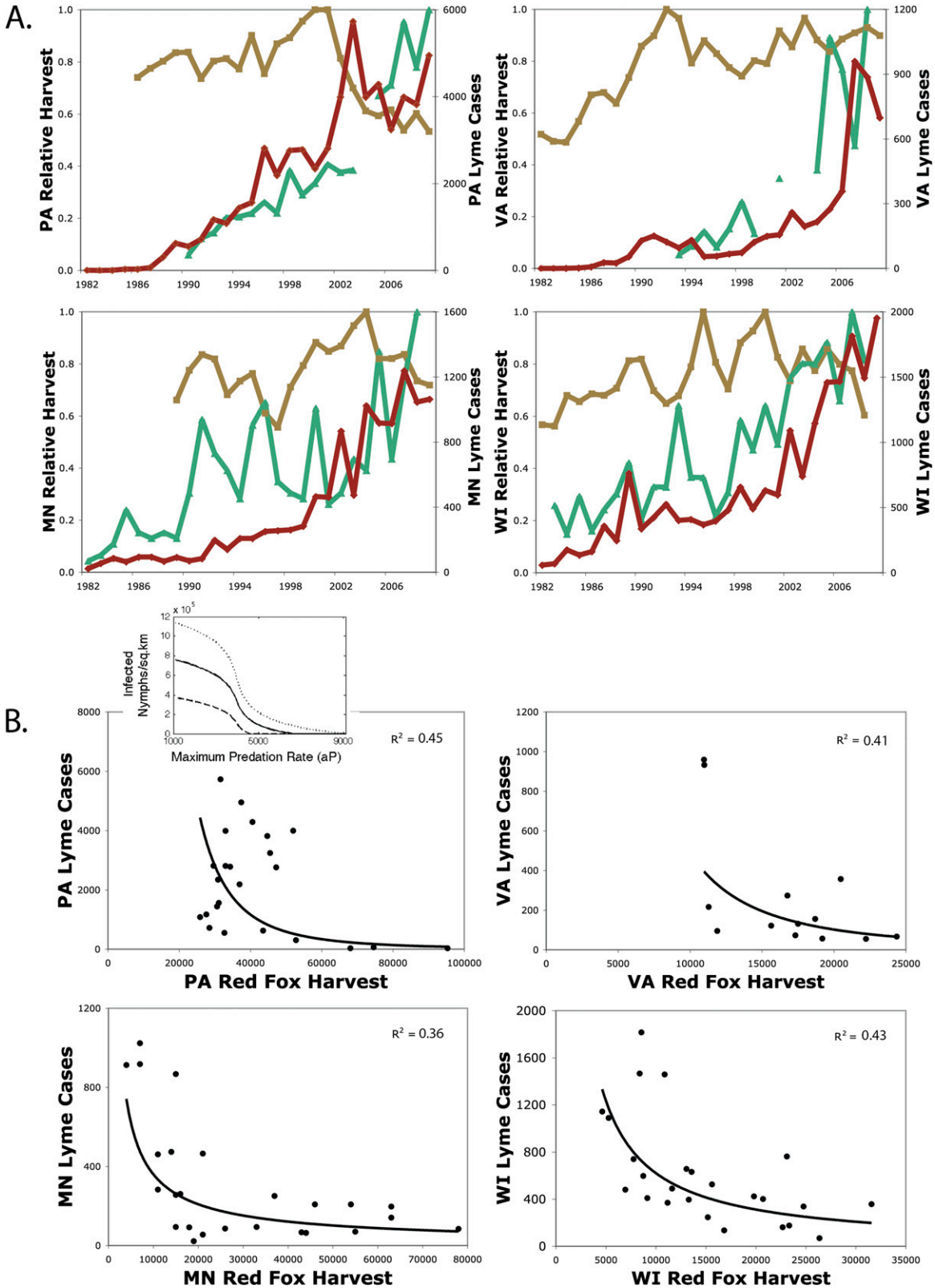

Fig. S1. (*A*) Annual Lyme disease cases (red diamonds) and the hunter harvests of coyotes (green diamonds) and antlered deer (brown squares) scaled to the fraction of maximum harvest in Pennsylvania (PA), Virginia (VA), Minnesota (MN), and Wisconsin (WI) (PA data includes trapper harvest). The maximum coyote harvest exceeds 20,000 in PA and VA, and 40,000 in WI and MN, and the maximum buck harvest exceeds 100,000 in all four states, and 200,000 in PA. (*B*) Lyme disease incidence vs. red fox abundance, fit with a power function, follows the relationship predicted by our theoretical model. (*Inset*) Steady-state density of infected nymphs as a function of the predation rate for low, medium, and high tick birth rates.

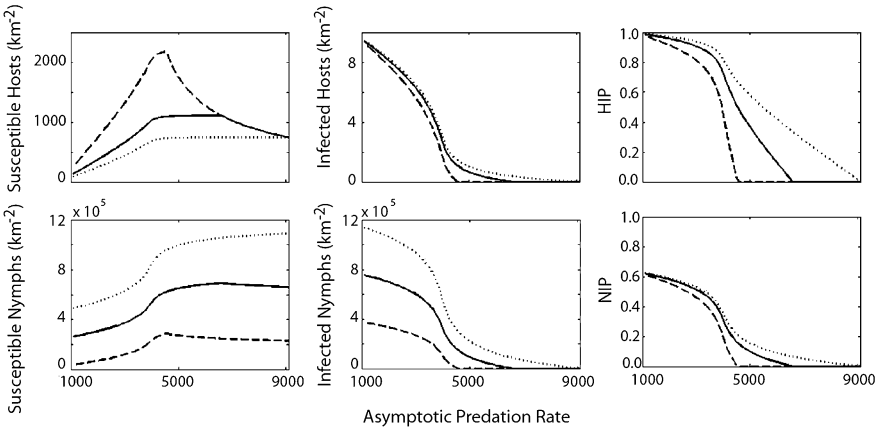

Fig. S2. Steady states of the different equations, and steady-state host and nymph infection prevalence (HIP and NIP) as a function of the asymptotic maximum predation rate. The dotted, solid, and dashed lines correspond to ν = 1.5, 1, 0.5 million larva born per km^2 per year.

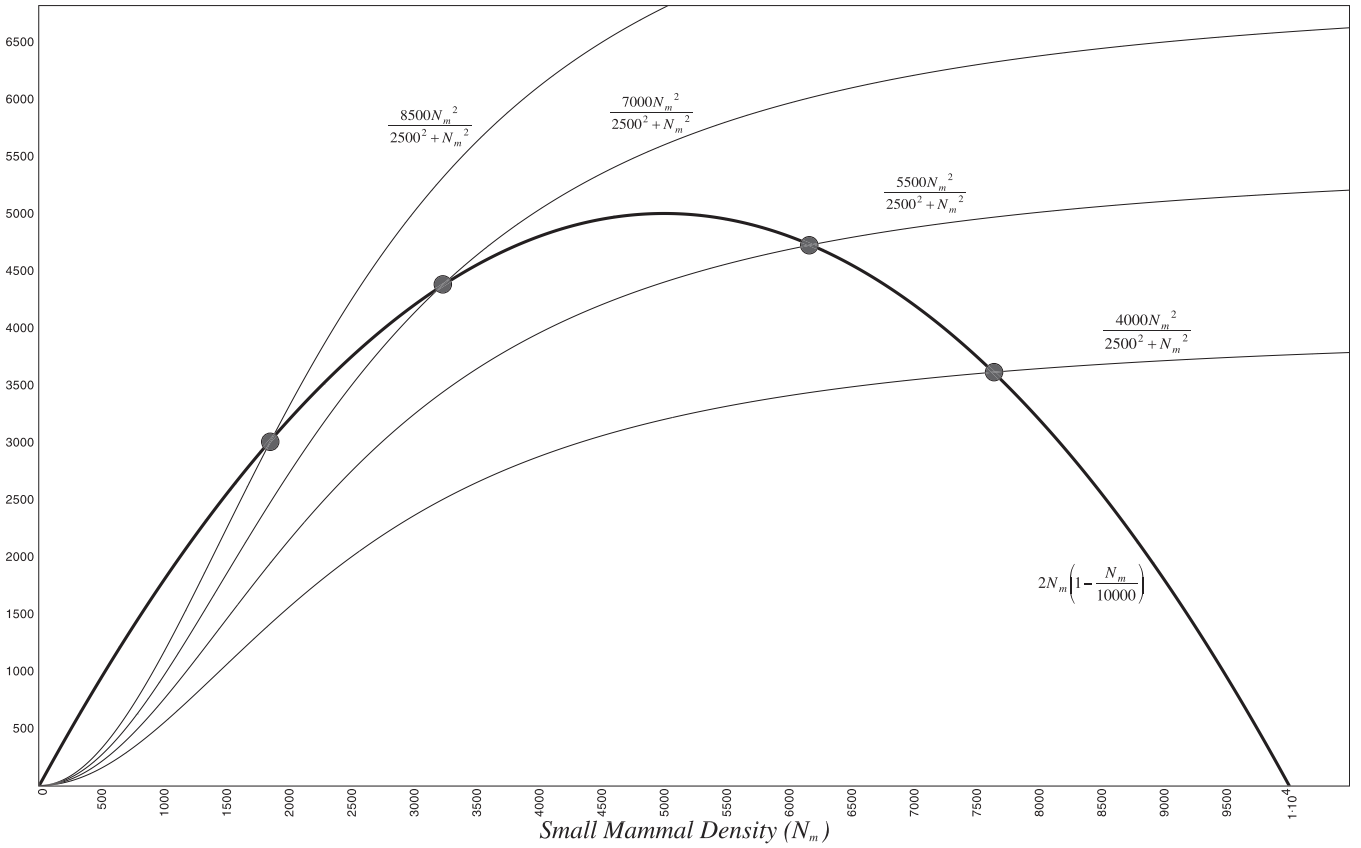

Fig. S3. The logistic growth and type III functional responses that are used in our model. There is a smooth transition to decreasing steady-state prey density as predator abundance increases. The low predation rate at low prey densities stabilizes the dynamics and prevents population extinction without requiring a model of the numerical response of predator populations.

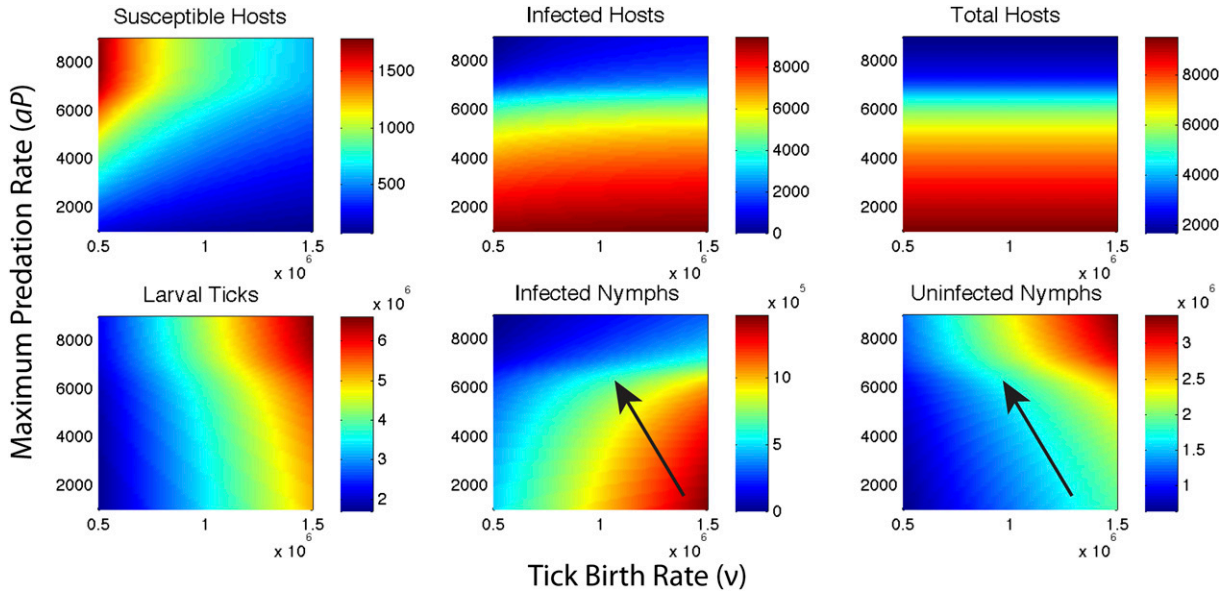

Fig. S4. Color plot of the steady states of the different equations as a function of the tick birth rate, ν, and the asymptotic maximum predation rate, aP. Black arrows signify the qualitative impact of predation on tick density when expected changes to the tick birth rate are accounted for. The density of infected nymphs is expected to decline substantially with the combined effect of predators on aP and ν.

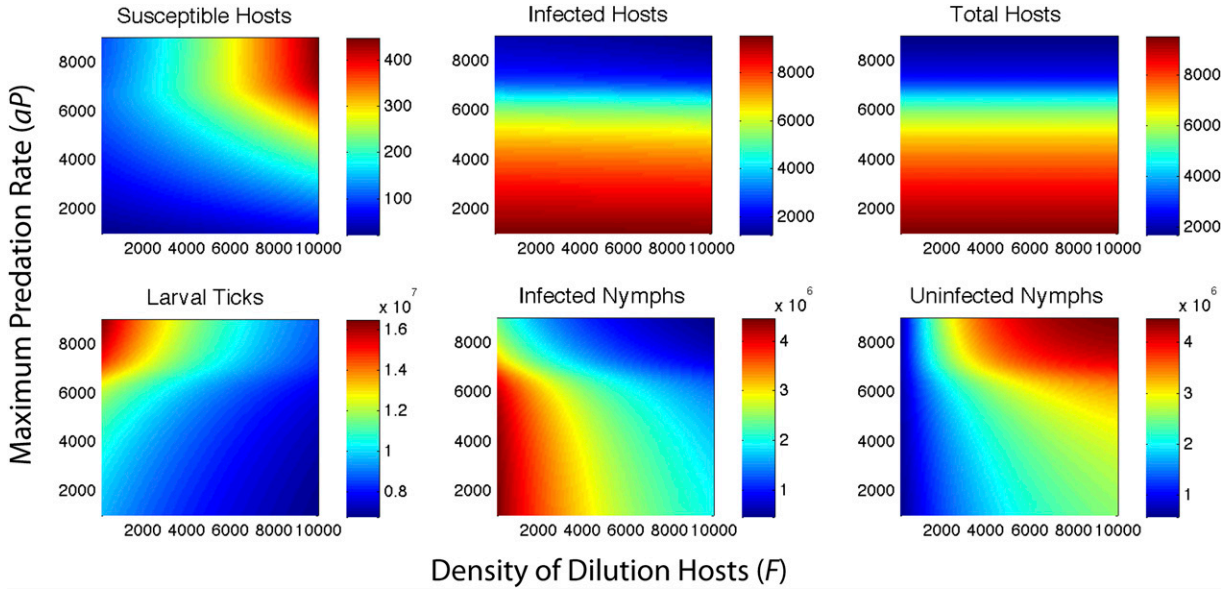

Fig. S5. Color plot of the steady states of the different equations as a function of F, dilution host density (km^2), and aP, the asymptotic maximum predation rate.

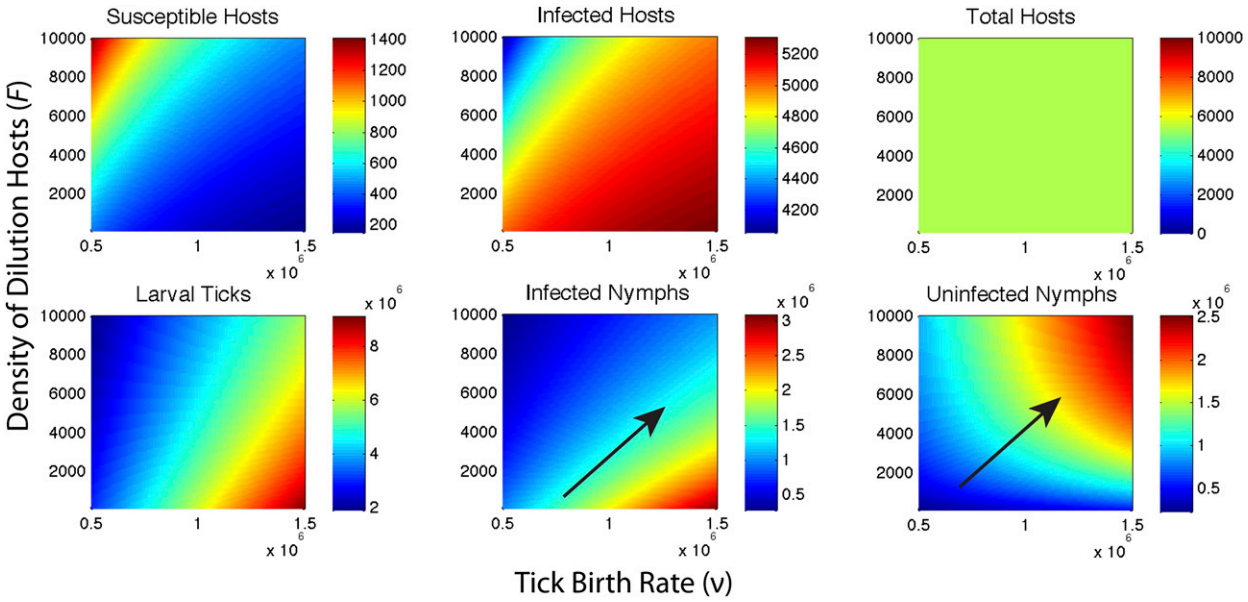

Fig. S6. Color plot of the steady states of the different equations as a function of F, dilution host density (km^2), and ν, the tick birth rate. Black arrows signify the qualitative impact of dilution hosts on tick density when expected changes to the tick birth rate are accounted for. The density of infected nymphs can remain constant, and the density of uninfected nymphs increases.

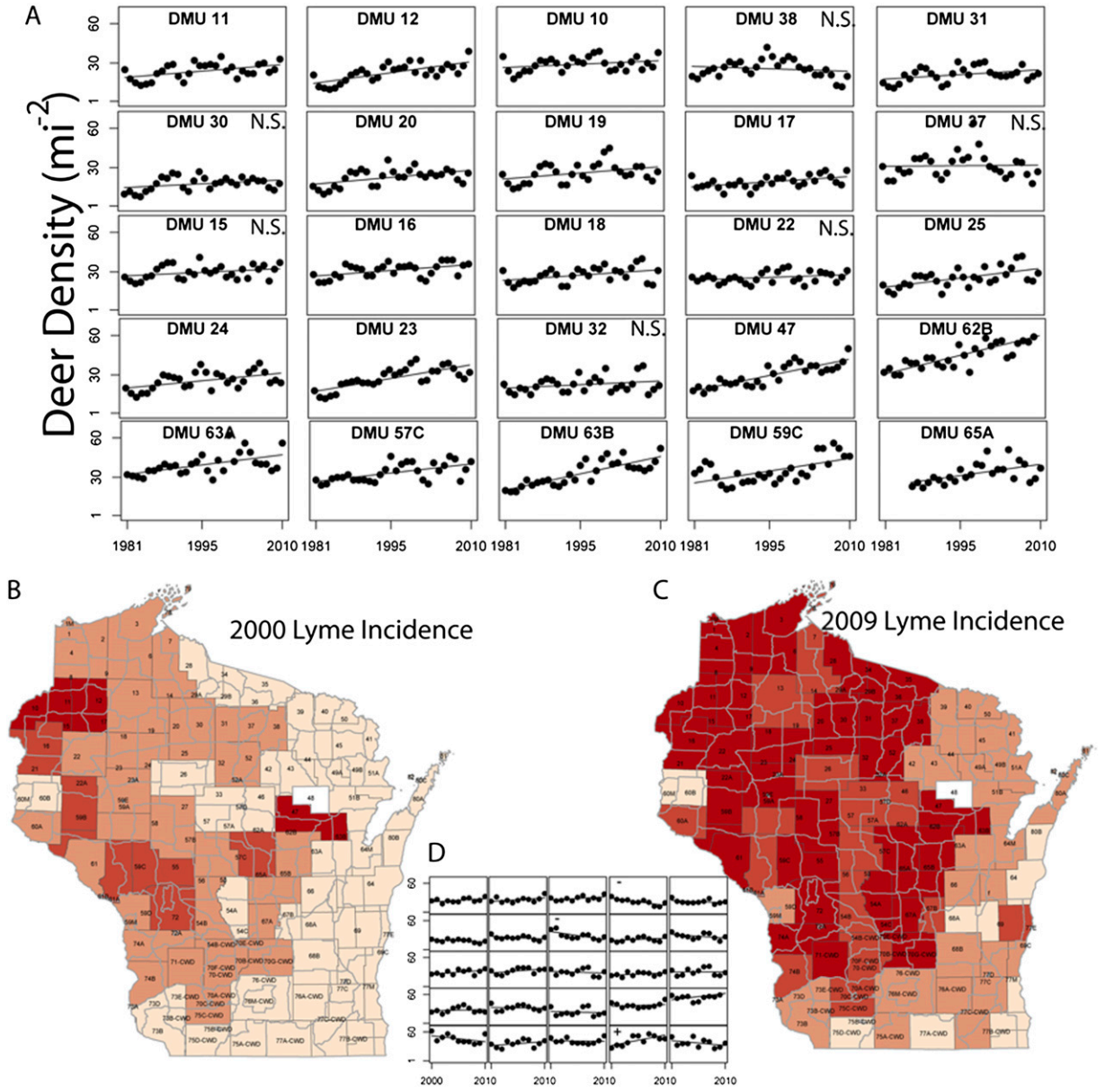

Fig. S7. (*A*) Deer density in a sample of 25 management units where Lyme disease incidence is highest in Wisconsin. Deer density has increased substantially in some cases, but deer have been abundant since the early 1980s, and in many units deer populations have been stable or only slightly increasing despite a great increase in incidence since 2000. The six units that have shown no significant increase since 1981 are labeled N.S. (*B* and *C*) Shades of red indicate Lyme incidence from 0 to 10, 10 to 50, 50 to 100, and >100 cases per 100,000. (*D*) In the same management units, there has been no change in deer densities over the past decade in 22 of the 25 units, a decrease in two, and an increase in one. Significant changes are labeled (+) and (−).

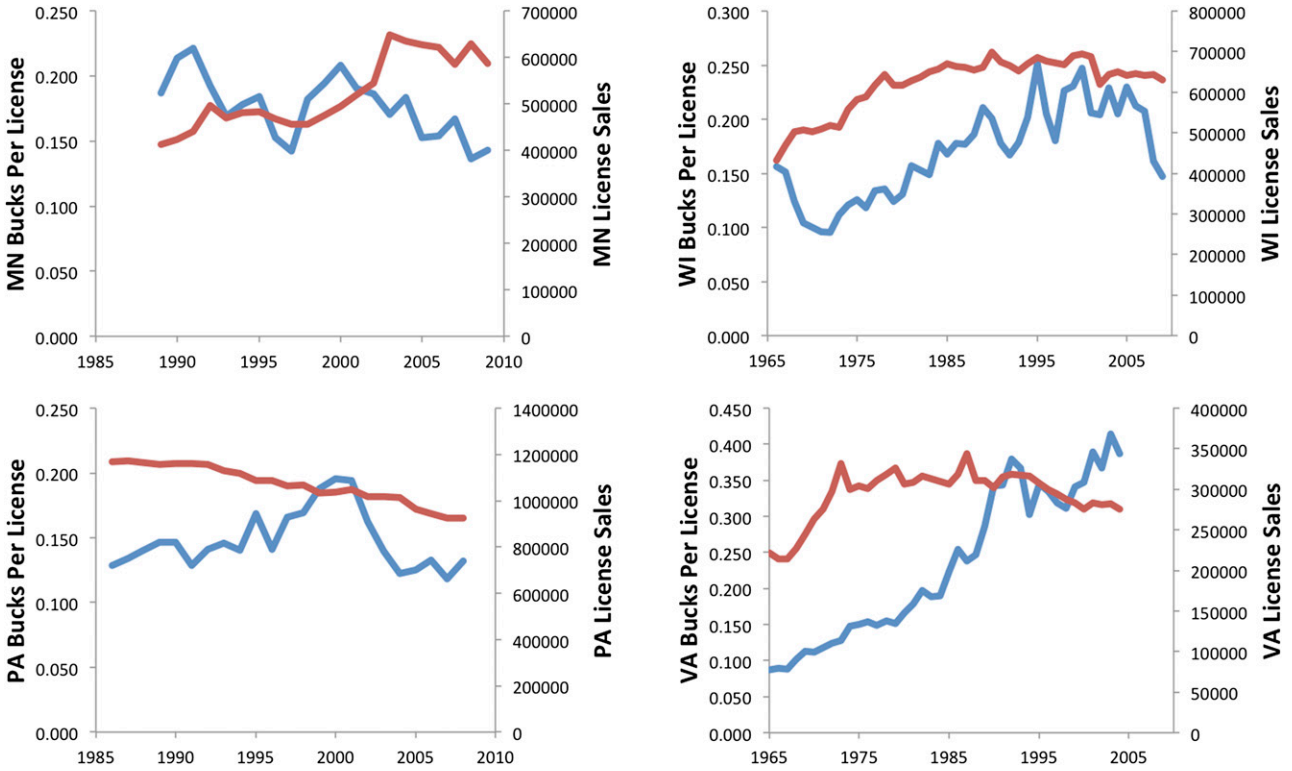

Fig. S8. Buck harvest per license (blue) and license sales (red) in MN, WI, PA, and VA. We have included data farther into the past from VA and WI so that the long period of deer population increase (particularly in VA) can be seen in the harvest data.

Table S1. Summary of studies measuring or manipulating deer populations and the corresponding response of ticks

Location	Island or mainland	Summary	Ref.
Montgomery County, MD	Mainland	Very low tick density found despite hyperabundant deer	1
Westchester County, NY	Mainland	After 25 y of deer exclosures, fewer nymphs inside most exclosures, but more nymphs inside in one site. No change in nymphal infection prevalence.	2
Westchester County, NY	Mainland	Differences in tick density inside and outside exclosure decline with successive tick developmental stages.	3
Ipswich, MA	Island	A 40% harvest rate of deer reduced population by 75% on an island. Larva per mouse falls substantially, and nymphs per mouse falls somewhat. Additionally, tick burdens on deer increase as deer density decreases.	4 (data presented in Fig. S1)
Long Island, NY	Island	*Ixodes scapularis* nymphs present at sites without deer but at low abundance.	5
Galway, Ireland	Mainland	Ticks much more abundant outside exclosure fence.	6
Sweden	Island	*Borrelia* and *Ixodes* ticks are both maintained in the absence of deer by hare populations.	7
Somerset County, NJ	Mainland	Deer culling by 47% produced no effect on tick abundance.	8
Monmouth County, NJ	Mainland	No relationship between ticks and deer pellet counts or browse damage.	9
Helsinki, Finland	Mainland	Ticks and *Borrelia* present without deer or any other ungulates.	10
Coastal Maine	Mainland	Deer pellet group and tick abundance are correlated.	11
Dutchess County, NY	Mainland	No relationship between deer and tick nymphs, but a strong relationship between ticks and rodents.	12
Italian Alps	Mainland	Small deer exclosure amplifies nymph intensity on rodents and increases infection prevalence but no change in larval intensity.	13
Various sites in Maine	Mainland	Adult tick abundance and deer pellet groups are positively correlated.	14
Monhegan Island, ME	Island	Complete removal of deer from small island with no other medium or large vertebrate hosts greatly reduced tick abundance.	15
Lyme, CT	Mainland	Deer exclosures greatly reduce larval and nymphal tick abundance. Adult tick results are mixed.	16
Bridgeport, CT, and Groton, CT	Mainland	Tick densities are reduced substantially by severe reduction in deer densities, but the effect saturates (Fig. S6).	17 (data presented in Fig. S2)
Mendocino, CA	Mainland	Nymphal density higher with deer at one site but not at another.	18
Great Island, MA	Island	A 70% reduction in deer did not reduce larval ticks per mouse the following year.	19
Great Island, MA	Island	On 13 islands, larval ticks are significantly correlated with deer but nymphs are not.	20
Great Island, MA	Island	Great reduction in larva and mild reduction in nymphs after complete removal of deer from island.	21

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Table S2. List of parameters and variables

	Interpretation	Value
Parameters		
μ_l, μ_n	Mortality rate of larva and nymphs	0.2
F	Density of dilution hosts	4,120
b_0	Half-saturation parameter of tick functional response	80,000
aP	Asymptotic number of hosts killed annually by predators with population, P	1,000–9,000
c	Mouse population where the predation rate reaches half of the maximum	2,500
T_{mt}	Probability that an infected tick biting a susceptible host transmits *Borrelia*	0.9
T_{tm}	Probability that an infected host bitten by a susceptible tick transmits *Borrelia*	0.9
r	Maximum intrinsic growth rate of hosts	2
K	Carrying capacity of hosts	10,000
ν	Birth rate of larval ticks	500,000, 1 million, 1.5 million
Variables		
S_m	Density of susceptible small mammals	
I_m	Density of infected small mammals	
N_m	Total density of small mammals	
S_t	Density of larval ticks, which are all susceptible	
I_t	Density of infected nymphal ticks	
J_t	Density of susceptible nymphal ticks	

Table S3. Model comparisons of three hunter-harvest predictors in explaining the number of annual Lyme disease cases (log transformed) in four states

State	Variable	R^2	AICc	ΔAICc	n	Model weight
MN	Deer + coyote + fox	0.85	30.21	0.00	21	0.89
	Coyote + fox	0.79	34.43	4.22	21	0.11
	Deer + fox	0.64	45.23	15.02	21	0.00
	Fox	0.58	45.45	15.25	21	0.00
	Deer + coyote	0.45	54.14	23.93	21	0.00
	Coyote	0.32	55.64	25.43	21	0.00
	Deer	0.13	60.69	30.48	21	0.00
WI	Coyote	0.73	39.66	0.00	27	0.37
	Coyote + fox	0.75	39.77	0.11	27	0.35
	Deer + coyote	0.74	41.13	1.47	27	0.18
	Deer + coyote + fox	0.76	42.21	2.55	27	0.10
	Fox	0.47	57.60	17.94	27	0.00
	Deer + fox	0.47	60.37	20.71	27	0.00
	Deer	0.02	74.04	34.37	27	0.00
PA	Coyote	0.53	28.62	0.00	18	0.51
	Deer + coyote	0.58	29.89	1.27	18	0.27
	Coyote + fox	0.54	31.30	2.68	18	0.13
	Deer + coyote + fox	0.60	32.71	4.09	18	0.07
	Fox	0.28	36.05	7.43	18	0.01
	Deer + fox	0.29	39.37	10.75	18	0.00
	Deer	0.09	40.31	11.69	18	0.00
VA	Coyote + fox	0.83	27.05	0.00	14	0.86
	Deer + coyote + fox	0.84	31.85	4.80	14	0.08
	Coyote	0.66	33.06	6.01	14	0.04
	Deer + coyote	0.72	34.57	7.53	14	0.02
	Fox	0.47	39.12	12.07	14	0.00
	Deer + fox	0.50	42.38	15.33	14	0.00
	Deer	0.25	44.02	16.97	14	0.00

Harvests are not scaled by license sales. MN, Minnesota; PA, Pennsylvania; VA, Virginia; WI, Wisconsin.

Table S4. Model comparisons of three predictors in explaining the number of annual Lyme disease cases (log transformed) in four states with deer, coyote, and fox scaled by big-game hunting license sales

State	Variable	R^2	AICc	ΔAICc	*n*	Model weight
MN	Coyote + fox	0.79	34.40	0.00	21	0.83
	Deer + coyote + fox	0.79	37.86	3.46	21	0.15
	Deer + fox	0.67	43.50	9.10	21	0.01
	Fox	0.63	42.67	8.27	21	0.01
	Deer	0.34	55.00	20.61	21	0.00
	Deer + coyote	0.36	57.36	22.96	21	0.00
	Coyote	0.12	61.05	26.65	21	0.00
WI	Coyote + fox	0.76	39.32	0.00	27	0.40
	Coyote	0.72	39.88	0.56	27	0.30
	Deer + coyote	0.74	40.93	1.61	27	0.18
	Deer + coyote + fox	0.76	41.62	2.30	27	0.13
	Fox	0.46	57.99	18.67	27	0.00
	Deer + fox	0.46	60.69	21.36	27	0.00
	Deer	0.07	72.85	33.53	27	0.00
PA	Deer + coyote	0.59	29.36	0.00	18	0.42
	Coyote	0.50	29.74	0.38	18	0.35
	Deer + coyote + fox	0.60	32.69	3.33	18	0.08
	Coyote + fox	0.50	32.93	3.57	18	0.07
	Fox	0.38	33.42	4.06	18	0.06
	Deer + fox	0.42	35.48	6.12	18	0.02
	Deer	0.00	42.07	12.71	18	0.00
VA	Coyote + fox	0.83	27.56	0.00	14	0.32
	Deer	0.76	28.19	0.63	14	0.24
	Deer + coyote	0.82	28.37	0.81	14	0.21
	Deer + coyote + fox	0.86	29.24	1.68	14	0.14
	Deer + fox	0.78	31.18	3.62	14	0.05
	Coyote	0.69	31.94	4.38	14	0.04
	Fox	0.29	43.23	15.67	14	0.00

MN, Minnesota; PA, Pennsylvania; VA, Virginia; WI, Wisconsin.