Reservoir host community and vector density predict human tick-borne diseases across the United States

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Summary Paragraph

Vector-borne disease incidences are increasing globally[1]. In the United States, tick-borne disease cases have tripled since the 1990s[2,3] and cost upwards of 10 billion USD annually[4,5]. Reactive antibiotics can be ineffective at preventing life-long symptoms of tick-borne diseases[6]; thus, disease prevention is vital[1,7,8]. Tick density and densities and diversity of non-human mammalian (i.e., reservoir) hosts are hypothesized to drive tick-borne disease dynamics[1,9,10] and are targets for interventions. Yet, support for these hypotheses has not integrated actual human disease incidence, so implications for human disease are unclear. Here, we relate human prevalence of four tick-borne diseases (Lyme Disease, Monocytic Ehrlichiosis, Granulocytic Anaplasmosis, and Babesiosis) to tick and reservoir host community data collected by the U.S. National Ecological Observatory Network (NEON) across the contiguous U.S. We show that human disease prevalence is correlated positively with tick and reservoir host densities and negatively with mammalian diversity for Lyme Disease and Ehrlichiosis, but positively for Anaplasmosis and Babesiosis. Reductions in tick and host density by a single individual could prevent, on average, ~1,850 and ~1,775 total tick-borne disease cases annually relative to 2017, respectively. Because of the non-monotonic diversity-disease relationship for total disease cases, conservation of small mammal species to 9-13 species per 10,000 m^2 could prevent an additional ~8,500 tick-borne disease cases in the U.S. relative to 2017 levels. Our results suggest that the efficacy of tick-borne disease interventions depends on tick and host densities and host diversity. Thus, policymakers and disease managers should consider these ecological contexts before implementing preventative measures.

Vector-borne diseases and tick-borne diseases, specifically, are on the rise globally[1]. In the United States, cases of tick-borne diseases have more than doubled since 2004 and cases of Lyme disease have tripled since the 1990s[2,3]. There are an estimated 240,000-440,000 new cases of Lyme Disease annually[11]. Lyme Disease costs the United States >$1 billion in healthcare costs[4], and $5 to $10 billion in economic and societal costs annually[5], and is only one of several major tick-borne disease in the U.S. Antibiotics are often ineffective at preventing life-long symptoms of bacterial tick-borne diseases (e.g. Lyme Disease, Anaplasmosis) if they are not prescribed soon after the tick bite[6] and, thus, effective prevention of tick-borne diseases is crucial. However, control of tick-borne zoonoses has been largely unsuccessful, because of a limited understanding of the ecological complexities and drivers of these diseases[1,7,8].

Tick densities, reservoir host densities, and reservoir host diversity are hypothesized drivers of tick-borne diseases (Fig. 1)[1,9,10]. In the majority of the United States, the causative agents of Lyme Disease (*Borrelia burgdorferi* [*sensu lato*]), Granulocytic Anaplasmosis (*Anaplasma phagocytophilum*; hereafter Anaplasmosis), and Babesiosis (*Babesia microti*) are transmitted by *Ixodes scapularis* (blacklegged ticks), while the causative agent of Monocytic Ehrlichiosis (*Ehrlichia chaffeensis*; hereafter Ehrlichiosis) is transmitted by *Amblyomma americanum* (lone star ticks)[12–14]. Larval ticks become infected following a blood-meal from an infected host, such as mammals (e.g. rodents, insectivores, and scurids[15]) that can serve as reservoir hosts (organisms that maintain and transmit pathogen populations[16]) for these pathogens. Importantly, reservoir competence (ability for a host to transmit pathogens to uninfected vectors[16]) of mammalian host species varies among these pathogens (Fig. 1)[15,17].

Although there is some support for the hypotheses that densities of ticks and densities and diversity of wildlife hosts drive Lyme Disease dynamics, most of the support is at local rather

than country or continental scales[14,15,18] and has not incorporated human disease (e.g. reported cases)[9,10,18] (but see ref[19]). Linking wildlife and densities of infected ticks to human disease is difficult, as human behavior, such as avoidance and chemical deterrents[20], can disrupt this link. Further, support for the hypotheses that tick densities and wildlife host densities and diversity drive tick-borne diseases is lacking in disease systems other than Lyme Disease. Up to this point, a major impediment to understanding the drivers of tick-borne diseases was a lack of broad-scale spatial datasets that combined reservoir host communities, tick densities, and tick infection prevalence with human disease incidence at the same scale[21]. With the establishment of the U.S. National Ecological Observatory Network (NEON), there are now data on reservoir host communities, tick densities, and tick infection prevalence from across the U.S. collected using standardized methodologies that can be coupled with U.S. Center for Disease Control (CDC) data on human tick-borne disease incidence to finally elucidate the role of wildlife factors on human prevalence of tick-borne diseases.

The objectives of our study are to: 1) identify the combination of host and vector community variables that best explain human tick-borne disease prevalence across space and time, 2) evaluate the direct and indirect effects of these variables on human disease prevalence mediated through density of infected ticks, and 3) evaluate the human health burden of tick-borne diseases across a mammalian diversity gradient. In accordance with the dilution effect hypothesis[14,22], we expect a positive biodiversity-disease relationship (dilution) when the most abundant hosts have the highest reservoir competence, because as rare host species are added to communities the mean competence of the community and, thus, disease risk decreases (assuming host community assembly is substitutive, i.e. competitive for niche space). We expect a positive biodiversity-disease relationship (amplification) when hosts do not differ or very weakly differ in

their reservoir competence because as rare hosts are added the mean competence of the host community would not change while host densities and disease risk could increase if assembly is additive (Fig. 1; see Supplement Text for further details)[10,14,22]. Hence, we predict that increasing small mammal richness will dilute Lyme Disease and Ehrlichiosis, because the most abundant mammal hosts are typically the most competent for these diseases, but we expect increasing small mammal richness to amplify Anaplasmosis and Babesiosis, because small mammal hosts exhibit similar competencies for these diseases.

To address these objectives, we linked ecological, tick density, and mammal density and diversity data collected by NEON from 2014-2018 to CDC data on human cases of Lyme Disease, Anaplasmosis, Babesiosis, and Ehrlichiosis gathered for the same counties and time period as the NEON data (see Supplementary Table 1 for specific county-year replicates included in analyses; Fig. S1). Because of differences in disease reporting and data availability, sample sizes for model selection were variable (n = 95 for Lyme Disease, n = 116 each for Anaplasmosis and Ehrlichiosis, and n = 57 for Babesiosis). To analyze correlations among ecological factors and human tick-borne disease prevalence, we used generalized linear mixed effects models with a binomial distribution and county as a random effect[23], and conducted model selection of all main effects and biologically relevant two-way interactions among wildlife variables (interactions between tick and host densities and host diversity and densities; see Supplementary Table 2 for competing models). To account for differences in questing behavior (height) of blacklegged ticks along a north-south gradient[24], we included latitude as a covariate for blacklegged-tick borne diseases. Further, to account for potential climate-related differences in relationships between wildlife variables and human disease incidence[19], all models included covariates of mean annual temperature and annual precipitation. The previously described

analyses did not include tick infection data because these data were collected at only 13 NEON sites, which would have reduced our statistical power to test our hypotheses. To investigate direct and indirect effects of tick and host community metrics on human disease prevalence mediated through density of infected ticks, we used sequential regressions (see Fig. 1 for *a priori* relationships). We chose sequential regressions because available data on tick infection prevalence (13 sites and <30 site-by-time replicates per pathogen) were insufficient for structural equation models.

Tick vector density, reservoir host density, and small mammal species richness predicted human disease prevalence, but the direction of effects and interactions among these variables differed across diseases (Table 1, Supplementary Table 3; Fig. 2). For Lyme Disease and Anaplasmosis, the relationship between reservoir host density and human disease prevalence became more positive as tick density increased (Fig. 2A,B). Thus, for Lyme Disease and Anaplasmosis, the reduction of a single tick per 1,000 m^2 would reduce incidence by 25.4% and 30.3% at median host densities, and 85.6% and 110.9% at high host densities, respectively. For Babesiosis and Ehrlichiosis, the reduction of tick density by a single individual per 1,000 m^2 would reduce disease incidence by 27.5% and 25.8%, respectively. Like reducing ticks, decreasing reservoir host densities also reduces human tick-borne diseases (Fig. 2). In fact, when other factors in the model are at median values, a reduction in one tick per 1,000 m^2 and a reduction of one reservoir host individual per 10,000 m^2 (lowering of deer density category for Ehrlichiosis) is predicted to prevent annually ~1,850 and ~2,180 total (across all four diseases) U.S. tick-borne disease cases, respectively, and ~230 and ~20 U.S. Lyme Disease disability-adjusted life years (DALYs)[25], respectively, relative to 2017 data. Unfortunately, DALY information is unavailable for the other three tick-borne diseases, which should be addressed in

future research given that tick-borne diseases vary in symptoms and virulence[2,7,25] and, thus, total cases of tick-borne diseases does not represent human disease burden. These results suggest that host-targeted interventions would prevent more human disease than tick-targeted interventions, if disease burden were similar across diseases. Although our results suggest that commonly employed host- and vector-targeted interventions should be effective at reducing disease, they often have not translated to fewer cases of human tick-borne diseases in practice[20,26]. Further, residential use of these control measures is limited, because host vaccines and acaricide-treated bait stations are often not logistically feasible for residents[27,28] and acaricides require semi-regular reapplication[29] and negatively affect non-target taxa[30].

An alternative or complementary approach to reducing ticks and reservoir hosts is to conserve or increase reservoir host species. However, this has been a controversial approach to managing tick-borne diseases for several reasons[10,18,21,22,31], such as concerns that it might increase certain tick-borne diseases while decrease others[10,18,21]. As predicted, for pathogens with variably competent hosts (Lyme Disease and Ehrlichiosis), human disease prevalence was negatively correlated with small mammal richness, supporting a dilution effect (Table 1, Fig. 2A,D). For Lyme Disease, the negative relationship between small mammal richness and human disease prevalence increased in magnitude as reservoir host (white-footed mouse) density increased, such that the conservation of a single mammal species would be predicted to reduce Lyme Disease incidence by 13.2% and 26.6% at median and high reservoir host densities, respectively (Table 1, Fig. 2A). For Ehrlichiosis, the conservation of a single mammal species would increase Anaplasmosis incidence by 19.8% across host densities (Table 1, Fig. 2D). Also in agreement with our predictions, human disease prevalence was positively correlated with small mammal richness for Anaplasmosis (a pathogen with similarly poor hosts), supporting an

amplification effect, such that the conservation of a single mammal species would increase Anaplasmosis incidence by 41.8% across host densities (Table 1, Fig. 2B). For Babesiosis, the positive relationship between small mammal richness and human disease prevalence decreased in magnitude as reservoir host (small mammal) density increased, such that the conservation of a single mammal species would be predicted to increase Babesiosis incidence by 34.2% and 6.0% at low and median reservoir host densities, respectively (Table 1, Fig. 2C).

Because of the opposing direction of the diversity-disease relationship for Lyme Disease and Anaplasmosis, a non-monotonic relationship emerged between small mammal richness and total tick-borne disease cases, such that human disease incidence was higher at the extremes of small mammal richness (<8 and >14 species per 10,000 m^2) than at intermediate numbers of species (9–13 species; Fig. 3). A similar pattern emerged when Babesiosis and Ehrlichiosis were included in the calculation of total human disease, likely due to low incidence of these diseases (Fig. S2). Thus, while mean mammal richness at NEON sites was 8 [7.6, 8.5] species per 10,000 m^2, our model suggests that 9-13 species per 10,000 m^2 should prevent the most human tick-borne disease cases regardless of tick and host densities (Fig. 3). More specifically, the conservation of small mammal species to 9-13 species per 10,000 m^2 is predicted to prevent annually ~8,500 additional total U.S. tick-borne disease cases and an average of 1,130, and as high as 5,300 (when tick and reservoir host densities are high), additional U.S. Lyme Disease-caused DALYs, relative to 2017 data (Fig. S3). These results suggest that conserving mammal species provides an alternative or complementary approach to reducing tick and/or reservoir host density for controlling tick-borne diseases, but caution must be taken when employing this approach given the non-monotonic relationship between mammalian richness and total tick-borne disease cases (Fig. 3).

Finally, to evaluate the contribution of changes to density of infected ticks on human disease prevalence (Fig. 1), we conducted sequential regressions. For Lyme Disease, Anaplasmosis, and Babesiosis, we found that human disease was positively associated with density of infected ticks (Fig. 4A,D,H), which was driven by the proportion of infected ticks (Fig. 4B,E,I, Supplementary Table 4). For Lyme Disease, the diluting relationship between small mammal diversity and human disease (Fig. 4, Supplementary Table 4) seemed to be mediated by the effect of small mammal richness on the association between the proportion of infected ticks and reservoir host density (Fig. 4C). When small mammal richness was low, the proportion of infected ticks was positively related to reservoir host density, but this relationship was not different from zero when small mammal richness was high (Fig. 4C). Conversely, the amplifying relationship between small mammal diversity and Anaplasmosis and Babesiosis was a function of reservoir host density increasing with small mammal richness (Fig. 4G,K), which in turn fueled an increase in the proportion of infected ticks (Fig. 4F,J; Supplementary Table 4). Despite observing a small mammal dilution effect and a positive effect of deer densities for Ehrlichiosis in the model selection analyses that included all sites, we did not find evidence for these effects in the sequential regressions (Supplementary Table 4), likely because subsetting the data resulted in both reduced samples sizes and the exclusion of sites with no deer.

The goal of our study was to evaluate how patterns of human tick-borne diseases are mediated by tick densities and the densities and diversity of reservoir hosts. We show that reservoir host and tick densities are correlated with human disease prevalence for all four diseases, likely because as tick and mammal densities increase, so do the density of infected ticks and transmission, as suggested by our sequential regressions. These results are consistent with previous studies showing positive relationships between reservoir host densities and densities of

infected ticks[14,15]. We also found support for our hypothesis that reservoir host richness is associated with human disease prevalence in directions that are predictable by reservoir competence of the host community. Specifically for pathogens with variably competent hosts (Lyme Disease and Ehrlichiosis)[15,17], host diversity dilutes disease, whereas for pathogens with reservoir host species that are weakly and similarly competent (Anaplasmosis and Babesiosis)[15], host diversity amplifies disease. These results imply that determining reservoir competence of a range of host species for vector-borne pathogens may provide valuable insights into whether or not biodiversity conservation would dilute or amplify disease risk[10,22,31]. Further, because of the lack of reservoir competence information for many small mammal species in North America for the pathogens examined in this study, we were unable to develop a quantitative estimate of host community competence. Thus, future work should investigate reservoir competency of small mammal species present in tick-borne disease endemic regions to better inform the relationship between host composition (integrated with reservoir competence) and human disease incidence. Alternatively, spatial genetic variation in pathogens could have altered the strength of the relationships among ecological variables and human disease incidence[19]. Yet, the inclusion of spatially explicit covariates likely account for much of this potential variation.

Antibiotic treatment of tick-borne diseases in humans can be ineffective at preventing life-long symptoms[6], and, thus, preventative measures, such as controlling ticks and reservoir host densities and diversity, are essential to reduce human tick-borne disease. While individual relationships among reservoir hosts, ticks, disease risk (e.g. density of infected ticks), and human disease incidence have been previously supported[9,15,18,19,21], our study is the first to support all links among these variables at broad spatial scales, supporting the promise of proactive rather than reactive approaches to tick-borne disease management. Although host- and vector-targeted

interventions reduce densities of infected ticks[27–29], the individual use of these interventions to reduce human disease has proved to have some shortcomings in practice[20,26,27,29,30]. Our findings suggest that conservation of small mammal species within a feasible but specific range of richness levels might offer an effective alternative and/or complementary approach to controlling vector or host densities for preventing tick-borne diseases. Consequently, future work should investigate the effectiveness of control measures targeting tick and reservoir host densities across a mammalian host richness gradient to determine what levels and combinations of interventions would be most effective at preventing human tick-borne diseases.

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Data availability
Ecological data that support the findings of this study are available from U.S. NEON (https://data.neonscience.org/data-products/explore, see Supplemental Table 2 for specific data products). Human health data are available from U.S. CDC, but restrictions apply to the availability of data on Anaplasmosis, Babesiosis, and Ehrlichiosis, which were used with permission for the current study, and so are not publically available. However, human health data on Lyme Disease are publically available from the U.S. CDC (https://www.cdc.gov/lyme/stats/survfaq.html).

Code availability
No custom code was used in this manuscript, but all R code is available from the authors by request.

Author contribution statement
MBM and JRR conceived the study; MBM organized data and performed analyses; MBM and JRR wrote the manuscript.

Conflict of interest
The authors declare they have no conflict of interest.

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Methods

Study area

Our analyses paired site-level estimates of vector tick density, tick-borne pathogen prevalence, and small mammal communities from 38 climatically and ecologically variable sites in the National Ecological Observatory Network (NEON; Supplementary Figure 1; Supplementary Table 1) with county-level human case counts of tick-borne diseases collected by the Center for Disease Control (CDC) Notifiable Diseases Surveillance System (NNDSS) and Division of Parasitic Diseases and Malaria (DPDM). The study area included 35 counties in 21 states across the continental United States (Riley County Kansas, Stutsman County North Dakota, and Boulder County Colorado each contained two NEON sites; Supplementary Table 1). Due to variability in data collection across NEON sites, not all sites or counties were included in each year of the data.

NEON Data

Sampling protocols for ticks, tick-borne pathogens, and small mammals in this section will be described briefly, as detailed information on sampling protocols are available through the appropriately cited NEON sampling protocols. NEON data used in this study were downloaded on 1 November 2019; datasets used in this study are outlined in Supplementary Table 2.

Tick sampling

Tick sampling occurred at 38 sites, starting in 2014[1]. At each site, tick sampling occurred in six plots that were iteratively sampled. Sampling frequency was dependent on whether ticks were detected. Sampling began with a sampling bout every six weeks, but collection of one or more ticks prompted sampling bouts every three weeks. The start of sampling at a given site coincided within two weeks of the onset of vegetation green-up and ended within two weeks of

senescence (typically April-September, but may be March-October depending on site and weather). Data availability between sites is variable, because of weather conditions and staff availability. Each tick sampling plot was 40 x 40m; the perimeter of each sampling plot was sampled with a 1 x 1m drag cloth. If vegetation within a sampling plot was too thick to allow dragging, flagging was used either instead of dragging or in conjunction with dragging. Larvae, nymph, and adult abundances were recorded and identified to species. For each species and sampling bout, nymph and adult abundances were pooled. Tick abundances were converted to densities based on area sampled (individuals per m^2) and scaled to individuals per 1000 m^2.

Tick-borne pathogen prevalence

Testing of pathogen prevalence in ticks occurred at 13 sites, starting in 2014[1]. At the time of data analysis, tick pathogen prevalence was only available for 2014-2017. At a given site, a subset (10-100 ticks per species-life stage combination) of sampled ticks were tested annually for the presence of zoonotic pathogens. *Ixodes scapularis* (blacklegged ticks) were tested for *Anaplasma phagocytophilum*, *Babesia microti*, and *Borrelia burgdorferi*. *Amblyomma americanum* (lone star ticks) were tested for *Ehrlichia chaffeensis*. Pathogens status in nymphal ticks was tested using next-generation sequencing and 16S rRNA primers. We excluded all pathogen status test results that did not also test positive for hard-tick DNA. Pathogen prevalence at a given site was estimated as the proportion of nymphal ticks that tested positive for a given pathogen.

Small mammal trapping

Small mammals trapping occurred at 38 sites, starting in 2014[2]. At each site, trapping plots were arranged in three to eight plots of 100 live traps (Sherman) arranged in a 10 x 10 grid, with 10 m spacing (100 x 100m area). Trapping plots were separated by at least 135 m. NEON

field technicians trapped, identified, and released small mammals from each grid either one or three nights (depending on whether sampling for diversity or pathogens, respectively) per month or every other month (depending on site designation as core or relocatable, respectively) during the growing season within a 21 day window centered on the new moon. Typical sampling occurs between May and September, but may extend into March and October, depending on site location and weather conditions. Ethical approval was obtained from IACUC[2].

Small mammal richness was the total number of unique species collected across all sampling plots at a given site in a given year. Small mammal abundance for a given site in a given year was calculated as the mean number of individuals collected per trap night per sampling plot (individuals per 10000 m^2). Similarly, abundance of white-footed mouse (*Peromyscus leucopus*), the main reservoir species for *Bo. burgdorferi*, was calculated as the mean number of white-footed mice individuals collected per trap night per sampling plot (individuals per 10000 m^2).

Deer density estimates

As white-tailed deer are the assumed main reservoir host for *E. chaffeensis*[3,4], we included deer densities as a predictor of HME. The most recent white-tailed deer density estimates for the United States cover 2001-2005, were compiled by the Quality Deer Management Association, and were compiled by the QDMA and are available through the United States Forest Service[5–7]. Deer densities were not meant to be a measure of absolute white-tailed deer density, but rather were meant to provide an estimate for relative densities across the continental United States. Therefore, we grouped deer density estimates into three categories: No Deer, Low Density, and High Density. The “No Deer” category represents areas where white-tailed deer are absent, the “Low Density” category represents deer densities (<11.6 deer/km^2)

that are below or on the cusp of ecologically damaging, and the "High Density" category represents deer densities (>11.6 deer/km^2) that are ecologically damaging and are greatly above historic, pre-European settlement densities[8,9].

Human cases of tick-borne diseases

We obtained the annual number of reported cases of Lyme Disease, Anaplasmosis, Ehrlichiosis caused by *E. chaffeensis*, and Babesiosis from the CDC NNDSS and DPDM from 2014 – 2018. At the time of analysis, Lyme Disease and Babesiosis cases were only available from 2014-2017. Lyme disease case definition by the CDC includes both confirmed and probable cases, to address under-reporting[10]**.** Due to differences in Lyme Disease case definitions between the CDC and Massachusetts Department of Health starting in 2016, most cases for Massachusetts are not reported to the CDC[10,11]; thus, Lyme Disease case data from Worcester, Massachusetts was limited to 2014 and 2015. Case definition of Anaplasmosis, Ehrlichiosis, and Babesiosis requires both clinical and laboratory evidence[12,13]. Confirmed cases of Anaplasmosis, Ehrlichiosis, and Babesiosis have both clinical and confirmatory laboratory evidence, while probable cases of Anaplasmosis, Ehrlichiosis, and Babesiosis have clinical evidence, but only supportive laboratory evidence (see ref[13] for further information). Anaplasmosis, Ehrlichiosis, and Babesiosis is not a reportable condition for all states in all years; thus, Anaplasmosis and Ehrlichiosis was not reported in Colorado and New Mexico across years, while Babesiosis was not reported in Arizona, Colorado, Georgia, Kansas, New Mexico, and Oklahoma across years. Further, Babesiosis was not reported from Virginia and Florida before 2017, so all sites in these states and years were not included in the analyses. Tick-borne disease cases are reported in the patient's county of residence, so reporting errors due to travel are possible, especially in non-endemic and non-emerging areas.

Statistical analyses

All statistical analyses were conducted in R version 3.6.1[14]. For the national-scale analyses, we used model selection and generalized linear mixed effects models with a binomial distribution to explore patterns among human cases of tick-borne disease prevalence, tick densities, small mammal diversity, and reservoir species abundance. Responses for each model were a binary county-level case count and population. For Lyme Disease and Anaplasmosis models, we used glmer() in the lme4 package[15] and for the Ehrlichiosis and Babesiosis models, we used bglmer()[16] with a gamma covariance prior, due to issues of singular fit related to low incidence of these diseases. Predictor variables included tick vector densities, year, reservoir host abundance, and small mammal richness. For Lyme Disease and Ehrlichiosis (diseases with primary competent reservoir hosts), we used abundance of white-footed mice and white-tailed deer, respectively, for the measure of reservoir host abundance. Conversely, for Anaplasmosis and Babesiosis (diseases with evenly poor reservoir hosts), we used abundance of all small mammals for the measure of host reservoir abundance. County was included as a random term in the model because annual observations within the same county are not independent.

We conducted model selection in which we fit all possible combinations of main effects and biologically relevant two-way interactions of all biotic variables (interactions between host density and tick density and between host density and host richness; dredge function in MuMIn R package)[17]. To account for differences in questing behavior (height) of blacklegged ticks along a north-south gradient[18], we included latitude as a covariate for blacklegged-tick borne diseases. Further, to account for potential climate-related differences in relationships between wildlife variables and human disease incidence[19], all models included covariates of mean annual temperature and annual precipitation. For Lyme Disease models, reporting definition type and

disease endemism were also included as covariates. To find best fitting models, we used a combination of the lowest Akaike's Information Criterion with bias-correction (AICc) and the law of parsimony; such that competing models (ΔAICc < 2) with fewer degrees of freedom than models with lowest AICc were selected as the best model for a given disease. Likelihood ratio tests against a null model containing only the random term of county and fixed effects of mean annual temperature and annual precipitation to determine overall p-values for best models. We also calculated AICc weights (*w*) as well as marginal (fixed) and conditional (fixed and random effects) R^2 values[20]. Residuals of best models were assessed for overdispersion, zero-inflation, heteroscedasticity, and spatial autocorrelation using the DHARMa R package[21]; residuals from all best models indicated all model assumptions had been met and no spatial autocorrelation present in residuals.

For models with significant effects of small mammal richness on human disease prevalence, we calculated the differences in case prevalence from 2017 (the year with the most recent complete data) by subtracting the median state-level prevalence for a given disease in 2017 from best model-predicted prevalence. We then multiplied this relative change in prevalence by the United States population from all states reporting Anaplasmosis cases to get total change in disease incidence across the U.S. These are reported in Figure 3 in the main text. Change in Lyme Disease incidence was then converted to change in annual disability-adjusted life years (DALYs) to provide an estimate of overall disease burden from changing disease incidence. Average case DALYs for Lyme Disease have been estimated for patients with different Lyme Disease outcomes: erythema migrans (0.005 DALYs), disseminated Lyme Disease (0.113 DALYs), and Lyme-related persisting symptoms (1.661 DALYs)[22]. Relative prevalence of these outcomes per Lyme Disease diagnosis are 82.8% for erythema migrans,

8.6% for disseminated LD, and 8.6% for persisting Lyme disease symptoms[23]. Thus, the estimated DALYs for a Lyme Disease case is 0.156.

To address our second objective of direct and indirect effects of tick density and reservoir host metrics on human disease prevalence, we used sequential regressions rather than traditional structural equation models (SEMs). Sequential regressions were used over SEMs due to limited data on tick infection prevalence (<30 replicates per pathogen) and sample size requirements for SEMs being at least 30 replicates for relatively simple models[24]. Models were fit using generalized linear models with either a normal error distribution (for tick and mammal density models) or a binomial error distribution (for tick and human disease prevalence models). All models were hypothesized *a priori* from national-scale analyses, the ecology of these pathogens, and previous studies[25–28]; see Fig. 1 in main text for *a priori* hypothesized links. Significance of relationships was found with type 3 error and p-values were adjusted using the Holm-Bonferroni sequential correction[29], by which k relationships are ranked (i) by their p-values (P_i) and p-values are adjust by the equation: $(k - i + 1) * P_i$. Significance levels of all models was $P < 0.05$.

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Tables

Table 1. Regression coefficients and statistics from best-fit models from model selection. Predictor variables were black-legged tick density, lone star tick density, white-footed mouse density, small mammal richness, small mammal density, and white-tailed deer density. Models included a random effect of county. All models included covariates of annual mean temperature and annual precipitation. Lyme Disease models included covariates of CDC reporting type and disease endemism. R^2 values represent marginal/conditional (fixed/random + fixed) R^2. Competing models are presented in Supplemental Table 3.

Model/Variable	Estimate (SE)	DF	Chisq	**P**
Lyme Disease (w = 0.81, R^2 = 0.15/0.78, n = 95, $X^2(5)$ = 33.45, p < 0.001)				
Black-legged tick density	-0.166 (0.12)	1	1.78	0.182
White-footed mouse density	0.277 (0.21)	1	1.74	0.187
Small mammal richness	0.026 (0.07)	1	0.13	0.722
Black-legged tick density*White-footed mouse density	0.196 (0.05)	1	16.04	< 0.001
White-footed mouse density*Small mammal richness	-0.084 (0.03)	1	9.35	0.002
Anaplasmosis (w = 0.63, R^2 = 0.19/0.75, n = 116, $X^2(4)$ = 22.41, p < 0.001)				
Black-legged tick density	-0.940 (0.47)	1	4.05	0.044
Small mammal density	-0.063 (0.04)	1	2.76	0.097
Small mammal richness	0.349 (0.12)	1	8.31	0.004
Black-legged tick density*Small mammal density	0.241 (0.07)	1	11.21	< 0.001
Babesiosis (w = 0.50, R^2 = 0.73/0.74, n = 57, $X^2(4)$ = 21.05, p < 0.001)				
Black-legged tick density	0.243 (0.12)	1	4.36	0.037
Small mammal density	0.749 (0.57)	1	1.75	0.186
Small mammal richness	0.649 (0.55)	1	1.37	0.242
Small mammal density *Small mammal richness	-0.118 (0.07)	1	3.29	0.070
Ehrlichiosis (w = 0.22, R^2 = 0.44/0.46, n = 116, $X^2(3)$ = 19.61, p < 0.001)				
Lone star tick density	0.230 (0.08)	1	7.44	0.006
Deer density	1.660 (0.46)	1	12.88	<0.001
Small mammal richness	-0.197 (0.11)	1	3.37	0.066

Figures

Fig. 1. Conceptual diagram for linkages connecting wildlife to human prevalence of tick-borne diseases, per the dilution effect hypothesis. (A), hypothesized links among wildlife and human disease prevalence for pathogens with hosts that differ in reservoir competence (ability to transmit pathogen to uninfected ticks). (B), hypothesized interaction between host diversity and competent host density, by which the effect of competent host density on tick infection prevalence is strongest when host diversity is low. (C), hypothesized links among wildlife and human disease prevalence for pathogens with hosts that are similarly poor reservoir hosts. (D), hypothesized additive relationship between host diversity and competent host density, such that the effect of increased host diversity in an area will increase reservoir density and, in turn, tick infection prevalence. In (A) and (C), links between density of infected ticks and human disease prevalence can be disrupted by human behavior (e.g. acaricides and avoidance). For each link in (A) and (C), signs are representative of whether relationship is positive (+), negative (−), or variable (±). Dotted arrows from host diversity to human disease prevalence in (A) and (C) are the overall, indirect effect of diversity on disease. Data for host reservoir competence in (A) and (C) are from ref[15].

Fig. 2. Human prevalence of tick-borne diseases is correlated with reservoir and vector metrics. Model predicted relationships among vector tick density, small mammal community metrics, and human prevalence of disease for A) Lyme Disease, B) Anaplasmosis, C) Babesiosis, and D) Ehrlichiosis. Facets of increasing small mammal richness along the top are 5, 8, and 11 species, respectively. For (A) Lyme Disease, facets of increasing host density down the right side are 0, 2, and 4 mice per 10000 m^2, respectively. For (B) Anaplasmosis and (C) Babesiosis, facets of increasing host density down the right side are 3, 5, and 7 small mammal individuals per 10000 m^2, respectively. For (D) Ehrlichiosis, the facets of increasing host density down the right side are no deer, low deer density, and high deer density. Coloration for reservoir host abundance is consistent across diseases: blue lines are no/low abundance, orange lines are mean abundance, and pink lines are high abundance. Models indicate positive correlations of vector tick density with human prevalence of disease, such that human disease prevalence is predicted to be highest in areas with high vector tick density. For Lyme Disease (A), Anaplasmosis (B), and Ehrlichiosis (D) there was a positive relationship between host abundance and disease prevalence. Accounting for differences in tick and reservoir host abundance, we found significant negative correlations between diversity and disease incidence for Lyme Disease (A) and Ehrlichiosis (D), but a significant positive correlation between diversity and disease for Anaplasmosis (B). The observed patterns are consistent with the dilution effect hypothesis, which posits that a diluting (negative) relationship between diversity and disease is expected when hosts differ in their ability to maintain and transmit pathogens (e.g. Lyme disease and Ehrlichiosis); when this condition is not met (e.g. Anaplasmosis), an amplifying (positive) relationship is expected.

Fig. 3. Changes in human disease incidence. Changes in disease incidence for Lyme Disease (green lines), Anaplasmosis (pink lines), and the sum of the two diseases (total; black solid lines) relative to incidence in the U.S. in 2017. Facets of low, medium, and high tick density along the top are 0.75, 2, and 4 ticks per 1000 m^2, respectively. Facets of host density down the right are 0, 2, and 4 mice per 10000 m^2 and 3, 5, and 7 small mammals per 10000 m^2 for Lyme Disease and Anaplasmosis respectively. Vertical dark grey line indicated median small mammal richness

across NEON sites. Light grey rectangle from 9 to 13 small mammal species represents the mammal richness required to maintain the lowest disease incidence across tick and reservoir host densities. Figures suggest that the magnitude of the relationship between small mammal richness and total tick-borne disease incidence is dependent on the densities of ticks and reservoir hosts. Similarly, figures suggest that the relationship between small mammal richness and total tick-borne disease incidence is non-monotonic and is driven by the reductions in Lyme Disease as small mammal richness increases from low (<8 species) to median (8 species), then by the increase in Anaplasmosis as small mammal richness increases from median (8 species) to high (>13 species).

Fig. 4. Mammal richness is indirectly correlated with human prevalence of tick-borne diseases. Relationships among (A) human prevalence of Lyme Disease and density of *Borrelia burgdorferi*-infected blacklegged ticks; (B) density of infected ticks and proportion of *Bo. burgdorferi*-infected ticks; (C) proportion of infected ticks, white-footed mouse density, and small mammal richness; (D) human prevalence of Anaplasmosis and density of *Anaplasma phagocytophilum*-infected blacklegged ticks; (E) density of infected ticks and proportion of *A. phagocytophilum*-infected ticks; (F) proportion of infected ticks and small mammal density; (G) small mammal density and small mammal richness; (H) human prevalence of Babesiosis and density of *Babesia microti*-infected blacklegged ticks; (I) density of infected ticks and proportion of *Ba. microti*-infected ticks; (J) proportion of infected ticks and small mammal density; and (K) small mammal density and small mammal richness. Figures indicate indirect effects of small mammal species richness on human prevalence mediated through increases in proportion of infected ticks, and, in turn, density of infected ticks. Figures support predictions of a negative diversity-disease relationship (dilution) for Lyme Disease (A-C), but positive diversity-disease relationships (amplification) for Anaplasmosis (D-G) and Babesiosis (H-K). Lines for all panels are generalized linear regression coefficients from sequential regressions (see Table S2); ribbons are model indicated standard error. Regression coefficients and statistics are described in Table S. Note: y-axis of (B), (E), (G), (I), and (K) are natural-log transformed. Points on panels are slightly jittered, but do not alter interpretation.

Fig. 1.

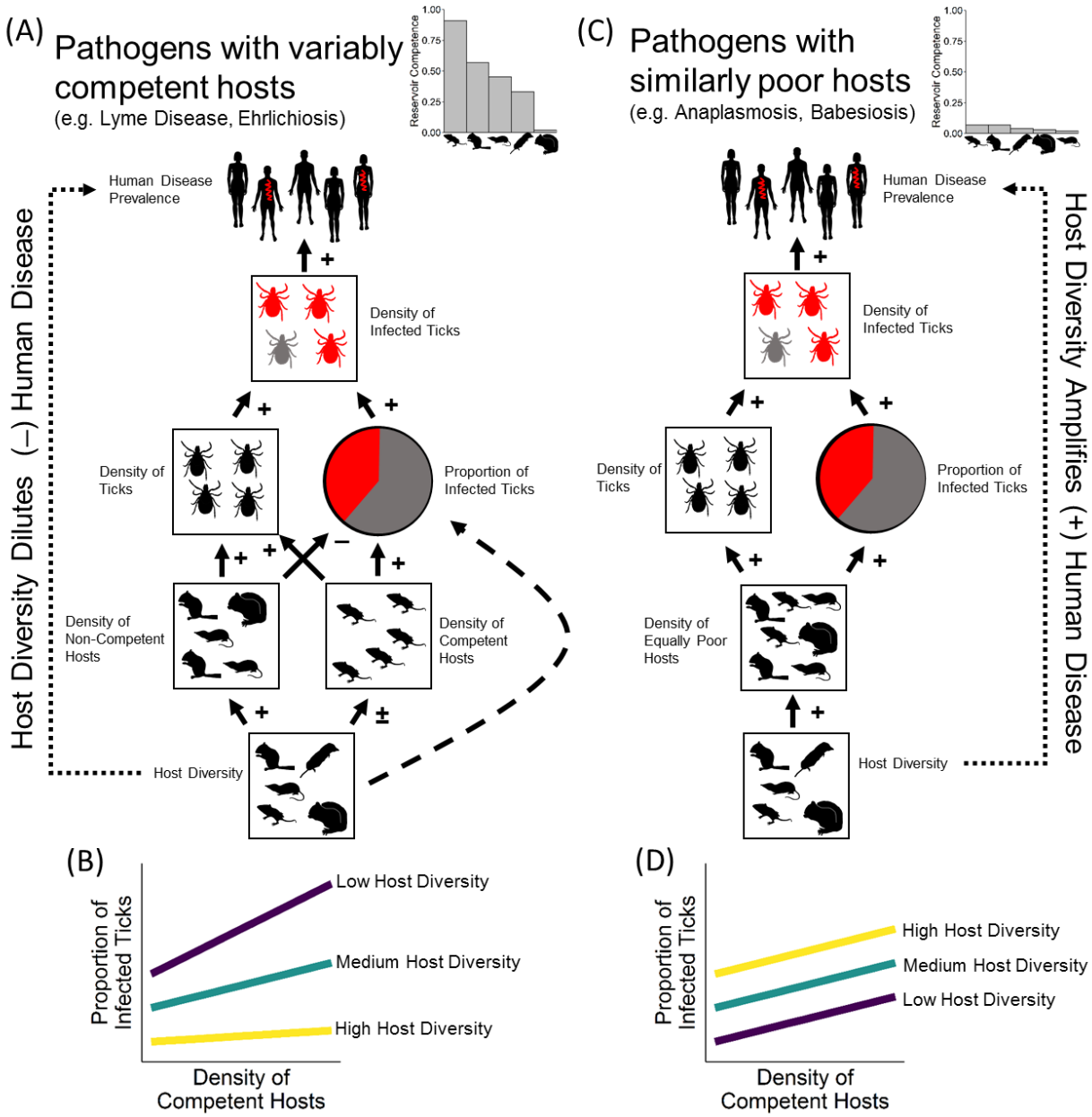

(A)
Pathogens with variably competent hosts
(e.g. Lyme Disease, Ehrlichiosis)
Reservoir Competence
1.00
0.75
0.50
0.25
0.00
Human Disease Prevalence
Density of Infected Ticks
Density of Ticks
Proportion of Infected Ticks
Density of Non-Competent Hosts
Density of Competent Hosts
Host Diversity
Host Diversity Dilutes (–) Human Disease
(B)
Proportion of Infected Ticks
Low Host Diversity
Medium Host Diversity
High Host Diversity
Density of Competent Hosts
(C)
Pathogens with similarly poor hosts
(e.g. Anaplasmosis, Babesiosis)
Reservoir Competence
1.00
0.75
0.50
0.25
0.00
Human Disease Prevalence
Density of Infected Ticks
Density of Ticks
Proportion of Infected Ticks
Density of Equally Poor Hosts
Host Diversity
Host Diversity Amplifies (+) Human Disease
(D)
Proportion of Infected Ticks
High Host Diversity
Medium Host Diversity
Low Host Diversity
Density of Competent Hosts

Fig. 2.

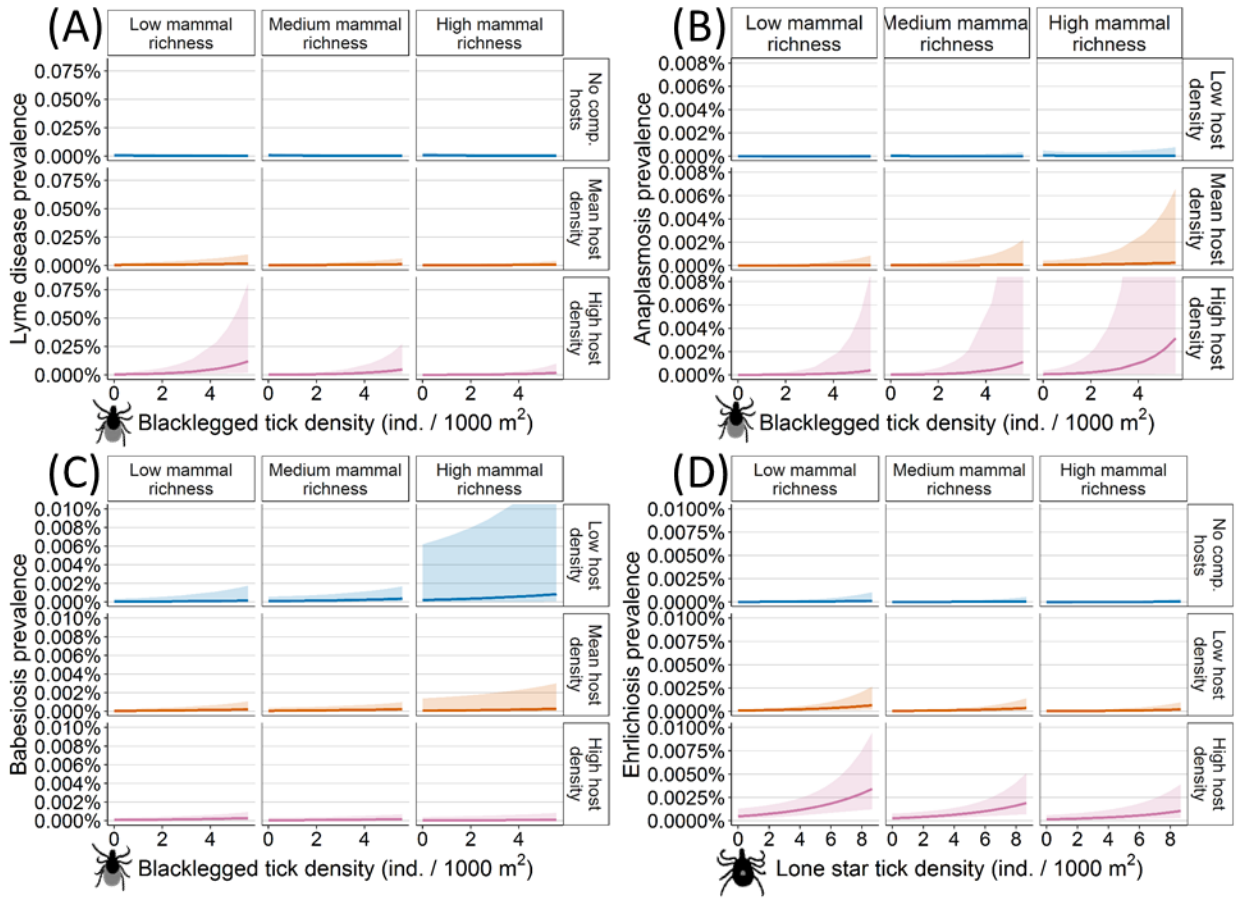

(A)
Low mammal richness
Medium mammal richness
High mammal richness
No comp. hosts
Mean host density
High host density
Lyme disease prevalence
0.075%
0.050%
0.025%
0.000%
0 2 4
Blacklegged tick density (ind. / 1000 m2)
(B)
Low mammal richness
Medium mamma richness
High mammal richness
Low host density
Mean host density
High host density
Anaplasmosis prevalence
0.008%
0.006%
0.004%
0.002%
0.000%
0 2 4
Blacklegged tick density (ind. / 1000 m2)
(C)
Low mammal richness
Medium mammal richness
High mammal richness
Low host density
Mean host density
High host density
Babesiosis prevalence
0.010%
0.008%
0.006%
0.004%
0.002%
0.000%
0 2 4
Blacklegged tick density (ind. / 1000 m2)
(D)
Low mammal richness
Medium mammal richness
High mammal richness
No comp. hosts
Low host density
High host density
Ehrlichiosis prevalence
0.0100%
0.0075%
0.0050%
0.0025%
0.0000%
0 2 4 6 8
Lone star tick density (ind. / 1000 m2)

Fig. 3.

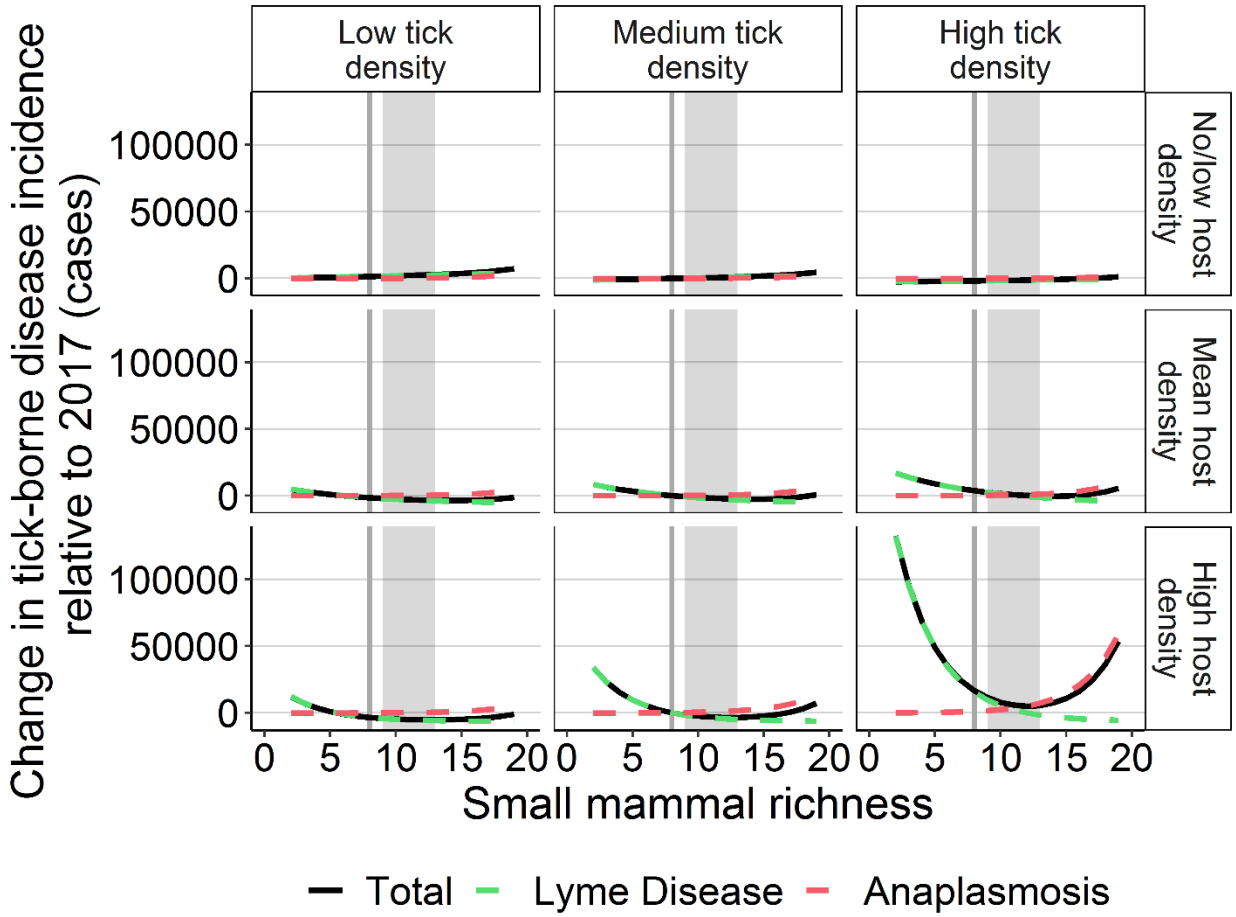
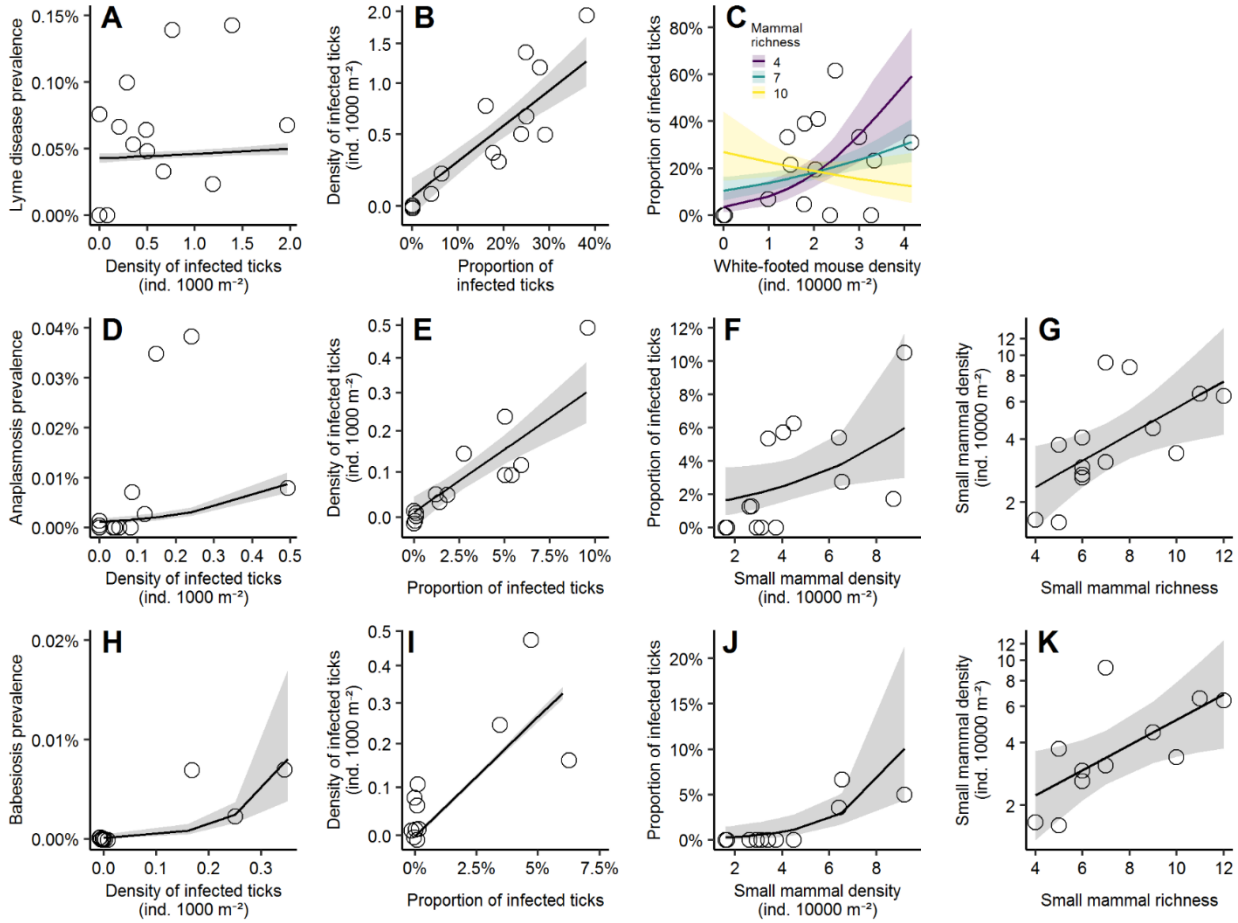

Low tick density
Medium tick density
High tick density
No/low host density
Mean host density
High host density
Change in tick-borne disease incidence relative to 2017 (cases)
100000
50000
0
0
5
10
15
20
Small mammal richness
Total
Lyme Disease
Anaplasmosis

Fig. 4.


A
Lyme disease prevalence
0.15%
0.10%
0.05%
0.00%
0.0 0.5 1.0 1.5 2.0
Density of infected ticks (ind. 1000 m⁻²)
B
Density of infected ticks (ind. 1000 m⁻²)
2.0 1.5 1.0 0.5 0.0
0% 10% 20% 30% 40%
Proportion of infected ticks
C
Mammal richness
4
7
10
Proportion of infected ticks
80% 60% 40% 20% 0%
0 1 2 3 4
White-footed mouse density (ind. 10000 m⁻²)
D
Anaplasmosis prevalence
0.04% 0.03% 0.02% 0.01% 0.00%
0.0 0.1 0.2 0.3 0.4 0.5
Density of infected ticks (ind. 1000 m⁻²)
E
Density of infected ticks (ind. 1000 m⁻²)
0.5 0.4 0.3 0.2 0.1 0.0
0% 2.5% 5% 7.5% 10%
Proportion of infected ticks
F
Proportion of infected ticks
12% 10% 8% 6% 4% 2% 0%
2 4 6 8
Small mammal density (ind. 10000 m⁻²)
G
Small mammal density (ind. 10000 m⁻²)
12 10 8 6 4 2
4 6 8 10 12
Small mammal richness
H
Babesiosis prevalence
0.02% 0.01% 0.00%
0.0 0.1 0.2 0.3
Density of infected ticks (ind. 1000 m⁻²)
I
Density of infected ticks (ind. 1000 m⁻²)
0.5 0.4 0.3 0.2 0.1 0.0
0% 2.5% 5% 7.5%
Proportion of infected ticks
J
Proportion of infected ticks
20% 15% 10% 5% 0%
2 4 6 8
Small mammal density (ind. 10000 m⁻²)
K
Small mammal density (ind. 10000 m⁻²)
12 10 8 6 4 2
4 6 8 10 12
Small mammal richness