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Research article

Idiosyncratic spatial scaling of biodiversity–disease relationships

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High host biodiversity is hypothesized to dilute the risk of vector-borne diseases if many host species are ‘dead ends’ that cannot effectively transmit the disease and low-diversity areas tend to be dominated by competent host species. However, many studies on biodiversity–disease relationships characterize host biodiversity at single, local spatial scales, which complicates efforts to forecast disease risk if associations between host biodiversity and disease change with spatial scale. Here, our objective is to evaluate the spatial scaling of relationships between host biodiversity and *Borrelia* (the bacterial taxon which causes Lyme disease) infection prevalence in small mammals. We compared the associations between infection prevalence and small mammal host diversity for local communities (individual plots) and metacommunities (multiple plots aggregated within a landscape) sampled by the National Ecological Observatory Network (NEON), an emerging continental-scale environmental monitoring program with a hierarchical sampling design. We applied a multispecies, spatially-stratified capture–recapture model to a trapping dataset to estimate five small mammal biodiversity metrics, which we used to predict infection status for a subset of trapped individuals. We found that relationships between *Borrelia* infection prevalence and biodiversity did indeed vary when biodiversity was quantified at different spatial scales but that these scaling behaviors were idiosyncratic among the five biodiversity metrics. For example, species richness of local communities showed a negative (dilution) effect on infection prevalence, while species richness of the small mammal metacommunity showed a positive (amplification) effect on infection prevalence. Our modeling approach can inform future analyses as data from similar monitoring programs accumulate and become increasingly available through time. Our results indicate that a focus on single spatial scales when assessing the influence of biodiversity on disease risk provides an incomplete picture of the complexity of disease dynamics in ecosystems.

Keywords: Bayesian modeling, biodiversity–function relationships, disease ecology, host competence, Lyme disease, National Ecological Observatory Network (NEON), spatial scaling



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Introduction

Links between biodiversity and ecosystem functions or services have long fascinated environmental scientists (Hooper et al. 2005). A general trend is that higher biodiversity promotes ecosystem functions such as primary productivity (Liang et al. 2016, Duffy et al. 2017). Despite a rich history of research, there remain many unresolved questions regarding the scaling of biodiversity–function relationships; specifically, we often do not know how these relationships vary when evaluated at different scales of space, time or biological organization (Thompson et al. 2018, Gonzalez et al. 2020). Because human wellbeing is dependent on ecosystem functions, a more comprehensive understanding of biodiversity–function scaling behaviors may enable more targeted conservation and protection of ecosystem functions, particularly since the scales at which decisions and laws are made (e.g. counties, provinces, states) are often mismatched with the scales (e.g. plots) at which biodiversity–function studies are conducted (Cumming et al. 2006, Meisinger et al. 2018, Winkler et al. 2021, Falco et al. 2022).

One ecosystem function of importance to human and wildlife populations is the regulation of disease dynamics in natural systems, especially vector-borne diseases with complex life cycles influenced by interactions of multiple species (Eisen et al. 2017). If certain ecosystem properties can reduce zoonotic disease risk (i.e. infection prevalence and/or intensity), understanding and promoting those properties can facilitate disease forecasts and trigger preventative management actions (Gardner et al. 2020). The biodiversity of hosts is hypothesized to affect disease risk, and, depending on the identities of the species in the host community, may either dilute or amplify that risk (Rohr et al. 2020, Keesing and Ostfeld 2021). Species composition of the host community determines the strength and direction of associations between host biodiversity and disease risk (Ostfeld 2011). In particular, the relative competence (i.e. the ability to transmit an infection to other individuals) of species in a community is the hypothesized mechanism underpinning biodiversity–disease relationships (Johnson and Thielges 2010, Johnson et al. 2019, Stewart Merrill et al. 2022). Dilution effects emerge when competence varies within the host community; in a species-rich community where most species are ‘dead-end’ hosts (ineffective disease spreaders, e.g. due to physical removal of parasites or rapid recovery from infection), biodiversity dilutes disease risk (Keesing et al. 2010, Ostfeld and Keesing 2012, Levi et al. 2016, Garrido et al. 2021). In contrast, species-poor communities – especially in areas that experience high anthropogenic disturbance – tend to be dominated by competent host species (Johnson et al. 2013, 2015, Han et al. 2015, Gibb et al. 2020). The Lyme disease system provides the canonical example of a dilution effect (Ostfeld 2011). Greater prevalence of *Borrelia burgdorferi* (the bacterium which causes Lyme disease) infection in vertebrate hosts is often observed in fragmented habitats in which small mammal communities are dominated by competent hosts,

especially *Peromyscus* mice, as compared to less-disturbed habitats in which host communities have greater proportions of non-competent hosts that deflect pathogens (termed ‘encounter reduction’; Johnson and Thielges 2010, Keesing and Ostfeld 2021). Alternatively, dilution effects may emerge through ‘vector regulation’ by which increased host diversity reduces vector abundance (Keesing et al. 2006). On the other hand, amplification effects – biodiversity associated with increases in disease risk – are predicted to emerge when species display similar levels of competence, or when changes in species richness are random in terms of the competence of species (Keesing et al. 2006, Halliday et al. 2017). However, like other ecosystem functions, different biodiversity–disease relationships may emerge when evaluated at different scales, even within the same system (Cohen et al. 2016, Liu et al. 2023).

Most biodiversity–disease research takes place at single scales, often a local scale as defined by the researchers. For example, some measure of disease risk (e.g. infection prevalence) is recorded within a plot and associated with some host biodiversity variable that is typically measured within that same plot (Halliday and Rohr 2019). However, several previous studies have investigated spatial scaling of biodiversity–disease relationships, some using meta-analyses and others using nested field designs (García-Peña et al. 2016, Cohen et al. 2016, Magnusson et al. 2020, Halle et al. 2022, Liu et al. 2023). In general, the expectation is that biotic variables (i.e. host diversity and composition) influence the distribution and abundance of host species and pathogens at fine spatial scales, while abiotic variables (i.e. climate) eclipse the influence of biotic ones at broad scales (the ‘Eltonian noise hypothesis’; Soberón and Nakamura 2009, McGill 2010, Cohen et al. 2016). In accordance with this expectation, Cohen et al. (2016) found that host diversity was only strongly associated with the distribution of three pathogens (*Batrachochytrium dendrobatidis*, West Nile virus and *Borrelia burgdorferi*) at finer 10^2 – 10^3 km² scales, while climate only showed significant associations with pathogen distributions at the broadest ($>10^4$ km²) scales considered. Focusing on a suite of much finer spatial scales (nested 0.02-, 0.06- and 0.25 m²-plots), Liu et al. (2023) found the strongest effect of plant diversity on foliar fungal disease severity at finer scales (0.02 m²), whereas associations between diversity and disease severity were weaker at broader scales (0.25 m²). In contrast to these two multiscale studies, Magnusson et al. (2020) performed a meta-analysis (considering multiple pathogen systems) to evaluate the influence of spatial scale (defined as the spatial extent over which diversity and disease metrics were estimated) on dilution effects and found similar strengths of dilution effects for spatial scales spanning several orders of magnitude. Finally, evaluating biological rather than spatial scale, Johnson et al. (2024) found that infection success for individual hosts (number of parasites per host) showed a negative relationship with host richness but that infection success for host communities (density of parasites across hosts in the community) was similar across the host richness gradient.

While most multiscale work assumes that biodiversity–disease relationships are strongest at fine spatial scales (Cohen et al. 2016, Magnusson et al. 2020, Liu et al. 2023), host biodiversity of the landscape surrounding a focal plot may complement or expand the explanatory power of biodiversity for disease risk (Johnson et al. 2015, 2016, García-Peña et al. 2016). This may occur if the diversity of the host metacommunity influences local disease risk in a different way than the diversity of the local host community. According to metacommunity theory, local communities do not exist in isolation but are enmeshed together, with dispersal of species between local communities forming the links (Leibold et al. 2004). Thus, movement of hosts and/or vectors likely underpins any effects of the host metacommunity on disease risk. For example, if a vector transmits a pathogen from a competent but sedentary host species that does not occur in a focal plot to another host species which then moves into the focal plot, the observed infection prevalence within that plot may be higher than expected based on the hosts that occur within the focal plot. Previous work indicates that prevalence of *Borrelia* infection is greater in fragmented habitats containing relatively depauperate local host communities, especially those dominated by competent hosts such as *Peromyscus* mice (Ostfeld 2011, Keesing and Ostfeld 2021). Although *Peromyscus* mice cannot disperse far, prevalence of *Borrelia* infections may be higher than predicted by presence or abundance of competent hosts within the local community if more vagile hosts (e.g. cervids or birds) transport infected ticks from an area with more competent hosts to an area with fewer competent hosts (Hasle et al. 2011, Leighton et al. 2012, Buck and Perkins 2018, VanAcker et al. 2023).

Previous investigations of biodiversity–disease relationships have varied in their methods from experimental manipulations of host community structure within mesocosms to macroecological analyses of disease and biodiversity data (Civitello et al. 2015, Wood et al. 2017). Each approach has its respective strengths and limitations, and there exist at least two key opportunities to expand inference regarding the links between biodiversity and disease risk. First, understanding the scaling of biodiversity–function relationships benefits from nested observations (i.e. multiple plots within sites), replicated over broad spatial extents (Thompson et al. 2018, Liu et al. 2023). Such practice allows fine-scale relationships to be quantified and compared to relationships of aggregated data representing broader spatial extents. Second, understanding biodiversity–disease relationships benefits from robustly estimating host biodiversity. In observational studies, biodiversity variables (e.g. abundance, species richness) are latent variables that cannot be directly observed; nevertheless, biodiversity–disease studies commonly use observed species richness (or other biodiversity variables) as predictors. Methods that account for imperfect detection in host surveys – such as capture–recapture and occupancy models – may thus provide a more accurate representation of biodiversity variables than counts that are not corrected for imperfect host detection (Kéry and Royle 2015).

Furthermore, performing model-based integration of biodiversity variables with disease data propagates the uncertainty in such biodiversity estimates, which may avoid overconfidence in associations between host biodiversity and disease risk (Behney 2020).

Here, our objective is to evaluate the spatial scaling behavior of biodiversity–disease relationships by comparing the relationships between host local diversity versus host metacommunity diversity on infection prevalence. We hypothesized that scale dependencies of biodiversity–disease relationships arise from movement of hosts and/or vectors (Wood and Johnson 2016). If host and/or vector movement is limited, we predict that diversity of the local host community will be more strongly associated with infection prevalence than the diversity of the host metacommunity. In other words, biodiversity's influence on disease risk is primarily a fine-scale process. Conversely, if host and/or vector movement is high, we predict that the diversity of the host metacommunity will show similar or stronger relationships with infection prevalence compared to the diversity of the local host community. In other words, biodiversity's influence on disease risk operates over multiple scales and is not necessarily strongest at the finest spatial scales.

Focusing on the Lyme disease system, we leverage continental datasets on small mammals and their *Borrelia* infection status from the National Ecological Observatory Network, or NEON (Keller et al. 2008, Thorpe et al. 2016, Barnett et al. 2019). Importantly, these data are collected at multiple plots nested within sites, allowing host biodiversity of local communities (plots; 0.008 km² in area) as well as metacommunities (all plots within a site; 9 km² area on average) to be quantified (Thorpe et al. 2016; Fig. 1). We focus inference on associations between infection prevalence and five host biodiversity metrics: 1) species richness (the canonical variable considered in biodiversity–disease literature), 2) *Peromyscus* dominance (the proportion of the total community abundance comprised of *Peromyscus* mice, the most competent hosts for *Borrelia burgdorferi*), 3) total small mammal abundance (relevant to disease risk if host competence shows limited among-species variation), 4) small mammal Shannon diversity (a measure of species' evenness; presumably more even communities are less likely to be dominated by competent hosts), and 5) small mammal phylogenetic diversity (if host competence is phylogenetically conserved, presumably communities comprised of distantly related species are associated with lower disease risk). We developed a two-stage modeling approach consisting of a multispecies capture–recapture model that enables estimation of host biodiversity metrics while accounting for imperfect detection of hosts followed by a second set of models in which these biodiversity metrics (calculated for local host communities as well as host metacommunities) were used to explain variation in *Borrelia* infection probability within individual small mammal hosts. This model provides a framework to guide future analyses as NEON data continue to be collected and will also be applicable to similar nested sampling scenarios.

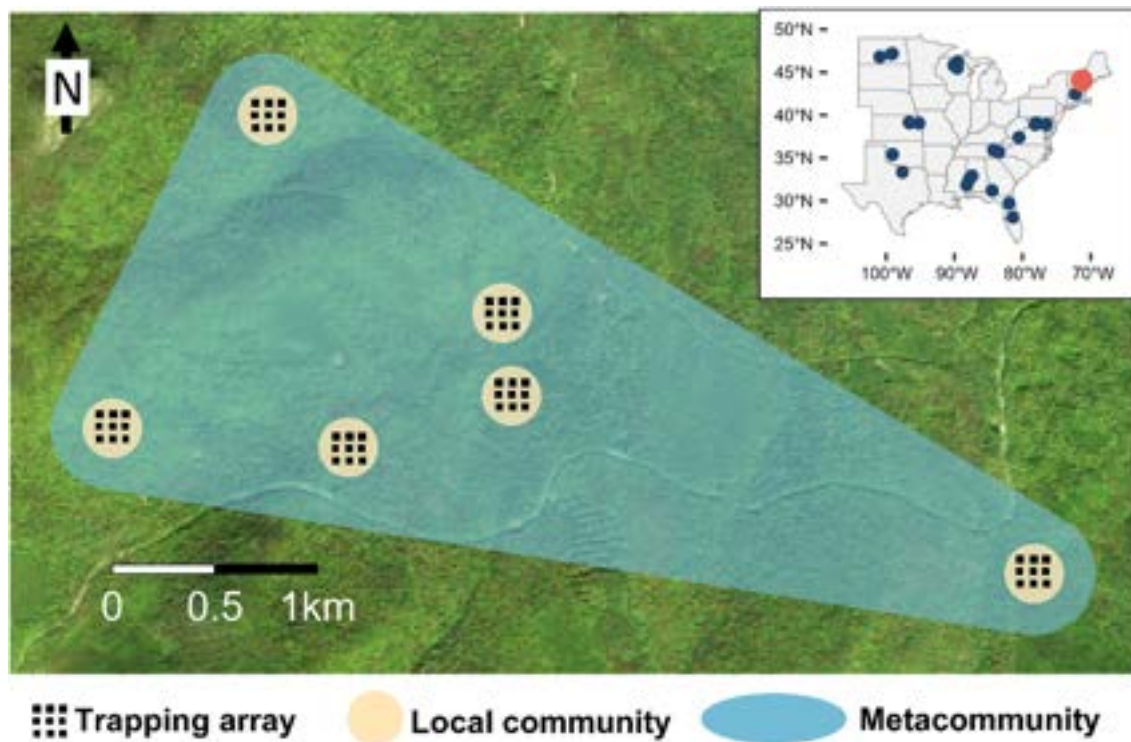


Figure 1. The National Ecological Observatory Network's (NEON) hierarchical sampling scheme permits quantification of small mammal biodiversity at local community and metacommunity levels. Arrays of box traps (black squares; 3×3 grids shown for simplicity but 10×10 grids are used) sample local small mammal communities (tan circles), and the collection of arrays characterizes the small mammal metacommunity (blue polygon) of the broader NEON site. Inset: the distribution of NEON sites in the eastern US that are used in the study (red circle is the Bartlett Experimental Forest site shown in the enlarged map; dark blue circles are the other 21 NEON sites used in the analysis).

Material and methods

Data description

Small mammal box trapping

Small mammals are monitored at 44 NEON sites across the US (Barnett et al. 2019); we focused on 22 NEON sites in the eastern half of continental North America (Fig. 1), where Lyme disease is most prevalent (Ostfeld 2011). Within each NEON site, small mammals are trapped at 1–8 (median: 6) plots (Fig. 1). Nearest-neighbor distances between plots within a site ranged from 0.25 to 1.85 km (median: 0.36 km); the maximum distance between plots within a site ranged from 1.2 to 17.8 km (median: 4.8 km). Each plot is 90×90 m (for an area of 8100 m^2) and contains 10 rows and columns of Sherman traps, separated by 10 m, such that there are 100 traps per plot (Kao et al. 2012). Thus, a plot is $\sim 2\text{--}20\times$ larger than the home range size of most of the small mammal species sampled (which have home ranges on the scale of several hundred to several thousand m^2), and most plots are separated by distances far greater than those typically traveled by small mammals (Fleaharty and Mares 1973, Cameron and Spencer 1985, Jones 1990, Lacher and Mares 1996, Ribble et al. 2002, Wilder et al. 2005, Getz and McGuire 2008, Tisell et al. 2019, Underhill et al. 2021).

However, a plot is at least one or two orders of magnitude smaller than the typical home range size of white-tailed deer *Odocoileus virginianus* (Stewart et al. 2011), another common host for ticks, and the spacing between plots is smaller than documented dispersal distances for deer (Long et al. 2005) and much smaller than distances over which migratory birds have transported ticks (Klich et al. 1996, Hasle et al. 2011).

There are up to six trapping bouts per year; each bout consists of 1–3 consecutive nights of trapping within 10 days of the new moon (to control for variation in small mammal activity due to moonlight, which increases perceived predation risk; Hinkelman et al. 2012). We used data from 2020 and 2021 only (June–November) because screening for tick-borne pathogens in small mammals only began in 2020. Trapped animals are tagged with either ear tags or passive integrated transponder (PIT) tags so that they can be individually identified upon recapture. We filtered the dataset to retain only animals within orders Rodentia (rodents) and Soricomorpha (shrews), thus omitting occasional bycatch of other taxa, such as weasels. Finally, we omitted individuals that were not identified to the species level. Given the short lifespans of the focal species and in the interest of keeping model structure relatively simple, we treat between-bout recaptures as separate individuals (i.e. we estimate closed-population

abundance for each bout and do not model survival, recruitment, etc. between bouts); 79% of individuals were captured during only one bout, 14% were captured during two bouts, 5% were captured during three bouts, and 1% were captured during four bouts. All told, the filtered dataset included 7902 captures of 4266 individuals of 36 species (see the Supporting information for a list of the species included and the number of captures per species).

Rodent pathogen screening

NEON scientists test blood and/or ear tissue samples for pathogens from a subset of small mammals captured via box trapping (Springer et al. 2016). Up to ~140 samples are tested per NEON site in a typical year. Blood samples are collected using the submandibular method for individuals weighting > 10 g and meeting criteria for good physical condition; ear samples are collected using a 2 mm biopsy punch (NEON 2024). Samples are stored on dry ice for transfer from the field and stored in a -80°C freezer until being transferred (on dry ice) to the University of Massachusetts Laboratory for Medical Zoology, where they are screened for pathogens using PCR (Springer et al. 2016, NEON 2024). We obtained 1391 pathogen screening tests for 1360 individuals (approximately a third of the captured individuals in our filtered dataset) representing 16 species. Thirty-one individuals, or approximately 2% of all individuals tested, were screened twice when captured during different trapping bouts. The number of tests per species ranged from 1 for *Reithrodontomys humulis* to 595 for *Peromyscus leucopus* (Supporting information). Most observations (775, or 56%) had both blood and ear tissue samples screened; 345 (25%) had only ear tissue screened, and 271 (19%) had only blood samples screened.

A supplemental analysis indicated that observations with both sample types were not more likely to be associated with a positive test result, and we thus did not include sample type in our analyses (Supporting information). The number of animals tested per NEON site ranged from 1 to 178 (mean: 63; SD: 50) and was relatively even across space, though fewer animals were screened in some of the southern sites (Fig. 2). We used data from 2020 onwards because screening for tick-borne pathogens only began in 2020. For the purposes of our analysis, we aggregated test results across *Borrelia burgdorferi* and *Borrelia* spp. categories to derive presence-absence of *Borrelia* infection for individual animals that were tested. Of the 48 samples which tested positive, 9 (18.8%) tested positive only for the *Borrelia* spp. category while the remainder tested positive for both categories.

Modeling biodiversity–disease relationships

Overview

We developed hierarchical Bayesian models to estimate the relationship between *Borrelia* infection prevalence and host biodiversity (Supporting information). We used a two-stage approach in which we first fit a multispecies capture–recapture model to estimate the abundance of each species for each plot, site, and trapping bout combination. We then used the posterior distributions of these abundance estimates to compute (with uncertainty propagated) small-mammal biodiversity metrics for NEON plots (i.e. local community scale) as well as aggregated biodiversity metrics for entire NEON sites (i.e. metacommunity scale; Fig. 1). Finally, we evaluated

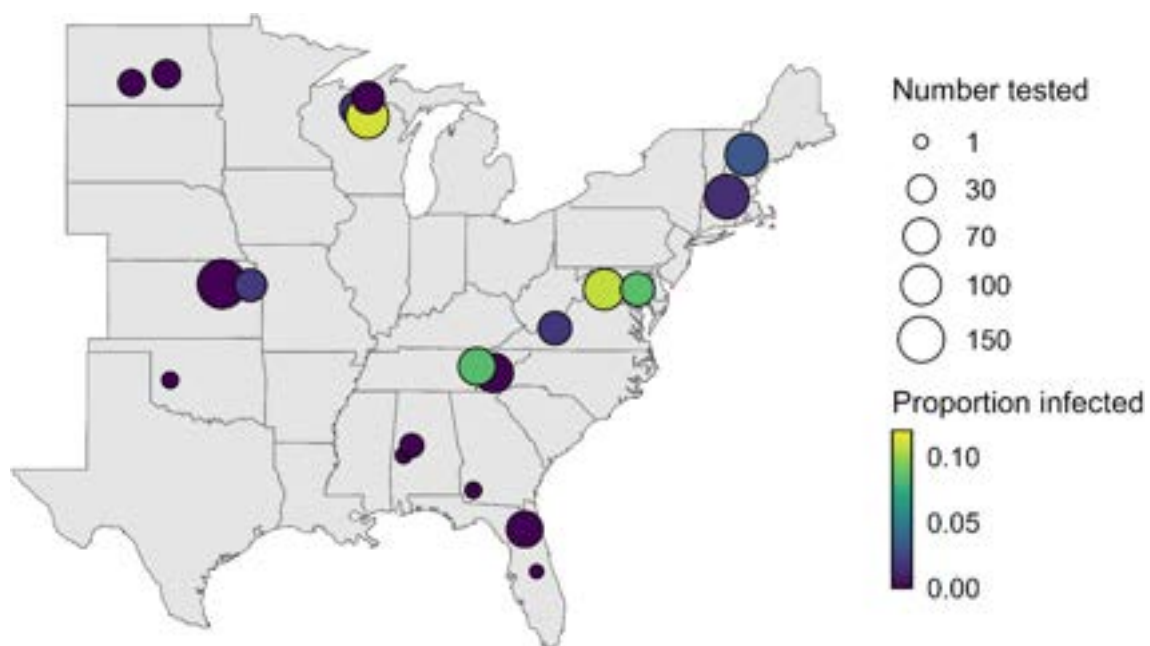


Figure 2. Map of *Borrelia* surveillance data showing the number of individual small mammals tested (size of point) and the proportion of individuals in which an infection was detected (fill of point) per NEON site across all trapping bouts.

secondary models that used these biodiversity metrics at local and metacommunity scales as predictors of *Borrelia* infection status of individual small mammal hosts (Table 1).

Stage 1: estimating small mammal biodiversity metrics

We fit a multispecies capture–recapture model to estimate species-specific abundances, which we then used to compute biodiversity metrics at the local and metacommunity scales. Importantly, in estimating species abundances, the model estimates species-specific detection probabilities from patterns of captures (and non-captures) of individuals within a trapping bout. Thus, the model accounts for imperfect detection of small mammals, providing more accurate estimates of abundance than indices such as the minimum number known alive (Parsons et al. 2022).

In the abundance portion of the multispecies capture–recapture model, we included a random intercept for each species–site–plot–bout combination to account for variation in abundance among species, sites, plot, and trapping bouts. We drew intercepts for each species–site–plot–bout combination from a species-specific distribution, the parameters for which were in turn drawn from a community-level distribution (Supporting information). We also included a quadratic effect of percent canopy cover – allowing effects to vary by species – to explain variation in abundance among plots because some species are likely to be most abundant in landscapes with intermediate levels of canopy cover (Supporting information). We calculated canopy cover as the mean percent canopy cover within a 500 m buffer of the center of each NEON plot using the National Land Cover Database canopy cover product (Wickham et al. 2021). We also constrained the model such that species' abundances were fixed to zero outside of their geographic ranges. We obtained this information by downloading range shapefiles from the International Union for Conservation of Nature website (IUCN 2016) and, for each species, calculating whether a NEON site was within or outside of the range polygon. In the detection portion of the multispecies capture–recapture model, we modeled detection probability as a logit-linear

function of an intercept (which varied by species), a linear effect of daily precipitation (the coefficient of which also varied by species), and linear and quadratic effects of daily minimum temperature (the coefficients for which varied by species). We used a linear effect for precipitation because we assumed that small mammal activity generally decreases with increasing precipitation but a quadratic effect for temperature because small mammal activity is likely to be reduced at both extremely low and extremely high temperatures. We derived these weather variables for each NEON site and day of sampling using Daymet accessed via the 'daymetr' R package (Hufkens et al. 2018), which provides interpolated daily temperature and precipitation data at 1 × 1 km spatial resolution from a national network of > 10 000 weather stations (Thornton et al. 2022).

The multispecies capture–recapture model produces estimates of abundance for each species, site, plot, and trapping bout in a four-dimensional array (species, NEON site, NEON plot, trapping bout). We used the posterior distributions of these abundance estimates to compute biodiversity metrics with full uncertainty (Kéry and Royle 2015, Supporting information). We calculated five biodiversity metrics for each plot (local community) and aggregated across plots within a NEON site (metacommunity): species richness, total abundance, *Peromyscus* dominance, Shannon diversity, and phylogenetic diversity. To calculate species richness, we summed the number of species with abundance greater than zero. We calculated total abundance as the abundance of all species within a plot or aggregated across a NEON site. We calculated *Peromyscus* dominance as the proportion of total small mammal abundance comprised of species in the mouse genus *Peromyscus*. Shannon diversity provides a measure of species diversity based on the proportional abundances of species in a sample, with larger values indicating more even and diverse communities. Finally, we used an abundance-weighted version of Faith's phylogenetic diversity, which is computed by summing the branch lengths of the phylogeny of the species occurring in a sample; larger values indicate higher phylogenetic diversity of the community (Tucker et al. 2017). We obtained a phylogeny of the species occurring in our dataset from VertLife (Upham et al. 2019, Supporting information).

Table 1. The 10 univariate logistic regressions to assess the influence of five host biodiversity metrics at two scales (local community: NEON plots; metacommunity: aggregated NEON plots within a NEON site) on *Borrelia* infection prevalence within individual small mammals.

Scale	Host biodiversity metric
Local community	Species richness
Metacommunity	Species richness
Local community	<i>Peromyscus</i> dominance
Metacommunity	<i>Peromyscus</i> dominance
Local community	Total abundance
Metacommunity	Total abundance
Local community	Shannon diversity
Metacommunity	Shannon diversity
Local community	Phylogenetic diversity
Metacommunity	Phylogenetic diversity

Stage 2: estimating biodiversity–disease relationships

For the second stage, we evaluated ten univariate logistic regressions: *Borrelia* infection status (infected: 1; not infected: 0) of individual small mammals predicted by small mammal species richness, *Peromyscus* dominance, total small mammal abundance, small mammal Shannon diversity, or small mammal phylogenetic diversity, fitting separate models for each biodiversity metric at the plot (local community) and NEON site (metacommunity) scales (Table 1). As a supplemental analysis, we also evaluated additional models that each contained two biodiversity variables and their interaction (Supporting information). We omitted five observations for the metacommunity-level models that came from

trapping bouts during which only one plot was sampled for a NEON site. We included a random intercept for species to account for differences in competence among species tested and included an additional random effect for NEON site to account for baseline differences in *Borrelia* prevalence across the sites (Supporting information). Importantly, we used the full distribution of each biodiversity metric obtained from the multispecies capture–recapture model in stage 1, rather than a point estimate, and thus propagated uncertainty in the biodiversity metrics (Behney 2020, Gilbert et al. 2023). We z-standardized (i.e. subtracted the mean and divided by the SD) the biodiversity metrics to facilitate comparisons between scales.

We fit all models using ‘NIMBLE’ in the R environment (see the Supporting information for full details; de Valpine et al. 2017, www.r-project.org). Below, we report the posterior means, 95% credible intervals (95% CI), and the proportion of the posterior greater than 0 (i.e. $p > 0$) for the coefficients relating biodiversity metrics to infection prevalence. To evaluate the scaling behavior of biodiversity–disease relationships, we compare the effect size of each biodiversity metric at the plot and site scales; we computed the differences between the effects’ posterior distributions (site – plot) and summarized the proportion of differences (across posterior samples) greater than zero, which can be interpreted as the probability that the metacommunity effect had a larger value (numerical value, not absolute magnitude) than the local community effect.

Results

Host biodiversity metrics showed dramatic spatial variation across NEON sites (Fig. 3). For an individual trapping bout, the posterior mean of metacommunity species richness ranged from a minimum of 1.5 [95% CI: 0, 4] species in October 2021 at Disney Wilderness Preserve in south-central Florida to a maximum of 11.6 [95% CI: 9, 14] species in July 2020 at Treehaven in northern Wisconsin (Fig. 3). *Peromyscus* dominance ranged from 0.13 [95% CI: 0.02,

0.29] in August 2021 at the Northern Great Plains Research Laboratory in west-central North Dakota to 0.93 [95% CI: 0.83, 0.99] in October 2021 at Chase Lake National Wildlife Refuge in east-central North Dakota (Fig. 3). Total small mammal abundance across all plots at NEON sites ranged from 2.3 individuals [95% CI: 0, 8] in October 2021 at Disney Wilderness Preserve in south-central Florida to 413 [95% CI: 337, 487] in August 2020 at Konza Prairie Biological Station in eastern Kansas (Fig. 3). Shannon diversity ranged from 0.36 [95% CI: 0.01, 1.24] in October 2021 at Disney Wilderness Preserve in south-central Florida to 2.01 [95% CI: 1.81, 2.19] in July 2020 at Harvard Forest in Massachusetts (Fig. 3). Finally, phylogenetic diversity ranged from 51.60 [95% CI: 1.95, 205] in October 2021 at Disney Wilderness Preserve to 432 [95% CI: 332, 582] in May 2021 at Mountain Lake Biological Station in southwestern Virginia (Fig. 3). The biodiversity metrics showed minor variation over time, though spatial variation among sites was much more apparent than temporal variation within sites (Supporting information). Within NEON sites, at the plot level, biodiversity metrics showed comparatively less variation than metacommunity biodiversity metrics among sites. For example, the SD among species richness estimates across iterations and plots for an individual trapping bout within a site ranged from 0.61 (Disney Wilderness Preserve in October 2021) to 3.29 (Treehaven in July 2020).

Associations between *Borrelia* infection prevalence and host biodiversity showed idiosyncratic evidence of scale dependence, i.e. the biodiversity metrics displayed different scaling behaviors (Fig. 4). The sign of species richness’ effect on infection prevalence switched from negative at the local community scale to positive at the metacommunity scale (Fig. 4). Infection prevalence showed a stronger, more negative relationship with *Peromyscus* dominance and total abundance at the local community scale than the metacommunity scale (Fig. 4). In contrast, infection prevalence showed stronger, more positive relationships with Shannon diversity and phylogenetic diversity at the metacommunity scale than the local community scale (Fig. 4). We found similar results for a model that accounted for phylogenetic relationships

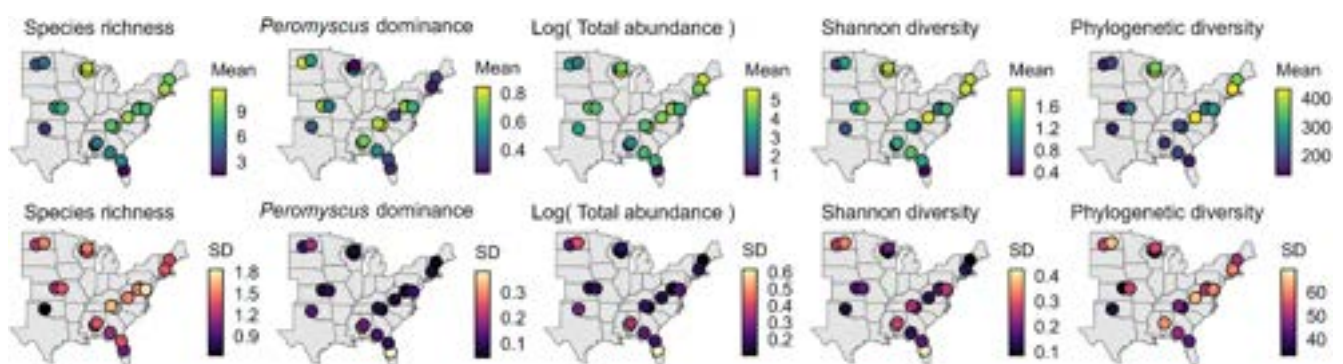


Figure 3. Estimated host biodiversity metrics across NEON sites (top row: posterior mean; bottom row: posterior SD). The plotted values are site-level (metacommunity) biodiversity metrics from the first trapping bout during 2021 (dates vary among sites; most are in late spring or summer).

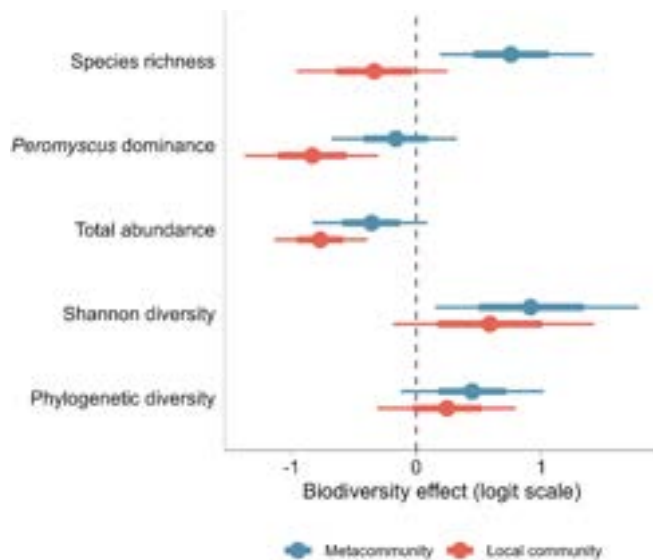


Figure 4. Associations between *Borrelia* infection probability in individual small mammals and five biodiversity metrics summarized at local community (NEON plots; red) and metacommunity (NEON sites; blue) scales. Points represent posterior means, while thin and thick bars represent 68 and 95% credible intervals, respectively. The dashed vertical line at zero indicates a lack of an effect.

between the species that were screened (Poulin et al. 2011), for models that accounted for variation in plot spacing within sites and for the number of plots sampled, and for models that included two biodiversity metrics and their interaction (Supporting information).

Species richness of the local community showed a weak negative relationship with infection prevalence (posterior mean and 95% credible interval of coefficient estimate on the logit scale: -0.34 [95% CI: $-0.96, 0.25$]; $p > 0.14$; Fig. 4–5). In contrast, species richness of the metacommunity showed a clear positive relationship with infection prevalence (0.76 [95% CI: $0.19, 1.42$]; $p > 0.99$; Fig. 4–5). The probability that the effect of metacommunity richness was larger than the effect of local community richness (the numerical value of the effect, not its absolute magnitude) was 0.99 (Supporting information). *Peromyscus* dominance showed a clear negative association with *Borrelia* infection at the local community scale (-0.83 [95% CI: $-1.38, -0.30$]; $p > 0.00$), but for the metacommunity scale, the weak negative association was not appreciably different from zero (-0.16 [95% CI: $-0.68, 0.33$]; $p > 0.27$; Fig. 4). The probability that the effect of metacommunity *Peromyscus* dominance was larger than the effect of local community *Peromyscus* dominance was 0.97 (Supporting information). However, an alternative metacommunity-level model that contained both species richness and *Peromyscus* dominance estimated a positive effect of *Peromyscus* dominance and a weak interaction with species richness such that *Peromyscus* dominance showed a stronger positive association with infection probability at low levels of species richness (Supporting information). Similarly, total small mammal abundance

showed a clear negative relationship with infection prevalence at the local scale (-0.77 [95% CI: $-1.14, -0.39$]; $p > 0.00$) but a comparatively weak negative relationship with infection prevalence at the metacommunity scale (-0.36 [95% CI: $-0.83, 0.09$]; $p > 0.05$). The probability that the effect of metacommunity abundance was larger than the effect of local community abundance was 0.92 (Supporting information). Shannon diversity showed a positive association with *Borrelia* infection probability (Fig. 4). This effect was stronger at the metacommunity scale (0.91 [95% CI: $0.15, 1.78$]; $p > 0.99$) than at the local community scale (0.59 [95% CI: $-0.19, 1.42$]; $p > 0.92$). The probability that the effect of metacommunity Shannon diversity was larger than the effect of local community Shannon diversity was 0.70 (Supporting information). Finally, phylogenetic diversity showed a positive association with *Borrelia* infection probability (Fig. 4). The effect was stronger for the metacommunity scale (0.45 [95% CI: $-0.12, 1.02$]; $p > 0.95$) than for the local community scale (0.25 [95% CI: $-0.31, 0.80$]; $p > 0.82$). The probability that the effect of metacommunity phylogenetic diversity was larger than the effect of local community phylogenetic diversity was 0.70 (Supporting information).

Borrelia infection prevalence showed considerable geographic variation beyond what was explained by the biodiversity metrics (Fig. 5). For example, focusing on estimates from the local-community species richness model with species richness held at its average value, *Borrelia* infection probably was estimated to be as high as 0.11 [0.05, 0.21] at Treehaven but as low as 0.00 [0.00, 0.02] at Konza Prairie (Fig. 5). In general, the proportion of individuals that tested positive for *Borrelia* infection was higher in the mid-Atlantic and upper Midwest compared to other locations in the eastern US (Fig. 2).

Discussion

We found idiosyncratic evidence of scale dependence in biodiversity–disease relationships (Fig. 4–5), with some relationships defying expectations. For example, while species richness of the local community had the expected negative (dilution) relationship with infection probability, species richness of the metacommunity showed an unexpected positive (amplification) relationship with infection probability (Fig. 4). While the sign of the relationship with infection prevalence did not change for any of the other biodiversity metrics (Fig. 4), both *Peromyscus* dominance and total abundance showed much stronger negative relationships with infection prevalence at the local community scale, contrasting with Shannon diversity and phylogenetic diversity which showed stronger positive relationships (an unexpected amplification effect) with infection prevalence at the metacommunity scale (Fig. 4). The results for species richness, phylogenetic diversity and Shannon diversity suggest that metrics of biodiversity in the local area may not reveal the full nature of biodiversity's influence on disease dynamics.

Dispersal of vectors and/or pathogens via unmonitored, more-vagile hosts may expand the spatial scale over which

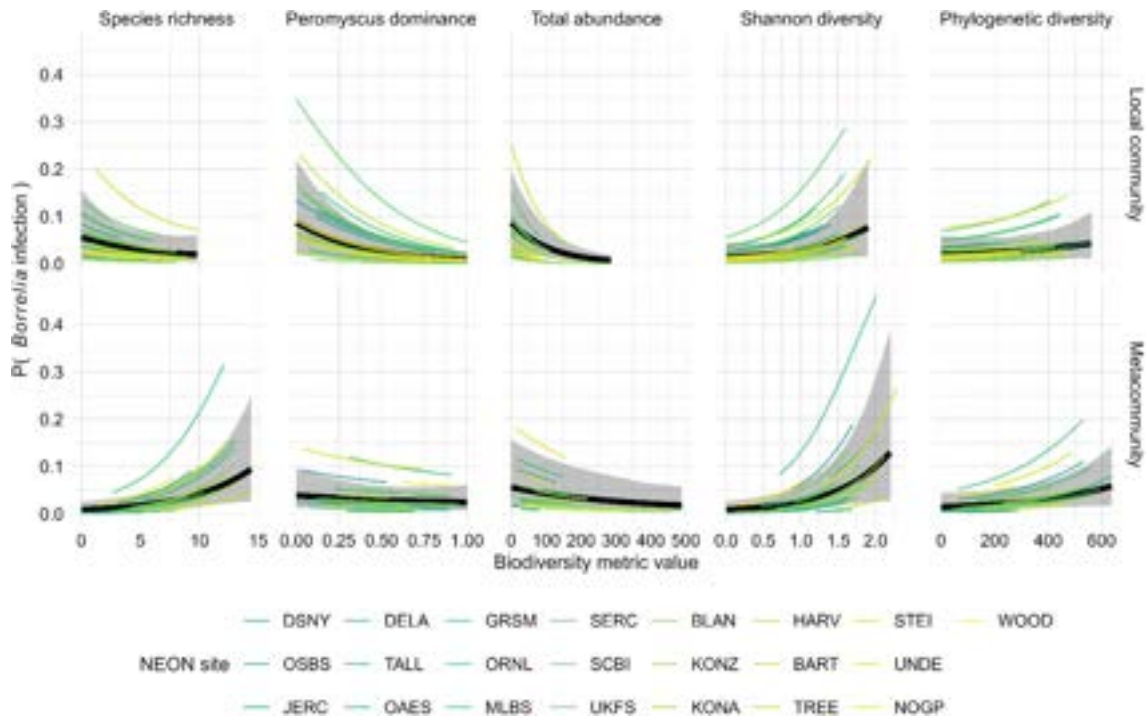


Figure 5. Relationships between *Borrelia* infection probability in individual small mammals and five host biodiversity metrics quantified at local community (top row) and metacommunity (bottom row) scales. Black lines and shaded regions are the mean and 95% credible interval, respectively, for the relationship averaged across sites. Colored lines are the estimated mean relationships for individual NEON sites. See the Supporting information for a key relating the NEON site abbreviations to their full names.

host diversity interacts with infection prevalence (Buck and Perkins 2018, Dougherty et al. 2018). While the members of the focal small mammal communities seldom move farther than a few hundred meters (Jones 1990, Wilder et al. 2005, Getz and McGuire 2008), species richness of small mammals over a broader spatial extent may influence local disease dynamics if ticks can be transported by comparatively mobile hosts such as cervids and birds (Hasle et al. 2011, VanAcker et al. 2023). For example, by pairing tick surveys and high-resolution tracking data of white-tailed deer *Odocoileus virginianus*, VanAcker et al. (2023) found that ticks were more likely to occur in urban backyards which were regularly visited by deer from nearby urban greenspaces than in backyards which were not visited by deer.

In contrast to the species richness, phylogenetic diversity and Shannon diversity results, *Borrelia* infection probability showed stronger associations with *Peromyscus* dominance and total abundance at the local scale than the metacommunity scale (Fig. 4). This suggests idiosyncrasies in scaling behavior of biodiversity–disease relationships such that different facets of biodiversity affect infection prevalence at different spatial scales (Ostfeld 2011). These scaling idiosyncrasies may arise partly because of how the biodiversity metrics themselves change with spatial scale (Supporting information). Species richness and total abundance either stay the same or – more commonly – increase with a shift from the local community to the metacommunity scale. Shannon diversity and (abundance-weighted) phylogenetic diversity can take on higher

values at the local community versus the metacommunity scale but usually increase with a shift from the local community to the metacommunity scale. By contrast, *Peromyscus* dominance is more likely to take on higher values at the local community scale versus the metacommunity scale because it is less likely for one or a few species to dominate over a larger area thanks to associated increases in environmental heterogeneity (Supporting information). Finally, habitat and landscape context may influence the scaling behavior of biodiversity–disease relationships; for example, García-Peña et al. (2016) found that metacommunity diversity of hosts (aquatic vertebrates and macroinvertebrates) showed a positive relationship with infection prevalence of *Mycobacterium ulcerans* in lotic habitats but a negative relationship in lentic habitats, which may be related to differences in movement behaviors of hosts in the two habitats.

Aside from the scaling behavior of the biodiversity–disease relationships, the direction of several of the estimated biodiversity–disease relationships were unexpected. For example, the positive relationship (amplification effect) between infection probability and metacommunity species richness contrasts with existing literature that has documented dilution effects of species richness in Lyme systems (Ostfeld 2011, Keesing and Ostfeld 2021). Moreover, the negative relationship between infection prevalence and *Peromyscus* dominance of the local community opposed our prediction that infection prevalence would increase as the proportion of competent hosts in the community rises (Johnson and Thielges 2010,

Keesing and Ostfeld 2021). We suggest three potential explanations (which are not mutually exclusive) of these surprising results. First, the shape of the underlying biodiversity–disease relationship, together with the breadth of biodiversity sampled (i.e. what portion of the x-axis is represented in the dataset) can determine the sign and magnitude of estimated biodiversity–disease relationships (Rohr et al. 2020). In this study, we focused on small mammal communities because of their importance as hosts for ticks and because of the availability of monitoring data. However, ticks feed on many other vertebrate taxa (e.g. lagomorphs, ungulates, birds) that are not included in our analysis (and in many cases not monitored by NEON), and thus small mammal biodiversity may not represent a broad enough spectrum of diversity to capture a dilution effect if the underlying biodiversity–disease relationship is right-skewed (Rohr et al. 2020). Second, unexpected results may have emerged due to the locations of NEON sites. NEON sites are situated in relatively natural landscapes: percent impervious cover within 5 km of NEON plots – a proxy for the degree of human influence in the landscape (Moll et al. 2019) – averaged only 1.7% (SD: 2.9%). Dilution effects have been described in studies comparing multiple locations that vary in their degree of landscape disturbance (which correlates with changes to species composition and richness), and thus, replicate NEON plots within a NEON site may not capture these dynamics for a given species pool, even though species richness did vary among NEON sites (Fig. 3). Third, and related to the previous point, the NEON sites cover a broad geographic region, and the prevalence of Lyme disease varies considerably over the eastern US due to factors not directly related to host biodiversity (e.g. climate, dispersal). For example, the unexpected negative relationship with *Peromyscus* dominance may have arisen because some of the NEON sites (e.g. Konza in Kansas and Chase Lake in North Dakota) had high levels of *Peromyscus* dominance but limited presence of *Borrelia*, while NEON sites with high presence of *Borrelia* detected happened to have low or moderate values of *Peromyscus* dominance (Fig. 2–3).

We describe a novel modeling approach that leverages emerging – and publicly available – data from the National Ecological Observatory Network to estimate biodiversity–disease relationships and how they scale over space. The hierarchical sampling design of NEON (i.e. plots nested within sites) enables the characterization of both local communities and metacommunities of small mammals, which are important hosts of tick-borne illnesses. The multispecies capture–recapture modeling approach we apply has several benefits, including 1) accounting for imperfect detection of hosts and 2) improving inference for rare species because information is shared across members of the community (Zipkin et al. 2023). By accounting for imperfect host detection, our model provides more complete estimates of abundance than one-off surveys (Parsons et al. 2022). In turn, these abundance estimates can be used to calculate biodiversity metrics with associated uncertainty, and propagating this uncertainty in estimating biodiversity–disease relationships helps avoid overconfidence

in estimated relationships between infection prevalence and biodiversity metrics (Behney 2020, Gilbert et al. 2023). The sampling effort required for capture–recapture data has historically restricted the spatiotemporal scope over which capture–recapture models are applied (Kéry and Schaub 2012, Parsons et al. 2022), but NEON’s standardized, continental sampling offers unprecedented opportunities to inform macroecological questions using standardized data. As NEON is still in its early years – especially the rodent pathogen screening data product, which only debuted in 2020 – our approach promises to inform future efforts to quantify small mammal biodiversity at continental scales and better understand variation in biodiversity–disease relationships.

Our work suggests that conclusions about biodiversity–disease relationships rest partially upon the spatial scale at which biodiversity metrics are measured. This is relevant to a human health perspective because, for example, local-scale management efforts intended to reduce infection prevalence (e.g. altering habitat structure to reduce abundance of competent hosts) may be spatially mismatched with the space use of the average human and thus have limited effectiveness in reducing disease risk for people. Our results also differ from previous studies which have found either stronger biodiversity–disease relationships at finer spatial scales (Cohen et al. 2016, Liu et al. 2023) or limited change in biodiversity–disease relationships among spatial scales (Magnusson et al. 2020). Moreover, the five biodiversity metrics we evaluated showed inconsistent scaling behaviors, suggesting that different biodiversity components (e.g. species richness versus total community abundance) influence infection prevalence through idiosyncratic pathways. Some of our unexpected findings (e.g. the negative relationship between *Peromyscus* dominance and infection probability) could be the result of NEON’s sampling scheme, highlighting future challenges for using these data to understand biodiversity–disease relationships. Our efforts are the first to analyze NEON small mammal data with a multispecies, multi-site capture–recapture model, which enables streamlined quantification of biodiversity metrics while accounting for imperfect host detection. Further accumulation of these data will facilitate better understanding of temporal variation in biodiversity–disease relationships, since temporal heterogeneity (e.g. phenology of vectors or interannual variation in environmental factors that influence host community structure) may strongly influence infection prevalence and interact with spatial mediators of disease risk (Halle et al. 2022). Such multi-species frameworks promise to facilitate future investigations into the spatiotemporal dynamics of biodiversity–disease relationships and other macroecological analyses of mammal biodiversity.

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Data availability statement

Data are available at: <https://doi.org/10.5066/P13P2VWG> (Gilbert et al. 2024) and at GitLab (<https://code.usgs.gov/cooperativeresearchunits/massachusetts/ideosyncratic-spatial-scaling>).

Supporting information

The Supporting information associated with this article is available with the online version.

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