Fine-scale spatial patterns of wildlife disease are common and understudied

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Abstract

All pathogens are heterogeneous in space, yet little is known about the prevalence and scale of this spatial variation, particularly in wild animal systems. To address this question, we conducted a broad literature search to identify datasets involving diseases of wild mammals in spatially distributed contexts. Across 31 such final datasets featuring 89 replicates and 71 host-parasite combinations, only 51% had previously been used to test spatial hypotheses. We analysed these datasets for spatial dependence within a standardised modelling framework using Bayesian linear models. We detected spatial autocorrelation in 44/89 model replicates (54%) across 21/31 datasets (68%), spread across parasites of all groups and transmission modes. Surprisingly, although larger sampling areas more easily detected spatial patterns, even some very small study areas (under 0.01km^2) exhibited substantial spatial heterogeneity. Parasites of all transmission modes had easily detectable spatial patterns, implying that structured contact networks and susceptibility effects are likely as important in spatially structuring disease as are environmental drivers of transmission efficiency. Our findings imply that fine-scale spatial patterns of infection often manifest in wild animal systems, whether or not the aim of the study is to examine environmentally varying processes. Given the widespread nature of these findings, studies should more frequently record and analyse spatial data, facilitating development and testing of spatial hypotheses in disease ecology.

Introduction

The maintenance and spread of pathogens are inherently spatially structured processes (Cross *et al.* 2005; Pullan *et al.* 2012; Kirby *et al.* 2017). Many pathogens are transmitted from one host individual to another via direct contact, which requires a degree of spatiotemporal overlap (Manlove *et al.* 2018), so that diseases are spatiotemporally staggered in waves of transmission across the population. Other pathogens transmit through persistence in the environment or depend upon arthropod vectors, so that exposure depends heavily on spatially varying abiotic conditions (Patz *et al.* 2000; Altizer *et al.* 2006; Jamison *et al.* 2015). Furthermore, host immunity and susceptibility can be affected by environmentally varying factors, with knock-on impacts on pathogen burden and transmission (Becker *et al.* 2018, 2020). All these and other processes will create spatial patterns of infection, which hold important ramifications for epidemiological dynamics and disease control efforts (Cross *et al.* 2005; Plowright *et al.* 2019; Becker *et al.* 2020). Yet, many epidemiological studies examine coarse spatial scales or assume that spatial patterns will be negligible compared to other hypothesized drivers. As such, it is unclear how often disease is spatially structured in wild systems, at what range this variation can manifest, and how host and parasite traits might alter the manifestation of spatial variation.

For logistical reasons, many studies of infectious disease in wild systems focus on either single populations or address between-population differences. However, recent work suggests that spatial patterns of infection may manifest at surprisingly fine spatial scales, within kilometres or even metres (Brooker *et al.* 2006; Wood *et al.* 2007; Abolins *et al.* 2018; Albery *et al.* 2019). Because wildlife disease studies often use a limited number of discrete sampling locations rather than distributing their sampling locations continuously or randomly in space (Plowright *et al.* 2019), the lower bound for the range at which spatial effects can act has yet to be established. Identifying the range of spatial dependence in wildlife disease systems is important for many reasons, including designing sampling regimes (Nusser *et al.* 2008; Vidal-Martínez *et al.* 2010; Plowright *et al.* 2019), building mechanistic models of pathogen evolution over space (Best *et al.* 2011; Débarre *et al.* 2014), examining how disease risk responds to anthropogenic activities such as urbanisation (Saito & Sonoda 2017), and directing public health and conservation schemes (Brooker *et al.* 2006; Gilbertson *et al.* 2016). Perhaps most importantly, identifying the range of spatial dependence can help to examine how pathogens spread over landscapes and to determine transmission mechanisms

(Reynolds 1988). For example, spatial dependence in infection across large scales can suggest the influence of major climatic correlates, whereas spatial dependence only between nearby locations can suggest a highly localized infection process (Pullan *et al.* 2012). In human disease systems, such work has shown that neighbouring districts of Thailand have more similar human malaria incidence, suggesting local similarities in abiotic conditions or vector control programs that could limit mosquito survival (Zhou *et al.* 2005). Similar analyses of wildlife disease could help pinpoint transmission routes and guide disease control effort. Lastly, the scale of spatial dependence also has implications for more general theoretical understandings of infectious disease. Most notably, links between biodiversity and vector-borne diseases (i.e., "dilution effects") are dependent on the spatial scale of sampling (Cohen *et al.* 2016; Rohr *et al.* 2020), and several rodent systems have also identified contrasting spatial trends for zoonotic diseases dependent on sampling scale (Luis *et al.* 2018; Morand *et al.* 2019).

Because the range of spatial variation in infection depends on environmental gradients across the host population, traits of hosts and parasites are also likely to determine the contexts under which spatial dependence will occur. For example, parasites that persist for longer in the environment are likely to experience stronger influences of environmental gradients than directly transmitted counterparts (Satterfield *et al.* 2017). Similarly, large, mobile species, such as large carnivores or nomadic bats, may more efficiently disseminate pathogens through the environment, reducing their spatial autocorrelation (Peel *et al.* 2013; Gilbertson *et al.* 2016). However, the relative contribution of host and parasite traits to shaping spatial variation in infection remains unknown. The range of spatial dependence is most commonly identified using spatial autocorrelation models (e.g. (Brooker *et al.* 2006; Wood *et al.* 2007; Gilbertson *et al.* 2016; Albery *et al.* 2019; Becker *et al.* 2019) or analyses that quantify the spatial buffer regions in which environmental variables are best-correlated with disease (e.g. (Saito & Sonoda 2017). Unfortunately, these approaches are almost always reactive and occur on a case-by-case basis. To establish general factors influencing the scale of spatial dependence in wildlife disease, multiple host-parasite systems must be analysed using comparable techniques. As well as revealing fundamental drivers of spatial heterogeneity, identifying general rules could facilitate development of predictive models for spatial structuring in host-pathogen systems with relatively poorly understood epidemiology (Gilbertson *et al.* 2016). Researchers could then predict how within- and between-population

processes will differ *a priori* before using empirical methods such as long-term studies at multiple scales (e.g. (Luis *et al.* 2018; Morand *et al.* 2019).

Prescriptive rules for examining geographic variation in wildlife disease are thin on the ground and hard to generalise. For example, where studies seek to quantify the impact of environmental drivers on parasitism, larger study extents may allow sampling the widest range of different environmental factors and thus increasing spatial variation (Cohen *et al.* 2016; Becker *et al.* 2020). Part of this methodological vacuum emerges from analytical complexity. A recent systematic review of ecoimmunology uncovered a surprising lack of spatial methods, with most studies fitting discrete fixed or random effects to control for spatial autocorrelation rather than directly examining continuous patterns in space or using spatially explicit statistics (Becker *et al.* 2020). Although the statistical competence of ecologists is high and increasing, particularly with regards to areas like movement ecology and network analysis (Jacoby & Freeman 2016; Dougherty *et al.* 2018; Webber & Vander Wal 2019), little empirical framework exists for establishing the presence or range of spatial variation in wildlife disease. Establishing general factors shaping spatial variation across wildlife disease systems could substantially improve mechanistic understandings of pathogen transmission, spatial sampling designs, and control efforts.

Here, we conducted a systematic synthesis of spatially distributed wildlife disease datasets across a range of different host and parasite taxa, geographic contexts, and sampling regimes. We analysed these datasets individually using a standardised modelling procedure, identifying how generalised host-, parasite-, and sampling-level factors affect the range and strength of spatial dependence. Specifically, we expected that studies would be most vulnerable to strong spatial effects in larger study areas, with greater sampling efforts, and when parasites exhibit indirect transmission mechanisms with extended environmental stages. We aimed to provide important general estimates for predicting the range of spatial autocorrelation from a wide range of different host-parasite systems, to inform sampling regimes, to be fitted in mechanistic models of movement and disease transmission, and to provide parameter estimates for host-pathogen systems with unknown spatial properties.

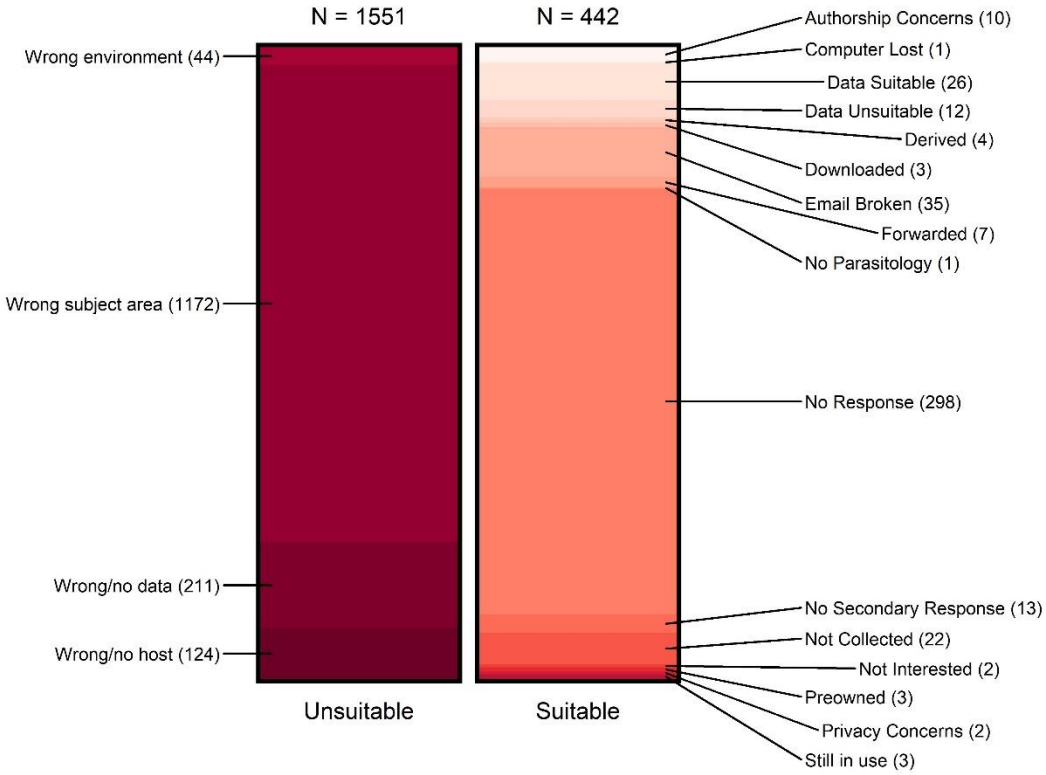

Figure 1: The outcomes of our literature search, expressed as proportions. Different outcomes have different (alphabetically assigned) fill colours. Numbers in brackets correspond to absolute numbers of results. The numbers at the top represent overall sample sizes for the studies deemed "suitable" and "unsuitable" based on their abstracts.

Methods

Data collection

To collect data we carried out a systematic literature search, emailed authors to request data, and searched data repositories for publicly available datasets (Figure 1). Our literature search used Web of Science to identify potential datasets with the following terms: "(parasit* OR infect* OR disease*)* AND (wild OR natural) AND (mammal)". We restricted the search to mammals to increase the generalisability of our findings within this group of animals, and because of their importance for human and livestock health (Han *et al.* 2016). Our search returned 1993 total studies.

We first screened studies based on their abstracts (SIFig), excluding studies of captive animals, review papers, meta-analyses (N=1172); publications without parasite data (N=81);

studies without hosts (i.e., only sampling parasites in the environment; N=43) and studies of non-mammals (N=84). Because our downstream analyses relied upon a standard spatial modeling procedure, we also excluded studies with few samples (N<35), very low prevalence (<10%), or very high prevalence (>90%), owing to likely failure in model convergence (N=130).

Of the remaining 442 studies, only three studies had suitable downloadable datasets, and another four included binary infection data in map figures, from which we derived approximate spatial locations and associated infection status (i.e., "heads up digitisation", HUD). For all 442 studies, we contacted corresponding authors using a standardized email template in September-December 2019 to request data. For 55 studies, we could not access the paper or find the corresponding authors, 35 email addresses failed (i.e., the address was incorrectly listed or no longer valid), and 298 did not receive a response.

We classified the remaining 157 studies for which we received a response into the following (Figure 1): System not suitable: the system was poorly suited to our questions (e.g., migratory host population; N=12). No parasitology: the system did not include disease measures (N=1). No spatial data collected: no sources of spatial data (grid references, GPS locations) were collected and associated with individuals or samples (N=22). Privacy concerns: 2 researchers were unable to share the data because they were collected on private land. 26 researchers kindly sent us their datasets. Data not suitable: once data were inspected, the genre of spatial data was found to be unsuitable (e.g. too few spatial replicates), or it was deemed unlikely that models would run (e.g., points very unevenly distributed, sample sizes too low; N=12). Data suitable (N=36).

We also searched the Dryad data repository (dryad.org) using the same search terms, revealing 43 datasets. Only one dataset concerned disease in wild mammals and included bivariate coordinates in the archived data (Tasmanian Devils, *Sarcophilus harrisii*; Lazenby et al., 2018). Finally, we supplemented our search with three pre-owned datasets that were also present in the literature search.

Within the datasets, each replicate was defined as a unique host-parasite-locality combination. Of the 36 wildlife disease datasets that we obtained, we excluded 10 spatial replicates with under 100 samples, to ensure convergence of our standardized spatial models (see below).

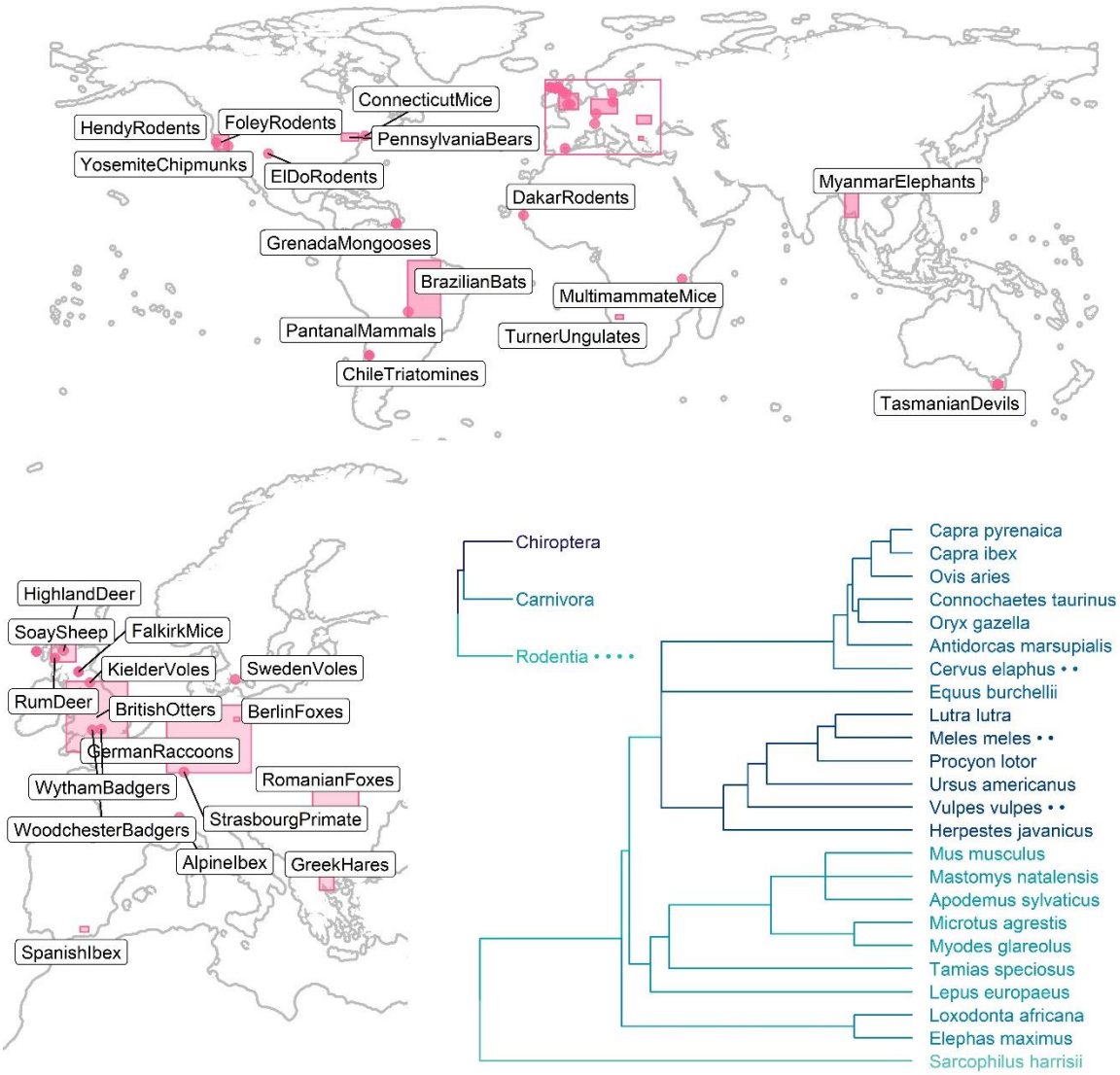

Figure 2: The geographic and taxonomic distribution of our spatial disease datasets. Our data were evenly spread across the earth, although with a notable cluster in Western Europe (see inset map in pink rectangle). Sampling areas greater than 5000 km^2 are displayed as rectangles; smaller sample areas are represented by dots. Study system names correspond to the names in Table 1. The datasets also included a wide range of different mammal orders and families. The inset phylogeny represents order-level summaries for studies that were not carried out at the species level. Dots next to species' names in the phylogenies denote that multiple datasets included samples from that species. Different colours correspond to different taxonomic groups used for meta-analysis: ungulates, carnivores, glires, elephants (Proboscidea), and carnivorous marsupials (Dasyuromorphia).

We concluded data collection with 31 datasets, including 89 spatial replicates, 72 parasites, and 90 host species (Figure 2). 67 replicates were species-level; the rest were conducted on selections of species in the same order (e.g., rodent trapping). The datasets were distributed

188 across all four continents (Figure 2), and included 7 different mammalian orders (TreeFig).
189 The parasites were similarly diverse, including viruses (N=6), bacteria (N=10), helminths
190 (N=25), arthropods (N=14), and one transmissible cancer (N=8). Infection measures included
191 counts of parasites or immune markers (N=30), binary assessment of infection status using
192 observation or seropositivity (N=52), and one study used parasite-associated mortality as a
193 proxy (Myanmar elephants, *Elephas maximus* (Lynsdale *et al.* 2017)). Study systems
194 included, for example: rodent trapping studies examining flea burdens and their associated
195 pathogens (e.g. rodents trapped in the Arizona hills (Kosoy *et al.* 2017) and chipmunks in
196 Yosemite National Park (Hammond *et al.* 2019)); long-term studies with parasite data
197 collected over the course of several decades (e.g. the Soay sheep of St Kilda, the Isle of Rum
198 red deer, and the badgers of Wytham Wood); and studies examining seropositivity of
199 mammals across a geographic range to identify endemic areas (e.g. British otters infected
200 with *Toxoplasma gondii* (Smallbone *et al.* 2017)). See Table 1 for a description of each study
201 system and the associated references and researchers that provided us with the data. The area
202 of the study systems varied widely, from 0.02 to 10^6 km^2 (Figure 3A). Generally, although
203 we principally aimed to quantify fine-scale, within-population spatial effects, we also
204 included several studies employing continuous or semi-continuous sampling at national and
205 county levels, to investigate whether the methods we used would operate well at these scales
206 and to establish an upper bound for sampling effects.

Study system	Country	Location	Species	Host order	N	Parasites	Samples	Sampling method	Tested spatial?	Contributors	Example Reference
AlpineIbex	Italy	Gran Paradiso National Park	*Capra ibex*	Ungulates	4	Helminths; *Nematodirus*; *Marshallagia*; Coccidia	145	Census	N	Alice Brambilla;	(Brambilla *et al.* 2015)
BerlinFoxes	Germany	Berlin	*Vulpes vulpes*	Carnivora	1	Canine distemper virus	762	Opportunistic	Y	Downloaded	(Gras *et al.* 2018)
BrazilianBats	Brazil		5+	Chiroptera	1	*Bartonella*	162	Noninvasive	Y	Keyla deSousa; Marcos Andre	
BritishOtters	UK		*Lutra lutra*	Carnivora	1	*Toxoplasma gondii*	583	Opportunistic	Y	Derived	(Smallbone *et al.* 2017)
ConnecticutMice	USA	Connecticut	*Peromyscus leucopus*	Glires	1	Ticks	105	Trapping	N	Danielle Tufts; Maria Diuk-Wasser	(Tufts & Diuk-Wasser 2018)
ChileTriatomines	Chile		5+	Glires	4	Trypanosomes	230	Trapping	Y	Antonella Bacigalupo	(Ihle-Soto *et al.* 2019)
DakarRodents	Senegal	Dakar	*Mus musculus*	Glires	1	*Toxoplasma gondii*	424	Trapping	N	Lokman Galal	(Galal *et al.* 2019)
ElDoRodents	USA	El Dorado	5+	Glires	2	Fleas; *Bartonella*	1612	Trapping	Y	Michael Kosoy	(Kosoy *et al.* 2017)
FalkirkMice	Scotland	Callendar Park	*Apodemus sylvaticus*	Glires	8	*Heligmosomoides polygyrus*; *Eimeria*; *Capillaria*; Ticks; Mites; Fleas	596	Trapping	N	Amy Sweeny; Amy Pedersen	(Sweeny *et al.* 2019)
FoleyRodents	United States	California	5+	Glires	3	*Anaplasma*; *Borrelia*; Ectoparasites	948	Trapping	N	Janet Foley	(Foley *et al.* 2016)
GermanRaccoons	Germany		*Procyon lotor*	Carnivora	1	*Toxoplasma gondii*	458	Opportunistic	Y	Mike Heddergott	(Heddergott *et al.* 2017)
GreekHares	Greece		*Lepus europaeus*	Glires	1	European brown hare syndrome virus	209	Hunted	Y	Derived	(Sokos *et al.* 2018)
GrenadaMongooses	Grenada		*Herpestes javanicus*	Carnivora	2	Rabies; *Salmonella*	157	Trapping	Y	Ulrike Ziegler; Bruno Chomel; David Jaffe	(Jaffe *et al.* 2018)
HendyRodents	USA	California	*Neotoma fuscipes; Peromyscus maniculatus; Tamias ochrogenys*	Glires	3	Ticks; Aphag; Borrelia	451	Trapping	N	Janet Foley	Unpublished
HighlandDeer	Scotland	Highlands	*Cervus elaphus*	Ungulates	1	*Fasciola hepatica*	103	Hunted	Y	Andrew French	(French *et al.* 2019)
KielderVoles	England	Kielder Woods, Liverpool	*Microtus agrestis*	Glires	9	Cowpoxvirus; *Anaplasma*; *Babesia*	3020	Trapping	N	Mike Begon; Sandra Telfer	(Davis *et al.* 2015)
MultimammateMice	Tanzania	Morogoro	*Mastomys natalensis*	Glires	1	MORV	5547	Trapping	Y	Lucinda Kirkpatrick	(Berkvens *et al.* 2019)
MyanmarElephants	Myanmar		*Elephas maximus*	Proboscidea	1	Parasite-associated mortality	1626	Opportunistic	N	Carly Lynsdale; Virpi Lummaa	(Lynsdale *et al.* 2017)

PantanalMammals	Brazil	Pantanal Wetlands	*Cerdocyon thous*	Carnivora	1	*Hepatozoon*	115	Trapping	N	Marcos Andre; Keyla de Sousa	(de Sousa *et al.* 2017)
PennsylvaniaBears	USA	Pennsylvania	*Ursus americanus*	Carnivora	1	*Toxoplasma gondii*	173	Hunted	N	Jitender Dubey; Justin Brown	(Dubey *et al.* 2016)
RomanianFoxes	Romania		*Vulpes vulpes*	Carnivora	1	Fleas	268	Opportunistic	Y	Janet Foley; Andrei Mihalca	(Foley *et al.* 2017)
RumDeer	Scotland	Isle of Rum	*Cervus elaphus*	Ungulates	8	Strongyles; *Nematodirus*; *Capillaria*; Coccidia; *Moniezia*; *Fasciola hepatica*; *Dictyocaulus*; *Elaphostrongylus cervi*	2068	Census	N	Greg Albery; Josephine Pemberton; Daniel Nussey	(Albery *et al.* 2019)
SoaySheep	Scotland	Isle of St. Kilda	*Ovis aries*	Ungulates	6	Strongyles; *Strongyloides*; Coccidia; *Nematodirus*; *Capillaria*; Keds	7197	Census	N	Josephine Pemberton; Daniel Nussey	(Hayward *et al.* 2014)
SpanishIbex	Spain	Sierra Nevada	*Capra ibex*	Ungulates	1	Mange	746	Hunted	Y	Joao Carvalho; José Granados	(Carvalho *et al.* 2015)
StrasbourgPrimate	France	Strasbourg Primatology Centre	5+	Carnivora	1	Taeniids	103	Noninvasive	N	Valentin Greigert	(Greigert *et al.* 2019)
SwedenVoles	Sweden	Revinge	*Myodes glareolus*	Glires	1	*Borrelia*	1999	Trapping	N	Lars Raberg	(Råberg *et al.* 2017)
TasmanianDevils	Tasmania		*Sarcophilus harrisii*	Dasyuro-morphia	7	Devil facial tumour disease	822	Trapping	Y	Downloaded	(Lazenby *et al.* 2018)
TurnerUngulates	Namibia	Etosha National Park	*Loxodonta africana; Oryx gazella; Connochaetes taurinus; Antidorcas marsupialis; Equus quagga*	Proboscidea; Ungulates; Artiodactyla; Proboscidea	10	Strongyles; *Eimeria melis*; *Strongyloides* sp.	1132	Census	Y	Wendy Turner; Wayne Getz	(Turner *et al.* 2010)
WoodchesterBadgers	England	Woodchester Park, Gloucestershire	*Meles meles*	Carnivora	1	Bovine Tuberculosis	3319	Trapping	N	Matthew Silk; David Hodgson; Dez Delahay	(Rozins *et al.* 2018)
WythamBadgers	England	Wytham Wood	*Meles meles*	Carnivora	4	Fleas; Lice; Ticks; *Eimeria melis*	7220	Trapping	Y	Chris Newman; Christina Buesching; David MacDonald	(Newman *et al.* 2001)
YosemiteChipmunks	USA	Yosemite National Park, California	*Tamias speciosus*	Glires	1	Fleas	1126	Trapping	Y	Tali Hammond	(Hammond *et al.* 2019)

Table 1: Summary table depicting the study systems used in the meta-analysis, including names, locations, host species, and sampling traits. The column "Tested spatial" denotes whether or not one of the study's stated aims was to investigate spatial or geographic variation, e.g. in environmental drivers. The column "N" refers to the number of spatial replicates and INLA models associated with the study system. For a similar table giving information on the INLA model replicates themselves, including host and parasite traits, see Table SI1.

Statistical Analysis

Data standardisation

Data were manipulated and analysed using R version (R Development Core Team 2011). All code is available at github.com/gfalbery/libra. Our data cleaning procedure aimed to minimise the probability of false positives and to restrict the data pool to a continuous spatial distribution of samples. All spatial coordinates were converted to the scale of kilometres or metres to allow comparison across systems. We removed spatial outliers and parasite count outliers; if parasite counts were very overdispersed and/or highly zero-inflated they were analysed as binomial (0/1) infection data rather than negative binomial. Categories with low replication (generally <10 samples) were removed. We removed specific classes that exhibited very low prevalence: e.g., adult Soay sheep and red deer had a very low prevalence of *Nematodirus* sp., which is primarily a parasite of young ungulates (Hoberg *et al.* 2001); hence only lambs/calves were analysed. Individual identity was fitted as a random effect if the dataset involved repeat measurements of the same individuals.

INLA Models

We based our analysis on a framework previously used in a study of spatial patterns of disease in wild red deer (Albery *et al.* 2019). Integrated Nested Laplace Approximation (INLA) models were fitted to each spatial dataset using the `inla` package. INLA is a deterministic Bayesian algorithm that allows fitting of a Stochastic Partial Differentiation Equation (SPDE) random effect to quantify and control for patterns of the response variable in space. This relies on detection of spatial autocorrelation, where samples closer in space are more similar than those further apart (Tobler 1970; Kirby *et al.* 2017). The model estimates how much variance is accounted for by autocorrelation, and models with and without the parameter can be compared to assess how it affects the fit of the model (Lindgren & Rue 2015; Zuur *et al.* 2017). The model also provides a "range" parameter, which estimates the

distance at which samples are autocorrelated. We took this parameter to represent a combination of sampling, transmission, and immune processes determining the scale of spatial variation in the focal population.

We first fitted a "base" model with disease burden (Gaussian or negative binomial) or presence/absence (binary) as a response variable and with any fixed and random covariates (SITable). To simplify our analyses, covariates usually included only temporal variables (month, year, both as categorical variables), age category, and sex. We then fitted a model featuring an SPDE random effect, with a penalised complexity prior (Fuglstad *et al.* 2019). We compared the base model with the SPDE model, identifying whether the latter had a lower Deviance Information Criterion (DIC), indicating improved model fit. We took a change in DIC (ΔDIC) of 2 to distinguish between the two models and calculated the DIC weight for the base and SPDE model, giving a proportion (0-1) that can be conceptualised as the confidence that the spatial model was the best-fitting (Wagenmakers & Farrell 2004). We also extracted the INLA range parameters. In total, we fitted INLA models to 89 host-locale-parasite combinations across 31 study systems.

Meta-analysis of INLA models

To identify factors driving general trends of spatial variation, we conducted a meta-analysis treating each unique parasite-system-site combination as a replicate, including parasite-, host-, and sampling-level traits as fixed effects. We constructed hierarchical models using the `metafor` package. Generally, meta-analyses typically focus on synthesizing effect sizes and their variances across multiple systems (e.g. Sánchez *et al.* 2018). However, as generalised spatial variation does not have a directional effect, we instead analysed measures of model fit, predictive capacity, and the autocorrelation range, which is bounded at 0 and infinity. To give a coarse measure of model predictive capacity that was easily standardised across all models, we calculated the Spearman's Rank correlation between the observed and predicted values for the model, using only the SPDE effect to predict (henceforth referred to as R^2). The measures of model fit give an impression of the detectability and importance of spatial patterns, while comparisons of the range estimate across systems will inform whether different host and parasite traits cause spatial patterns to vary more sharply in space. We used the *escalc* function to derive logit-transformed proportions (R^2) and sampling variances for DIC weight and the INLA range (using the point estimate and 95% confidence interval).

Our hierarchical models included each replicate nested within study as a random effect to account for within- and between-study heterogeneity (Konstantopoulos 2011). We also included a random effect for host family, for which the covariance structure used the phylogenetic correlation matrix (Nakagawa & Santos 2012); we obtained our phylogeny from the Open Tree of Life with the rotl and ape packages (Paradis *et al.* 2004; Michonneau *et al.* 2016). All models used the `rma.mv` function and weighting by sampling variance. We first assessed heterogeneity in each of our three response variables by fitting a random-effects model (REM; intercept only). We used restricted maximum likelihood to obtain unbiased estimates of the variance components, from which we derived I^2 to quantify the contribution of true heterogeneity to the total variance in each INLA model output (Senior *et al.* 2016). We used Cochran's *Q* to test if such heterogeneity was greater than expected by sampling error alone (Borenstein *et al.* 2009).

We next used mixed-effects models (MEMs) to test how sampling-, host-, and parasite-level factors affected our INLA model outputs. Sampling variables included: Number of samples; Sampling area (total rectangular extent between the furthest points on the X- and Y-coordinates, in km^2); Sampling method (3 levels: trapping, censusing, and necropsy/convenience sampling); Spatial encoding method (4 levels: GPS; trapping grid; locality; Easting/Northing); Spatial hypothesis testing (binary – i.e., did the study aim to quantify spatial variation in some way?). We interpreted this latter variable as a combination of study design and publication bias, where studies that are intended to pick up spatial variation are both more likely to identify spatial patterns because of their sampling design, and then more likely to be published if they do. Parasite traits included Transmission mode (4 levels: direct; faecal-oral; vector-borne; environmentally transmitted) and Taxon (8 levels: arthropod, nematode, trematode, cestode, protozoan, bacterium, virus, other). Host traits included: Home Range size (in km^2; log-transformed); Body Mass (in grams; log-transformed); Host order (5 levels: Carnivora, Chiroptera, Ungulates, Glires, Proboscidea). There was only one lagomorph, so rodents and lagomorphs were lumped together into the "glires" clade. The same was true of odd-toed ungulates (Perissodactyla), so they were lumped with Artiodactyla into an "ungulates" clade. For species for which a phenotypic measure (e.g. body mass) was unavailable, we used the value for the closest relative for which the data were available, according to a mammalian supertree (Fritz *et al.* 2009).

To identify important drivers among these many correlated drivers, we conducted a model addition process using Akaike Information Criterion corrected for sample size (AICc) to

determine model fit. Each of our meta-analytical explanatory variables was added in turn, and the best-fitting variable (i.e., the one that most decreased AICc) was kept for the following round. This process was repeated with the remaining variables, until no variables decreased model fit by more than 2 AICc. We report the final model, with the minimal number of variables that improved model fit.

Spatiotemporal INLA models

Finally, we constructed spatiotemporal INLA models to assess the consistency of spatial hotspots from year to year, and to investigate evidence of ephemeral waves of transmission across the study systems. Of our 89 replicates, 44 replicates had more than one year of sampling, with more than 100 spatial points per year, facilitating fitting spatiotemporal models. For these replicates, we first reran the original models with the reduced dataset that only included years with more than 100 replicates. We then fitted a spatiotemporal model with a different field for each year, with no autocorrelation between the fields. Improved model fit for this model would imply that the spatial distribution of the parasite varied notably from year to year. Second, we fitted a similar spatiotemporal model with an "exchangeable" autocorrelation specification between years. This model format allows correlation between spatial fields, but without enforcing a time sequence: that is, all fields were correlated by the same parameter ("Rho") regardless of how far apart in time they were. The Rho parameter, which is bounded between -1 and 1, was then interpreted to give an impression of the spatiotemporal consistency of the parasite distribution. Parasites with high rho coefficients had very similar hotspots from year to year, while those with low coefficients did not.

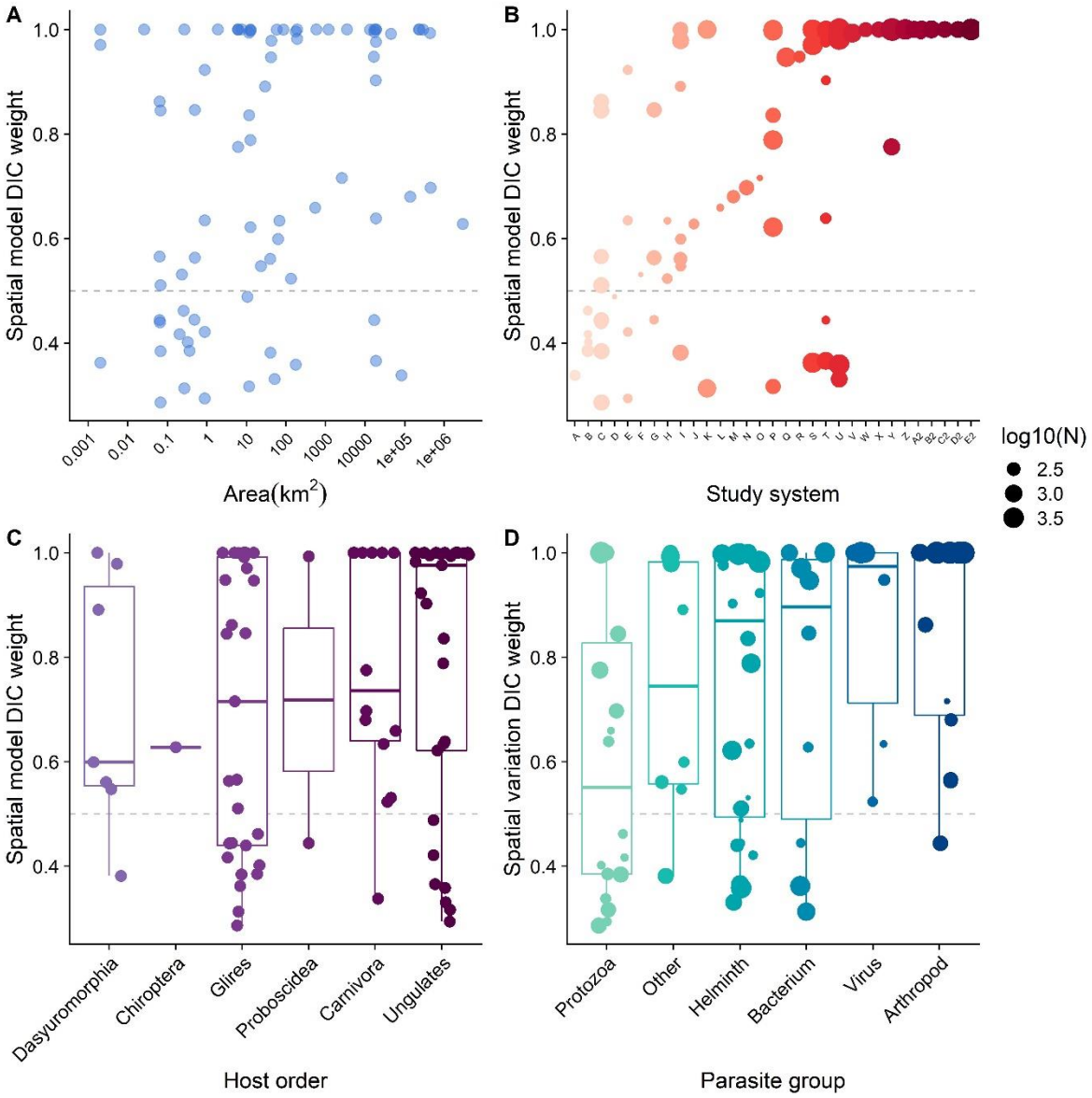

Figure 3: The spatial autocorrelation term (SPDE) improved models across host-parasite systems and sampling regimes. The Y axis displays the degree of confidence that the spatial autocorrelation term improved model fit (Deviance Information Criterion weight), where models at the top of the figure fitted better than those at the bottom. A: larger study areas more often revealed spatial patterns. B: most of our 31 study systems exhibited at least one spatially structured host-parasite combination. Study systems have been assigned arbitrary letters to anonymise them, and are arranged in order of increasing DIC weight. C: multiple mammalian host taxa exhibited spatial effects. D: multiple parasite taxa exhibited spatial effects. The points in panels C and D are sized according to the number of samples in the replicate. None of the terms displayed here had significant effects in our meta-analysis.

Results

Our literature review revealed that very few studies take and archive continuous, within-population spatial data. Only 3/496 studies (0.6%) had such data ready to download, and 4 further studies had maps of samples from which we could easily digitise sufficient data. When we emailed the corresponding authors of the studies we identified, 22/157 responders (14.01%) indicated that they had not collected any within-population spatial data as part of their study (Figure 1). After navigating a number of other obstacles to data sharing, followed by initial data triage, 26 authors kindly offered to provide us with spatial data, resulting in 36 total viable datasets when supplemented with 3 pre-owned datasets. Of these 36 datasets, only 31 had at least one replicate with >100 samples.

Most authors that responded were happy to share data, and the vast majority of studies for which we did not receive data were due to a lack of response or secondary response (Figure 1). 15 authors responded but declined to share data due to privacy concerns, ongoing data usage, or authorship concerns. Comparing this to the 22 responders that had not collected spatial data and the >300 that did not respond, it appears that the main reason researchers do not share spatial data, either in open data repositories or when requested, is that they did not collect it. Notably, studies that investigated spatial variation tended to be larger than those that did not (Figure SI1), implying that larger study areas motivate researchers to more often consider spatial variation in their analyses.

Our INLA models applied across datasets consistently revealed strong spatial patterns of disease (Figure 3-4). The mean DIC change across all study systems was -14.5 (median -3.3), and the spatial model fit better than the base model for 65/89 models (73%; DIC weight>0.5). Using a conventional change of 2ΔDIC as a cutoff for improved model fit, 54% of models across 21 study systems displayed detectable spatial patterns (Figure 3). Although most study systems were spatially structured, our meta-analyses revealed that few host-, parasite-, or sampling factors were predictive of spatial effects. The best-fitting model for DIC weight included only the study duration (years), revealing that long-term studies were slightly more likely to uncover spatial effects (ΔAIC=3.38; for all other variables ΔAIC<1.56). The INLA range parameter increased with study area (ΔAIC=74.44) but was not affected by any other variables (ΔAIC<0.09). There was no variation accounted for by host or parasite taxon, or

host size or ranging behaviour. Most notably, there was no variation in spatial range across transmission ranges (Figure 4A-B).

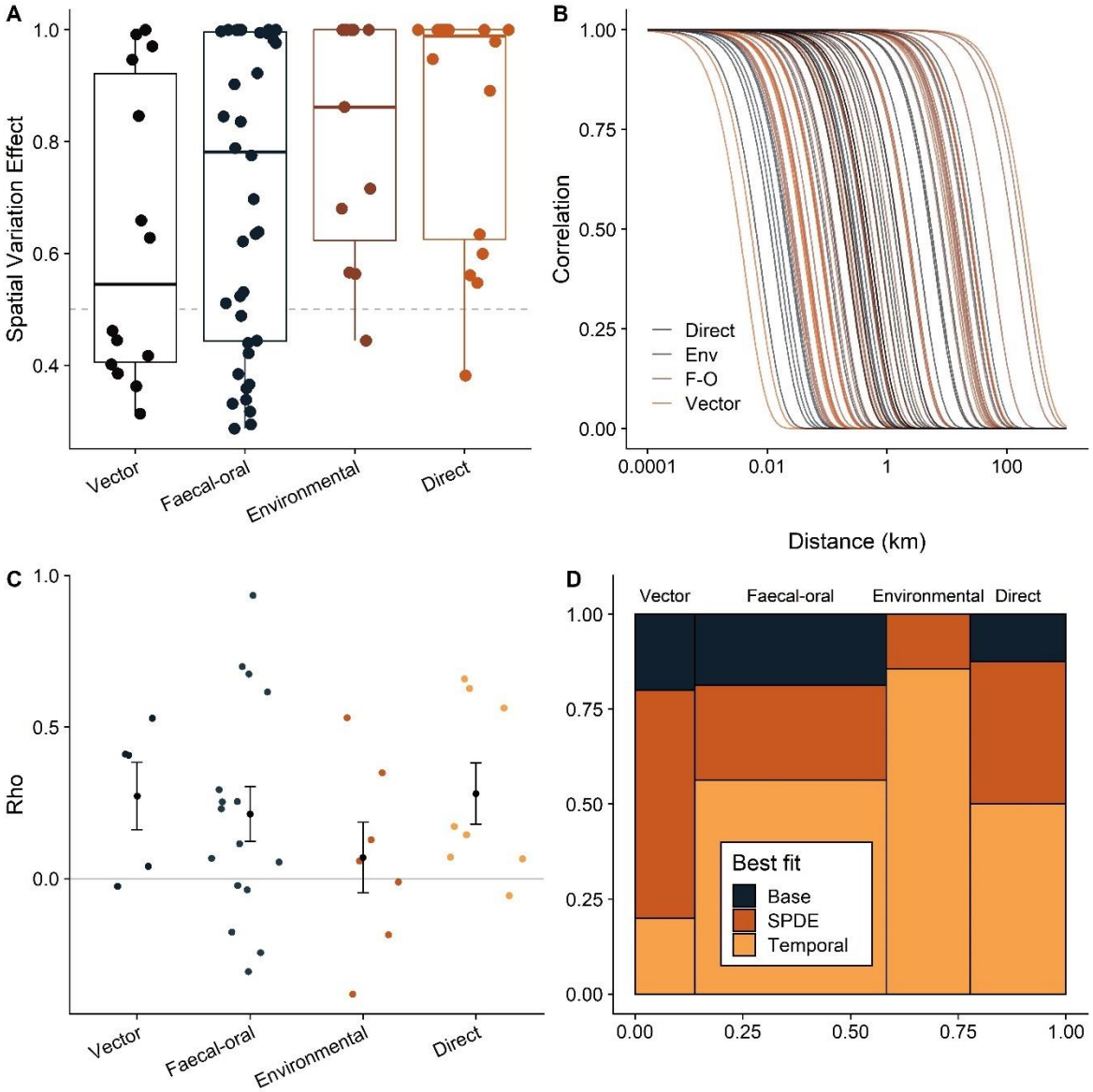

Figure 4: Parasites of diverse transmission modes exhibit spatial autocorrelation effects. We display A) spatial model DIC weight, with points representing the outcome of each replicate INLA model. Boxplots represent range, interquartile range, and median for parasites of each transmission mode. B) INLA autocorrelation ranges; each line represents the autocorrelation decay of a different replicate INLA model. C) Temporal autocorrelation (Rho) component demonstrating inter-annual correlations between spatial fields. Points represent a different replicate INLA model; black dots represent means, and error bars represent standard errors. D) Mosaic plot displaying the proportions of best-fitting models according to DIC changes, across our spatiotemporal replicates.

Spatiotemporal models examining a subset of multi-year studies consistently improved model fit over static equivalents. The best-fitting model for many examined replicates was a spatiotemporal model, but the findings did not differ notably across transmission modes (Figure 4D). Rho (temporal autocorrelation of the spatial field) estimates for these models were moderate, and did not vary notably across transmission modes (Figure 4C). Most (36/44, 82%) had 95% credibility intervals that overlapped with zero, and 8 (18%) were significantly positive.

Discussion

We uncovered strong, pervasive spatial variation across an expansive diversity of mammal-parasite systems. By collating datasets covering many different hosts, parasites, and study systems, our results indicate that spatial variation manifests regularly and unpredictably in disease ecology, whether or not the study in question aims to quantify spatial variation or environmental drivers. Contrary to expectations, spatial heterogeneity was equally common and short-ranged for all transmission modes, implying that spatially structured contact networks are at least as important in driving spatial heterogeneity as are environmental drivers of susceptibility and transmission efficiency (Albery et al., in revision). We impress that our sample represents a vanishingly small proportion of spatially distributed disease studies, and is unlikely to be a random sample, being only 31 of over 1000 studies in our search alone. Our findings therefore best represent a proof-of-principle that disease ecology studies are commonly spatially structured, and that these cryptic patterns should be more commonly investigated, for all kinds of hosts and parasites. We recommend that wild animal studies in disease ecology more regularly collect and share data on spatial behaviours and sampling locations where possible, regardless of host, parasite, or sampling regime.

Our methodology differed from that used in many other studies by investigating generalised spatial dependence rather than by quantifying specific environmental drivers which might drive this dependence. The only similar study that we know of (Gilbertson *et al.* 2016) used 48 parasite-locality replicates of cougar (*Puma concolor*) and bobcat (*Lynx rufus*) populations and found little evidence of spatial autocorrelation in parasite infection. In contrast to their approach, we used a wide set of different hosts, and our replicates all had between 100 and 10,000 samples (Table 1), whereas only a few of their replicates had >100 samples, and none had >200 (Gilbertson *et al.* 2016). Additionally, they used Mantel tests, which do not account

for fixed covariates, while the INLA analyses we employed are more suited to controlling for this variation. As such, we interpret our contrasting findings to represent a difference in the power of our analyses, and the absence of large carnivores from our dataset. Owing to its generality, similar methodology could be used in a range of ecological contexts as a useful hypothesis-generating exercise: after uncovering strong spatial structuring, researchers could follow up on this finding by investigating possible biotic or abiotic drivers. We hope that more disease ecology studies in wild animals will make use of similar methodology to bolster our understanding of disease dynamics in wild settings.

Surprisingly, neither larger study systems nor those that had previously been used to study spatial hypotheses were more likely to exhibit detectable spatial patterns. Some very small spatial replicates exhibited strong spatial effects, and the smallest area demonstrating a strong spatial trend was 0.002km^2 (Figure 3). Similarly, some very large, well-sampled areas showed no detectable spatial patterns: anti-*Toxoplasma gondii* antibodies in almost 200 Pennsylvania black bears (*Ursus americanus*) did not (Dubey *et al.* 2016), while prevalence of *T. gondii* exhibited very strong spatial patterns in otters (*Lutra lutra*) across the United Kingdom (Smallbone *et al.* 2017), and in house mice (*Mus musculus*) within the Senegalese city of Dakar (Galal *et al.* 2019). However, larger study extents unsurprisingly exhibited more long-range spatial autocorrelation effects. These areas inevitably contain within them a multitude of smaller spatial effects and gradients, so that the findings of a specific study will depend critically on the spatial sampling scale it employs (Pullan *et al.* 2012; Cohen *et al.* 2016; Luis *et al.* 2018; Morand *et al.* 2019). Notably, the studies that did attempt to quantify spatial variation tended to have substantially larger spatial extent than those that did not (Figure SI1); this may represent a perception bias, where researchers operating in larger study areas tend to anticipate spatial variation as being more important to account for – or, *vice versa*, researchers asking spatial questions tend to sample across a wider range to incorporate as much testable variation as possible (Becker *et al.* 2019). The finding that larger study systems do not tend to more commonly exhibit detectable spatial patterns in disease demonstrates that this perception bias is perhaps unwarranted, and researchers at all scales should be able to incorporate spatial components and hypotheses about infection processes.

Despite the ubiquity and unpredictability of spatial effects, we discovered a very low frequency of spatial data collection and sharing. Across our extremely broad literature search which identified over 1000 potentially relevant studies, only 3 studies had suitable bivariate

spatial data readily available for download, 4 had them in published maps, and 26 had access to (and provided) within-population spatial data of some sort when we requested it (Figure 1). The responses that we received indicated that alongside concerns about privacy and the desire to control the data associated with one's study system, the main reason for not sharing spatial data was that the data were not collected in the first place.

Privacy is an issue of considerable ethical concern in epidemiology (Kirby *et al.* 2017). Sharing spatial data risks connecting individuals with their disease status, which is particularly unwelcome in the case of stigmatised diseases such as HIV/AIDS; indeed, although we did not examine human diseases, several of the researchers we contacted opted not to share data because they were concerned that their results could be traced to specific households or individuals. Researchers may overcome this issue by jittering points, or by masking the actual GPS locations, replacing them with relative locations which are the same distance away (Kirby *et al.* 2017). Unfortunately, the first option will reduce precision and the latter precludes investigation of specific geographic hypotheses, but this is a small price to pay in the cases where data are potentially sensitive.

Location data may evade collection in some contexts where GPS signals are hard to receive, precluding spatial data collection and investigation of spatial questions. GPS instruments that function in remote environments can be expensive, and for studies that do not explicitly aim to identify spatial patterns this may seem an unnecessary expenditure. However, smartphones that can receive GPS data are now widely available and can be used in all but the most remote locations. As many researchers carry the means to collect spatial data in their pocket on a daily basis, it might take little alteration to collection protocols to include location data in many cases. Future studies should capitalise on the increasing availability of spatial telemetry and biologging technology, and associated analytical capacity (Long *et al.* 2014; Kays *et al.* 2015; Williams *et al.* 2020) to more frequently record, analyse, and share spatial data in disease ecology (Kirby *et al.* 2017; Albery *et al.* 2019). This practice and the associated calls to "let go of your data" (Noy & Noy 2020) will facilitate testing of related hypotheses.

We foresee a range of potential uses for our curated dataset and others like it. Although we quantified some ecological and sampling-level drivers here, the dataset was still relatively small, and subject to covarying factors: for example, most analyses of nematode infection

were conducted on even-toed ungulates, so that it was difficult to disentangle their implications for spatial variation. Future data collection and kind contributions from researchers may allow us to bolster this dataset to include a greater number of replicates, increasing the power and diversity of our analyses, bringing predictive models of spatial variation within our grasp. Further analysis on this dataset could investigate a number of disease drivers such as population density or environmental heterogeneity, informing how they drive spatial patterns of infection within and across systems. Similar methodology could be applied to other animal groups such as birds and reptiles, whose nest and burrow locations offer ideal spatial context (e.g. Wood et al., 2007), or to marine mammals that are regularly subject to behavioural censuses and disease surveillance (e.g. (Leu *et al.* 2020). Finally, immunity is often quantified alongside parasite burden and prevalence, and it would be interesting to see whether spatial variation in immunity manifests on the same scale, and whether it predicts disease risk (Becker *et al.* 2020).

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References

Abolins, S., Lazarou, L., Weldon, L., Hughes, L., King, E.C., Drescher, P., *et al.* (2018). The ecology of immune state in a wild mammal, Mus musculus domesticus. *PLoS Biol.*, 16, e2003538.

Albery, G.F., Becker, D.J., Kenyon, F., Nussey, D.H. & Pemberton, J.M. (2019). The fine-scale landscape of immunity and parasitism in a wild ungulate population. *Integr. Comp. Biol.*, icz016, 1–11.

Altizer, S., Dobson, A., Hosseini, P., Hudson, P., Pascual, M. & Rohani, P. (2006). Seasonality and the dynamics of infectious diseases. *Ecol. Lett.*, 9, 467–484.

Becker, D.J., Albery, G.F., Kessler, M.K., Lunn, T.J., Falvo, C.A., Czirják, G.Á., *et al.* (2020). Macroimmunology: the drivers and consequences of spatial patterns in wildlife immune defense. *J. Anim. Ecol.*, 89, 972–995.

Becker, D.J., Czirják, G.Á., Volokhov, D. V., Bentz, A.B., Carrera, J.E., Camus, M.S., *et al.* (2018). Livestock abundance predicts vampire bat demography, immune profiles, and bacterial infection

520 risk. *Philos. Trans. R. Soc. B*, 373, 20170089.

521 Becker, D.J., Nachtmann, C., Argibay, H.D., Botto, G., Escalera-Zamudio, M., Carrera, J.E., *et al.*
522 (2019). Leukocyte Profiles Reflect Geographic Range Limits in a Widespread Neotropical Bat.
523 *Integr. Comp. Biol.*, icz007.

524 Berkvens, R., Olivares, I.H., Mercelis, S., Kirkpatrick, L. & Weyn, M. (2019). Contact Detection for
525 Social Networking of Small Animals. In: *Advances on P2P, Parallel, Grid, Cloud and Internet*
526 *Computing*. Springer International Publishing, pp. 405–414.

527 Best, A., Webb, S., White, A. & Boots, M. (2011). Host resistance and coevolution in spatially
528 structured populations. *Proc. R. Soc. B Biol. Sci.*, 278, 2216–2222.

529 Borenstein, M., Hedges, L. V., Higgins, J.P.T. & Rothstein, H. (2009). *Introduction to meta-analysis*.
530 John Wiley & Sons.

531 Brambilla, A., Biebach, I., Bassano, B., Bogliani, G., von Hardenberg, A., Hardenberg, A. Von, *et al.*
532 (2015). Direct and indirect causal effects of heterozygosity on fitness-related traits in Alpine
533 ibex. *Proc. Biol. Sci.*, 282, 20141873.

534 Brooker, S., Alexander, N., Geiger, S., Moyeed, R.A., Stander, J., Fleming, F., *et al.* (2006).
535 Contrasting patterns in the small-scale heterogeneity of human helminth infections in urban and
536 rural environments in Brazil. *Int. J. Parasitol.*, 36, 1143–1151.

537 Carvalho, J., Granados, J.E., López-Olvera, J.R., Cano-Manuel, F.J., Pérez, J.M., Fandos, P., *et al.*
538 (2015). Sarcoptic mange breaks up bottom-up regulation of body condition in a large herbivore
539 population. *Parasites and Vectors*, 8, 1–9.

540 Cohen, J.M., Civitello, D.J., Brace, A.J., Feichtinger, E.M., Ortega, C.N., Richardson, J.C., *et al.*
541 (2016). Spatial scale modulates the strength of ecological processes driving disease distributions,
542 3359–3364.

543 Cross, P.C., Lloyd-Smith, J.O., Johnson, P.L.F. & Getz, W.M. (2005). Duelling timescales of host
544 movement and disease recovery determine invasion of disease in structured populations. *Ecol.*
545 *Lett.*, 8, 587–595.

546 Davis, S., Abbasi, B., Shah, S., Telfer, S., Begon, M. & Davis, S. (2015). Spatial analyses of wildlife
547 contact networks. *J. R. Soc. Interface*, 12, 20141004.

548 Débarre, F., Hauert, C. & Doebeli, M. (2014). Social evolution in structured populations. *Nat.*
549 *Commun.*, 5, 1–7.

550 Dougherty, E.R., Seidel, D.P., Carlson, C.J., Spiegel, O. & Getz, W.M. (2018). Going through the
551 motions : incorporating movement analyses into disease research. *Ecol. Lett.*, 21, 588–604.

552 Dubey, J.P., Brown, J., Ternent, M., Verma, S.K., Hill, D.E., Cerqueira-Cézar, C.K., *et al.* (2016).
553 Seroepidemiologic study on the prevalence of Toxoplasma gondii and Trichinella spp. infections
554 in black bears (Ursus americanus) in Pennsylvania, USA. *Vet. Parasitol.*, 229, 76–80.

555 Foley, J., Rejmanek, D., Foley, C. & Matocq, M. (2016). Fine-scale genetic structure of woodrat
556 populations (Genus: Neotoma) and the spatial distribution of their tick-borne pathogens. *Ticks*
557 *Tick. Borne. Dis.*, 7, 243–253.

558 Foley, P., Foley, J., Sándor, A.D., Ionică, A.M., Matei, I.A., D'Amico, G., *et al.* (2017). Diversity of
559 flea (siphonaptera) parasites on red foxes (Vulpes vulpes) in Romania. *J. Med. Entomol.*, 54,
560 1243–1250.

561 French, A.S., Zadoks, R.N., Skuce, P.J., Mitchell, G., Gibbs, D.K.G. & Taggart, M.A. (2019). Habitat
562 and host factors associated with liver fluke (*Fasciola hepatica*) diagnoses in wild red deer
563 (*Cervus elaphus*) in the Scottish Highlands. *Parasit. Vectors*, 1–14.

564 Fritz, S.A., Bininda-Emonds, O.R.P. & Purvis, A. (2009). Geographical variation in predictors of
565 mammalian extinction risk: big is bad, but only in the tropics. *Ecol. Lett.*, 12, 538–549.

566 Fuglstad, G.A., Simpson, D., Lindgren, F. & Rue, H. (2019). Constructing Priors that Penalize the
567 Complexity of Gaussian Random Fields. *J. Am. Stat. Assoc.*, 114, 445–452.

568 Galal, L., Schares, G., Stragier, C., Vignoles, P., Brouat, C., Cuny, T., *et al.* (2019). Diversity of
569 Toxoplasma gondii strains shaped by commensal communities of small mammals. *Int. J.*
570 *Parasitol.*, 49, 267–275.

571 Gilbertson, M.L.J., Carver, S., Vandewoude, S., Crooks, K.R., Lappin, M.R. & Craft, M.E. (2016). Is
572 pathogen exposure spatially autocorrelated? Patterns of pathogens in puma (Puma concolor) and
573 bobcat (Lynx rufus). *Ecosphere*, 7, 1–12.

574 Gras, P., Knuth, S., Börner, K., Marescot, L., Benhaiem, S., Aue, A., *et al.* (2018). Landscape
575 structures affect risk of canine distemper in urban wildlife. *Front. Ecol. Evol.*, 6, 1–16.

576 Greigert, V., Brion, N., Lang, C., Regnard, P., Pfaff, A.W., Abou-Bacar, A., *et al.* (2019). Cestode
577 infections in non-human primates suggest the existence of zoonotic cycles in the area
578 surrounding the Strasbourg primatology center. *Parasite*, 26.

579 Hammond, T.T., Hendrickson, C.I., Maxwell, T.L., Petrosky, A.L., Palme, R., Pigage, J.C., *et al.*
580 (2019). Host biology and environmental variables differentially predict flea abundances for two
581 rodent hosts in a plague-relevant system. *Int. J. Parasitol. Parasites Wildl.*, 9, 174–183.

582 Han, B.A., Kramer, A.M. & Drake, J.M. (2016). Global Patterns of Zoonotic Disease in Mammals.
583 *Trends Parasitol.*, 32, 565–577.

584 Hayward, A.D., Nussey, D.H., Wilson, A.J., Berenos, C., Pilkington, J.G., Watt, K. a., *et al.* (2014).
585 Natural Selection on Individual Variation in Tolerance of Gastrointestinal Nematode Infection.
586 *PLoS Biol.*, 12, 1–13.

587 Heddergott, M., Frantz, A.C., Stubbe, M., Stubbe, A., Ansorge, H. & Osten-Sacken, N. (2017).
588 Seroprevalence and risk factors of Toxoplasma gondii infection in invasive raccoons (Procyon
589 lotor) in Central Europe. *Parasitol. Res.*, 116, 2335–2340.

590 Hoberg, E.P., Kocan, A.A. & Rickard, L.G. (2001). Gastrointestinal strongyles in wild ruminants.
591 *Dis. Wild Mamm.*, 193–227.

592 Ihle-Soto, C., Costoya, E., Correa, J.P., Bacigalupo, A., Cornejo-Villar, B., Estadella, V., *et al.*
593 (2019). Spatio-temporal characterization of Trypanosoma cruzi infection and discrete typing
594 units infecting hosts and vectors from non-domestic foci of chile. *PLoS Negl. Trop. Dis.*, 13, 1–
595 19.

596 Jacoby, D.M.P. & Freeman, R. (2016). Emerging Network-Based Tools in Movement Ecology.
597 *Trends Ecol. Evol.*, 31, 301–314.

598 Jaffe, D.A., Chomel, B.B., Kasten, R.W., Breitschwerdt, E.B., Maggi, R.G., McLeish, A., *et al.*
599 (2018). Bartonella henselae in small Indian mongooses (Herpestes auropunctatus) from Grenada,
600 West Indies. *Vet. Microbiol.*, 216, 119–122.

601 Jamison, A., Tuttle, E., Jensen, R., Bierly, G. & Gonser, R. (2015). Spatial ecology, landscapes, and
602 the geography of vector-borne disease: A multi-disciplinary review. *Appl. Geogr.*, 63, 418–426.

603 Kays, R., Crofoot, M.C., Jetz, W. & Wikelski, M. (2015). Terrestrial animal tracking as an eye on life
604 and planet. *Science (80-.).*, 348, aaa2478.

605 Kirby, R.S., Delmelle, E. & Eberth, J.M. (2017). Advances in spatial epidemiology and geographic
606 information systems. *Ann. Epidemiol.*, 27, 1–9.

607 Konstantopoulos, S. (2011). Fixed effects and variance components estimation in three-level meta-
608 analysis. *Res. Synth. Methods*, 2, 61–76.

609 Kosoy, M., Reynolds, P., Bai, Y., Sheff, K., Enscore, R.E., Montenieri, J., *et al.* (2017). Small-Scale
610 Die-Offs in Woodrats Support Long-Term Maintenance of Plague in the U.S. Southwest.
611 *Vector-Borne Zoonotic Dis.*, 17, 635–644.

612 Lazenby, B.T., Tobler, M.W., Brown, W.E., Hawkins, C.E., Hocking, G.J., Hume, F., *et al.* (2018).
613 Density trends and demographic signals uncover the long-term impact of transmissible cancer in
614 Tasmanian devils. *J. Appl. Ecol.*, 55, 1368–1379.

615 Leu, S.T., Sah, P., Krzyszczyk, E., Jacoby, A. & Mann, J. (2020). Sex, synchrony, and skin contact:

integrating multiple behaviors to assess pathogen transmission risk. *Behav. Ecol.*, 1–10.

Lindgren, F. & Rue, H. (2015). Bayesian Spatial Modelling with R-INLA. *J. Stat. Softw.*, 63, 1–25.

Long, J.A., Nelson, T.A., Webb, S.L. & Gee, K.L. (2014). A critical examination of indices of dynamic interaction for wildlife telemetry studies. *J. Anim. Ecol.*, 83, 1216–1233.

Luis, A.D., Kuenzi, A.J. & Mills, J.N. (2018). Species diversity concurrently dilutes and amplifies transmission in a zoonotic host–pathogen system through competing mechanisms. *Proc. Natl. Acad. Sci.*, 115, 7979–7984.

Lynsdale, C.L., Mumby, H.S., Hayward, A.D., Mar, K.U. & Lummaa, V. (2017). Parasite-associated mortality in a long-lived mammal: Variation with host age, sex, and reproduction. *Ecol. Evol.*, 7, 10904–10915.

Manlove, K., Aiello, C., Sah, P., Cummins, B., Hudson, P.J. & Cross, P.C. (2018). The ecology of movement and behaviour: a saturated tripartite network for describing animal contacts. *Proceedings. Biol. Sci.*, 285, 20180670.

Michonneau, F., Brown, J.W. & Winter, D.J. (2016). rotl: an R package to interact with the Open Tree of Life data. *Methods Ecol. Evol.*, 7, 1476–1481.

Morand, S., Blasdell, K., Bordes, F., Buchy, P., Carcy, B., Chaisiri, K., *et al.* (2019). Changing landscapes of Southeast Asia and rodent-borne diseases: decreased diversity but increased transmission risks. *Ecol. Appl.*, 29, 1–15.

Nakagawa, S. & Santos, E.S.A. (2012). Methodological issues and advances in biological meta-analysis. *Evol. Ecol.*, 26, 1253–1274.

Newman, C., Macdonald, D.W. & Anwar, M.A. (2001). Coccidiosis in the European badger, Meles meles in Wytham Woods: Infection and consequences for growth and survival. *Parasitology*, 123, 133–142.

Noy, N. & Noy, A. (2020). Let go of your data. *Nat. Mater.*, 19, 128–128.

Nusser, S.M., Clark, W.R., Otis, D.L. & Huang, L. (2008). Sampling Considerations for Disease Surveillance in Wildlife Populations. *J. Wildl. Manage.*, 72, 52–60.

Paradis, E., Claude, J. & Strimmer, K. (2004). APE: Analyses of phylogenetics and evolution in R language. *Bioinformatics*, 20, 289–290.

Patz, J.A., Graczyk, T.K., Geller, N. & Vittor, A.Y. (2000). Effects of environmental change on emerging parasitic diseases. *Int. J. Parasitol.*, 30, 1395–1405.

Peel, A.J., Sargan, D.R., Baker, K.S., Hayman, D.T.S., Barr, J.A., Crameri, G., *et al.* (2013).

647 Continent-wide panmixia of an African fruit bat facilitates transmission of potentially zoonotic
648 viruses. *Nat. Commun.*, 4.

649 Plowright, R.K., Becker, D.J., McCallum, H. & Manlove, K.R. (2019). Sampling to elucidate the
650 dynamics of infections in reservoir hosts. *Philos. Trans. R. Soc. B Biol. Sci.*, 374.

651 Pullan, R.L., Sturrock, H.J.W., Soares Magalhaes, R.J., Clements, A.C.A. & Brooker, S.J. (2012).
652 Spatial parasite ecology and epidemiology: a review of methods and applications. *Parasitology*,
653 139, 1870–1887.

654 R Development Core Team, R. (2011). R: A Language and Environment for Statistical Computing. *R*
655 *Found. Stat. Comput.*, R Foundation for Statistical Computing.

656 Råberg, L., Hagström, Andersson, M., Bartkova, S., Scherman, K., Strandh, M., *et al.* (2017).
657 Evolution of antigenic diversity in the tick-transmitted bacterium Borrelia afzelii: a role for host
658 specialization? *J. Evol. Biol.*, 30, 1034–1041.

659 Reynolds, K.M. (1988). Analysis of Epidemics Using Spatio-Temporal Autocorrelation.
660 *Phytopathology*, 78, 240.

661 Rohr, J.R., Civitello, D.J., Halliday, F.W., Hudson, P.J., Lafferty, K.D., Wood, C.L., *et al.* (2020).
662 Towards common ground in the biodiversity– disease debate. *Nat. Ecol. Evol.*, 4, 24–33.

663 Rozins, C., Silk, M.J., Croft, D.P., Delahay, R.J., Hodgson, D.J., McDonald, R.A., *et al.* (2018).
664 Social structure contains epidemics and regulates individual roles in disease transmission in a
665 group-living mammal. *Ecol. Evol.*, 1–12.

666 Saito, M.U. & Sonoda, Y. (2017). Symptomatic Raccoon Dogs and Sarcoptic Mange Along an Urban
667 Gradient. *Ecohealth*, 14, 318–328.

668 Sánchez, C.A., Becker, D.J., Teitelbaum, C.S., Barriga, P., Brown, L.M., Majewska, A.A., *et al.*
669 (2018). On the relationship between body condition and parasite infection in wildlife: a review
670 and meta-analysis. *Ecol. Lett.*, 21, 1869–1884.

671 Satterfield, D.A., Altizer, S., Williams, M.K. & Hall, R.J. (2017). Environmental persistence
672 influences infection dynamics for a butterfly pathogen. *PLoS One*, 12, 1–16.

673 Senior, A.M., Grueber, C.E., Kamiya, T., Lagisz, M., O'Dwyer, K., Santos, E.S.A., *et al.* (2016).
674 Heterogeneity in ecological and evolutionary meta-analyses: Its magnitude and implications.
675 *Ecology*, 97, 3293–3299.

676 Smallbone, W.A., Chadwick, E.A., Francis, J., Guy, E., Perkins, S.E., Sherrard-Smith, E., *et al.*
677 (2017). East-West Divide: Temperature and land cover drive spatial variation of Toxoplasma
678 gondii infection in Eurasian otters (Lutra lutra) from England and Wales. *Parasitology*, 144,

1433–1440.

Sokos, C., Touloudi, A., Giannakopoulos, A., Papaspyropoulos, K.G., Tsokana, C.N., Birtsas, P., *et al.* (2018). Population density, roads and altitude influences on spatial distribution of hares positive to EBHSV. *Basic Appl. Ecol.*, 31, 82–91.

de Sousa, K.C.M., Fernandes, M.P., Herrera, H.M., Benevenute, J.L., Santos, F.M., Rocha, F.L., *et al.* (2017). Molecular detection of Hepatozoon spp. in domestic dogs and wild mammals in southern Pantanal, Brazil with implications in the transmission route. *Vet. Parasitol.*, 237, 37–46.

Sweeny, A.R., Clerc, M., Pontifes, P.A., Venkatesan, S., Babayan, S.A. & Pedersen, A.B. (2019). Supplemented nutrition increases immunity and drug efficacy in a natural host-helminth system, 1–43.

Tobler, W.R. (1970). A Computer Movie Simulating Urban Growth in the Detroit Region. *Econ. Geogr.*, 46, 234.

Tufts, D.M. & Diuk-Wasser, M.A. (2018). Transplacental transmission of tick-borne Babesia microti in its natural host Peromyscus leucopus. *Parasites and Vectors*, 11, 1–9.

Turner, W.C., Cizauskas, C. a & Getz, W.M. (2010). Variation in faecal water content may confound estimates of gastro-intestinal parasite intensity in wild African herbivores. *J. Helminthol.*, 84, 99–105.

Vidal-Martínez, V.M., Pech, D., Sures, B., Purucker, S.T. & Poulin, R. (2010). Can parasites really reveal environmental impact? *Trends Parasitol.*, 26, 44–51.

Wagenmakers, E.J. & Farrell, S. (2004). AIC model selection using Akaike weights. *Psychon. Bull. Rev.*, 11, 192–196.

Webber, Q.M.R. & Vander Wal, E. (2019). Trends and perspectives on the use of animal social network analysis in behavioural ecology: a bibliometric approach. *Anim. Behav.*, 149, 77–87.

Williams, H.J., Taylor, L.A., Benhamou, S., Bijleveld, A.I., Clay, T.A., de Grissac, S., *et al.* (2020). Optimizing the use of biologgers for movement ecology research. *J. Anim. Ecol.*, 89, 186–206.

Wood, M.J., Cosgrove, C.L., Wilkin, T.A., Knowles, S.C.L., Day, K.P. & Sheldon, B.C. (2007). Within-population variation in prevalence and lineage distribution of avian malaria in blue tits, <i>Cyanistes caeruleus</ i>. *Mol. Ecol.*, 16, 3263–3273.

Zhou, G., Sirichaisinthop, J., Sattabongkot, J., Jones, J., Bjørnstad, O.N., Yan, G., *et al.* (2005). Spatio-temporal distribution of Plasmodium falciparum and p. Vivax malaria in Thailand. *Am. J. Trop. Med. Hyg.*, 72, 256–62.

711 Zuur, A.F., Ieno, E.N. & Saveliev, A.A. (2017). *Beginner's guide to spatial, temporal, and spatial-*
712 *temporal ecological data analysis with R-INLA*. Highstat Ltd.

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