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Abstract

1. All parasites 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 sought to identify and examine spatial dependence of wildlife disease across a wide range of systems.

2. Conducting a broad literature search, we collated 31 such datasets featuring 89 replicates and 71 unique host-parasite combinations, only 51% of which had previously been used to test spatial hypotheses. We analysed these datasets for spatial dependence within a standardised modelling framework using Bayesian linear models, and we then meta-analysed the results to identify generalised determinants of the scale and magnitude of spatial autocorrelation.

3. We detected spatial autocorrelation in 48/89 model replicates (54%) across 21/31 datasets (68%), spread across parasites of all groups. Even some very small study areas (under 0.01km^2) exhibited substantial spatial variation.

4. Despite the common manifestation of spatial variation, our meta-analysis was unable to identify host-, parasite-, or sampling-level determinants of this heterogeneity across systems. Parasites of all transmission modes had easily detectable spatial patterns, implying that structured contact networks and susceptibility effects are potentially as important in spatially structuring disease as are environmental drivers of transmission efficiency.

5. Our findings demonstrate that fine-scale spatial patterns of infection manifest frequently and across a range of wild animal systems, and many studies are able to investigate them – whether or not the original aim of the study was to examine spatially 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. Ultimately, this may pave the way for an *a priori* predictive framework for spatial variation in novel host-parasite systems.

Keywords: Wildlife disease; parasite transmission; spatial analysis; meta-analysis

Introduction

The maintenance and spread of parasites are inherently spatially structured (Cross, Lloyd-Smith, Johnson, & Getz, 2005; Kirby, Delmelle, & Eberth, 2017; Pullan, Sturrock, Soares Magalhaes, Clements, & Brooker, 2012), which holds important ramifications for epidemiological dynamics and disease control efforts (Becker et al., 2020; Cross et al., 2005; Plowright, Becker, McCallum, & Manlove, 2019). Spatial structure can arise through a wide variety of processes (Albery, Kirkpatrick, Firth, & Bansal, 2021): for example, many parasites are transmitted from one host individual to another via direct contact, which requires a degree of spatiotemporal coincidence between individuals (Manlove et al., 2018), so that infections are spatiotemporally staggered in waves of transmission across the population. Other parasites transmit through persistent environmental stages or arthropod vectors whose viability depends on spatially varying abiotic conditions, creating spatial patterns of exposure and therefore of infection (Altizer et al., 2006; Jamison, Tuttle, Jensen, Bierly, & Gonser, 2015; Patz, Graczyk, Geller, & Vittor, 2000). Finally, host immunity and susceptibility can be influenced by environmentally varying factors like resource availability and climatic conditions, with knock-on impacts on parasite burden and transmission (Becker et al., 2020, 2018). These diverse processes should produce spatial patterns of infection across a wide range of wildlife systems, yet many wildlife disease studies examine coarse spatial scales or assume that spatial patterns will be negligible compared to other hypothesised drivers. As such, it is unclear how often infection is spatially structured in these systems, at what range this variation can manifest, and how host and parasite traits might alter its manifestation.

For logistical reasons, many studies of spatial drivers of infectious disease focus on discrete between-population differences across large distances, often using a limited number of discrete sampling locations rather than distributing their sampling locations continuously in space (Plowright et al., 2019). Nevertheless, some work suggests that spatial patterns of infection may manifest at surprisingly fine spatial scales, within kilometres or even metres (Abolins et al., 2018; Albery, Becker, Kenyon, Nussey, & Pemberton, 2019; Brooker et al., 2006; Wood et al., 2007). This observation begs the question: what is the lower bound for the range at which spatial effects can act? Identifying the range of spatial dependence (or autocorrelation, meaning that data points that are closer together in space tend to be more similar) is important for many reasons, including designing sampling regimes (Nusser, Clark,

64   Otis, & Huang, 2008; Plowright et al., 2019; Vidal-Martínez, Pech, Sures, Purucker, &

65   Poulin, 2010), building mechanistic models of parasite evolution over space (Best, Webb,

66   White, & Boots, 2011; Débarre, Hauert, & Doebeli, 2014), examining how disease risk

67   responds to anthropogenic activities like urbanisation (Saito & Sonoda, 2017), and directing

68   public health and conservation schemes (Brooker et al., 2006; Gilbertson et al., 2016).

69

70   Identifying the range of spatial dependence can also help to examine how parasites spread

71   over landscapes and to determine their transmission mechanisms (Reynolds, 1988). For

72   example, spatial dependence across large distances might suggest the influence of major

73   climatic correlates, while spatial dependence between nearby locations implies a highly

74   localised infection process (Pullan et al., 2012). In human disease systems, such work has

75   shown that neighbouring districts of Thailand have more similar human malaria incidence,

76   suggesting local similarities in abiotic conditions or vector control programs that could limit

77   mosquito survival (Zhou et al., 2005). Similar analyses of wildlife disease could help pinpoint

78   transmission routes and guide disease control efforts: for example, if researchers find that a

79   zoonotic disease has a long range of dependence in its wildlife reservoir, this could motivate

80   the use of widely placed sampling locations when trying to identify environmental drivers

81   (Becker, Crowley, Washburne, & Plowright, 2019; Plowright et al., 2019). Lastly, the scale

82   of spatial dependence has implications for more general theoretical understanding of

83   infectious disease dynamics. For example, links between biodiversity and disease dynamics

84   (e.g. "dilution effects") are dependent on the spatial scale of sampling (Cohen et al., 2016;

85   Rohr et al., 2020), and several rodent systems have identified contrasting spatial trends for

86   zoonotic diseases dependent on sampling scale (Luis, Kuenzi, & Mills, 2018; Morand et al.,

87   2019).

88

89   The strength and range of spatial dependence are also likely to depend on the traits of the

90   hosts and parasites involved. For example, parasites that persist for longer in the environment

91   are likely to experience stronger influences of environmental gradients than directly

92   transmitted counterparts (Satterfield, Altizer, Williams, & Hall, 2017). Similarly, highly

93   mobile species such as large carnivores or nomadic bats may more efficiently disseminate

94   parasites through the environment, reducing spatial autocorrelation (Gilbertson et al., 2016;

95   Peel et al., 2013). The range of spatial dependence is most commonly identified using spatial

96   autocorrelation models (e.g. Albery et al., 2019; Becker, Nachtmann, et al., 2019; Brooker et

97   al., 2006; Gilbertson et al., 2016; Wood et al., 2007) or analyses that quantify the spatial

98   buffer regions in which environmental variables are best-correlated with disease (e.g. Saito &
99   Sonoda, 2017). Unfortunately, these approaches are almost always reactive rather than
100   proactive, and they occur on a case-by-case basis rather than being founded on general rules
101   or *a priori* understanding. As such, the relative contribution of host and parasite traits to
102   shaping spatial variation in infection remains unknown. To establish general factors
103   influencing the scale of spatial dependence in wildlife disease, a variety of host-parasite
104   systems must be analysed using comparable techniques and then synthesised. As well as
105   revealing fundamental drivers of spatial heterogeneity, identifying general rules in this way
106   could facilitate the development of predictive models for spatial structuring in host-parasite
107   systems with relatively poorly understood epidemiology. Researchers could then predict how
108   within- and between-population processes will differ *a priori*, before using empirical methods
109   such as long-term studies at multiple scales (e.g. Luis et al., 2018; Morand et al., 2019).
110

111   Prescriptive rules for examining geographic variation in wildlife disease are rare and hard to
112   generalise, partly due to the analytical complexity of identifying them. For example, a recent
113   systematic review of ecoimmunology studies uncovered a surprising lack of spatial methods,
114   with most studies fitting discrete fixed or random effects to control for spatial autocorrelation
115   rather than directly examining continuous patterns in space or using spatially explicit
116   statistics (Becker et al., 2020). Nevertheless, the statistical competence of ecologists is high
117   and increasing, particularly with regards to areas like movement ecology and network
118   analysis (Albery et al., 2021; Dougherty, Seidel, Carlson, Spiegel, & Getz, 2018; Jacoby &
119   Freeman, 2016; Webber & Vander Wal, 2019). The increase in such studies over time has led
120   to a few general rules to guide spatial sampling: for example, where studies seek to quantify
121   the impact of environmental drivers on parasitism, larger study extents may allow sampling
122   the widest range of different environmental factors and thus increasing spatial variation
123   (Becker et al., 2020; Cohen et al., 2016). Nevertheless, no standardised empirical framework
124   yet exists for identifying and comparing the presence or range of spatial variation across
125   wildlife disease systems. Establishing such a framework could help to identify general factors
126   shaping spatial variation across systems, improving mechanistic understanding of parasite
127   transmission, spatial sampling designs, and control efforts.
128

129   Here, we conducted a synthesis of spatially distributed wildlife disease datasets across a wide
130   range of different host and parasite taxa, geographic contexts, and sampling regimes. We
131   analysed these datasets individually using a standardised modelling procedure, identifying

how generalised host-, parasite-, and sampling-level factors affect the prevalence and range 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 the range of spatial autocorrelation from a wide range of different host-parasite systems, laying the groundwork for *a priori* predictions about host-parasite systems with unknown spatial properties.

Materials and methods

Data collection

To obtain a wide variety of raw datasets we carried out a literature search, emailed authors to request data, and searched data repositories for publicly available datasets (Supplementary Figure 1). Our literature search used Web of Science to identify potential datasets published between 2009 and 28th August 2019, 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, Kramer, & Drake, 2016).

We screened a random subset of studies based on their abstracts, excluding studies of captive animals, review papers, and meta-analyses; publications without parasite data; studies without hosts (i.e., only sampling parasites in the environment); and studies of non-mammals. Because our downstream analyses relied upon a standard spatial modelling 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.

If a study had openly available datasets we downloaded them, and for those that included binary infection data in map figures, we derived approximate spatial locations and associated infection status (i.e., "heads up digitisation", HUD). We also searched the Dryad data repository (https://datadryad.org) using the same search terms to find publicly available datasets.

For all other studies, we contacted corresponding authors using a standardised email template in September-December 2019 to request data. We classified the authors' responses into the following categories (Supplementary Figure 1): System not suitable: the system was poorly

162   suited to our questions (e.g., migratory host population). No parasitology: the system did not

163   include disease measures. No spatial data collected: no sources of spatial data (grid

164   references, GPS locations) were collected and associated with individuals or samples. Privacy

165   concerns: researchers were unable to share the data because they were collected on private

166   land. Data not suitable: once data were inspected, the genre of spatial data was found to be

167   unsuitable (e.g. too few spatial replicates), or it was deemed unlikely that models would run

168   (e.g., points very unevenly distributed, sample sizes too low).

169   Some of the datasets contained multiple spatial sites that were each defined as a distinct

170   population. Therefore, within the datasets, each replicate was defined as a unique host-

171   parasite-locality combination examining a contiguous population. We excluded replicates

172   with under 100 samples, to ensure convergence of our standardised spatial models (see

173   below).

174   Although we principally aimed to quantify fine-scale, within-population spatial effects, we

175   included several studies employing continuous or semi-continuous sampling at county and

176   national levels, to investigate whether the methods we used would operate well at these scales

177   and to establish an upper bound for sampling effects.

178

Statistical Analysis

Data standardisation

181   Data were manipulated and analysed using R version 3.6.3 (R Development Core Team,

182   2011). All code is available at github.com/gfalbery/libra. Our data cleaning procedure aimed

183   to minimise the probability of false positives and to restrict the data pool to a continuous

184   spatial distribution of samples. All spatial coordinates were converted to the scale of

185   kilometres or metres to allow comparison across systems. We removed spatial outliers and

186   parasite count outliers; if parasite counts were very overdispersed and/or highly zero-inflated

187   they were analysed as binomial (0/1) infection data rather than negative binomial. Categories

188   with low replication (generally <10 samples) were removed. We removed specific classes

189   that exhibited very low prevalence: e.g., adult Soay sheep and red deer had a very low

190   prevalence of *Nematodirus* sp., which is primarily a parasite of young ungulates (Hoberg,

191   Kocan, & Rickard, 2001); hence only lambs/calves were analysed. Individual identity was

192   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 (Kirby et al., 2017; Tobler, 1970). The model estimates how much variance is accounted for by autocorrelation, and models with and without the SPDE effect can be compared to assess how it affects the fit of the model (Lindgren & Rue, 2015; Zuur, Ieno, & Saveliev, 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 parasite burden (Gaussian or negative binomial) or presence/absence (binary) as a response variable and with any fixed and random covariates. 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, Simpson, Lindgren, & Rue, 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 "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 spatial replicate, each of which comprised a different host-locale-parasite combination, generated from 31 different study systems.

Meta-analysis of INLA models

To identify factors driving general trends of spatial variation, we conducted a meta-analysis treating each unique host-locale-parasite combination as a replicate, including parasite-, host-,

226 and sampling-level traits as fixed effects. We constructed hierarchical models using the

227 `metafor` package. Generally, meta-analyses typically focus on synthesizing effect sizes and

228 their variances across multiple systems (e.g. Sánchez *et al.* 2018). However, as generalised

229 spatial variation does not have a directional effect, we instead analysed measures of model fit,

230 predictive capacity, and the autocorrelation range, which is bounded at 0 and infinity. To give

231 a coarse measure of model predictive capacity that was easily standardised across all models,

232 we calculated the Spearman's Rank correlation between the observed and predicted values

233 for the model, using only the SPDE effect to predict (henceforth referred to as R). The

234 measures of model fit give an impression of the detectability and importance of spatial

235 patterns, while comparisons of the range estimate across systems will inform whether

236 different host and parasite traits cause spatial patterns to vary more sharply in space. We used

237 the *escalc* function to derive sampling variances for DIC weight and the INLA range (using

238 the point estimate and 95% confidence interval).

239 Our hierarchical models included each replicate as a random effect to account for within- and

240 between-study heterogeneity (Konstantopoulos, 2011). We also included a random effect for

241 host family, for which the covariance structure used the phylogenetic correlation matrix

242 (Nakagawa & Santos, 2012); we obtained our phylogeny from the Open Tree of Life with the

243 rotl and ape packages (Michonneau, Brown, & Winter, 2016; Paradis, Claude, & Strimmer,

244 2004). All models used the `rma.mv` function and weighting by sampling variance. We first

245 assessed heterogeneity in each of our response variables by fitting a random-effects model

246 (REM; intercept only) with restricted maximum likelihood and then used Cochran's Q to test

247 if such heterogeneity was greater than expected by sampling error alone (Borenstein, Hedges,

248 Higgins, & Rothstein, 2009).

249 We next used mixed-effects models (MEMs) to test how sampling-, host-, and parasite-level

250 factors affected our INLA model outputs. Sampling variables included: Number of samples;

251 Sampling area (total rectangular extent between the furthest points on the X- and Y-

252 coordinates, in km^2); Sampling method (3 levels: trapping, censusing, and

253 necropsy/convenience sampling); Spatial encoding method (4 levels: GPS; trapping grid;

254 locality; Easting/Northing); Spatial hypothesis testing (binary – i.e., did the study aim to

255 quantify spatial variation in some way?). We interpreted this latter variable as a combination

256 of study design and publication bias, where studies that are intended to pick up spatial

257 variation are both more likely to identify spatial patterns because of their sampling design,

258 and then more likely to be published if they do. Parasite traits included Transmission mode (4

259   levels: direct; faecal-oral; vector-borne; environmentally transmitted) and Taxon (8 levels:

260   arthropod, nematode, trematode, cestode, protozoan, bacterium, virus, other). Host traits

261   included: Home Range size (in km^2; log-transformed); Body Mass (in grams; log-

262   transformed); Host order (5 levels: Carnivora, Chiroptera, Ungulates, Glires, Proboscidea).

263   There was only one lagomorph, so rodents and lagomorphs were lumped together into the

264   "glires" clade. The same was true of odd-toed ungulates (Perissodactyla), so they were

265   lumped with Artiodactyla into an "ungulates" clade. For species for which a phenotypic

266   measure (e.g. body mass) was unavailable, we used the value for the closest relative for

267   which the data were available, according to a mammalian supertree (Fritz, Bininda-Emonds,

268   & Purvis, 2009).

269   To identify important drivers among these many correlated drivers, we conducted a model

270   addition process using maximum likelihood and Akaike Information Criterion corrected for

271   sample size (AICc) to determine model fit. Each of our meta-analytical explanatory variables

272   was added in turn, and the best-fitting variable (i.e., the one that most decreased AICc) was

273   kept for the following round. This process was repeated with the remaining variables, until no

274   variables improved model fit by more than 2 AICc. We report the final model, with the

275   minimal number of variables that improved model fit.

Spatiotemporal INLA models

277   Finally, we constructed spatiotemporal INLA models to assess the consistency of spatial

278   hotspots from year to year, and to investigate evidence of ephemeral waves of transmission

279   across the study systems. Of our 89 replicates, 44 replicates had more than one year of

280   sampling, with more than 100 spatial points per year, facilitating fitting spatiotemporal

281   models. For these replicates, we first reran the original models with the reduced dataset that

282   only included years with more than 100 replicates. We then fitted a spatiotemporal model

283   with a different spatially distributed effect (i.e. "spatial field") for each year, with no

284   autocorrelation between the fields. Improved model fit for this model would imply that the

285   spatial distribution of the parasite varied notably from year to year. Second, we fitted a similar

286   spatiotemporal model with an "exchangeable" autocorrelation specification between years.

287   This model format allows correlation between spatial fields, but without enforcing a time

288   sequence: that is, all fields were correlated by the same parameter ("Rho") regardless of how

289   far apart in time they were. The Rho parameter, which is bounded between -1 and 1, was then

290   interpreted to give an impression of the spatiotemporal consistency of the parasite distribution.

291 Parasites with high rho coefficients had very similar hotspots from year to year, while those
292 with low coefficients did not.

Results

294 Our literature review returned 3399 studies, and we screened a random selection of 1993
295 abstracts (over two weeks) to expedite data collection. 1151 of these were unsuitable because
296 they were in the wrong environment, host, or subject area, or had no data. This left 496
297 studies, for which we assessed data availability. Very few studies publicly archived
298 continuous, within-population spatial data. Only 3/496 studies (0.6%) had such data ready to
299 download, and 4 further studies had maps of samples from which we could easily digitise
300 sufficient data (Supplementary Figure 1). We also already owned 3 datasets. We then emailed
301 432 authors to request data if unavailable (Supplementary Figure 1). When we emailed them,
302 92 responded, 22 of which (23.9%) indicated that they had not collected any within-
303 population spatial data as part of their study (Supplementary Figure 1). After navigating a
304 number of other obstacles to data sharing, followed by initial data triage, 26 authors kindly
305 offered to provide us with spatial data, resulting in 36 total viable datasets. Of these 36
306 datasets, 31 had at least one continuous spatial population with >100 samples to which we
307 could apply INLA models.
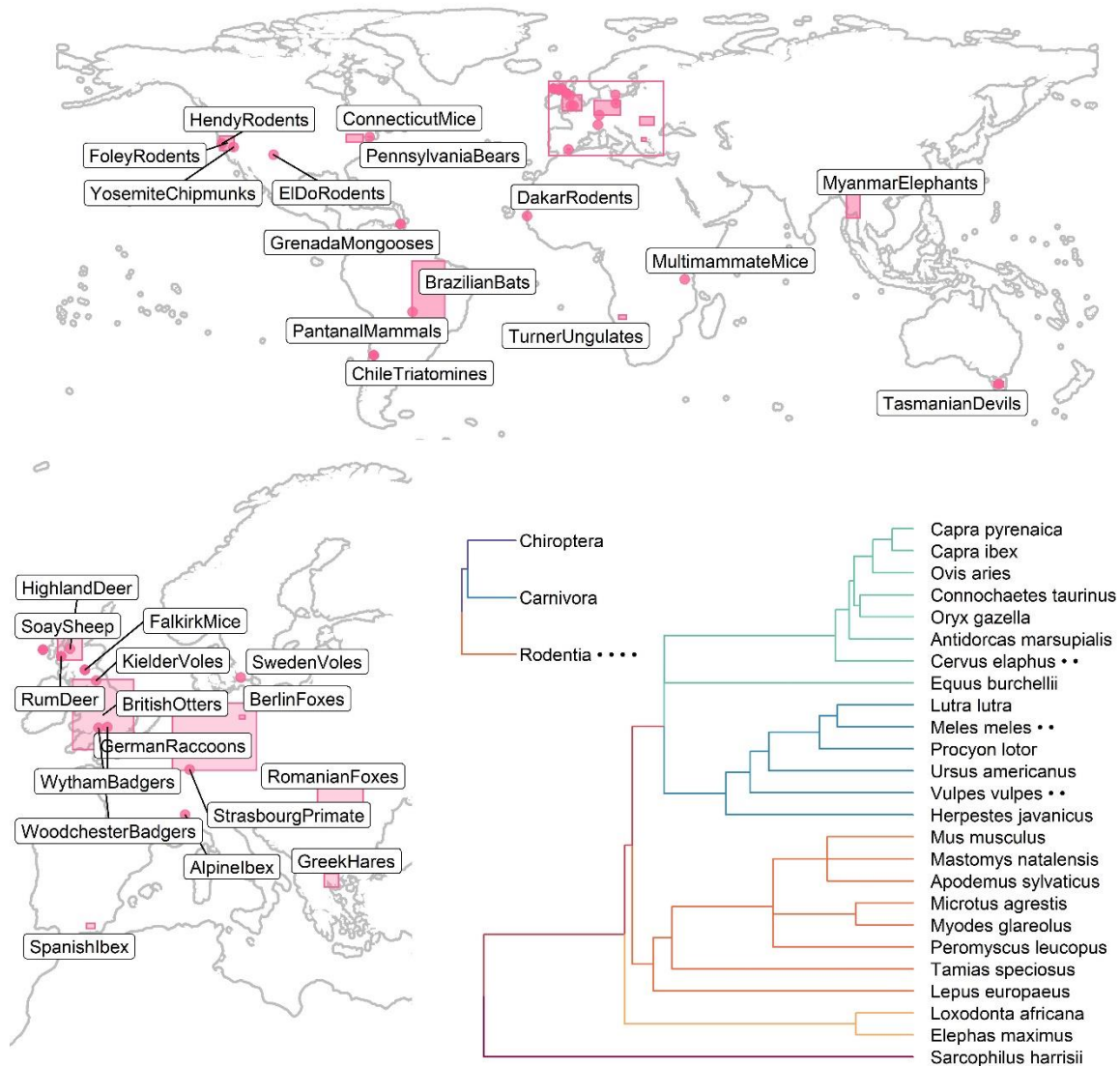
Figure 1: The geographic and taxonomic distribution of the 31 datasets that we included in our final meta-analysis. Our data were evenly spread across the earth (Panel A), although with a notable cluster in Western Europe (see inset map in pink rectangle, Panel B). Sampling areas greater than 5000 km^2 are displayed as rectangles; smaller sample areas are represented by dots. Study system names correspond to the names in Supplementary Table 1. The datasets also included a wide range of different mammal orders and families (Panel C). 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, and carnivorous marsupials.

Most authors that responded (and had collected spatial data) were happy to share data with us, and the vast majority of studies for which we did not receive data were due to a lack of

323 response or secondary response (Supplementary Figure 1). 15 authors responded but declined

324 to share data due to privacy concerns, ongoing data usage, or authorship concerns.

325 Comparing this to the 22 responders that had not collected spatial data implies that the main

326 reason researchers do not share spatial data is that they did not collect it; however, given that

327 >300 researchers did not respond (and they may not have been a random subset of the total),

328 our ability to infer this confidently is diminished. Notably, studies that investigated spatial

329 variation tended to be larger than those that did not (Supplementary Figure 2), implying that

330 larger study areas motivate researchers to more often consider spatial variation in their

331 analyses.

332

333 We concluded data collection with 31 datasets, including 89 spatial replicates and 90 host

334 species (Figure 1). 67 replicates were species-level; the rest were conducted on selections of

335 species in the same order (e.g., rodent trapping, bat sampling, carnivore faecal sampling). The

336 datasets were distributed across five continents (Figure 1), and included 7 different

337 mammalian orders (Figure 1). The studies examined 41 different parasites, across a diverse

338 selection including viruses (N=6), bacteria (N=10), helminths (N=25), arthropods (N=14),

339 and one transmissible cancer (N=8). Infection measures included counts of parasites or

340 immune markers (N=30), binary assessment of infection status using observation or

341 seropositivity (N=52), and one study used parasite-associated mortality as a proxy (Myanmar

342 elephants, *Elephas maximus* (Lynsdale, Mumby, Hayward, Mar, & Lummaa, 2017)). Study

343 systems included, for example: rodent trapping studies examining flea burdens and flea-borne

344 pathogens (e.g. rodents trapped in the Arizona hills (Kosoy et al., 2017) and chipmunks in

345 Yosemite National Park (Hammond et al., 2019)); long-term studies with parasite data

346 collected over the course of several decades (e.g. the Soay sheep of St Kilda (Hayward et al.,

347 2014), the Isle of Rum red deer (Albery et al., 2019), and the badgers of Wytham Wood

348 (Albery et al., 2020)); and studies examining seropositivity of mammals across a geographic

349 range to identify endemic areas (e.g. British otters infected with *Toxoplasma gondii*

350 (Smallbone et al., 2017)). See Supplementary Table 1 for a description of each study system

351 and the associated references and researchers that provided us with the data. The area of the

352 study systems varied widely, from 0.02 to 10^6 km^2 (Figure 2A).

353

354
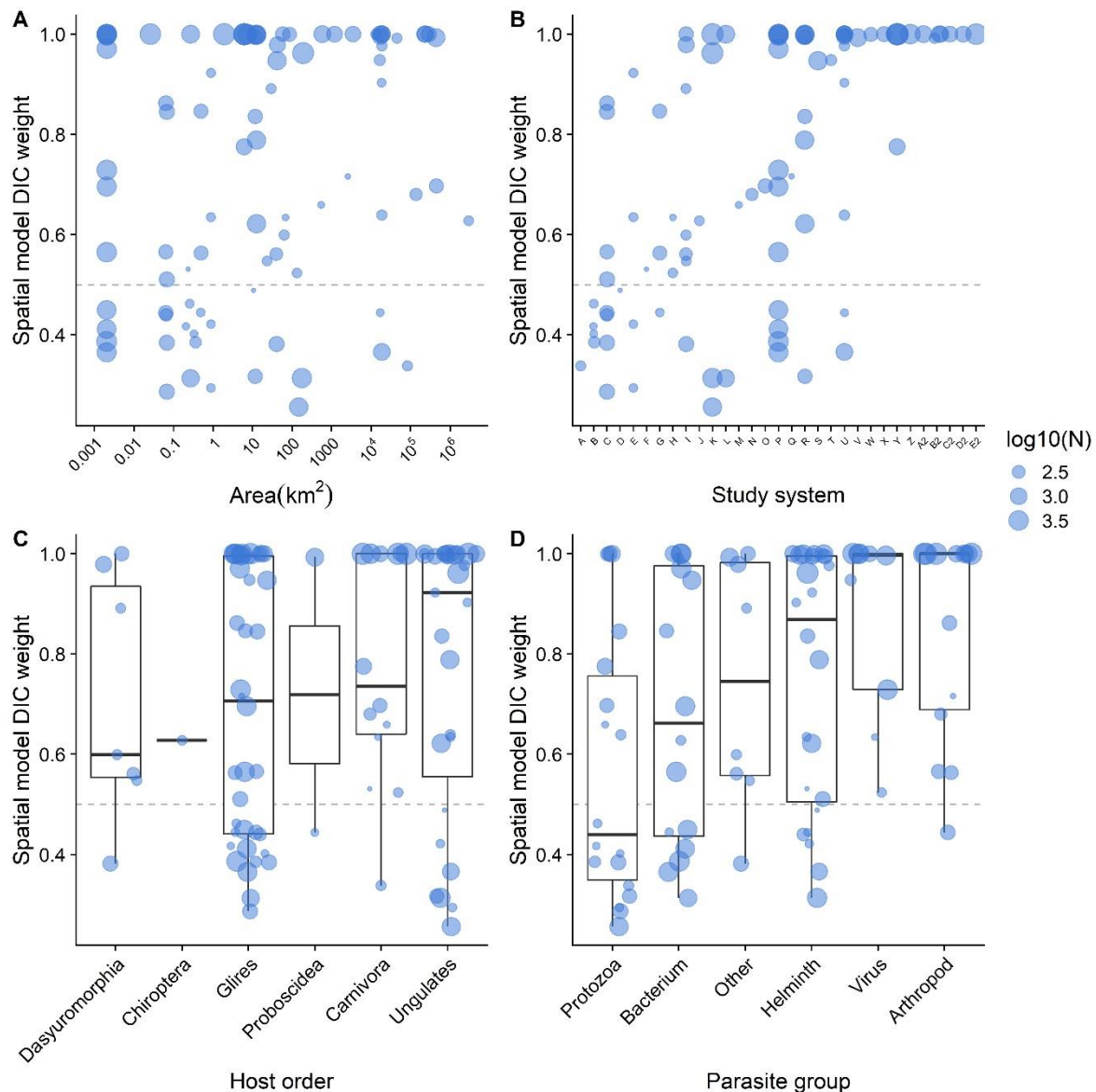
Figure 2: The spatial autocorrelation term (SPDE) improved models across host-parasite
systems and sampling regimes. The Y axis displays the degree of confidence that the spatial
autocorrelation term improved model fit (Deviance Information Criterion weight), where
models at the top of the panel fitted better than those at the bottom. The dashed line at DIC
weight=0.5 denotes the point at which spatial and non-spatial models were equally supported.
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.

367

368   Our INLA models applied across datasets consistently revealed strong spatial patterns of

369   disease (Figure 2-3). The mean DIC change across all study systems was -14.5 (median -3.3),

370   and the spatial model fit better than the base model for 65/89 models (73%; DIC weight>0.5).

371   Using a conventional change of 2ΔDIC as a cutoff for improved model fit, 54% of models

372   across 21 study systems displayed detectable spatial patterns (Figure 2). Cochran's *Q*

373   revealed very low heterogeneity between systems in terms of their DIC weight (*Q(df=86)* =

374   46.89, P=0.9998), but extreme heterogeneity in terms of the range of autocorrelation

375   (*Q(df=86)* = 3823, P<.0001).

376

377   Although half of the systems were spatially structured, our meta-analyses revealed that few

378   host-, parasite-, or sampling factors were predictive of spatial effects (see Supplementary

379   Table 2). The best-fitting model for DIC weight included only the study duration (years),

380   revealing that long-term studies were slightly more likely to uncover spatial effects

381   (ΔAIC=3.38; for all other variables ΔAIC<1.56). The INLA range parameter increased with

382   study area (ΔAIC=74.44) but was not affected by any other variables (ΔAIC<0.09). No

383   variation was accounted for by host or parasite taxon, host size, or host ranging behaviour.

384   Most notably, there was no significant variation in spatial range or DIC changes across

385   parasite transmission modes (Figure 3A-B).
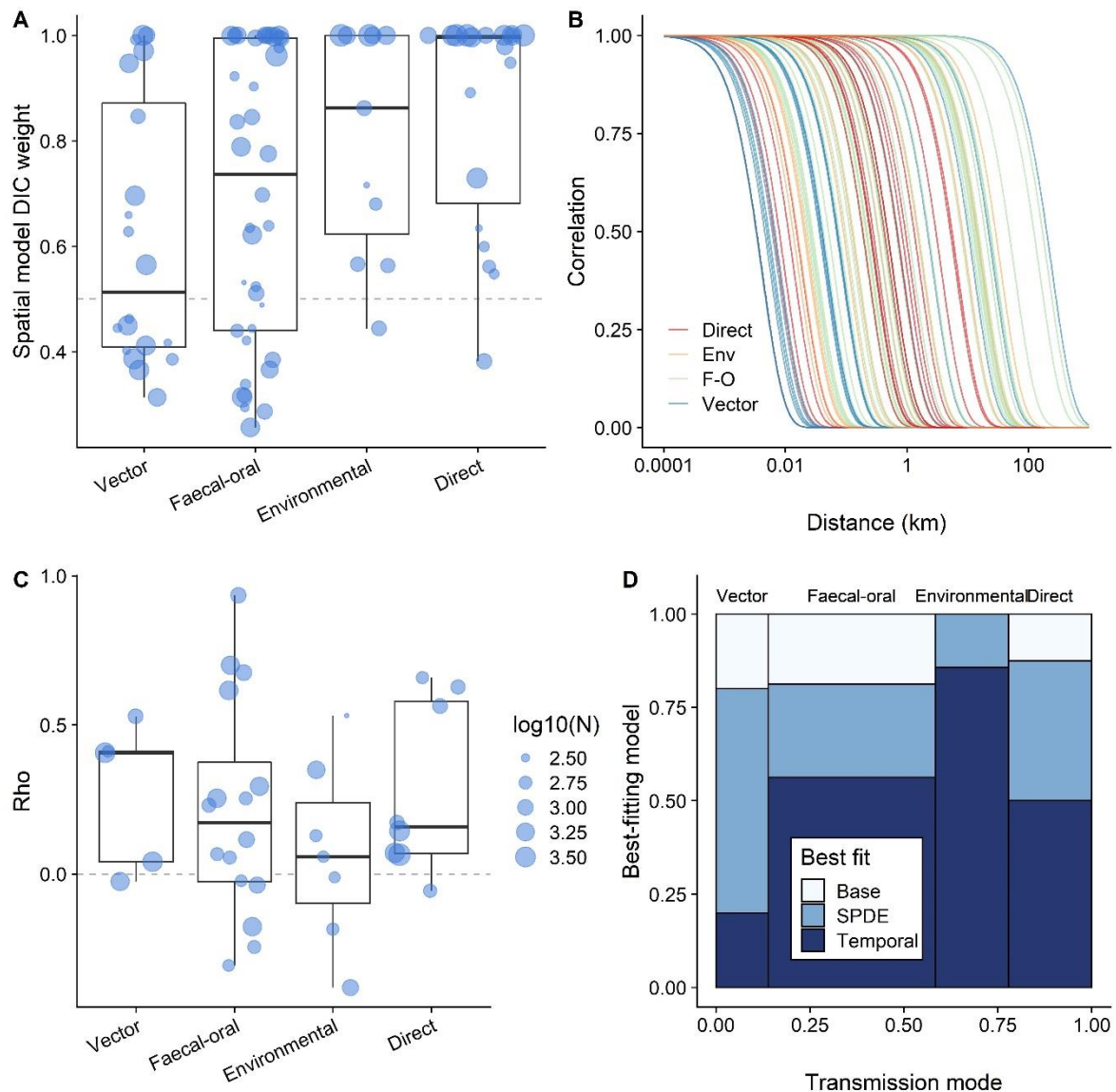
Figure 3: Parasites of diverse transmission modes exhibit spatial autocorrelation effects. We display A) spatial model DIC (deviance information criterion) weight, with points representing the outcome of each replicate INLA (integrated nested laplace approximation) model. Boxplots represent the range, interquartile range, and median for parasites of each transmission mode. The dashed line at DIC weight=0.5 denotes the point at which spatial and non-spatial models were equally supported; points above the line display host-parasite systems for which the spatial model was better supported than the non-spatial model. B) INLA autocorrelation ranges; each line represents the autocorrelation decay of a different replicate INLA model. The colours correspond to different transmission modes, demonstrating substantial mixing of the range estimates for parasites of different transmission modes. C) Temporal autocorrelation (Rho) component demonstrating inter-annual correlations between spatial fields, for the subset of model replicates that had multiple sampling years. Points represent a different replicate INLA model. The dashed line at Rho=0 represents the point of no correlation; points above the line had a positive correlation, while

401 points below the line had a negative correlation. D) Mosaic plot displaying the proportions of
402   best-fitting models according to DIC changes, across our spatiotemporal replicates.
403

404 Spatiotemporal models examining a subset of multi-year studies consistently improved model
405 fit over static equivalents. The best-fitting model for many examined replicates was a
406 spatiotemporal model, but the findings did not differ notably across transmission modes
407 (Figure 3D). Rho (temporal autocorrelation of the spatial field) estimates for these models
408 were moderate, and did not vary notably across transmission modes (Figure 3C). Most
409 (36/44, 82%) had 95% credibility intervals that overlapped with zero, and 8 (18%) were
410 significantly positive.

Discussion

412 We uncovered strong, pervasive spatial heterogeneity manifesting within an expansive
413 diversity of mammal-parasite systems. Contrary to expectations, spatial heterogeneity was
414 equally common and short-ranged for all transmission modes, despite our prediction that
415 parasites with longer environmental stages would be more likely to exhibit spatial patterns.
416 There are therefore three main takeaways from our findings: first, many study systems are
417 spatially structured, likely by a combination of drivers, whether or not the study in question
418 aims to quantify spatial variation or environmental drivers. Second, these drivers are
419 relatively rarely investigated, but many systems currently have the spatial power and ability
420 to investigate them if they wish, irrespective of the host-parasite system involved. Third, we
421 were unable to develop a predictive framework for spatial dependence using the data
422 available, but given more data across a wider range of host-parasite systems, such a
423 framework may be possible to develop in the future. We therefore recommend that wild
424 animal studies in disease ecology more regularly collect and share data on spatial behaviours
425 and sampling locations where possible, regardless of host, parasite, or sampling regime.
426

427 Our methodology differed from that used in many other studies by investigating generalised
428 spatial dependence rather than by quantifying specific environmental drivers that might drive
429 this dependence. The only similar study that we know of (Gilbertson et al., 2016) used 48
430 parasite-locality replicates of cougar (*Puma concolor*) and bobcat (*Lynx rufus*) populations
431 and found little evidence of spatial autocorrelation in parasite infection. In contrast to their
432 approach, we used a wide set of different hosts, and our replicates all had between 100 and

433   10,000 samples (Supplementary Table 1), whereas only a few of their replicates had >100

434   samples, and none had >200 (Gilbertson et al., 2016). Additionally, they used Mantel tests,

435   which do not account for fixed covariates, while the INLA analyses we employed are more

436   suited to controlling for this variation. As such, we interpret our contrasting findings to

437   represent a difference in the power of our analyses, and the absence of large carnivores from

438   our dataset. Owing to its generality, similar methodology could be used in a range of

439   ecological contexts as a useful hypothesis-generating exercise: after uncovering strong spatial

440   structuring, researchers could follow up on this finding by investigating possible biotic or

441   abiotic drivers. We hope that more disease ecology studies in wild animals will make use of

442   similar methodology to ours to bolster our understanding of disease dynamics in wild

443   settings.

444

445   Surprisingly, neither larger study systems nor those that had previously been used to study

446   spatial hypotheses were more likely to exhibit detectable spatial patterns. Some very small

447   spatial replicates exhibited strong spatial effects, and the smallest area demonstrating a strong

448   spatial trend was 0.002km^2 (Figure 2). On the other hand, some very large, well-sampled

449   areas showed no detectable spatial patterns: for example, anti-*Toxoplasma gondii* antibodies

450   in almost 200 Pennsylvania black bears (*Ursus americanus*) were not autocorrelated (Dubey

451   et al., 2016) even though the prevalence of *T. gondii* exhibited very strong spatial patterns in

452   otters (*Lutra lutra*) across the United Kingdom (Smallbone et al., 2017), and in house mice

453   (*Mus musculus*) within the Senegalese city of Dakar (Galal et al., 2019). However, larger

454   study extents unsurprisingly exhibited more long-range spatial autocorrelation effects. These

455   areas inevitably contain within them a multitude of smaller spatial effects and gradients, so

456   the findings of a specific study will depend critically on the spatial sampling scale it employs

457   (Cohen et al., 2016; Luis et al., 2018; Morand et al., 2019; Pullan et al., 2012). Notably, the

458   studies that did attempt to quantify spatial variation tended to have substantially larger spatial

459   extent than those that did not (Supplementary Figure 2); this may represent a perception bias,

460   where researchers operating in larger study areas tend to anticipate spatial variation as being

461   more important to account for – or, *vice versa*, researchers asking spatial questions tend to

462   sample across a wider range to incorporate as much testable variation as possible (Becker,

463   Nachtmann, et al., 2019). The finding that larger study systems do not tend to more

464   commonly exhibit detectable spatial patterns in disease demonstrates that this perception bias

465   is perhaps unwarranted, and researchers at all scales should be able to incorporate spatial

466   components and hypotheses about infection processes.

467

468   Despite the ubiquity of spatial effects, we discovered a very low frequency of spatial data

469   collection and sharing: only 3 publicly available datasets included spatial data, and 22/92

470   responders said they had not collected any spatial data. The responses that we received

471   implied that, alongside concerns about privacy and the understandable desire to control the

472   data associated with one's study system, the main reason for not sharing spatial data was that

473   the data were not collected in the first place. Location data may evade collection in some

474   contexts where GPS signals are hard to receive, precluding spatial data collection and

475   investigation of spatial questions. GPS instruments that function in remote environments can

476   be expensive, and for studies that do not explicitly aim to identify spatial patterns this may

477   seem an unnecessary expenditure. However, smartphones that can receive GPS data are now

478   widely available and can be used in all but the most remote locations. As many researchers

479   carry the means to collect spatial data in their pocket on a daily basis, it might take little

480   alteration to collection protocols to include location data in many cases. Future studies should

481   capitalise on the increasing availability of spatial telemetry and biologging technology, and

482   associated analytical capacity (Kays, Crofoot, Jetz, & Wikelski, 2015; Long, Nelson, Webb,

483   & Gee, 2014; Williams et al., 2020) to more frequently record, analyse, and share spatial data

484   in disease ecology (Albery et al., 2019; Kirby et al., 2017). This practice will facilitate easier

485   testing of the hypotheses that we outline above, as well as informing sampling regimes and

486   mechanistic models of disease dynamics, and allowing *a priori* prediction of host-parasite

487   systems' spatial properties. Moreover, large-scale, integrative analyses of disease processes

488   across systems are increasingly being used to inform on the epidemiological consequences of

489   global change (e.g. (Cohen, Sauer, Santiago, Spencer, & Rohr, 2020); increased availability

490   of geographically-tied disease samples could profoundly help our ability to carry out such

491   analyses, perhaps ultimately moving us towards developing a "weather system" for infectious

492   disease outbreaks.

493

494   Privacy is an issue of considerable ethical concern in epidemiology (Kirby et al., 2017), and

495   we contend that this concern may be contributing to a lack of open data sharing in wildlife

496   disease ecology. Sharing spatial data risks connecting individuals with their disease status,

497   which is particularly unwelcome in the case of stigmatised diseases such as HIV/AIDS;

498   indeed, although we did not examine human diseases, several of the researchers we contacted

499   opted not to share data because they were concerned that their results could be traced to

500   specific households or individuals. Researchers could 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 may preclude investigation of specific geographic hypotheses or environmental drivers, but this is a small price to pay in the cases where data are potentially sensitive.

We foresee a range of potential uses for curated datasets like ours. For example, further analysis on this dataset could investigate a number of general drivers such as population density or environmental heterogeneity, informing how they drive spatial patterns of infection within and across systems. Moreover, 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 like dolphins that are regularly subject to behavioural censuses and disease surveillance (e.g. Leu, Sah, Krzyszczyk, Jacoby, & Mann, 2020). This approach could also be applied to intensively and widely spatially distributed sampling locations, either for smaller animals such as insects (Wallace et al., 2021) or for immobile organisms like plants (Halliday 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). Given these diverse and widespread opportunities, popularising the breadth and frequency of open spatial data sharing is likely to open the door to a wide range of interesting studies and, ultimately, to the development of predictive *a priori* frameworks for spatial processes in disease ecology.

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