



Swansea University  
Prifysgol Abertawe



## Cronfa - Swansea University Open Access Repository

---

This is an author produced version of a paper published in:  
*Global Change Biology*

Cronfa URL for this paper:  
<http://cronfa.swan.ac.uk/Record/cronfa45072>

---

### **Paper:**

Wells, K., Gibson, D., Clark, N., Ribas, A., Morand, S. & McCallum, H. (2018). Global spread of helminth parasites at the human-domestic animal-wildlife interface. *Global Change Biology*, 24(7), 3254-3265.  
<http://dx.doi.org/10.1111/gcb.14064>

---

This item is brought to you by Swansea University. Any person downloading material is agreeing to abide by the terms of the repository licence. Copies of full text items may be used or reproduced in any format or medium, without prior permission for personal research or study, educational or non-commercial purposes only. The copyright for any work remains with the original author unless otherwise specified. The full-text must not be sold in any format or medium without the formal permission of the copyright holder.

Permission for multiple reproductions should be obtained from the original author.

Authors are personally responsible for adhering to copyright and publisher restrictions when uploading content to the repository.

<http://www.swansea.ac.uk/library/researchsupport/ris-support/>



26

27 **Keywords:** Global spread of parasites, helminth parasites, human–wildlife interface, parasite  
28 biodiversity, parasite host shifting, zoonoses

29

### 30 **Abstract**

31 Changes in species distributions open novel parasite transmission routes at the human–  
32 wildlife interface, yet the strength of biotic and biogeographical factors that prevent or  
33 facilitate parasite host shifting are not well understood.

34 We investigated global patterns of helminth parasite (Nematoda, Cestoda, Trematoda)  
35 sharing between mammalian wildlife species and domestic mammal hosts (including  
36 humans) using > 24,000 unique country-level records of host-parasite associations. We used  
37 hierarchical modelling and species trait data to determine possible drivers of the level of  
38 parasite sharing between wildlife species and either humans or domestic animal hosts. We  
39 found the diet of wildlife species to be a strong predictor of levels of helminth parasite  
40 sharing with humans and domestic animals, followed by a moderate effect of  
41 zoogeographical region and minor effects of species' habitat and climatic niches. Combining  
42 model predictions with the distribution and ecological profile data of wildlife species, we  
43 projected global risk maps that uncovered strikingly similar patterns of wildlife parasite  
44 sharing across geographical areas for the different domestic host species (including humans).  
45 These similarities are largely explained by the fact that widespread parasites are commonly  
46 recorded infecting several domestic species.

47 If the dietary profile and position in the trophic chain of a wildlife species largely drives its  
48 level of helminth parasite sharing with humans/domestic animals, future range shifts of host  
49 species that result in novel trophic interactions may likely increase parasite host shifting and  
50 have important ramifications for human and animal health.

51

**52 Introduction**

53 The emergence of parasitic diseases is largely a consequence of the exploitation of novel host  
54 species by parasites capable of shifting hosts (Lloyd-Smith *et al.*, 2009). A central goal in  
55 disease ecology is thus to identify factors that enable parasite sharing, especially since  
56 determinants of parasite sharing can influence the spread of parasites to new habitats and  
57 biogeographic regions. For zoonotic diseases (i.e. infectious diseases of humans caused by  
58 parasites that have an animal reservoir) a key determinant of emergence is overlapping  
59 environmental conditions and biological traits that enable parasites to be shared by human  
60 and animal hosts. Along early human migration pathways, increased physical contact with  
61 endemic animal and plant species led to increased exposure to novel parasites (Pedersen &  
62 Davies, 2009; Pulliam, 2008), especially those acquired through ingestion of wild animal  
63 meat (Reinhard *et al.*, 2013). Anthropogenic land use, conversion of natural habitats into  
64 production landscapes, and intensification of international travel and wildlife trades continue  
65 to diminish or shift former geographical barriers between humans and wildlife, likely  
66 facilitating exposure to novel parasite pools (Murray *et al.*, 2015; Patz *et al.*, 2008). In  
67 contrast, decreasing wildlife populations and the isolation of populations through habitat  
68 fragmentation (through construction of roads or other barriers that prevent animal movement)  
69 may effectively decrease contact between humans and wildlife.

70         While direct human-wildlife parasite sharing is a topic of major importance, domestic  
71 animals that occur in close proximity to humans may also act as key hosts for wildlife  
72 parasites. Domestic animals (hereafter including domesticated animals, such as dogs, but also  
73 animals that live in close proximity to humans, such as commensal rats) have colonised  
74 almost all terrestrial environments (Hoberg & Brooks, 2008; Matisoo-Smith *et al.*, 1998). In  
75 turn, domestic animals commonly share subsets of their parasite fauna with humans. This

76 subset increases the longer a species has been domesticated (Morand *et al.*, 2014; Wolfe *et*  
77 *al.*, 2007). Parasite host shifting at the interface between humans, domestic animals and  
78 wildlife is a multifaceted and multidirectional problem, with potential effects for human and  
79 wildlife health (Daszak *et al.*, 2000). Yet, while previous studies found 60 % of human  
80 diseases to be of zoonotic origin (Taylor *et al.*, 2001; Woolhouse & Gowtage-Sequeria,  
81 2005), global patterns in parasite sharing at the human–domestic animal–wildlife interface  
82 are poorly resolved.

83         Predicting zoonotic disease risk requires understanding wildlife characteristics that  
84 enable host shifting at local and global scales (Hoberg & Brooks, 2008). Host attributes, such  
85 as phylogenetic relatedness or overlap in habitat use, are useful for predicting whether hosts  
86 share the same parasite species through ecological fitting (Streicker *et al.*, 2010; Wells *et al.*,  
87 2015) or how invasions into novel environments may result in novel host-parasite  
88 associations (Agosta & Klemens, 2008; Clark *et al.*, 2017). Conversely, knowledge of  
89 whether species attributes such as demography, body size or diet increase the likelihood of  
90 sharing parasites with humans, and whether zoonotic disease burdens in humans or domestic  
91 animals exhibit biogeographical structure, remains sparse (Han *et al.*, 2015; Just *et al.*, 2014;  
92 Stephens *et al.*, 2016).

93         A key gap in our understanding of zoonotic disease emergence is information on how  
94 patterns of wildlife parasite sharing differ among domestic host species or across  
95 biogeographical regions. Despite persisting in close spatial proximity, humans and domestic  
96 animals differ in habitat use, diet and other ecological traits. This may have consequences for  
97 determining subsets of parasites that humans and domestic animals share with wildlife.  
98 Humans and dogs, for example, each consume a large range of invertebrate and vertebrate  
99 species (many of which may be relevant reservoir hosts) and can access almost any type of  
100 terrestrial habitat. Other domestic species, such as cows, are confined to relatively few

101 habitats and food items (e.g. grassland vegetation). One may expect that different domestic  
102 animals will exhibit different patterns of wildlife parasite sharing and, consequently, different  
103 potential roles as carriers of zoonotic parasites. Globally, wildlife communities occur in  
104 distinct species assemblages according to biogeographical history, speciation events and  
105 habitat biomes (Holt *et al.*, 2013; Kraft *et al.*, 2007; Wallace, 1876). Such biogeographical  
106 structure may lead to spatial gradients in wildlife parasite sharing for humans and domestic  
107 animals.

108         Here, we investigate possible drivers of helminth parasite (Nematoda, Cestoda,  
109 Trematoda) sharing between wildlife and focal domestic host species (including humans) at a  
110 global scale. Using a large database of mammalian host-parasite associations, we addressed  
111 two key questions: 1) Which species traits make wildlife most prone to share helminth  
112 parasites with humans or domestic species? 2) Do patterns of wildlife parasite sharing exhibit  
113 biogeographical structure across the globe? Given that humans share parasites most  
114 intensively with domestic species, we expect to find similar patterns of wildlife parasite  
115 sharing among humans and domestic animals. We expect this to be especially true when  
116 comparing patterns for humans and dogs, as dogs have a long domestication history and share  
117 a broad range of habitats with humans. We also expect biogeographical structure in wildlife  
118 assemblages to drive global patterns in wildlife–human and wildlife–domestic animal parasite  
119 exchange, as different wildlife traits may facilitate or impede parasite transmission cycles and  
120 host shifting abilities.

121

## 122 **Materials and Methods**

### 123 **Host-parasite database**

124 We compiled a global database of mammalian host–parasite associations from the publicly  
125 available Natural History Museum (NHM), London’s Host-Parasite Database (Gibson *et al.*,

2005). This database has been used as a backbone for the comprehensive Fauna Europaea biodiversity inventories of parasitic worms (Gibson & Bray, 1994; Gibson *et al.*, 2014) and is arguably the largest publicly available compilation of country-level records for helminth host associations to date. In humans, for example, previous estimates suggested > 300 helminth species infecting humans (Crompton, 1999), whereas our database reports a total of 397 helminth species (Nematoda, Cestoda, Trematoda) to be associated with humans. We downloaded all host-parasite data from the database using web-scraping tools implemented in the package *xml* in the software R (R Development Core Team, 2017). The data of interest for our study were country-specific combinations of parasite-mammal species associations, which included information from wild and domestic mammals as well as humans. We excluded all records from captive animals or experiments, and considered only records that included full binomial species names (scientific genus and species names). As the original database records were not specified in detail, records may include reports of molecular identification of parasite species and also dead-end hosts, from which parasites are not transmitted to other species. Mammal species synonyms were standardised using the taxonomy of Wilson & Reeder (2005) and the IUCN Red List. Parasite names were standardised using the WoRMS database (<http://www.marinespecies.org>), the tapeworm database at the University of Connecticut (<http://tapewormdb.uconn.edu/>) and GBIF ([www.gbif.org/](http://www.gbif.org/)). Location names were standardised to country names of the current world geopolitical map and assigned to one of 11 global zoogeographical regions according to Holt *et al.* (2013). Since China covers different zoogeographical regions, and not all records from China could be assigned to any particular region, we classified these unspecified records into an extra category (“China unspecified”).

Our dataset consisted of 24,486 unique combinations of host–parasite–country records for selected helminth species (Nematoda, Cestoda, Trematoda), of which 1,737 involved humans

151 as a host, from a total of 4,507 selected parasite species. Of the 1,366 total mammalian host  
152 species in our dataset, we considered 21 species as ‘domestic’ (including humans and  
153 commensal murids) and all others as ‘wildlife’. Domestic species were banteng (*Bos*  
154 *javanicus*), yak (*B. mutus*), cow (*B. taurus*), bactrian camel (*Camelus bactrianus*), dromedary  
155 (*C. dromedarius*), dog (*Canis familiaris*), goat (*Capra aegagrus*), guinea pig (*Cavia*  
156 *porcellus*), wild ass (*Equus africanus*), donkey (*E. asinus*), horse (*E. caballus*), cat (*Felis*  
157 *catus*), human (*Homo sapiens*), guanaco (*Lama guanicoe*), house mouse (*Mus musculus*),  
158 rabbit (*Oryctolagus cuniculus*), sheep (*Ovis aries*), brown rat (*Rattus norvegicus*), black rat  
159 (*R. rattus*), pig (*Sus scrofa*) and vicugna (*Vicugna vicugna*). From these domestic species, we  
160 selected parasite assemblages (Nematoda, Cestoda, Trematoda) from seven focal host species  
161 (hereafter termed focal species) to examine associations with wildlife: man, dog, cat, cow,  
162 pig, black rat and brown rat. Focal host species were selected because they are among the  
163 most cosmopolitan host species and are represented with enough records in the database to  
164 facilitate statistical inference of wildlife parasite sharing patterns.

165         We are aware that this dataset is incomplete in that it lacks recently described parasite  
166 species and recent records of host-parasite associations in different locations; while this limits  
167 inference about absolute species numbers, we believe this dataset provides meaningful  
168 insights into the relative strength of how wildlife species share parasites with domestic  
169 species in relation to ecological traits and projected global maps, which were the main  
170 interests of this study.

171

## 172 **Host ecological traits**

173 We selected nine ecological traits of terrestrial mammals, based on the PanTHERIA (Jones *et*  
174 *al.*, 2009) and EltonTraits 1.0 (Wilman *et al.*, 2014) databases, to include a broad range of  
175 attributes likely to distinguish hosts in terms of their suitability for a parasite’s life and



176 transmission cycles. Selected traits included: body mass, which is a key feature of mammals  
177 in terms of their metabolism and adaptation to environments; average longevity, litter size  
178 and the average number of litters per year as demographic parameters that could be relevant  
179 for allowing parasites to complete parts of their life cycles in a host; diet breadth (calculated  
180 as a Shannon diversity index based on the proportional use of 10 diet categories as presented  
181 in EltonTraits) and diet class ('invertebrate predator', 'herbivore', 'omnivore' or 'carnivore')  
182 as trophic interactions traits; range area, which we expect to affect the exposure to other  
183 mammalian host species; average temperature within range as an indicator of climate niche;  
184 and habitat as multiple binary indicators of whether a species uses 1) forest, 2) open  
185 vegetation, and/or 3) artificial/anthropogenic habitats. Information about specific habitat  
186 utilisation was compiled from the International Union for the Conservation of Nature (IUCN)  
187 database (IUCN, 2014). We did not include a larger set of ecological traits in our analysis to  
188 avoid trait autocorrelation and colinearity issues in the modelling.

189 We accounted for phylogenetic distances between wildlife species and focal domestic species  
190 based on a correlation matrix (Paradis *et al.*, 2004) of phylogenetic branch lengths, which  
191 was built using a recent mammalian phylogenetic supertree (Bininda-Emonds *et al.*, 2007).  
192 We further considered the orders Carnivora, Rodentia and Primates as binary (categorical)  
193 indicator variables for the major taxonomic groups that are suspected to share parasites with  
194 the focal species; we used these as indicators to account for possible group-level taxonomic  
195 effects additional to the phylogenetic branching. To account for sampling bias among wildlife  
196 species, we queried the number of published references for each binomial wildlife species  
197 name from the Scopus literature database (accessed 25/02/2017); we used this measure as  
198 more refined searches, such as the number of references linked only to parasites, included  
199 large proportions of zeros and information on the true number of sampled individuals (which  
200 should determine the chance parasites are detected if prevalence is low) was not available.

201

202 **Statistical analysis**

203 The primary goal of this study was to identify drivers of parasite sharing between focal  
 204 domestic species and wildlife. We addressed this aim using logistic hierarchical regressions  
 205 to analyse the relative strength of covariates that could determine the probability of parasites  
 206 from either humans or domestic species to be found in wildlife species (calibrated on host  
 207 species from the NHM database). For each focal domestic host species  $d$ , we constructed  
 208 presence-absence vectors  $Y_d(w_r, p_d)$  that encompassed all combinations of mammalian  
 209 wildlife species  $w_r$  (i.e. non-domestic species in the database) surveyed for parasites from any  
 210 zoogeographical region  $r$  and all parasite species  $p_d$  from one of the selected parasite groups  
 211 (Nematoda, Cestoda, Trematoda) recorded in the respective focal species. Here, we assume  
 212 that any wildlife species recorded in our database has been potentially examined for all  
 213 parasite species  $p_d$  known to occur in the respective region; the absence of such records are  
 214 set to 0. These ‘absence records’ likely include false zeros due to missing observations and  
 215 hence underestimate the link of parasite species from focal hosts to wildlife; however, we  
 216 prefer this approach to presence-only modelling, as the true proportion of wildlife species  
 217 infected remains unknown, and we thus expect techniques such as data imputation not to  
 218 improve our analysis.

219 We assumed the resulting data vectors  $Y_d(w_r, p_d)$  are random draws from the  
 220 underlying association probability  $\Psi_d(w_r, p_d)$  of a wildlife species sharing a parasite with a  
 221 focal species according to a Bernoulli distribution, as commonly used in logistic regression.

222 We modelled the probability  $\Psi_d(w_r, p_d)$  further using a logit-link function such that

$$223 \quad \text{logit}[\Psi_d(w_r, p_d)] = \mu_{Parasite}(p_d) + \mu_{Region}(r) + B ET_{wr}$$

224 where  $\mu_{Parasite}(p_d)$  is the parasite-specific intercept,  $\mu_{Region}$  are coefficient estimates that  
 225 account for variation across zoogeographical regions  $r$ , and  $B$  is a vector of coefficient

226 estimates that accounts for variations in the association risk linked to the matrix of covariates  
227  $ET_{wr}$  of the nine host ecological traits, the phylogenetic distance of wildlife to the focal host  
228 species and the number of publications, as specified above.

229 We used a hierarchical model with a common hyperprior  $\eta_\mu$  for the intercept as

$$230 \quad \mu(p_d) = N(\eta_\mu, \varepsilon_\mu).$$

231 where  $\varepsilon_\mu$  is a random Gaussian variance term that allows species-specific intercepts to vary  
232 from the hyperprior (no group specific hyperprior was specified as we ran models separately  
233 for the three parasite groups). We fitted the model in a Bayesian framework with Markov  
234 Chain Monte Carlo (MCMC) sampling based on the Gibbs sampler in the freeware  
235 OpenBUGS (Lunn *et al.*, 2012). We used a Gibbs variable selection procedure (GVS) to only  
236 include variables in the model if sufficiently supported by the fit to data and joint sampling of  
237 the most likely coefficient values (selection frequencies were typically high for covariates  
238 with significant effects, except for the categorical effects of ‘region’). We used normal priors  
239 with mean = 0 and variance  $\sigma \sim Exp(1)$  for intercepts and all regression coefficients if selected  
240 as part of the GVS, and  $\sigma \rightarrow 0$  otherwise. This prior gives close approximation to a logistic  
241 distribution and is appropriate for logit-scale estimates (Lunn *et al.*, 2012). Convergence of  
242 MCMC chains was assessed visually and with Gelman-Rubin diagnostics (all values < 1.2)  
243 after burn-ins > 50,000 MCMC samples. Parameter estimates were calculated as posterior  
244 modes and 95% highest posterior density credible intervals (CI) from 5,000 samples.  
245 Posterior predictive checks assessed whether model assumptions were reasonable  
246 approximations of the data generating process, with Bayesian  $P$ -values around 0.5 indicating  
247 a good fit. This model checking approach essentially compares whether the observed data  
248 resemble data simulated from the posterior distribution (Gelman *et al.*, 1996). All covariates  
249 were scaled (dividing centred values by one SD) and log-transformed if featuring  
250 overdispersion (body mass, range area, number of publications) to facilitate comparison of

251 effect sizes. Missing values of ecological trait covariates were imputed during MCMC  
252 updates, randomly drawing values from priors according to the mean and variance of all  
253 known trait values (considering all information in the trait databases) from species in the  
254 same orders. Specific trait data are currently not available for a considerable diversity of  
255 mammalian species; consequently, because of our ability to meaningfully impute missing  
256 data using a Bayesian sampling approach, account for uncertainty in parameter estimates, and  
257 make reasonably accurate parameter estimates, we preferred this approach over others, such  
258 as machine learning techniques, which are commonly used to more flexibly model nonlinear  
259 and interaction relationships (Elith *et al.*, 2008). Significance of model effects was  
260 determined by examining whether the 95% CI of regression coefficients did not overlap zero  
261 for continuous covariates or were clearly distinguished from each other for categorical  
262 covariates.

263         We computed the relative risk that a wildlife species will share parasites with each of  
264 the focal host species for all 5,289 terrestrial mammal species in the IUCN database by  
265 entering species' ecological traits into equations from the fitted models above (using posterior  
266 modes of parameter estimates). We hereafter refer to this relative risk as the association risk,  
267 which is appropriate in this case since the analysed data vectors included all combinations of  
268 parasites from a focal host and wildlife species. Thus, the association risk would be '1' if a  
269 wildlife species is likely to share all parasite species known from a particular focal species.  
270 We set the respective parameter values to zero if trait variables were missing (i.e. assuming  
271 an 'average' effect of the respective covariate for computing the respective species-level  
272 association risk).

273         The second aim of this study was to examine whether patterns of wildlife parasite  
274 sharing among domestic hosts exhibit biogeographical structure, which could be informative  
275 for understanding the future spread of zoonotic parasites. We addressed this goal by

276 exploring global patterns in observed parasite associations for focal host species and  
277 forecasted associations of wildlife infestation with parasites shared with the focal host  
278 species. First, for each focal host species, we used our model outputs to generate a series of  
279 maps (10km<sup>2</sup> raster cell sizes) to forecast global patterns in both wildlife parasite association  
280 risks and parasite assemblage structure. Using IUCN geographical range maps for wildlife  
281 species, we projected the respective parasite association risks on a global raster and, for each  
282 cell, computed average species-level and cumulative community-level geographical  
283 association risks for local wildlife assemblages. We were not able to account for possible  
284 regional variation in realized host-parasite interactions (which could arise due to variation in  
285 local conditions that enable host-parasite interactions) within the given wildlife range maps  
286 and, for simplicity, assumed homogenous association risks throughout species' given ranges.

287         Next, to explore variation in parasite assemblage structure across zoogeographical  
288 regions, we computed for each cell the hypothetical presence of focal host parasites in local  
289 wildlife by assuming that a parasite species occurred throughout the range of its associated  
290 wildlife host species. We then aggregated the presence-absence of these parasites at the  
291 zoogeographical region level and calculated parasite species turnover across regions using the  
292 *βsim* index, a basic turnover index that is based on the number of shared and unique species  
293 and is relatively unbiased by species richness (Lennon *et al.*, 2001). As an index of parasite  
294 assemblage distinctiveness in each region, we calculated the mean of all region-specific  
295 pairwise *βsim* indices. We explored geographical sampling bias by computing the number of  
296 wildlife species examined for helminths (including species not found in domestic host  
297 species) and wildlife species richness for each cell.

298         Spearman rank correlation tests were used to compare biogeographical patterns. First,  
299 we assessed whether infestation of a greater number of focal host species leads to broader  
300 biogeographical spread by testing the correlation strength between the Shannon index of

301 biogeographical spread and the total number of associated focal host species. We then  
 302 explored whether wildlife species show similar biogeographical patterns in the risk for  
 303 sharing parasites with different focal host species by testing all pairwise correlations between  
 304 the geographical association risks for parasites from the different focal host species.

305 We quantified the biogeographical spread of parasites (Nematoda, Cestoda,  
 306 Trematoda) found in any focal species. We did this by calculating a Shannon index  $H_p$  for  
 307 each parasite species  $p$  to account for both ‘richness’, according to the number of  
 308 zoogeographical regions where a species was recorded, and ‘evenness’, according to the  
 309 proportion  $\varphi_p(r)$  of wildlife species infected with the respective parasite species in each  
 310 zoogeographical region  $r$  (Magurran, 2004). We calculated the index as

$$311 \quad H_p = \sum_{r=1}^R \varphi_p(r) \ln[\varphi_p(r)].$$

312 Larger values indicate higher proportions of wildlife species infected and a more even spread  
 313 by the parasite across zoogeographical regions.

314 All statistical analyses and distributional map constructions were conducted separately for the  
 315 three groups of Nematoda, Cestoda and Trematoda using R (R Development Core Team,  
 316 2017) for data preparation and summary statistics.

317

## 318 **Results**

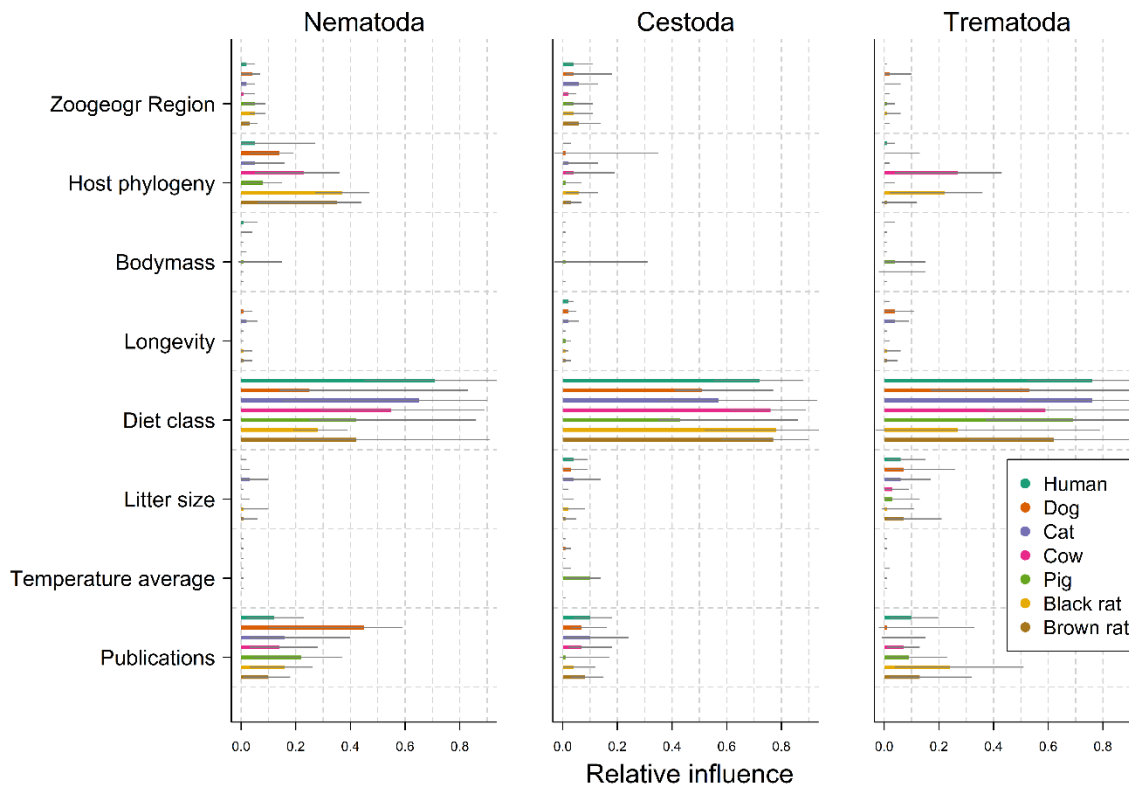
319 Of the 1,345 mammalian wildlife species in our host-parasite database, 41 % (n = 553 spp.)  
 320 were infected with helminth parasite species (Nematoda, Cestoda and Trematoda) also found  
 321 in humans. For humans, in turn, 49 % (195 of 397 spp.) of all helminth parasite species were  
 322 also found in wildlife and 45 % (182 spp.) in at least one other domestic host species. The  
 323 wildlife species associated with the highest numbers of zoonotic parasites were *Vulpes vulpes*  
 324 (red fox, 51 spp.), *Canis lupus* (grey wolf, 33 spp.) and *Nyctereutes procyonoides* (raccoon  
 325 dog, 29 spp.). For the other focal domestic host species, proportions of examined wildlife

326 species that shared parasites ranged from 21 – 31 % (**Table S1, Supplementary**  
327 **Information**).

328 Diet class was the strongest predictor of sharing parasites with focal host species for all  
329 combinations of parasites (Nematoda, Cestoda, Trematoda) and focal host species, explaining  
330 25 – 78 % of variation in wildlife infestation risk (all 95 % credible intervals, CIs = 13 – 96  
331 %) (**Fig. 1**). Wild insectivorous and omnivorous mammals were at significantly lower risk of  
332 sharing parasites with humans than were herbivores and carnivores, a pattern that was also  
333 true for other domestic host species (with a few exceptions; **Fig. S1**). Risks of wildlife  
334 species sharing parasites with the focal species also differed across zoogeographical regions.  
335 Overall, risks were relatively high in the Palaearctic region (**Fig. S2**), though some  
336 combinations of parasite and domestic host species exhibited other informative  
337 zoogeographical patterns. Wildlife had increased risk of sharing trematodes with cows and  
338 black rats in the Neotropical region and increased risk of sharing nematodes with humans,  
339 dogs and cats in the Nearctic region. In contrast, the risk for wildlife sharing cestodes with  
340 focal host species was generally low in the Neotropical region (**Fig. S2**). Nevertheless, the  
341 overall effect of zoogeographical region was weaker than the effect of diet class (**Fig. 1**).

342 Coefficient estimates for all other covariates are presented in **Table S2**; notably, various  
343 coefficient estimates were significantly different from zero, though they explained much less  
344 variance than diet and zoogeographical region. Bayesian  $p$  values ranged from 0.43 to 0.79  
345 for the various models.

346



347

348 **Figure 1.** Relative influence (% variance explained) of wildlife host ecological traits,  
 349 zoogeographical region and number of publications (a surrogate of sampling effort) on the  
 350 probability that wildlife species shared helminth parasite species with humans or selected  
 351 domestic species. Coloured bars represent posterior modes, grey bars show 95 % credible  
 352 intervals based on the statistical sampling approach. The trait variables habitat, range area,  
 353 diet breadth and litters per years were excluded from the plot because of their negligible  
 354 effects.

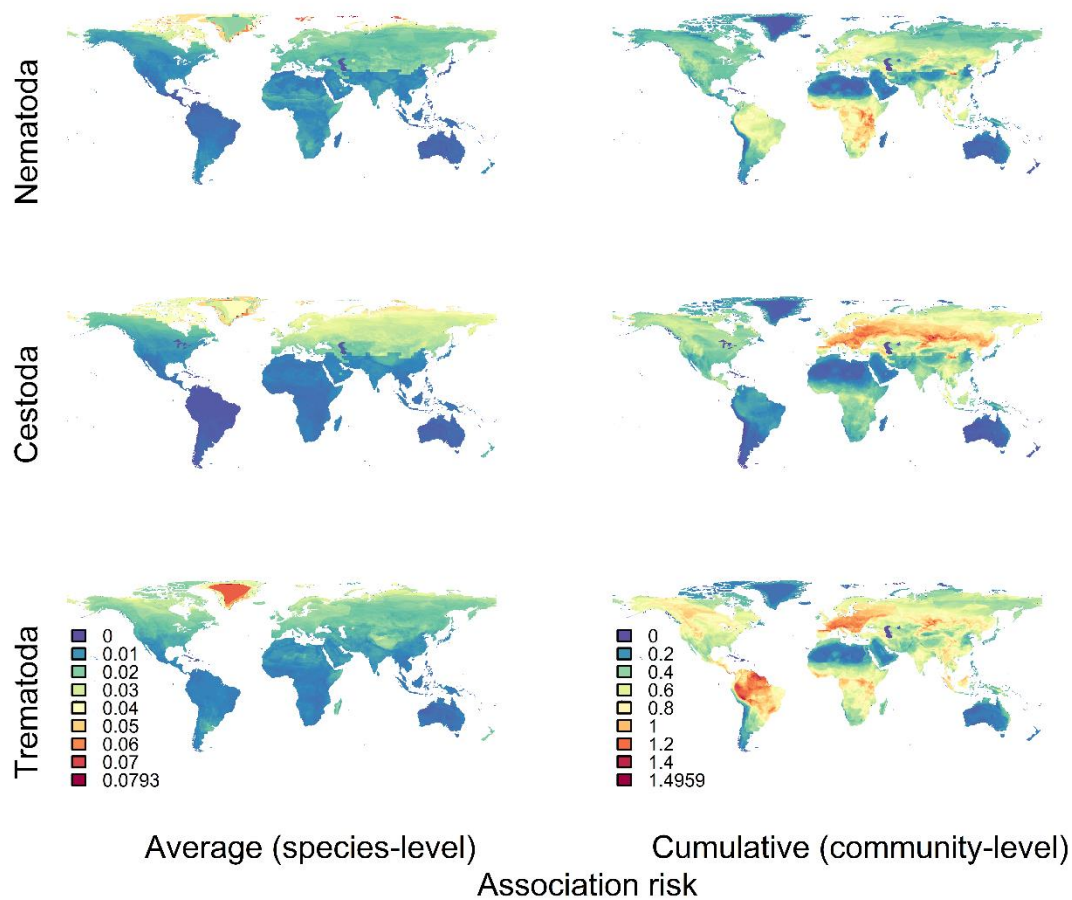
355

356 Model-based predictions of association risk revealed two prominent patterns: first, bat  
 357 species (Chiroptera) are predicted to show a low risk of sharing parasites with focal hosts  
 358 (**Fig. S3**). Second, wildlife association risks were often strongly correlated across different  
 359 focal hosts. The strongest of these correlations were between the risk of wildlife species  
 360 sharing human cestodes and dog cestodes, human trematodes and dog trematodes, and human  
 361 cestodes and dog trematodes (Spearman's  $r = 0.97, 0.98,$  and  $0.96,$  respectively) (**Fig. S4**).



362 Wild mammals occurring in the Nearctic and Palaearctic regions were predicted to  
363 show high association risk for sharing parasites with humans, a pattern that held across all  
364 three parasite groups (**Fig. 2**). In contrast, this predicted risk was remarkably low when  
365 considering human-associated cestodes in the Neotropical region and human-associated  
366 parasites from all three parasite groups in the Australian region (**Fig. 2**). Cumulative  
367 community-level association risks (summed over all wildlife species in local species pools)  
368 resulted in some different patterns. The risk of sharing human parasites was high for wildlife  
369 communities occurring in the Nearctic region (particularly for cestodes and trematodes) and  
370 in mammalian diversity hotspots such as the Panamanian and Neotropical (especially for  
371 trematodes) and Afrotropical (nematodes and trematodes) regions (**Fig. 2**). Note that  
372 relationships between observed proportions of shared parasites and the trait-based prediction  
373 of association risks exhibited some uncertainty (**Fig. S5**). Nevertheless, correlations in  
374 community-level association risks were even stronger than were species-level correlations,  
375 suggesting broad-scale patterns in parasite sharing are predictable (**Fig. S6**). We did not  
376 identify any major global patterns in parasite assemblage distinctiveness (mean turnover in  
377 shared parasite species across zoogeographical regions), though this metric appeared to be  
378 relatively higher in trematodes than in cestodes, and relatively moderate in nematodes (**Fig.**  
379 **S7**).

380



381

382 **Figure 2.** Predicted average (species-level) and cumulative (community-level) geographical  
 383 association risks for local wildlife assemblages sharing helminth parasites (Nematoda,  
 384 Cestoda, Trematoda) with humans. The risk of wildlife species sharing parasites with humans  
 385 were computed using data on host-parasite associations and ecological profiles for 1,345  
 386 wildlife species. Projections of model-based predictions on a global map are based on  
 387 computed wildlife species-level association risks for all extant mammals, rasterised at 10 km<sup>2</sup>  
 388 resolution and respective IUCN range maps.

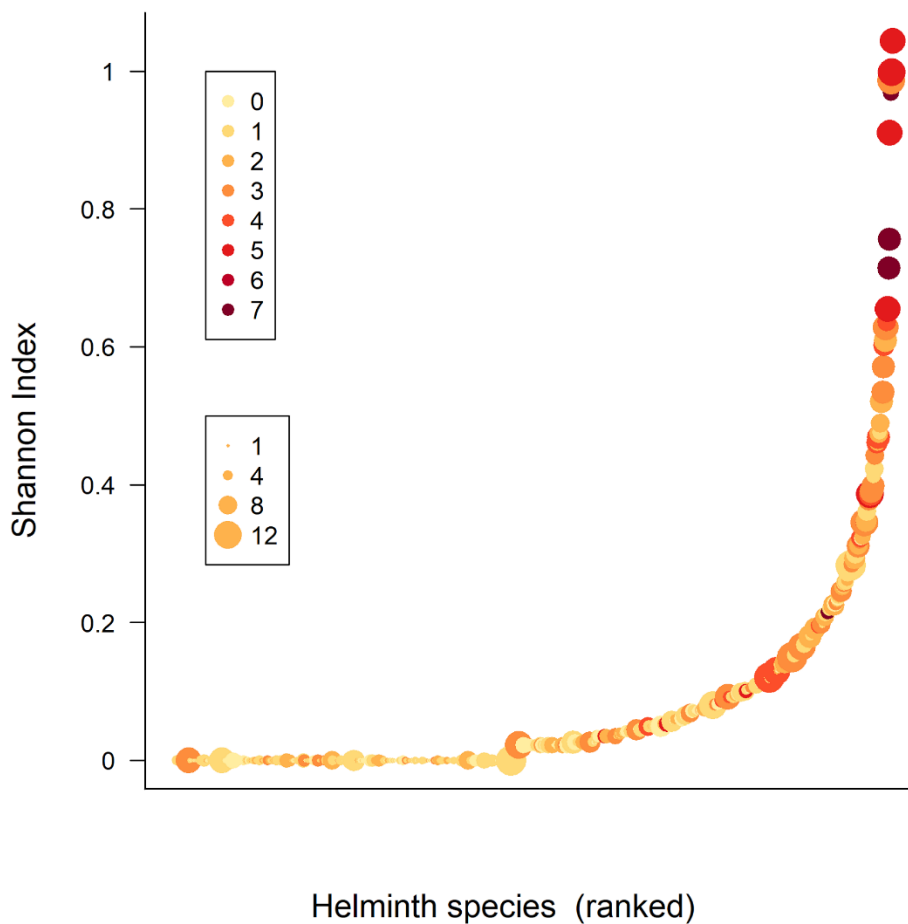
389

390 At the parasite species level, Shannon indices describing the biogeographical spread  
 391 of the 1,103 recorded helminth species followed an exponential distribution (**Fig. 3**). The  
 392 most globally widespread parasite species were *Calodium hepaticum* (Nematoda),

393 *Echinococcus granulosus* (Cestoda), *E. multilocularis* (Cestoda), *Hydatigera taeniaeformis*  
 394 (Cestoda) and *Hymenolepis diminuta* (Cestoda), all of which infected 50 – 73 wildlife species  
 395 and were recorded in at least three of the focal host species (**Fig. 4**). The correlation between  
 396 the index of parasite biogeographical spread and the total number of associated focal host  
 397 species, however, was only moderate (Spearman's  $r = 0.5$ ,  $p < 0.01$ ).

398

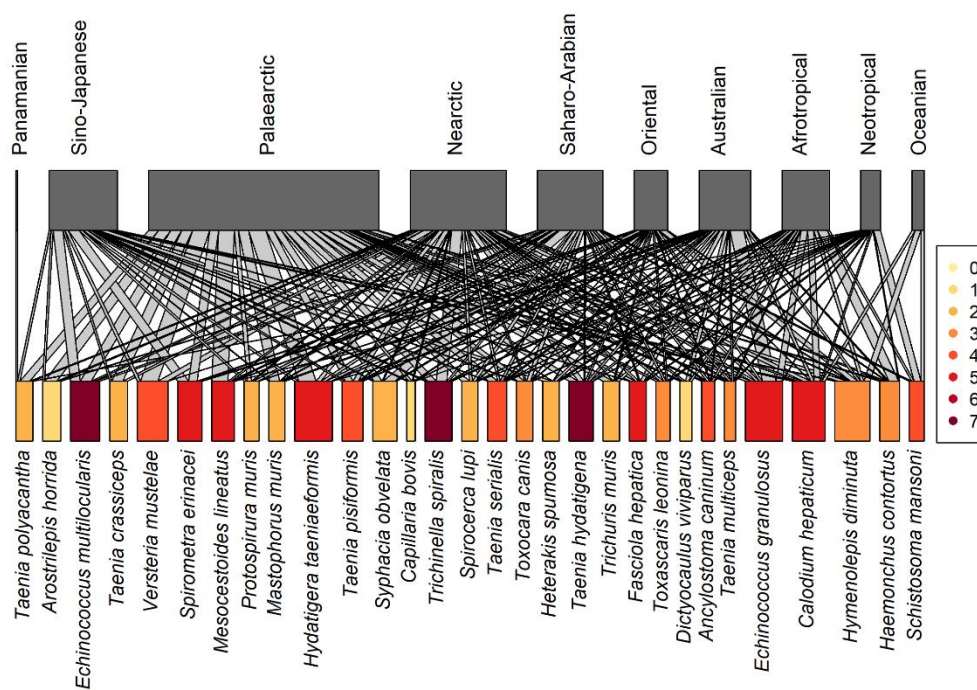
399



401 **Figure 3.** Rank distribution plot of Shannon indices for 1,103 helminth parasite species  
 402 (Nematoda, Cestoda and Trematoda) recorded in domestic host species, indicating the  
 403 relative global spread and linkage to wildlife for each parasite species (Shannon indices are  
 404 based on the proportion of wildlife species associated with the parasites in different

405 zoogeographical regions). Colours represent the number of focal domestic host species  
 406 (human, dog, cat, cow, pig, black rat, brown rat) also associated with that parasite species.  
 407 The size of points reflects the number of zoogeographical realms in which the respective  
 408 helminth species have been recorded (1 – 12, including one class of unspecified records from  
 409 China).

410



411

412 **Figure 4.** Bipartite network plot of the most globally widespread helminth parasite species  
 413 associations with wildlife species pools in different zoogeographical regions. Upper nodes  
 414 represent zoogeographical regions and lower nodes parasite species. The widths of links  
 415 represent the relative proportion of wildlife species from the regional species pool associated  
 416 with the respective parasites. Colours of lower nodes (parasites) represent the number of focal  
 417 domestic host species (humans, dog, cat, cow, pig, black rat, brown rat) also associated with  
 418 that parasite species, illustrating that the majority of globally spread parasites are linked to

419 multiple domestic host species. Widespread parasites species ( $n = 31$ ) were identified as  
420 those with the highest Shannon index scores, accounting for the associated proportions of  
421 sampled wildlife species in different zoogeographical realms. Note the Madagascan region is  
422 not shown, as no wildlife species were associated with the displayed parasites.

423

## 424 **Discussion**

425 Global biodiversity change will affect human and animal health in many ways, but potential  
426 shifts in disease burden at the human–animal interface are largely unexplored (Myers *et al.*,  
427 2013), particularly at the macro-ecological scale (Stephens *et al.*, 2016). We show that diet is  
428 a key driver of the risk that wild mammal species share helminth parasites with humans.  
429 Carnivores and herbivores, in particular, are at high risk of sharing parasites with humans,  
430 while insectivores are generally at low risk. Relatively weaker effects of a wildlife species’  
431 climatic and habitat niches indicate that zoonotic parasite spread will not be contained if  
432 contacts between wildlife and humans continue to increase. Crucially, these same patterns  
433 hold when assessing the risk of wildlife sharing helminths with important domestic animals.  
434 While parasite sharing is a multifaceted one-health issue, we show that decomposing risk of  
435 parasite sharing based on species’ ecological and climatic niches is an important first step  
436 towards predicting future parasite emergence.

437

### 438 **Diet as a key driver of helminth parasite sharing**

439 Our study focuses on terrestrial mammalian species, of which many interact in predator–prey  
440 relationships. The completion of life cycles for some of the most globally widespread  
441 helminths, such as *Echinococcus* spp. and *Fasciola* spp., which are also of significant health  
442 concern (Garcia *et al.*, 2007), depend on such trophic interactions among mammalian hosts.  
443 Unlike microparasites (viruses, bacteria, protozoa, fungi), the majority of parasitic helminth

444 species do not replicate in the definitive vertebrate host, with many species requiring  
445 transmission through a diversity of invertebrates to complete their life cycles. For wildlife  
446 insectivores, the low risks of carrying domestic animal helminths found by our study suggest  
447 there is a transmission disruption that prevents host shifting (e.g. if humans consume  
448 insectivorous species such as bats or shrews, but these species do not in turn ingest  
449 contaminated material from humans or other infected species). Alternatively, domestic  
450 animals and insectivorous wildlife species may not adequately share resources, such as  
451 invertebrate food items or particular habitats, which would enable parasite transmission.

452 The majority of parasitic nematode species undergo free-living life-history stages in the  
453 environment; some are transmitted by direct skin penetration into the definite host, whereas  
454 others are transmitted through trophic interactions that may involve the ingestion of  
455 intermediate invertebrate hosts (Anderson, 2000). This environmental transmission may play  
456 an important role in governing nematode host sharing. Wild and domestic ungulate species,  
457 for example, may share considerable proportions of their nematode fauna through grazing on  
458 common grounds (Walker & Morgan, 2014). Importantly, although one might expect host-  
459 shifting of parasites with free-living stages to be susceptible to environmental conditions, our  
460 results suggest host sharing is more strongly linked to the diet strategy of the host species.

461 Focusing just on helminth parasites, we found notable differences compared to previous  
462 studies examining zoonotic disease risk and reservoir potential for wildlife species. A recent  
463 study on the zoonotic reservoir potential of rodents for both helminths and microparasites  
464 (viruses, bacteria, protozoa, fungi), for example, predicted that the rather fast-paced life  
465 history strategies of rodents should be linked to a higher reservoir potential for zoonotic  
466 diseases (Han *et al.*, 2015). Furthermore, Luis *et al.* (2013) reported both bats and rodents to  
467 be major natural reservoirs for viral zoonoses. In contrast, we predicted the majority of bat  
468 species are less likely to share parasites with humans and domestic species (see **Fig. S4**).

469 Different mechanisms may apply in relation to how helminths and microparasites are spread  
470 through multi-species systems at the human–domestic animal–wildlife interface, warranting  
471 future research.

472

### 473 **Roles of domestic animal hosts at the human – wildlife interface**

474 Consistent with our expectations, we found strong correlations between the risk that wildlife  
475 will share cestodes with human and the risk that wildlife will share cestodes with dogs.

476 Previous work has suggested that dogs and humans share a considerable number of parasites  
477 (Morand *et al.*, 2014). We extend these findings to show that, concomitant with man’s long  
478 association with dogs and the collective exploitation of environments, both humans and dogs  
479 share a considerable number of their helminth parasites with wildlife. However, this pattern is  
480 not restricted to dogs, but can be also seen in patterns of parasite sharing for various domestic  
481 species at a global scale. We found generally strong correlations in the spatially projected  
482 wildlife associations risks – both at the species-level and the community-level – across  
483 domestic host species (**Fig. S6, S7**). This emphasises, that for helminth parasites, the human–  
484 wildlife interface is not independent of domestic species.

485 Our findings support previous calls for multi-species and community-level  
486 approaches to understand parasite and disease spread (Fenton *et al.*, 2015; Johnson *et al.*,  
487 2015; Viana *et al.*, 2014). Notably, we provide a starting point for explaining how  
488 overlapping distributions and contact patterns between humans, domestic animals and  
489 wildlife may impact zoonotic helminth spread at a global scale. Based on our results, future  
490 geographical spread of helminth parasites will likely be facilitated through infection of  
491 multiple domestic hosts (and possibly also invasive mammal species) that show similar  
492 trophic relationships.

493 We demonstrate clear zoogeographical structure in predicted risks that wildlife will

494 share parasites with humans and domestic host species. The highest risk is consistently found  
495 in the Palaearctic and Nearctic regions. Similar global patterns have been reported for rodent-  
496 borne zoonotic diseases, for example by Han *et al.* (2015). In previous work, we found the  
497 two commensal rat species included in our study generally share helminth parasites with  
498 wildlife species of least conservation concern (Wells *et al.*, 2015), which are likely those  
499 species well adapted to anthropogenically modified landscapes. Possibly, strong adaptation to  
500 anthropogenically modified landscapes by many wildlife species in the Palaearctic and  
501 Nearctic regions, in combination with relevant ecological profiles, could contribute to the  
502 strong geographical gradients in risks of parasite sharing.

503         Unfortunately, it is very difficult to discern historical host shifts by parasites, and thus  
504 any possible spill-over and spill-back events, unless adequate molecular data for ancestral  
505 state reconstruction are available (Hoberg *et al.*, 2001; Terefe *et al.*, 2014). Our analysis does  
506 not determine whether wildlife hosts have acquired parasites from humans and domestic  
507 animals, or vice versa. This is especially challenging for humans and domestic species, which  
508 hardly exist in isolation from each other.

509

### 510 **Future parasite spread through mechanisms of parasite sharing**

511 Our finding that trophic interactions are important for interspecific helminth sharing  
512 indicates the need for quantitative approaches that predict whether potential host species may  
513 interact locally in predator-prey relationships. Our predictions can foster a better  
514 understanding of how future domestic animal and wildlife assemblages might impact  
515 potential parasite host shifting through ecological fitting and changed biotic interactions (e.g.  
516 predator-prey relationships). Zoonotic disease risk caused by helminths, for example, could  
517 then be refined to sophisticated measures that take multi-species networks of trophic  
518 interactions into account, rather than only considering the number of wildlife species in local



519 assemblages (Karesh *et al.*, 2005). Given the variable sensitivities among wildlife species to  
520 climate change, such work could also account for shifting trophic interactions among  
521 potential parasite hosts through regionally altered community assemblages (Lurgi *et al.*,  
522 2012).

523         The wildlife and domestic animal trade, together with species invasions and shifting  
524 species ranges, will continue to mix formerly disjunctive host species assemblages and cause  
525 biotic homogenisation (Hobbs *et al.*, 2009). However, future climate-induced range shifts,  
526 decreasing population sizes or newly arising barriers that prevent wildlife movement can also  
527 decrease contact intensity between humans and some wildlife species. This may serve to  
528 inhibit the sharing of parasites. We nevertheless believe that very few wildlife species will be  
529 sufficiently 'left alone' by humans to prevent parasite exchange unless such wildlife species  
530 are extremely rare.

531

### 532 **Host-parasite interactions and sampling bias**

533 Based on records of presence-only host-parasite associations, we consider the results of our  
534 study to be indicative for unravelling general patterns, rather than for providing precise  
535 predictions. Several challenges are associated with studying species interactions and macro-  
536 ecological patterns from presence-only data. First, it is well known from sampling and  
537 probability theory that parasites are likely overlooked in host species sampled with relatively  
538 low intensity (Little, 2004). This will be especially true when low parasite prevalence  
539 prevents detection in a limited number of examined host individuals. Helminth species  
540 richness in freshwater fish, for example, was found to be highly correlated with the number  
541 of individuals examined per host species (Walther *et al.*, 1995). An obstacle to accounting for  
542 this sampling bias is that the true sampling effort, that is the number individuals per host  
543 species examined, was not available for our study; this would have enabled us to better

544 correct for sampling bias when making inferences about host-parasite associations (Wells *et*  
545 *al.*, 2013). Moreover, spatial bias, both in the species sampled and in species–species  
546 interactions, is generally known to strongly bias inference of macro-ecological patterns  
547 (Boakes *et al.*, 2010; Meyer *et al.*, 2015). Bias may be also linked to parasite size, if large  
548 species are more likely to be detected. Our trait-based approach may leverage (to some  
549 extent) poorly sampled species and we used the number of publications for each host species  
550 as a simplified proxy of sampling bias. Limitations in the currently available data on host-  
551 parasite associations and infectious disease prevent concise mapping of the majority of  
552 parasites and diseases (Hay *et al.*, 2013). Considering further sampling bias – as far as  
553 relevant data are available – could be of especial interest for inferring large-scale global  
554 patterns. The proportion of wildlife species examined for parasites, for example, exhibits  
555 considerable gradients across zoogeographical regions (**Fig. S8**). This warrants future  
556 research and a critical revision of whether the particularly strong linkage of human parasites  
557 to wildlife in temperate Europe and North America, as found in this study and others (Han *et*  
558 *al.*, 2015; Murray *et al.*, 2015), is a true biological phenomenon or a consequence of uneven  
559 survey efforts. Moreover, improving the spatial resolution to understand whether host-  
560 parasite interactions and disease emergence are constrained to only those parts of a species  
561 range where enabling conditions are met would improve predictions and our understanding of  
562 how natural barriers may prevent disease emergence. This is of particular importance as,  
563 ultimately, ecological and epidemiological dynamics are driving the interaction between  
564 hosts and parasites and possible parasite spill-over among hosts (Plowright *et al.*, 2017).

565

566         Anticipating and mitigating future changes in parasite host shifting at the human–  
567 wildlife interface may require quantitative approaches that consider novel transmission  
568 pathways. These shifting pathways could be caused by the ongoing decline and/or extinction

569 of native species (Schipper *et al.*, 2008), the introduction of invasive species (Clavero &  
570 García-Berthou, 2005) and/or the increasing density of domestic livestock species (Jones *et*  
571 *al.*, 2013). Novel trophic interactions at the human–wildlife interface may also be largely  
572 driven by human behaviour, such as expanding the menu of consumed animal species, or the  
573 exposure of domestic species to potentially contaminated food waste (Macpherson, 2005).  
574 Disentangling the roles of trophic and other biotic interactions versus environmental  
575 conditions in driving parasite host sharing will improve public and wildlife health measures.

576

## 577 **Acknowledgements**

578 We are grateful to the Natural History Museum, London, for access to records of host-  
579 parasite associations and for making these data publicly available. We thank three  
580 anonymous reviewers and the editor for comments that considerably improved the paper.

581

## 582 **REFERENCES**

- 583 Agosta SJ, Klemens JA (2008) Ecological fitting by phenotypically flexible genotypes:  
584 implications for species associations, community assembly and evolution. *Ecology*  
585 *Letters*, **11**, 1123-1134.
- 586 Anderson RC (2000) *Nematode parasites of vertebrates: their development and transmission*,  
587 Wallingford, Oxon, U.K., CABI Publishing.
- 588 Bininda-Emonds ORP, Cardillo M, Jones KE *et al.* (2007) The delayed rise of present-day  
589 mammals. *Nature*, **446**, 507-512.
- 590 Boakes EH, McGowan PJK, Fuller RA, Chang-Qing D, Clark NE, O'Connor K, Mace GM  
591 (2010) Distorted views of biodiversity: spatial and temporal bias in species  
592 occurrence data. *Plos Biology*, **8**, e1000385.
- 593 Clark NJ, Clegg SM, Sam K, Goulding W, Koane B, Wells K (2017) Climate, host

- 594 phylogeny and the connectivity of host communities govern regional parasite  
595 assembly. *Diversity and Distributions*, doi: 10.1111/ddi.12661.
- 596 Clavero M, García-Berthou E (2005) Invasive species are a leading cause of animal  
597 extinctions. *Trends in Ecology and Evolution*, **20**, 110.
- 598 Crompton DWT (1999) How much human helminthiasis is there in the world? *Journal of*  
599 *Parasitology*, **85**, 397-403.
- 600 Daszak P, Cunningham AA, Hyatt AD (2000) Emerging infectious diseases of wildlife -  
601 threats to biodiversity and human health. *Science*, **287**, 443-449.
- 602 Elith J, Leathwick JR, Hastie T (2008) A working guide to boosted regression trees. *Journal*  
603 *of Animal Ecology*, **77**, 802-813.
- 604 Fenton A, Streicker DG, Petchey OL, Pedersen AB (2015) Are all hosts created equal?  
605 Partitioning host species contributions to parasite persistence in multihost  
606 communities. *The American Naturalist*, **186**, 610-622.
- 607 Garcia HH, Moro PL, Schantz PM (2007) Zoonotic helminth infections of humans:  
608 echinococcosis, cysticercosis and fascioliasis. *Current Opinion in Infectious Diseases*,  
609 **20**, 489-494.
- 610 Gelman A, Meng XL, Stern H (1996) Posterior predictive assessment of model fitness via  
611 realized discrepancies. *Statistica Sinica*, **6**, 733-760.
- 612 Gibson DI, Bray RA (1994) The evolutionary expansion and host-parasite relationships of the  
613 Digenea. *International Journal for Parasitology*, **24**, 1213-1226.
- 614 Gibson DI, Bray RA, Harris EA (2005) Host-parasite database of the Natural History  
615 Museum, London. pp Page, Natural History Museum.
- 616 Gibson DI, Bray RA, Hunt D *et al.* (2014) Fauna Europaea: helminths (animal parasitic).  
617 *Biodiversity Data Journal*, **2**, e1060.
- 618 Han BA, Schmidt JP, Bowden SE, Drake JM (2015) Rodent reservoirs of future zoonotic

- 619 diseases. *Proceedings of the National Academy of Sciences*, **112**, 7039-7044.
- 620 Hay SI, Battle KE, Pigott DM *et al.* (2013) Global mapping of infectious disease.
- 621 *Philosophical Transactions of the Royal Society B-Biological Sciences*, **368**, 0120250.
- 622 Hobbs RJ, Higgs E, Harris JA (2009) Novel ecosystems: implications for conservation and
- 623 restoration. *Trends in Ecology and Evolution*, **24**, 599-605.
- 624 Hoberg EP, Alkire NL, Queiroz AD, Jones A (2001) Out of Africa: origins of the *Taenia*
- 625 tapeworms in humans. *Proceedings of the Royal Society of London B: Biological*
- 626 *Sciences*, **268**, 781-787.
- 627 Hoberg EP, Brooks DR (2008) A macroevolutionary mosaic: episodic host-switching,
- 628 geographical colonization and diversification in complex host–parasite systems.
- 629 *Journal of Biogeography*, **35**, 1533-1550.
- 630 Holt BG, Lessard J-P, Borregaard MK *et al.* (2013) An update of Wallace’s zoogeographic
- 631 regions of the world. *Science*, **339**, 74-78.
- 632 Johnson PTJ, Ostfeld RS, Keesing F (2015) Frontiers in research on biodiversity and disease.
- 633 *Ecology Letters*, **18**, 1119-1133.
- 634 Jones BA, Grace D, Kock R *et al.* (2013) Zoonosis emergence linked to agricultural
- 635 intensification and environmental change. *Proceedings of the National Academy of*
- 636 *Sciences*, **110**, 8399-8404.
- 637 Jones KE, Bielby J, Cardillo M *et al.* (2009) PanTHERIA: a species-level database of life
- 638 history, ecology, and geography of extant and recently extinct mammals. *Ecology*, **90**,
- 639 2648-2648.
- 640 Just MG, Norton JF, Traud AL *et al.* (2014) Global biogeographic regions in a human-
- 641 dominated world: the case of human diseases. *Ecosphere*, **5**, 1-21.
- 642 Karesh WB, Cook RA, Bennett EL, Newcomb J (2005) Wildlife trade and global disease
- 643 emergence. *Emerging Infectious Diseases*, **11**, 1000-1002.

- 644 Kraft Nathan jB, Cornwell William k, Webb Campbell o, Ackerly David d (2007) Trait  
645 evolution, community assembly, and the phylogenetic structure of ecological  
646 communities. *The American Naturalist*, **170**, 271-283.
- 647 Lennon JJ, Koleff P, Greenwood JJD, Gaston KJ (2001) The geographical structure of British  
648 bird distributions: diversity, spatial turnover and scale. *Journal of Animal Ecology*,  
649 **70**, 966-979.
- 650 Little RJ (2004) To model or not to model? Competing modes of inference for finite  
651 population sampling. *Journal of the American Statistical Association*, **99**, 546-556.
- 652 Lloyd-Smith JO, George D, Pepin KM *et al.* (2009) Epidemic dynamics at the human-animal  
653 interface. *Science*, **326**, 1362-1367.
- 654 Lunn D, Jackson C, Best N, Thomas A, Spiegelhalter D (2012) *The BUGS Book - A practical*  
655 *introduction to Bayesian analysis*, London, CRC Press / Chapman and Hall.
- 656 Lurgi M, López BC, Montoya JM (2012) Novel communities from climate change.  
657 *Philosophical Transactions of the Royal Society B-Biological Sciences*, **367**, 2913-  
658 2922.
- 659 Macpherson CNL (2005) Human behaviour and the epidemiology of parasitic zoonoses.  
660 *International Journal for Parasitology*, **35**, 1319-1331.
- 661 Magurran AE (ed) (2004) *Measuring biological diversity*, Oxford, Blackwell.
- 662 Matisoo-Smith, E. , Roberts, R. M., Irwin, G. J. , Allen, J. S., Penny, D. , Lambert, D. M.  
663 (1998) Patterns of prehistoric human mobility in Polynesia indicated by mtDNA from  
664 the Pacific rat. *Proceedings of the National Academy of Sciences*, **95**, 15145-15150.
- 665 Meyer C, Kreft H, Guralnick R, Jetz W (2015) Global priorities for an effective information  
666 basis of biodiversity distributions. *Nature Communications*, **6**, 8221.
- 667 Morand S, McIntyre KM, Baylis M (2014) Domesticated animals and human infectious  
668 diseases of zoonotic origins: domestication time matters. *Infection, Genetics and*

- 669           *Evolution*, **24**, 76-81.
- 670 Murray KA, Preston N, Allen T, Zambrana-Torrel C, Hosseini PR, Daszak P (2015) Global  
671           biogeography of human infectious diseases. *Proceedings of the National Academy of*  
672           *Sciences*, **112**, 12746-12751.
- 673 Myers SS, Gaffikin L, Golden CD *et al.* (2013) Human health impacts of ecosystem  
674           alteration. *Proceedings of the National Academy of Sciences*, **110**, 18753-18760.
- 675 Paradis E, Claude J, Strimmer K (2004) APE: Analyses of phylogenetics and evolution in R  
676           language. *Bioinformatics*, **20**, 289-290.
- 677 Patz JA, Olson SH, Uejio CK, Gibbs HK (2008) Disease emergence from global climate and  
678           land use change. *Medical Clinics of North America*, **92**, 1473–1491.
- 679 Pedersen AB, Davies TJ (2009) Cross-species pathogen transmission and disease emergence  
680           in primates. *Ecohealth*, **6**, 496-508.
- 681 Plowright RK, Parrish CR, McCallum H, Hudson PJ, Ko AI, Graham AL, Lloyd-Smith JO  
682           (2017) Pathways to zoonotic spillover. *Nature Review Microbiology*, doi:  
683           10.1038/nrmicro.2017.45.
- 684 Pulliam JRC (2008) Viral host jumps: moving toward a predictive framework. *Ecohealth*, **5**,  
685           80-91.
- 686 R Development Core Team (2017) R: A language and environment for statistical computing,  
687           Vienna, Austria, R Foundation for Statistical Computing.
- 688 Reinhard KJ, Ferreira LF, Bouchet F *et al.* (2013) Food, parasites, and epidemiological  
689           transitions: a broad perspective. *International Journal of Paleopathology*, **3**, 150-157.
- 690 Schipper J, Chanson JS, Chiozza F *et al.* (2008) The status of the world's land and marine  
691           mammals: diversity, threat, and knowledge. *Science*, **322**, 225-230.
- 692 Stephens PR, Altizer S, Smith KF *et al.* (2016) The macroecology of infectious diseases: a  
693           new perspective on global-scale drivers of pathogen distributions and impacts.

- 694 *Ecology Letters*, **19**, 1159–1171.
- 695 Streicker DG, Turmelle AS, Vonhof MJ, Kuzmin IV, Mccracken GF, Rupprecht CE (2010)
- 696 Host phylogeny constrains cross-species emergence and establishment of rabies virus
- 697 in bats. *Science*, **329**, 676-679.
- 698 Taylor LH, Latham SM, Woolhouse MEJ (2001) Risk factors for human disease emergence.
- 699 *Philosophical Transactions of the Royal Society of London B Biological Sciences*,
- 700 **356**, 983-989.
- 701 Terefe Y, Hailemariam Z, Menkir S *et al.* (2014) Phylogenetic characterisation of *Taenia*
- 702 tapeworms in spotted hyenas and reconsideration of the “Out of Africa” hypothesis of
- 703 *Taenia* in humans. *International Journal for Parasitology*, **44**, 533-541.
- 704 Viana M, Mancy R, Biek R, Cleaveland S, Cross PC, Lloyd-Smith JO, Haydon DT (2014)
- 705 Assembling evidence for identifying reservoirs of infection. *Trends in Ecology &*
- 706 *Evolution*, **29**, 270-279.
- 707 Walker JG, Morgan ER (2014) Generalists at the interface: nematode transmission between
- 708 wild and domestic ungulates. *International Journal for Parasitology: Parasites and*
- 709 *Wildlife*, **3**, 242-250.
- 710 Wallace AR (1876) *The geographical distributions of animals, with a study of the relations of*
- 711 *living and extinct faunas as elucidating the past changes of the Earth’s surface*,
- 712 London, Macmillan.
- 713 Walther BA, Cotgreave P, Price RD, Gregory RD, Clayton DH (1995) Sampling effort and
- 714 parasite species richness. *Parasitology Today*, **11**, 306-310.
- 715 Wells K, O’Hara RB, Morand S, Lessard J-P, Ribas A (2015) The importance of parasite
- 716 geography and spillover effects for global patterns of host–parasite associations in
- 717 two invasive species. *Diversity and Distributions*, **21**, 477-486.
- 718 Wells K, O’Hara RB, Pfeiffer M, Lakim MB, Petney TN, Durden LA (2013) Inferring host



- 719 specificity and network formation through agent-based models: tick–mammal  
720 interactions in Borneo. *Oecologia*, **172**, 307-316.
- 721 Wilman H, Belmaker J, Simpson J, De La Rosa C, Rivadeneira MM, Jetz W (2014)  
722 EltonTraits 1.0: species-level foraging attributes of the world's birds and mammals.  
723 *Ecology*, **95**, 2027-2027.
- 724 Wolfe ND, Dunavan CP, Diamond J (2007) Origins of major human infectious diseases.  
725 *Nature*, **447**, 279-283.
- 726 Woolhouse MEJ, Gowtage-Sequeria S (2005) Host range and emerging and reemerging  
727 pathogens. *Emerging Infectious Diseases*, **11**, 1842-1847.