Paper:
Global spread of helminth parasites at the human – domestic animal – wildlife interface

Running head: Parasites at the human–animal interface

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Abstract

Changes in species distributions open novel parasite transmission routes at the human–wildlife interface, yet the strength of biotic and biogeographical factors that prevent or facilitate parasite host shifting are not well understood. We investigated global patterns of helminth parasite (Nematoda, Cestoda, Trematoda) sharing between mammalian wildlife species and domestic mammal hosts (including humans) using > 24,000 unique country-level records of host-parasite associations. We used hierarchical modelling and species trait data to determine possible drivers of the level of parasite sharing between wildlife species and either humans or domestic animal hosts. We found the diet of wildlife species to be a strong predictor of levels of helminth parasite sharing with humans and domestic animals, followed by a moderate effect of zoogeographical region and minor effects of species’ habitat and climatic niches. Combining model predictions with the distribution and ecological profile data of wildlife species, we projected global risk maps that uncovered strikingly similar patterns of wildlife parasite sharing across geographical areas for the different domestic host species (including humans). These similarities are largely explained by the fact that widespread parasites are commonly recorded infecting several domestic species. If the dietary profile and position in the trophic chain of a wildlife species largely drives its level of helminth parasite sharing with humans/domestic animals, future range shifts of host species that result in novel trophic interactions may likely increase parasite host shifting and have important ramifications for human and animal health.
Introduction

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

While direct human-wildlife parasite sharing is a topic of major importance, domestic animals that occur in close proximity to humans may also act as key hosts for wildlife parasites. Domestic animals (hereafter including domesticated animals, such as dogs, but also animals that live in close proximity to humans, such as commensal rats) have colonised almost all terrestrial environments (Hoberg & Brooks, 2008; Matisoo-Smith et al., 1998). In turn, domestic animals commonly share subsets of their parasite fauna with humans. This
subset increases the longer a species has been domesticated (Morand et al., 2014; Wolfe et al., 2007). Parasite host shifting at the interface between humans, domestic animals and wildlife is a multifaceted and multidirectional problem, with potential effects for human and wildlife health (Daszak et al., 2000). Yet, while previous studies found 60% of human diseases to be of zoonotic origin (Taylor et al., 2001; Woolhouse & Gowtage-Sequeria, 2005), global patterns in parasite sharing at the human–domestic animal–wildlife interface are poorly resolved.

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

A key gap in our understanding of zoonotic disease emergence is information on how patterns of wildlife parasite sharing differ among domestic host species or across biogeographical regions. Despite persisting in close spatial proximity, humans and domestic animals differ in habitat use, diet and other ecological traits. This may have consequences for determining subsets of parasites that humans and domestic animals share with wildlife. Humans and dogs, for example, each consume a large range of invertebrate and vertebrate species (many of which may be relevant reservoir hosts) and can access almost any type of terrestrial habitat. Other domestic species, such as cows, are confined to relatively few
habitats and food items (e.g. grassland vegetation). One may expect that different domestic animals will exhibit different patterns of wildlife parasite sharing and, consequently, different potential roles as carriers of zoonotic parasites. Globally, wildlife communities occur in distinct species assemblages according to biogeographical history, speciation events and habitat biomes (Holt et al., 2013; Kraft et al., 2007; Wallace, 1876). Such biogeographical structure may lead to spatial gradients in wildlife parasite sharing for humans and domestic animals.

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

**Materials and Methods**

**Host-parasite database**

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

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

**Host ecological traits**

We selected nine ecological traits of terrestrial mammals, based on the PanTHERIA (*Jones et al.*, 2009) and EltonTraits 1.0 (*Wilman et al.*, 2014) databases, to include a broad range of attributes likely to distinguish hosts in terms of their suitability for a parasite’s life and
transmission cycles. Selected traits included: body mass, which is a key feature of mammals

in terms of their metabolism and adaptation to environments; average longevity, litter size

and the average number of litters per year as demographic parameters that could be relevant

for allowing parasites to complete parts of their life cycles in a host; diet breadth (calculated

as a Shannon diversity index based on the proportional use of 10 diet categories as presented

in EltonTraits) and diet class (‘invertebrate predator’, ‘herbivore’, ‘omnivore’ or ‘carnivore’)

as trophic interactions traits; range area, which we expect to affect the exposure to other

mammalian host species; average temperature within range as an indicator of climate niche;

and habitat as multiple binary indicators of whether a species uses 1) forest, 2) open

vegetation, and/or 3) artificial/anthropogenic habitats. Information about specific habitat

utilisation was compiled from the International Union for the Conservation of Nature (IUCN)
database (IUCN, 2014). We did not include a larger set of ecological traits in our analysis to

avoid trait autocorrelation and colinearity issues in the modelling.

We accounted for phylogenetic distances between wildlife species and focal domestic species

based on a correlation matrix (Paradis et al., 2004) of phylogenetic branch lengths, which

was built using a recent mammalian phylogenetic supertree (Bininda-Emonds et al., 2007).

We further considered the orders Carnivora, Rodentia and Primates as binary (categorical)
indicator variables for the major taxonomic groups that are suspected to share parasites with
the focal species; we used these as indicators to account for possible group-level taxonomic
effects additional to the phylogenetic branching. To account for sampling bias among wildlife
species, we queried the number of published references for each binomial wildlife species
name from the Scopus literature database (accessed 25/02/2017); we used this measure as
more refined searches, such as the number of references linked only to parasites, included
large proportions of zeros and information on the true number of sampled individuals (which
should determine the chance parasites are detected if prevalence is low) was not available.
Statistical analysis

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

We assumed the resulting data vectors $Y_d(w_r, p_d)$ are random draws from the underlying association probability $\Psi_d(w_r, p_d)$ of a wildlife species sharing a parasite with a focal species according to a Bernoulli distribution, as commonly used in logistic regression. We modelled the probability $\Psi_d(w_r, p_d)$ further using a logit-link function such that

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

where $\mu_{Parasite}(p_d)$ is the parasite-specific intercept, $\mu_{Region}$ are coefficient estimates that account for variation across zoogeographical regions $r$, and $B$ is a vector of coefficient
estimates that account for variations in the association risk linked to the matrix of covariates

$ET_{wr}$ of the nine host ecological traits, the phylogenetic distance of wildlife to the focal host

species and the number of publications, as specified above.

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

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

where $\varepsilon_\mu$ is a random Gaussian variance term that allows species-specific intercepts to vary

from the hyperprior (no group specific hyperprior was specified as we ran models separately

for the three parasite groups). We fitted the model in a Bayesian framework with Markov

Chain Monte Carlo (MCMC) sampling based on the Gibbs sampler in the freeware

OpenBUGS (Lunn et al., 2012). We used a Gibbs variable selection procedure (GVS) to only

include variables in the model if sufficiently supported by the fit to data and joint sampling of

the most likely coefficient values (selection frequencies were typically high for covariates

with significant effects, except for the categorical effects of ‘region’). We used normal priors

with mean = 0 and variance $\sigma \sim Exp(1)$ for intercepts and all regression coefficients if selected

as part of the GVS, and $\sigma \to 0$ otherwise. This prior gives close approximation to a logistic

distribution and is appropriate for logit-scale estimates (Lunn et al., 2012). Convergence of

MCMC chains was assessed visually and with Gelman-Rubin diagnostics (all values $< 1.2$)

after burn-ins $> 50,000$ MCMC samples. Parameter estimates were calculated as posterior

modes and 95% highest posterior density credible intervals (CI) from 5,000 samples.

Posterior predictive checks assessed whether model assumptions were reasonable

approximations of the data generating process, with Bayesian $P$-values around 0.5 indicating

a good fit. This model checking approach essentially compares whether the observed data

resemble data simulated from the posterior distribution (Gelman et al., 1996). All covariates

were scaled (dividing centred values by one SD) and log-transformed if featuring

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

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

The second aim of this study was to examine whether patterns of wildlife parasite sharing among domestic hosts exhibit biogeographical structure, which could be informative for understanding the future spread of zoonotic parasites. We addressed this goal by
exploring global patterns in observed parasite associations for focal host species and
generated forecasts of wildlife infestation with parasites shared with the focal host
species. First, for each focal host species, we used our model outputs to generate a series of
maps (10km² raster cell sizes) to forecast global patterns in both wildlife parasite association
risks and parasite assemblage structure. Using IUCN geographical range maps for wildlife
species, we projected the respective parasite association risks on a global raster and, for each
cell, computed average species-level and cumulative community-level geographical
association risks for local wildlife assemblages. We were not able to account for possible
regional variation in realized host-parasite interactions (which could arise due to variation in
local conditions that enable host-parasite interactions) within the given wildlife range maps
and, for simplicity, assumed homogenous association risks throughout species’ given ranges.

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

Spearman rank correlation tests were used to compare biogeographical patterns. First,
we assessed whether infestation of a greater number of focal host species leads to broader
biogeographical spread by testing the correlation strength between the Shannon index of
biogeographical spread and the total number of associated focal host species. We then explored whether wildlife species show similar biogeographical patterns in the risk for sharing parasites with different focal host species by testing all pairwise correlations between the geographical association risks for parasites from the different focal host species.

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

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

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

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

**Results**

Of the 1,345 mammalian wildlife species in our host-parasite database, 41 % (n = 553 spp.) were infected with helminth parasite species (Nematoda, Cestoda and Trematoda) also found in humans. For humans, in turn, 49 % (195 of 397 spp.) of all helminth parasite species were also found in wildlife and 45 % (182 spp.) in at least one other domestic host species. The wildlife species associated with the highest numbers of zoonotic parasites were *Vulpes vulpes* (red fox, 51 spp.), *Canis lupus* (grey wolf, 33 spp.) and *Nyctereutes procyonoides* (raccoon dog, 29 spp.). For the other focal domestic host species, proportions of examined wildlife
species that shared parasites ranged from 21 – 31 % (Table S1, Supplementary Information).

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

Coefficient estimates for all other covariates are presented in Table S2; notably, various coefficient estimates were significantly different from zero, though they explained much less variance than diet and zoogeographical region. Bayesian $p$ values ranged from 0.43 to 0.79 for the various models.
Figure 1. Relative influence (% variance explained) of wildlife host ecological traits, zoogeographical region and number of publications (a surrogate of sampling effort) on the probability that wildlife species shared helminth parasite species with humans or selected domestic species. Coloured bars represent posterior modes, grey bars show 95% credible intervals based on the statistical sampling approach. The trait variables habitat, range area, diet breadth and litters per years were excluded from the plot because of their negligible effects.

Model-based predictions of association risk revealed two prominent patterns: first, bat species (Chiroptera) are predicted to show a low risk of sharing parasites with focal hosts (Fig. S3). Second, wildlife association risks were often strongly correlated across different focal hosts. The strongest of these correlations were between the risk of wildlife species sharing human cestodes and dog cestodes, human trematodes and dog trematodes, and human cestodes and dog trematodes (Spearman’s r = 0.97, 0.98, and 0.96, respectively) (Fig. S4).
Wild mammals occurring in the Nearctic and Palaearctic regions were predicted to show high association risk for sharing parasites with humans, a pattern that held across all three parasite groups (Fig. 2). In contrast, this predicted risk was remarkably low when considering human-associated cestodes in the Neotropical region and human-associated parasites from all three parasite groups in the Australian region (Fig. 2). Cumulative community-level association risks (summed over all wildlife species in local species pools) resulted in some different patterns. The risk of sharing human parasites was high for wildlife communities occurring in the Nearctic region (particularly for cestodes and trematodes) and in mammalian diversity hotspots such as the Panamanian and Neotropical (especially for trematodes) and Afrotropical (nematodes and trematodes) regions (Fig. 2). Note that relationships between observed proportions of shared parasites and the trait-based prediction of association risks exhibited some uncertainty (Fig. S5). Nevertheless, correlations in community-level association risks were even stronger than were species-level correlations, suggesting broad-scale patterns in parasite sharing are predictable (Fig. S6). We did not identify any major global patterns in parasite assemblage distinctiveness (mean turnover in shared parasite species across zoogeographical regions), though this metric appeared to be relatively higher in trematodes than in cestodes, and relatively moderate in nematodes (Fig. S7).
Figure 2. Predicted average (species-level) and cumulative (community-level) geographical association risks for local wildlife assemblages sharing helminth parasites (Nematoda, Cestoda, Trematoda) with humans. The risk of wildlife species sharing parasites with humans were computed using data on host-parasite associations and ecological profiles for 1,345 wildlife species. Projections of model-based predictions on a global map are based on computed wildlife species-level association risks for all extant mammals, rasterised at 10 km\(^2\) resolution and respective IUCN range maps.

At the parasite species level, Shannon indices describing the biogeographical spread of the 1,103 recorded helminth species followed an exponential distribution (Fig. 3). The most globally widespread parasite species were *Calodium hepaticum* (Nematoda),
Echinococcus granulosus (Cestoda), *E. multilocularis* (Cestoda), *Hydatigera taeniaeformis* (Cestoda) and *Hymenolepis diminuta* (Cestoda), all of which infected 50–73 wildlife species and were recorded in at least three of the focal host species (Fig. 4). The correlation between the index of parasite biogeographical spread and the total number of associated focal host species, however, was only moderate (Spearman’s r = 0.5, p < 0.01).

**Figure 3.** Rank distribution plot of Shannon indices for 1,103 helminth parasite species (Nematoda, Cestoda and Trematoda) recorded in domestic host species, indicating the relative global spread and linkage to wildlife for each parasite species (Shannon indices are based on the proportion of wildlife species associated with the parasites in different...
zoogeographical regions). Colours represent the number of focal domestic host species (human, dog, cat, cow, pig, black rat, brown rat) also associated with that parasite species. The size of points reflects the number of zoogeographical realms in which the respective helminth species have been recorded (1 – 12, including one class of unspecified records from China).

Figure 4. Bipartite network plot of the most globally widespread helminth parasite species associations with wildlife species pools in different zoogeographical regions. Upper nodes represent zoogeographical regions and lower nodes parasite species. The widths of links represent the relative proportion of wildlife species from the regional species pool associated with the respective parasites. Colours of lower nodes (parasites) represent the number of focal domestic host species (humans, dog, cat, cow, pig, black rat, brown rat) also associated with that parasite species, illustrating that the majority of globally spread parasites are linked to...
multiple domestic host species. Widespread parasites species (n = 31) were identified as those with the highest Shannon index scores, accounting for the associated proportions of sampled wildlife species in different zoogeographical realms. Note the Madagascan region is not shown, as no wildlife species were associated with the displayed parasites.

Discussion

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

Diet as a key driver of helminth parasite sharing

Our study focuses on terrestrial mammalian species, of which many interact in predator–prey relationships. The completion of life cycles for some of the most globally widespread helminths, such as *Echinococcus* spp. and *Fasciola* spp., which are also of significant health concern (Garcia et al., 2007), depend on such trophic interactions among mammalian hosts. Unlike microparasites (viruses, bacteria, protozoa, fungi), the majority of parasitic helminth
species do not replicate in the definitive vertebrate host, with many species requiring transmission through a diversity of invertebrates to complete their life cycles. For wildlife insectivores, the low risks of carrying domestic animal helminths found by our study suggest there is a transmission disruption that prevents host shifting (e.g. if humans consume insectivorous species such as bats or shrews, but these species do not in turn ingest contaminated material from humans or other infected species). Alternatively, domestic animals and insectivorous wildlife species may not adequately share resources, such as invertebrate food items or particular habitats, which would enable parasite transmission.

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

Focusing just on helminth parasites, we found notable differences compared to previous studies examining zoonotic disease risk and reservoir potential for wildlife species. A recent study on the zoonotic reservoir potential of rodents for both helminths and microparasites (viruses, bacteria, protozoa, fungi), for example, predicted that the rather fast-paced life history strategies of rodents should be linked to a higher reservoir potential for zoonotic diseases (Han et al., 2015). Furthermore, Luis et al. (2013) reported both bats and rodents to be major natural reservoirs for viral zoonoses. In contrast, we predicted the majority of bat species are less likely to share parasites with humans and domestic species (see Fig. S4).
Different mechanisms may apply in relation to how helminths and microparasites are spread through multi-species systems at the human–domestic animal–wildlife interface, warranting future research.

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

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

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

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

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

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

Our finding that trophic interactions are important for interspecific helminth sharing indicates the need for quantitative approaches that predict whether potential host species may interact locally in predator-prey relationships. Our predictions can foster a better understanding of how future domestic animal and wildlife assemblages might impact potential parasite host shifting through ecological fitting and changed biotic interactions (e.g. predator-prey relationships). Zoonotic disease risk caused by helminths, for example, could then be refined to sophisticated measures that take multi-species networks of trophic interactions into account, rather than only considering the number of wildlife species in local
assemblages (Karesh et al., 2005). Given the variable sensitivities among wildlife species to climate change, such work could also account for shifting trophic interactions among potential parasite hosts through regionally altered community assemblages (Lurgi et al., 2012).

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

Host-parasite interactions and sampling bias

Based on records of presence-only host-parasite associations, we consider the results of our study to be indicative for unravelling general patterns, rather than for providing precise predictions. Several challenges are associated with studying species interactions and macro-ecological patterns from presence-only data. First, it is well known from sampling and probability theory that parasites are likely overlooked in host species sampled with relatively low intensity (Little, 2004). This will be especially true when low parasite prevalence prevents detection in a limited number of examined host individuals. Helminth species richness in freshwater fish, for example, was found to be highly correlated with the number of individuals examined per host species (Walther et al., 1995). An obstacle to accounting for this sampling bias is that the true sampling effort, that is the number individuals per host species examined, was not available for our study; this would have enabled us to better...
correct for sampling bias when making inferences about host-parasite associations (Wells et al., 2013). Moreover, spatial bias, both in the species sampled and in species–species interactions, is generally known to strongly bias inference of macro-ecological patterns (Boakes et al., 2010; Meyer et al., 2015). Bias may also be linked to parasite size, if large species are more likely to be detected. Our trait-based approach may leverage (to some extent) poorly sampled species and we used the number of publications for each host species as a simplified proxy of sampling bias. Limitations in the currently available data on host-parasite associations and infectious disease prevent concise mapping of the majority of parasites and diseases (Hay et al., 2013). Considering further sampling bias – as far as relevant data are available – could be of especial interest for inferring large-scale global patterns. The proportion of wildlife species examined for parasites, for example, exhibits considerable gradients across zoogeographical regions (Fig. S8). This warrants future research and a critical revision of whether the particularly strong linkage of human parasites to wildlife in temperate Europe and North America, as found in this study and others (Han et al., 2015; Murray et al., 2015), is a true biological phenomenon or a consequence of uneven survey efforts. Moreover, improving the spatial resolution to understand whether host-parasite interactions and disease emergence are constrained to only those parts of a species range where enabling conditions are met would improve predictions and our understanding of how natural barriers may prevent disease emergence. This is of particular importance as, ultimately, ecological and epidemiological dynamics are driving the interaction between hosts and parasites and possible parasite spill-over among hosts (Plowright et al., 2017).

Anticipating and mitigating future changes in parasite host shifting at the human–wildlife interface may require quantitative approaches that consider novel transmission pathways. These shifting pathways could be caused by the ongoing decline and/or extinction
of native species (Schipper et al., 2008), the introduction of invasive species (Clavero &
García-Berthou, 2005) and/or the increasing density of domestic livestock species (Jones et
al., 2013). Novel trophic interactions at the human–wildlife interface may also be largely
driven by human behaviour, such as expanding the menu of consumed animal species, or the
exposure of domestic species to potentially contaminated food waste (Macpherson, 2005).
Disentangling the roles of trophic and other biotic interactions versus environmental
conditions in driving parasite host sharing will improve public and wildlife health measures.

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