PROCEEDINGS B

royalsocietypublishing.org/journal/rspb

Research

**Cite this article:** Martins PM, Poulin R. 2024 Universal versus taxon-specific drivers of helminth prevalence and intensity of infection. *Proc. R. Soc. B* **291**: 20241673.
https://doi.org/10.1098/rspb.2024.1673

Received: 15 April 2024
Accepted: 16 September 2024

**Subject Category:**
Ecology

**Subject Areas:**
ecology, evolution

**Keywords:**
amphibians, climate, dilution effect, epidemiology, host–parasite interaction, wildlife diseases

**Author for correspondence:**
Robert Poulin
e-mail: robert.poulin@otago.ac.nz

†All authors contributed equally to the study.

Electronic supplementary material is available online at https://doi.org/10.6084/m9.figshare.c.7492637.

THE ROYAL SOCIETY PUBLISHING



# Universal versus taxon-specific drivers of helminth prevalence and intensity of infection

Paulo Mateus Martins[1,†] and Robert Poulin[2,†]

[1]Department of Biological Sciences, University of Notre Dame, Notre Dame, IN 46556, USA
[2]Department of Zoology, University of Otago, PO Box 56, Dunedin 9054, New Zealand

PMM, 0000-0003-2780-2604; RP, 0000-0003-1390-1206

Two key epidemiological parameters, prevalence and mean intensity of infection, together capture the abundance of macroparasite populations, the strength of density-dependent effects they experience, their potential impact on host population dynamics and the selective pressures they exert on their hosts. Yet, the drivers of the extensive variation observed in prevalence and mean intensity of infection, even among related parasite taxa infecting related hosts, remain mostly unknown. We performed phylogenetically grounded Bayesian modelling across hundreds of amphibian populations to test the effects of various predictors of prevalence and intensity of infection by six families of helminth parasites. We focused on the potential effects of key host traits and environmental factors pertinent to focal host populations, i.e. the local diversity of the amphibian community and local climatic variables. Our analyses revealed several important determinants of prevalence or intensity of infection in various parasite families, but none applying to all families. Our study uncovered no universal driver of parasite infection levels, even among parasite taxa from the same phylum, or with similar life cycles and transmission modes. Although local variables not considered here may have effects extending across taxa, our findings suggest the need for a taxon-specific approach in any attempt to predict disease dynamics and impacts in the face of environmental and climatic changes.

## 1. Introduction

On both ecological and evolutionary time scales, the interaction strength between host and parasite populations depends on the frequency at which their individuals encounter and act on each other [1–3]. However, large-scale drivers of both intraspecific (geographical) and interspecific variation in interaction strength among host–parasite systems remain mostly unknown. The interface and overlap between a macroparasite population and a host population have long been quantified by two simple epidemiological parameters [4]: the proportion of infected host individuals, or *prevalence* of infection, and the average number of parasite individuals per infected host individuals, or *mean intensity*. The product of prevalence and mean intensity yields the average number of parasite individuals per host individuals including uninfected hosts, or *mean abundance*, another useful measure. Although empirical data from natural populations indicate that prevalence and mean intensity of infection are generally positively correlated with each other [5,6], they nevertheless capture slightly different aspects of host–parasite population associations and dynamics.

The strength of trophic interactions between hosts and parasites in real time and the coevolutionary pressures they exert on each other over evolutionary time are tightly linked to prevalence, intensity and/or abundance of

infection. From the host perspective, these parameters measure the proportion of the host population that experiences the direct effects of parasite infection, as well as the severity of these effects. Parasite-induced host mortality and other reductions in host fitness are more pronounced at high intensities of infection [7,8]. The impact of a parasite population on host population dynamics, and the selective pressures it exerts on host traits, are therefore greater if it occurs at high prevalence and intensity of infection [9,10]. From the parasite perspective, prevalence, intensity and/or abundance of infection capture the size of the parasite population at a given life stage, and its distribution among host individuals. Because individual hosts represent suitable habitat patches, prevalence quantifies the proportion of patches occupied, whereas intensity of infection is equivalent to the density of individual parasites per occupied patch. All else being equal, competition among individual parasites for space and nutrients increases with intensity of infection, with *per capita* growth and fecundity of many helminths being lower at high intensities [11–14]. Thus for a given total parasite population size, total egg output can vary greatly depending on how individual parasites are distributed among hosts, either crowded in a few hosts (low prevalence, high intensity) or spread across the host population (high prevalence, low intensity). Inequalities in body sizes and egg output among conspecific helminths also become more pronounced at high intensities, such that a few individuals can account for a disproportionate genetic contribution to the next generation [15,16]. Low prevalence combined with high mean intensity can exacerbate these inequalities. Thus, for a given total parasite population size, prevalence and intensity of infection can determine the effective population size ($N_e$ in population genetics) and consequently the efficiency of selection for beneficial alleles [17].

Despite their central importance in host–parasite population ecology and coevolution, the factors driving variation in prevalence, intensity and/or abundance of infection are poorly known. Furthermore, it remains unclear whether these driving factors are universal across parasite taxa, or instead differ among parasite taxa. Why are different populations of the same parasite species, or of closely related parasite species, achieving different prevalence or infection intensity in different host populations? There are at least four, non-mutually exclusive explanations. First, different parasite taxa may have evolved distinct taxon-specific traits that affect their transmission efficiency and the population sizes they achieve. The more closely related two parasite species are, the more similar they are likely to be with respect to key life history traits such as prepatency period, adult body size, resource requirements and fecundity [18]. As a consequence, all else being equal, the similarity in prevalence and intensity of infection achieved by different parasite species should be proportional to their phylogenetic relatedness. Possible phylogenetic influences must, therefore, be taken into account when testing for drivers of variation in prevalence or intensity of infection [19]. Second, host traits may also influence the prevalence or intensity of infection achieved by a parasite independently of the parasite's own traits [20,21]. For instance, populations of longer lived and larger bodied host species may be characterized by higher values of prevalence or intensity of infection by a given parasite than populations of short-lived and small-bodied hosts [22–24]. Variation among host populations or species in other properties, such as diet, microhabitat preferences or investment into reproduction, may also affect their exposure to infection or ability to mount immune responses, thus accounting for differences in epidemiological parameters [25,26].

Third, the local biotic community may determine what prevalence or intensity a generalist parasite achieves in a particular host population. If the focal host coexists locally with multiple other species of hosts that are suitable for the parasite, in many situations infection by the latter may be diluted across the community, resulting in lower prevalence and intensity values in the focal host [27–29]. The diversity of non-suitable host species in the local community also matters, as these non-hosts may reduce infection rates in suitable hosts, for example by acquiring parasites as transmission dead ends [28,30]. Fourth, the local abiotic conditions can modulate the transmission success and infection levels of many parasites, by directly affecting the survival or activity of eggs, larvae or other infective stages exposed to the external environment [31]. Both the average and temporal variation in temperature or precipitation can impact the survival and infectivity of helminth larval stages [32–34]. Local thermal conditions may also influence the activity levels and feeding rates of ectothermic hosts, as well as the efficiency of their immune responses [35–37], thereby affecting their exposure and/or resistance to infection.

Here, using a large dataset on the prevalence and intensity of infection of several taxa of helminth parasites recorded in multiple distinct populations of amphibian hosts across the world, we account for the first explanation above (phylogenetic conservatism of key parasite traits) while testing for the relative importance of the other three. By performing analyses among closely related parasite species (same family), we minimize any phylogenetic differences among parasites and achieve strong tests for the roles of host traits, local community diversity and local abiotic factors in shaping infection levels. Further, we conduct comparative analyses within vastly different families of helminth parasites, with different life cycles and modes of transmission. Our study, therefore, addresses an unresolved but fundamental issue in disease ecology: it aims to determine whether (i) the determinants of infection level are universal, transcending differences among parasite species and applying to all, (ii) they are instead idiosyncratic and taxon-dependent, or (iii) their effect falls between these extremes, for instance by applying only to parasite taxa with a particular mode of transmission.

## 2. Methods

### (a) Parasite data

We used a large, publicly available dataset on helminth parasites of amphibians used in two previous studies [38,39]. This dataset was assembled through a systematic search of literature published between 1970 and 2020 in the Google Scholar and Web of Science databases [38,39]. Studies were found through a combination of the keywords 'Helminth* OR Parasite*' and keywords associated with different anuran groups: 'Amphibia* OR Anura* OR Frog* OR Toad* OR Caudata* OR Urodela* OR Salamander* OR Newt* OR Gymnophiona OR Caecilian*' [38,39].

**Table 1.** Life cycle information and number of observations (i.e. host populations) for each helminth family included in the analyses of variation in prevalence and mean intensity of infection.

| parasite taxon | number of observations[a] | life cycle | hosts[b] | infection mode of amphibian host |
|---|---|---|---|---|
| **Nematoda** | | | | |
| Cosmocercidae | $P = 354, I = 251$ | direct | amphibian (H) | skin penetration |
| Rhabdiasidae | $P = 129, I = 104$ | direct | amphibian (H) | skin penetration |
| Molineidae | $P = 118, I = 83$ | direct | amphibian (H) | ingestion of infective larvae |
| **Trematoda** | | | | |
| Gorgoderidae | $P = 98, I = 50$ | complex | mollusc (IH1), insect (IH2), amphibian (DH) | ingestion of infected IH2 |
| Haematoloechidae | $P = 80, I = 53$ | complex | mollusc (IH1), insect (IH2), amphibian (DH) | ingestion of infected IH2 |
| **Cestoda** | | | | |
| Nematotaeniidae | $P = 71, I = 59$ | direct | amphibian (H) | ingestion of eggs/larvae[c] |

[a]$P$ = analyses of prevalence; $I$ = analyses of mean intensity.
[b]H = only host; IH1 or IH2 = first or second intermediate host; DH = definitive host.
[c]Based on the very limited information available for this taxon.

For the present analyses, we only included studies that examined the entire helminth community of adult amphibians in a given host community or population and provided data on host sample size and geographical location [38,39]. Each entry in the final dataset represents the interaction between one helminth species and one host population in one locality. Furthermore, we focused our analyses on helminth families for which at least 30 host individuals per population were examined for prevalence and mean intensity of infection across at least 50 host populations. Six parasite families met these requirements: the nematode families Cosmocercidae, Rhabdiasidae, and Molineidae; the trematode families Gorgoderidae and Haematoloechidae; and the cestode family Nematotaeniidae (table 1).

## (b) Spatial clustering and site-level predictor variables

We employed the density-based spatial clustering of applications with noise (DBSCAN) algorithm [40] to generate 5 km clusters that served to group data from nearby populations of the same host species. For each 5 km cluster, we derived coordinates weighted by the number of individual hosts examined for each population within the cluster and, subsequently, used these coordinates to obtain site-level predictor variables. For local climate, the latter were maximum temperature of the warmest month (Bio 05), minimal temperature of the coldest month (Bio 06), mean temperature of the wettest quarter (Bio 08), annual precipitation (Bio 12) and precipitation seasonality (Bio 15). These climatic variables were chosen for their likely importance for host and parasite biology and were obtained from WorldClim's v. 2.0 [41] at a spatial resolution of 2.5 min (approx. 5 km).

Local host species richness was obtained from a global raster of amphibian species richness with a spatial resolution of 30 arc-seconds (approx. 1 km) [42]. Prior to data extraction, we reprojected the amphibian richness raster to ensure that the predictor variables were all at the same spatial resolution.

## (c) Amphibian traits and missing data

As predictors related to amphibian host species, we obtained data on host habitat (categorical), maximum litter size (egg number) and body size (snout–vent length in mm) from the AMPHIBIO database [43]. In our dataset, species that use both the aquatic and terrestrial environments were classified as 'semi-aquatic', while the others were classified as 'terrestrial'. We worked with these two categories as a further split considering arboreal and fossorial habitats as separate categories would have compromised our sample sizes and statistical power.

Because trait values were unavailable for certain amphibian species, we used the 'impute' function from the R package 'funspace' [44] to impute missing information for body size and maximum litter size. This function uses the Random Forest approach while also allowing for the inclusion of phylogenetic information when computing missing trait information [44]. We used VertNet (http://vertnet.org/) to generate 99 amphibian phylogenetic trees [45] that were used for trait imputation and also for constructing the phylogenetic covariance matrix used in the Bayesian multilevel models (see §2d).

## (d) Data analysis

All analyses were conducted in the R environment [46]. To test potential drivers of helminth prevalence and mean intensity of infection among amphibian hosts, we fitted a series of Bayesian generalized linear multilevel models using the 'brms' R package [47–49]. First, to explore possible universal drivers of prevalence and intensity overriding any differences among parasite taxa,

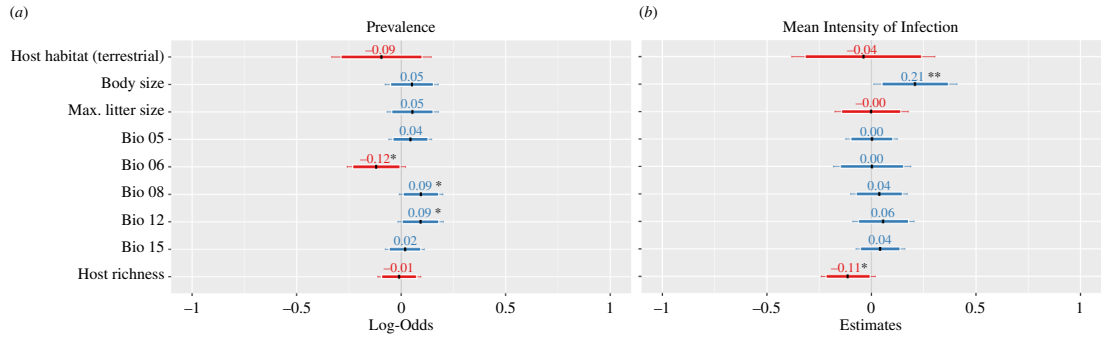


**Figure 1.** Posterior median estimates and high-density intervals (bars, 95% CI; whiskers, 89% CI) for the relationship between each predictor and helminth prevalence (*a*) and mean intensity of infection (*b*) in amphibians, across all parasite taxa pooled. Inner probabilities for CIs were set to 0.5, while outer probabilities were set to 0.95. Blue represents positive estimates and red indicates negative estimates. Asterisks indicate the probability of direction (pd): *, pd > 95% (roughly equivalent to $p < 0.10$); **, pd > 97.5% (roughly equivalent to $p < 0.05$). Climatic variables: Bio 05 = maximum temperature of the warmest month; Bio 06 = minimum temperature of the coldest month; Bio 08 = mean temperature of the wettest quarter; Bio 12 = annual precipitation; and Bio 15 = precipitation seasonality.

we fitted two models pooling all data for the six helminth families that matched our criteria (50 host populations sampled and 30 individuals examined per population), one model for prevalence and the other for intensity of infection. Following that, to test for family-specific infection drivers, we fitted separate models for each helminth family, separately for prevalence and mean intensity of infection. This yielded a total of 14 models. Full equations for our models, showing their detailed structure, are given in the electronic supplementary material. All models included host habitat (terrestrial or semi-aquatic), host body size (snout–vent length in mm), maximum litter size (number of eggs), maximum temperature of the warmest month, minimal temperature of the coldest month, mean temperature of the wettest quarter, annual precipitation, precipitation seasonality and local amphibian species richness. In addition to the phylogenetic correlation structure in the form of a variance–covariance phylogenetic matrix, all models also included a random intercept related to the zoogeographical realm of the host populations following [50]. The two models using all available data also included a random intercept for each parasite family. We scaled all numerical predictor variables by subtracting each value from the mean and dividing by the standard deviation. None of the pairwise correlations among predictors exceeded 0.7 (electronic supplementary material, figure S1).

Values of prevalence and mean intensity may be consistently low or high across host species within certain amphibian clades, for reasons other than the host traits considered here. To assess the phylogenetic signal ($\lambda$), we followed the recommendations of Bürkner [49]. Following [51], we applied the 'hypothesis' method, substituting the residual variance with $\pi 2/3$.

Although our models account for host phylogeny, we make no attempt to account for parasite phylogeny, for two reasons. First, although there are published within-family phylogenies for some of the parasite taxa we investigate (e.g. Rhabdiasidae [52]), they include only a fraction of the species in our dataset. Second, for a large number of entries in our dataset, the parasite is only identified to the genus level, rarely only to the family level. The separate family-level models described above assume a high level of biological similarity (ecological equivalence) across the parasite species included, which is a reasonable expectation for species within a family.

When modelling prevalence, the response variable was set as a binary matrix of 'successes' and 'failures', respectively, related to the number of infected individuals and sample size. When modelling the mean intensity of infection, we used the 5 km cluster average mean infection for a given host population. We used the beta-binomial distribution for modelling prevalence and the exponential distribution to model the mean intensity of infection; the latter was chosen over the gamma distribution as it produced much better model convergence. Models were estimated using Markov chain Monte Carlo (MCMC) sampling with five chains of 5000 iterations and a 2500-iteration warmup, with uniform priors for all population-level parameters (fixed effects). The only exception was the mean intensity of infection model for Nematotaeniidae, which, due to low effective sample sizes (ESS), used five chains of 10 000 interactions and normal distributions with mean 0 and standard deviation 1 as population-level priors. For the group-level parameters (random effects), we used the package's default weakly informative prior, which has a half Student's-*t* prior with three degrees of freedom and a scale parameter that is dependent upon the standard deviation of the response after applying the link function [47–49].

We evaluated the convergence and stability of Bayesian sampling using R-hat and ESS. The former should be less than 1.01 [53], and the latter higher than 1000 [48]. Additionally, we present posterior plots and convergence chains for each parameter and predictive checks for the whole model (electronic supplementary material, figures S2–S15). We also checked for evidence of spatial autocorrelation in our models using Moran's I test for distance-based autocorrelation via the 'testSpatialAutocorrelation' function of the 'DHARMa' package [54].

For each parameter, we obtained its probability of direction (pd) as well as the percentage of its posterior probability that falls within the region of practical equivalence (ROPE). In a nutshell, the pd quantifies the certainty an effect is either positive or negative depending on the proportion of the posterior distribution that falls in a particular direction [55,56]. The index has a strong correlation with the frequentist *p*-value, with pd greater than 95% roughly corresponding to $p < 0.10$ and pd greater than 97.5% roughly corresponding to $p < 0.05$ [55,56]. On the other hand, the ROPE is a user-defined range that encompasses values deemed equivalent to a null hypothesis of 'negligible effect' [55]. The percentage in ROPE is the proportion of samples that fall within this null-equivalent region, indicating the strength of the effect [55]. Following the package's recommendations, we used the percentage of the full posterior distribution to obtain the proportion of posterior draws within the ROPE region [55]. According to [55], when using this approach, the null hypothesis is rejected when the percentage that falls within the ROPE

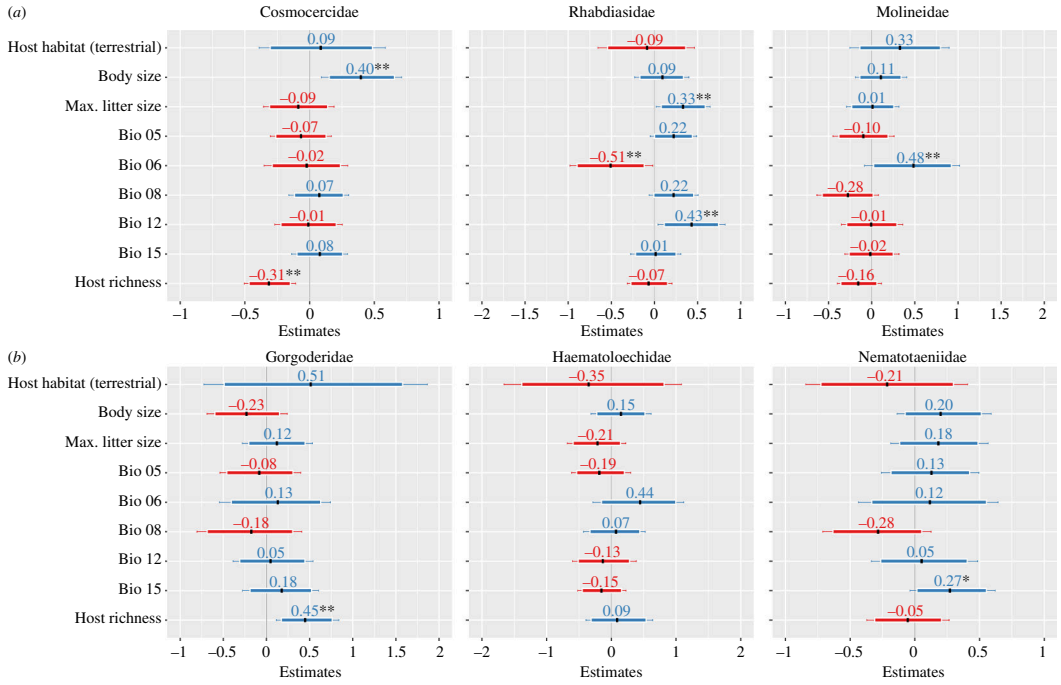


**Figure 2.** Posterior median estimates and high-density intervals (bars, 95% CI; whiskers, 89% CI) for the relationship between each predictor and helminth mean intensity of infection, for each parasite family separately. Inner probabilities for CIs were set to 0.5, while outer probabilities were set to 0.95. Blue represents positive estimates and red indicates negative estimates. Asterisks indicate the probability of direction (pd): *, pd > 95% (roughly equivalent to $p < 0.10$); **, pd > 97.5% (roughly equivalent to $p < 0.05$). Climatic variables: Bio 05 = maximum temperature of the warmest month; Bio 06 = minimum temperature of the coldest month; Bio 08 = mean temperature of the wettest quarter; Bio 12 = annual precipitation; and Bio 15 = precipitation seasonality.

region is lower than 2.5% and accepted when this number exceeds 97.5%. Any value in between is inconclusive [55]. Using the package's defaults, we defined our ROPE region for the mean intensity of infection models as [−0.1, 0.1], and for the prevalence models as [−0.18, 0.18].

## 3. Results

Our dataset included data on 747 host populations of 224 species (200 anurans and 24 salamanders) for prevalence models, and data on 613 host populations of 188 species (173 anurans and 15 salamanders) for models of mean intensity of infection (table 1). The majority of the data in both models came from studies conducted in the Neotropics, followed by the Nearctic and Palearctic. Electronic supplementary material, tables S1 and S2, provides detailed sample sizes for each parasite family and realm. Variation in infection parameters was extensive among host populations in our dataset: for each parasite family, prevalence ranged from well below 10% to well over 50%, while mean intensity varied 10-fold or more (e.g. over 50-fold in Cosmocercidae). Model diagnostics indicate good convergence and stability across all models and estimated parameters (electronic supplementary material, tables S3 and S4; figures S2–S15). We also did not find any evidence of spatial autocorrelation in any of our models (electronic supplementary material, table S5). We report the median of the posterior distribution and its 89% and 95% CI (highest density interval) in figures 1–3, as well as the pd and percentage of posteriors within the ROPE (electronic supplementary material, tables S3 and S4).

In the model of mean intensity of infection encompassing data pooled across all six parasite families, we observed a positive relationship between host body size and intensity of infection (figure 1; electronic supplementary material, table S4). In terms of the strength of the effect, the positive effect of body size has a relatively high probability of being strong (only approximately 13.75% of posterior falls within ROPE; electronic supplementary material, table S4). Additionally, there is a high probability that host richness is negatively associated with the mean intensity of infection across the six parasite groups (pd = 0.95%; figure 1; electronic supplementary material, table S4); however, almost half of the posterior distribution falls within ROPE, which is inconclusive in relation to the strength of the effect (electronic supplementary material, table S4). In the corresponding model of prevalence across all parasite families, none of the variables appear to be related to prevalence (figure 1; electronic supplementary material, table S3). Even though the minimum temperature of the wettest month (Bio 06), the mean temperature of the wettest quarter (Bio 08) and annual precipitation (Bio 12) have high probabilities of direction (pd > 95%), more than 80% of their posteriors fall within the ROPE (electronic supplementary material, table S3), implying a weak effect. In both models, we found strong indications that the parasite family explains a high amount of variability in parasite prevalence and intensity of infection (electronic supplementary material, tables S3 and S4), implying that the relationship between predictors and infection is family specific. Finally, for both models, the phylogenetic component showed strong indications for negligible effects, with more than 97.5% of the posterior distribution falling within the ROPE (electronic supplementary material, tables S3 and S4).

In accordance with what was indicated by the importance of the random family component of the previous models, the family-level models yielded family-specific outcomes. In the models focused on Cosmocercidae, we found a positive association

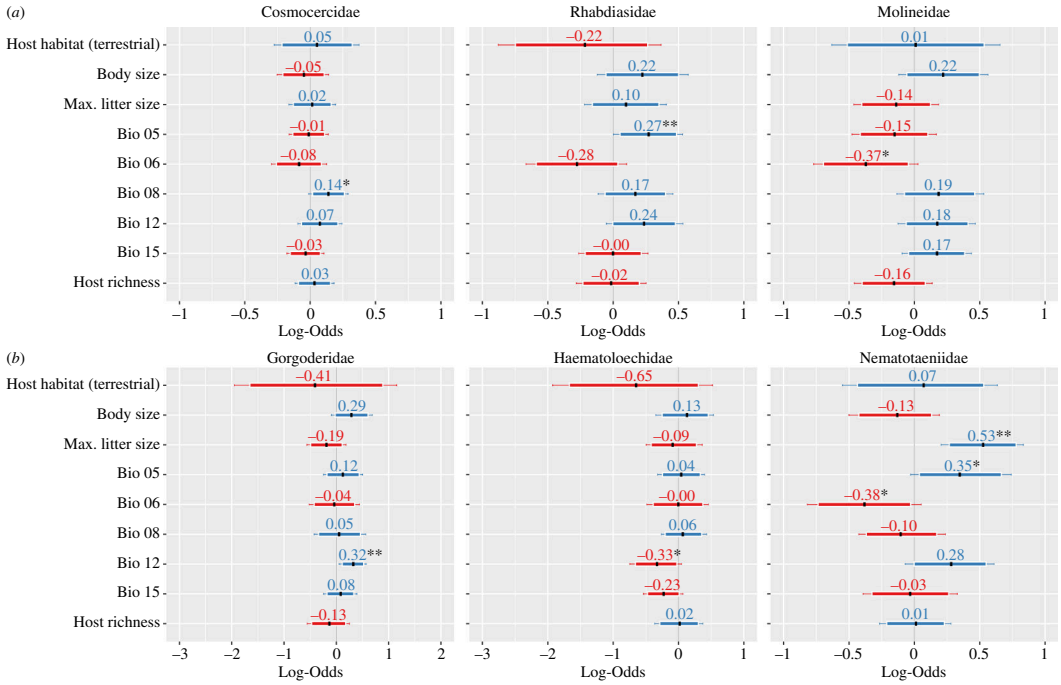


**Figure 3.** Posterior median estimates and high-density intervals (bars, 95% CI; whiskers, 89% CI) for the relationship between each predictor and helminth prevalence, for each parasite family separately. Inner probabilities for CIs were set to 0.5, while outer probabilities were set to 0.95. Blue represents positive estimates and red indicates negative estimates. Asterisks indicate the probability of direction (pd): *, pd > 95% (roughly equivalent to $p < 0.10$); **, pd > 97.5% (roughly equivalent to $p < 0.05$). Climatic variables: Bio 05 = maximum temperature of the warmest month; Bio 06 = minimum temperature of the coldest month; Bio 08 = mean temperature of the wettest quarter; Bio 12 = annual precipitation; and Bio 15 = precipitation seasonality.

between intensity of infection and host body size (figure 2; electronic supplementary material, table S4), as well as a negative association between intensity of infection and host richness (figure 2; electronic supplementary material, table S4); both with a high probability of being strong (figure 2; electronic supplementary material, table S4). On the other hand, none of the predictors seem to be strongly associated with Cosmocercidae prevalence, despite a high probability of a weak positive association between minimal temperature of the coldest month and prevalence (figure 3; electronic supplementary material, table S3).

For Rhabdiasidae, we found that intensity of infection was positively associated with the maximum litter size of hosts and annual precipitation (figure 2; electronic supplementary material, table S4) and negatively associated with the minimum temperature of the coldest month (figure 2; electronic supplementary material, table S4); all with a relatively high chance of being strong (percentage of posterior in ROPE < 7%; electronic supplementary material, table S4). None of the predictors were associated with Rhabdiasidae prevalence (figure 3; electronic supplementary material, table S3), despite a high probability of a positive relationship between the maximum temperature of the warmest month and prevalence.

For Molineidae, we found that there is a high probability (pd > 95%; figure 3) of a negative association between the minimum temperature of the coldest month (Bio 06) and prevalence, though the strength of the effect is inconclusive (electronic supplementary material, table S3). Interestingly, we found that this same climatic variable has a high probability of being positively associated with Molineidae mean intensity of infection (figure 2; electronic supplementary material, table S3), with a relatively high probability of being strong (electronic supplementary material, table S4).

For Gorgoderidae, we found a positive association between prevalence and annual precipitation (figure 3; electronic supplementary material, table S3). The posterior within ROPE is around 13.8%, which points towards a relatively high probability of a strong effect. Additionally, we found a strong positive association between host richness and intensity of infection (figure 2; electronic supplementary material, table S4).

For Haematoloechidae, we found a possible weak association between annual precipitation and prevalence (figure 3; electronic supplementary material, table S3), while none of the variables seemed important for intensity of infection (figure 2; electronic supplementary material, table S4).

Finally, we found a positive association between Nematotaeniidae prevalence and host maximum litter size (figure 2; electronic supplementary material, table S3). Additionally, we found that the maximum temperature of the warmest month (Bio 05) and the minimum temperature of the coldest month (Bio 06) might be also, respectively, positively and negatively related to prevalence (figure 2; electronic supplementary material, table S3). For the mean intensity of infection, there is a relatively high probability that precipitation seasonality is positively associated with intensity of infection (electronic supplementary material, table S4).

In terms of the effect of host phylogeny in our models, despite the probability of direction being high, almost all posterior draws for phylogenetic standard deviation fell within the ROPE (electronic supplementary material, tables S3 and S4), and all estimates of phylogenetic signal ($\lambda$) had confidence intervals overlapping zero.

## 4. Discussion

The search for universal determinants of animal abundance has uncovered some nearly universal rules, such as the well-established negative interspecific relationship between body size and population density observed within most free-living higher taxa [57,58]. In contrast, the search for universal rules in parasite ecology has revealed very few general patterns applying across parasite taxa, and as yet no universal predictor of parasite prevalence or mean intensity of infection, the two components of parasite abundance [59]. Here, using a large dataset comprising population-level data from several hundred amphibian populations, we found that the determinants of parasite abundance are taxonomically idiosyncratic: host traits or local external factors that are associated with infection levels by parasites in one helminth family are unrelated to infection levels in other helminth families. Our findings indicate that the drivers of parasite prevalence and intensity of infection are taxon specific.

The strongest candidate for a universal driver of helminth infection levels was host body size. For simplicity, here and elsewhere in the discussion that follows, we consider as significant only the predictors with estimates whose CIs did not overlap with zero; these were generally well supported by the other indices of effect certainty and strength (pd and ROPE) we report. Across the pooled data from all parasite families combined, we observed a positive relationship between host body size and intensity of infection. Other interspecific analyses have reported similar associations, explained by the greater space and energy provided by larger hosts allowing higher parasite burdens [22–24,60,61]. However, our analysis of the pooled data considered vastly different helminth taxa, potentially creating a spurious relationship. When the analyses were restricted to parasites from the same family, which are comparable in terms of transmission mode, site of infection, adult worm sizes, etc., host body size emerged as significantly associated with the mean intensity of infection in only one of six helminth families. Thus, we did not detect any universal effect of the availability of host resources, as estimated by average host body size, as either a determinant or constraint of parasite abundance among the host populations in our dataset.

Among the other host traits we considered, litter size (number of eggs laid per clutch) was positively associated with prevalence for one helminth family (Nematotaeniidae) and with intensity of infection for another family (Rhabdiasidae). These relationships suggest a potential trade-off between investments in reproduction versus those in defence against parasites [62]. Thus, the production of many offspring may come at the cost of greater susceptibility to infection by certain parasites. Again, however, no universal reproduction–infection connection was uncovered by our analyses among the host populations considered here, instead, family-specific effects were observed. We found no association between host habitat (semi-aquatic versus fully terrestrial) and either prevalence or intensity of infection, for any of the six helminth families. This is unexpected, given that the mode of infection of parasites in some of the helminth families considered here suggests that association with water or water-saturated soil should favour infection success.

There are other host traits, not considered here, that may affect infection levels by helminths. Our models accounted for phylogenetic relatedness among the amphibian species included in our analyses, and therefore indirectly accounted for ecological and life history similarities and differences among host species. However, no significant phylogenetic signal was detected in any of our models. This could be due to the limited size of our phylogenetic trees since our analyses included only a subset of extant amphibian species. With this caveat, we found no evidence that higher or lower infection values are overrepresented in some branches of the amphibian phylogenetic tree, and so no evidence of differential susceptibility to infection among host clades. To our knowledge, only one previous study quantified the phylogenetic signal in infection levels among host species, that of [51], who report a weak but significant overall phylogenetic signal for the prevalence of haemosporidian parasites in birds. Clade differences in susceptibility may be more likely in the case of haemosporidians, given the greater role of the host immune system in combating these parasites compared to helminths.

Local factors can also influence infection levels achieved by a particular parasite species in a given host species. Helminth prevalence and intensity of infection have been shown to be species properties of host species, as infection levels by particular parasites often vary less among populations of the same vertebrate host species than among populations of different host species [63,64]. Yet they do vary intraspecifically, suggesting the action of local variables. Of the site-level predictors included in our models, the species richness of the local amphibian community was negatively related with intensity of infection for Cosmocercidae, but positively related with intensity of infection for Gorgoderidae. In the former case, the negative association between infection levels in a focal amphibian species and the local diversity of amphibians is compatible with the dilution hypothesis [27–29]. Cosmocercid nematodes infect their amphibian hosts via skin penetration; the greater the number of alternative amphibian hosts available locally, the greater the chances alternative hosts make contact with and acquire parasite larvae from the environment, leaving fewer larvae to infect the focal host. In contrast, the pattern observed for trophically transmitted gorgoderid trematodes suggests an amplification effect, whereby the presence of many alternative host species boosts transmission rates and subsequently the reproductive success of parasites, leading to higher infections of the focal host species. In this case, a locally diverse suite of suitable hosts may support a greater adult worm population, and lead to higher infection levels of trematode juvenile stages in the insect second intermediate hosts. Usually associated with biological invasions and the spillback effect [65,66], amplification is also possible for a generalist parasite within a diverse host community.

Finally, of the local climatic variables included as predictors in our models, annual precipitation emerged as influential, being positively associated with intensity of infection for Rhabdiasidae and with prevalence for Gorgoderidae. The minimum temperature of the coldest month was also negatively associated with intensity of infection for Rhabdiasidae. In the case of Rhabdiasidae, greater soil humidity may favour the survival and eventual host penetration of rhabdiasid infective larvae, whereas cold temperatures may have the opposite effect. For gorgoderids, greater rainfall may lead to more aquatic habitats for the snail's first intermediate host. Based on the indices pd and ROPE, other climatic variables show some associations with infection parameters, but inconsistently across parasite families. Overall, the main outcome of our analyses is the demonstration

that site-level drivers of parasite infection levels, just as host traits, are highly idiosyncratic and taxon-specific, with little evidence of universal determinants.

Detecting drivers of parasite abundance across multiple host populations and species is challenging. First, although relatively large, the size of our dataset may nevertheless have been too small to allow small-to-modest effect sizes to emerge from the noise in the data, thus limiting the statistical power of our analyses. Second, the type of data used in this study is the only one available across large spatial and taxonomic scales. However, these data on parasite abundance are based on single sampling points in time: they do not account for variation in abundance across seasons or years. In temperate regions, amphibian collections are constrained to the warmer months, a period during which infection levels increase predictably [67]; most field collections target the month(s) when higher infection levels are expected. On longer time scales, host and/or parasite populations may experience cyclic fluctuations and may not be at equilibrium. The few long-term (5–12 years) surveys of helminth infections in amphibians, however, suggest relatively consistent year-to-year parasite prevalence and intensity. In some cases, helminth prevalence and intensity in local frog populations show moderate annual variation [68], whereas in other cases infection levels in amphibians show remarkable stability, varying by 10% or less from year to year [69], even in response to experimental parasite removal [70] or changes in infections of intermediate hosts [71]. Therefore, the 'snapshot' samples making up our dataset are likely to provide representative patterns. Third, the original studies from which we obtained the data also do not report several other local factors of potential importance. For example, they do not provide usable data on local host population density, a factor known to affect parasite transmission [20], as well as other potentially influential variables such as the diversity of available invertebrate prey or the extent of anthropogenic habitat modification. Finally, parasite abundance is the outcome of exposure to parasites on the one hand and susceptibility to infection on the other. It is not possible to tease apart these two processes and how they may have been independently affected by the predictors considered in our analyses. Had this been possible, perhaps universal drivers of either exposure or susceptibility would have been apparent.

While acknowledging these caveats, we conclude that among the *a priori* important host traits and local factors investigated here in a global comparative analysis, none could consistently (i.e. across parasite taxa) explain inter-population and interspecific variation in prevalence and mean intensity of infection, the two components of parasite abundance and the two key parameters shaping host–parasite interaction strength. This is true even among parasite taxa from the same phylum, or with similar life cycles and modes of transmission. In contrast, the same predictors, e.g. host body size, local host diversity and climatic variables, generally correlate well with interspecific variation in parasite species richness among amphibian species [38,72], as well as more broadly across metazoans [73]. With prevalence and intensity of infection for a given parasite genus or family varying widely among host species or localities, the potential of parasites to regulate host populations is itself highly variable [74]. Notwithstanding the many interacting factors that also affect host population dynamics, our findings suggest that the abundance of parasites is not predictably and consistently linked with host traits and local factors. Instead, at least based on the factors tested in this study, the determinants of parasite infection levels appear idiosyncratic and taxon specific, preventing any generalization, including general predictions of the responses of local parasite abundance to climate change.

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. The dataset, phylogeny files, and R code used in this study are available as electronic supplementary material [75].

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. P.M.M.: conceptualization, data curation, formal analysis, methodology, visualization, writing—original draft; R.P.: conceptualization, methodology, writing—original draft.

Both authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

Funding. This research received no specific funding.

Acknowledgements. We thank Prof. Diogo Borges Provete for advice regarding Bayesian modelling and two anonymous reviewers for comments on an earlier version.

## References

1. Vázquez DP, Melián CJ, Williams NM, Blüthgen N, Krasnov BR, Poulin R. 2007 Species abundance and asymmetric interaction strength in ecological networks. *Oikos* **116**, 1120–1127.
2. Canard EF, Mouquet N, Mouillot D, Stanko M, Miklisova D, Gravel D. 2014 Empirical evaluation of neutral interactions in host-parasite networks. *Am. Nat.* **183**, 468–479. (doi:10.1086/675363)
3. Runghen R, Poulin R, Monlleó-Borrull C, Llopis-Belenguer C. 2021 Network analysis: ten years shining light on host-parasite interactions. *Trends Parasitol.* **37**, 445–455. (doi:10.1016/j.pt.2021.01.005)
4. Margolis L, Esch GW, Holmes JC, Kuris AM, Schad GA. 1982 The use of ecological terms in parasitology (report of an ad hoc committee of the American Society of Parasitologists). *J. Parasitol.* **68**, 131. (doi:10.2307/3281335)
5. Morand S, Guégan JF. 2000 Distribution and abundance of parasite nematodes: ecological specialisation, phylogenetic constraint or simply epidemiology? *Oikos* **88**, 563–573. (doi:10.1034/j.1600-0706.2000.880313.x)
6. Simková A, Kadlec D, Gelnar M, Morand S. 2002 Abundance-prevalence relationship of gill congeneric ectoparasites: testing the core satellite hypothesis and ecological specialisation. *Parasitol. Res.* **88**, 682–686. (doi:10.1007/s00436-002-0650-3)
7. Anderson RM, Gordon DM. 1982 Processes influencing the distribution of parasite numbers within host populations with special emphasis on parasite-induced host mortalities. *Parasitology* **85**, 373–398. (doi:10.1017/s0031182000055347)

8. Benesh DP. 2011 Intensity-dependent host mortality: what can it tell us about larval growth strategies in complex life cycle helminths? *Parasitology* **138**, 913–925. (doi:10.1017/S0031182011000370)
9. Hudson PJ, Dobson AP, Newborn D. 1998 Prevention of population cycles by parasite removal. *Science* **282**, 2256–2258. (doi:10.1126/science.282.5397.2256)
10. Stien A, Irvine RJ, Ropstad E, Halvorsen O, Langvatn R, Albon SD. 2002 The impact of gastrointestinal nematodes on wild reindeer: experimental and cross-sectional studies. *J. Anim. Ecol.* **71**, 937–945. (doi:10.1046/j.1365-2656.2002.00659.x)
11. Shostak AW, Scott ME. 1993 Detection of density-dependent growth and fecundity of helminths in natural infections. *Parasitology* **106**, 527–539. (doi:10.1017/s0031182000076836)
12. Heins DC, Baker JA, Martin HC. 2002 The 'crowding effect' in the cestode *Schistocephalus solidus*: density-dependent effects on plerocercoid size and infectivity. *J. Parasitol.* **88**, 302–307. (doi:10.1645/0022-3395(2002)088[0302:TCEITC]2.0.CO;2)
13. Lowrie FM, Behnke JM, Barnard CJ. 2004 Density-dependent effects on the survival and growth of the rodent stomach worm *Protospirura muricola* in laboratory mice. *J. Helminthol.* **78**, 121–128. (doi:10.1079/joh2003230)
14. Chylinski C, Boag B, Stear MJ, Cattadori IM. 2009 Effects of host characteristics and parasite intensity on growth and fecundity of *Trichostrongylus retortaeformis* infections in rabbits. *Parasitology* **136**, 117–123. (doi:10.1017/S0031182008005143)
15. Shostak AW, Dick TA. 1987 Individual variability in reproductive success of *Triaenophorus crassus* Forel (Cestoda: Pseudophyllidea), with comments on use of the Lorenz curve and Gini coefficient. *Can. J. Zool.* **65**, 2878–2885. (doi:10.1139/z87-437)
16. Szalai AJ, Dick TA. 1989 Differences in numbers and inequalities in mass and fecundity during the egg-producing period for *Raphidascaris acus* (Nematoda: Anisakidae). *Parasitology* **98**, 489–495. (doi:10.1017/S0031182000061588)
17. Criscione CD, Blouin MS. 2005 Effective sizes of macroparasite populations: a conceptual model. *Trends Parasitol.* **21**, 212–217. (doi:10.1016/j.pt.2005.03.002)
18. Krasnov BR, Morand S, Poulin R. 2015 Phylogenetic signals in ecological properties of parasites. In *Parasite diversity and diversification: evolutionary ecology meets phylogenetics* (eds S Morand, B Krasnov, D Littlewood), pp. 351–359. Oxford, UK: Oxford University Press.
19. Morand S, Poulin R. 2003 Phylogenies, the comparative method and parasite evolutionary ecology. *Adv. Parasitol.* **54**, 281–302. (doi:10.1016/s0065-308x(03)54006-4)
20. Arneberg P, Skorping A, Grenfell B, Read AF. 1998 Host densities as determinants of abundance in parasite communities. *Proc. R. Soc. B* **265**, 1283–1289. (doi:10.1098/rspb.1998.0431)
21. Johnson PTJ, Hoverman JT. 2014 Heterogeneous hosts: how variation in host size, behaviour and immunity affects parasite aggregation. *J. Anim. Ecol.* **83**, 1103–1112. (doi:10.1111/1365-2656.12215)
22. Poulin R. 1999 Body size vs abundance among parasite species: positive relationships? *Ecography* **22**, 246–250. (doi:10.1111/j.1600-0587.1999.tb00499.x)
23. Braicovich PE, Ieno EN, Sáez M, Despos J, Timi JT. 2016 Assessing the role of host traits as drivers of the abundance of long-lived parasites in fish-stock assessment studies. *J. Fish Biol.* **89**, 2419–2433. (doi:10.1111/jfb.13127)
24. Cassin Sackett L. 2018 Does the host matter? Variable influence of host traits on parasitism rates. *Int. J. Parasitol.* **48**, 27–39. (doi:10.1016/j.ijpara.2017.07.006)
25. Vignon M, Sasal P. 2010 Multiscale determinants of parasite abundance: a quantitative hierarchical approach for coral reef fishes. *Int. J. Parasitol.* **40**, 443–451. (doi:10.1016/j.ijpara.2009.09.010)
26. Breiner DJ, Whalen MR, Worthington AM. 2022 The developmental high wire: balancing resource investment in immunity and reproduction. *Ecol. Evol.* **12**, e8774. (doi:10.1002/ece3.8774)
27. Keesing F, Holt RD, Ostfeld RS. 2006 Effects of species diversity on disease risk. *Ecol. Lett.* **9**, 485–498. (doi:10.1111/j.1461-0248.2006.00885.x)
28. Johnson PTJ, Thieltges DW. 2010 Diversity, decoys and the dilution effect: how ecological communities affect disease risk. *J. Exp. Biol.* **213**, 961–970. (doi:10.1242/jeb.037721)
29. Rohr JR, Civitello DJ, Halliday FW, Hudson PJ, Lafferty KD, Wood CL, Mordecai EA. 2020 Towards common ground in the biodiversity-disease debate. *Nat. Ecol. Evol.* **4**, 24–33. (doi:10.1038/s41559-019-1060-6)
30. Thieltges DW, Jensen KT, Poulin R. 2008 The role of biotic factors in the transmission of free-living endohelminth stages. *Parasitology* **135**, 407–426. (doi:10.1017/S0031182007000248)
31. Pietrock M, Marcogliese DJ. 2003 Free-living endohelminth stages: at the mercy of environmental conditions. *Trends Parasitol.* **19**, 293–299. (doi:10.1016/s1471-4922(03)00117-x)
32. Froeschke G, Harf R, Sommer S, Matthee S. 2010 Effects of precipitation on parasite burden along a natural climatic gradient in southern Africa: implications for possible shifts in infestation patterns due to global changes. *Oikos* **119**, 1029–1039. (doi:10.1111/j.1600-0706.2009.18124.x)
33. Selbach C, Poulin R. 2020 Some like it hotter: trematode transmission under changing temperature conditions. *Oecologia* **194**, 745–755. (doi:10.1007/s00442-020-04800-y)
34. Díaz-Morales DM, Bommarito C, Vajedsamiei J, Grabner DS, Rilov G, Wahl M, Sures B. 2022 Heat sensitivity of first host and cercariae may restrict parasite transmission in a warming sea. *Sci. Rep.* **12**, 1174. (doi:10.1038/s41598-022-05139-5)
35. Raffel TR, Rohr JR, Kiesecker JM, Hudson PJ. 2006 Negative effects of changing temperature on amphibian immunity under field conditions. *Funct. Ecol.* **20**, 819–828. (doi:10.1111/j.1365-2435.2006.01159.x)
36. Terrell KA, Quintero RP, Murray S, Kleopfer JD, Murphy JB, Evans MJ, Nissen BD, Gratwicke B. 2013 Cryptic impacts of temperature variability on amphibian immune function. *J. Exp. Biol.* **216**, 4204–4211. (doi:10.1242/jeb.089896)
37. Scharsack JP, Franke F. 2022 Temperature effects on teleost immunity in the light of climate change. *J. Fish Biol.* **101**, 780–796. (doi:10.1111/jfb.15163)
38. Martins PM, Poulin R, Gonçalves-Souza T. 2021 Integrating climate and host richness as drivers of global parasite diversity. *Glob. Ecol. Biogeogr.* **30**, 196–204. (doi:10.1111/geb.13213)
39. Martins PM, Poulin R, Gonçalves-Souza T. 2021 Drivers of parasite β-diversity among anuran hosts depend on scale, realm and parasite group. *Phil. Trans. R. Soc. B* **376**, 20200367. (doi:10.1098/rstb.2020.0367)
40. Hahsler M, Piekenbrock M, Doran D. 2019 Dbscan: fast density-based clustering with R. *J. Stat. Softw.* **91**, 1–30. (doi:10.18637/jss.v091.i01)
41. Fick SE, Hijmans RJ. 2017 WorldClim 2: new 1-km spatial resolution climate surfaces for global land areas. *Int. J. Climatol.* **37**, 4302–4315. (doi:10.1002/joc.5086)
42. International Union for Conservation of Nature—IUCN, Center for International Earth Science Information Network—CIESIN, Columbia University. 2015 Gridded species distribution: global amphibian richness grids, 2015 release. Palisades, NY: NASA Socioeconomic Data and Applications Center (SEDAC). See https://doi.org/10.7927/H4RR1W66.
43. Oliveira BF, São-Pedro VA, Santos-Barrera G, Penone C, Costa GC. 2017 AmphiBIO, a global database for amphibian ecological traits. *Sci. Data* **4**, 170123. (doi:10.1038/sdata.2017.123)
44. Pavanetto N, Puglielli G. 2024 Funspace: creating and representing functional trait spaces. R package version 02.1. See https://CRAN.R-project.org/package=funspace.
45. Jetz W, Pyron RA. 2018 The interplay of past diversification and evolutionary isolation with present imperilment across the amphibian tree of life. *Nat. Ecol. Evol.* **2**, 850–858. (doi:10.1038/s41559-018-0515-5)

46. R Core Team. 2024 R: a language and environment for statistical computing. See https://www.r-project.org.
47. Bürkner P-C. 2017 brms: an R package for Bayesian multilevel models using Stan. *J. Stat. Softw*. **80**, 1–28. (doi:10.18637/jss.v080.i01)
48. Bürkner PC. 2018 Advanced Bayesian multilevel modeling with the R package brms. *R J*. **10**, 395–411. (doi:10.32614/RJ-2018-017)
49. Bürkner P-C. 2021 Bayesian item response modeling in R with brms and Stan. *J. Stat. Softw*. **100**, 1–54. (doi:10.18637/jss.v100.i05)
50. Holt BG *et al*. 2013 An update of Wallace's zoogeographic regions of the world. *Science* **339**, 74–78. (doi:10.1126/science.1228282)
51. Barrow LN, McNew SM, Mitchell N, Galen SC, Lutz HL, Skeen H, Valqui T, Weckstein JD, Witt CC. 2019 Deeply conserved susceptibility in a multi-host, multi-parasite system. *Ecol. Lett*. **22**, 987–998. (doi:10.1111/ele.13263)
52. Zeng JL, Chen HX, Ni XF, Kang JY, Li L. 2024 Molecular phylogeny of the family Rhabdiasidae (Nematoda: Rhabditida), with morphology, genetic characterization and mitochondrial genomes of *Rhabdias kafunata* and *R. bufonis*. *Parasit. Vectors* **17**, 100. (doi:10.1186/s13071-024-06201-z)
53. Vehtari A, Gelman A, Simpson D, Carpenter B, Bürkner PC. 2021 Rank-normalization, folding, and localization: an improved $\check{R}$ for assessing convergence of MCMC (with discussion). *Bayesian Anal.* **16**, 667–718. (doi:10.1214/20-BA1221)
54. Hartig F. 2022 DHARMa: residual diagnostics for hierarchical (multi-level/mixed) regression models. R package version 0.4.6. See https://CRAN.R-project.org/package=DHARMa.
55. Makowski D, Ben-Shachar M, Lüdecke D. 2019 BayestestR: describing effects and their uncertainty, existence and significance within the Bayesian framework. *J. Open Source Softw.* **4**, 1541. (doi:10.21105/joss.01541)
56. Makowski D, Ben-Shachar MS, Chen SHA, Lüdecke D. 2019 Indices of effect existence and significance in the Bayesian framework. *Front. Psychol*. **10**, 2767. (doi:10.3389/fpsyg.2019.02767)
57. Peters RH, Wassenberg K. 1983 The effect of body size on animal abundance. *Oecologia* **60**, 89–96. (doi:10.1007/BF00379325)
58. Blackburn TM, Gaston KJ. 1999 The relationship between animal abundance and body size: a review of the mechanisms. *Adv. Ecol. Res.* **28**, 181–210. (doi:10.1016/S0065-2504(08)60033-1)
59. Poulin R. 2007 Are there general laws in parasite ecology? *Parasitology* **134**, 763–776. (doi:10.1017/S0031182006002150)
60. Grutter AS, Poulin R. 1998 Intraspecific and interspecific relationships between host size and the abundance of parasitic larval gnathiid isopods on coral reef fishes. *Mar. Ecol. Progr. Ser.* **164**, 263–271. (doi:10.3354/MEPS164263)
61. Del Mar Labrador M *et al*. 2024 Host space, not energy or symbiont size, constrains feather mite abundance across passerine bird species. *J. Anim. Ecol.* **93**, 393–405. (doi:10.1111/1365-2656.14032)
62. French SS, DeNardo DF, Moore MC. 2007 Trade-offs between the reproductive and immune systems: facultative responses to resources or obligate responses to reproduction? *Am. Nat*. **170**, 79–89. (doi:10.1086/518569)
63. Arneberg P, Skorping A, Read AF. 1997 Is population density a species character? Comparative analyses of the nematode parasites of mammals. *Oikos* **80**, 289. (doi:10.2307/3546597)
64. Poulin R. 2006 Variation in infection parameters among populations within parasite species: intrinsic properties versus local factors. *Int. J. Parasitol*. **36**, 877–885. (doi:10.1016/j.ijpara.2006.02.021)
65. Kelly DW, Paterson RA, Townsend CR, Poulin R, Tompkins DM. 2009 Parasite spillback: a neglected concept in invasion ecology? *Ecology* **90**, 2047–2056. (doi:10.1890/08-1085.1)
66. Telfer S, Bown K. 2012 The effects of invasion on parasite dynamics and communities. *Funct. Ecol.* **26**, 1288–1299. (doi:10.1111/j.1365-2435.2012.02049.x)
67. Poulin R. 2020 Meta-analysis of seasonal dynamics of parasite infections in aquatic ecosystems. *Int. J. Parasitol*. **50**, 501–510. (doi:10.1016/j.ijpara.2020.03.006)
68. Lunde KB, Resh VH, Johnson PTJ. 2012 Using an ecosystem-level manipulation to understand host-parasite interactions and how they vary with study venue. *Ecosphere* **3**, 1–18. (doi:10.1890/ES12-00001.1)
69. Zelmer DA, Wetzel EJ, Esch GW. 1999 The role of habitat in structuring *Halipegus occidualis* metapopulations in the green frog. *J. Parasitol*. **85**, 19–24.
70. Zelmer DA, Esch GW. 2000 Relationship between structure and stability of a *Halipegus occidualis* component population in green frogs: a test of selective treatment. *J. Parasitol*. **86**, 233–240. (doi:10.1645/0022-3395(2000)086[0233:RBSASO]2.0.CO;2)
71. Esch GW, Wetzel EJ, Zelmer DA, Schotthoefer AM. 1997 Long-term changes in parasite population and community structure: a case history. *Am. Midl. Nat.* **137**, 369. (doi:10.2307/2426856)
72. Campião KM, Ribas AC de A, Morais DH, da Silva RJ, Tavares LER. 2015 How many parasites species a frog might have? Determinants of parasite diversity in South American anurans. *PLoS ONE* **10**, e0140577. (doi:10.1371/journal.pone.0140577)
73. Kamiya T, O'Dwyer K, Nakagawa S, Poulin R. 2014 What determines species richness of parasitic organisms? A meta-analysis across animal, plant and fungal hosts. *Biol. Rev.* **89**, 123–134. (doi:10.1111/brv.12046)
74. Tompkins DM, Dunn AM, Smith MJ, Telfer S. 2011 Wildlife diseases: from individuals to ecosystems. *J. Anim. Ecol*. **80**, 19–38. (doi:10.1111/j.1365-2656.2010.01742.x)
75. Martins PM, Poulin R. 2024 Data from: Universal versus taxon-specific drivers of helminth prevalence and intensity of infection. Figshare. (doi:10.6084/m9.figshare.c.7492637)