

LETTER

Habitat heterogeneity drives the host-diversity-begets-parasite-diversity relationship: evidence from experimental and field studies

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Abstract

Despite a century of research into the factors that generate and maintain biodiversity, we know remarkably little about the drivers of parasite diversity. To identify the mechanisms governing parasite diversity, we combined surveys of 8100 amphibian hosts with an outdoor experiment that tested theory developed for free-living species. Our analyses revealed that parasite diversity increased consistently with host diversity due to habitat (i.e. host) heterogeneity, with secondary contributions from parasite colonisation and host abundance. Results of the experiment, in which host diversity was manipulated while parasite colonisation and host abundance were fixed, further reinforced this conclusion. Finally, the coefficient of host diversity on parasite diversity increased with spatial grain, which was driven by differences in their species–area curves: while host richness quickly saturated, parasite richness continued to increase with neighbourhood size. These results offer mechanistic insights into drivers of parasite diversity and provide a hierarchical framework for multi-scale disease research.

Keywords

Biodiversity, biogeography, dilution effect, disease ecology, emerging disease, macroecology.

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INTRODUCTION

Why biodiversity is concentrated in some areas and not others remains one of the most central questions in community ecology, for which three explanations are often advanced (Connell 1978; Rosenzweig 1995; Allouche *et al.* 2012). First, species richness may increase with *resource availability*, as suggested by positive correlations between productivity and species richness (Waide *et al.* 1999; Mittelbach *et al.* 2001; Jetz *et al.* 2009; but see Adler *et al.* 2011). Increased energy availability is hypothesised to increase opportunities for ‘species packing’ while decreasing the risk of local extinction by increasing population size (MacArthur 1970). Second, some habitats may support a large number of species because they offer more *colonisation opportunities*, due to large patch size or close proximity to a propagule source (i.e. island biogeography theory, MacArthur & Wilson 1967; and neutral theory, Hubbell 2001). Finally, *resource heterogeneity* can promote coexistence and species richness by offering a greater diversity of niches, thereby limiting interspecific competition (habitat-heterogeneity hypothesis, Hutchinson 1959; MacArthur & MacArthur 1961; Rosenzweig 1995). For instance, variation in both structural habitat complexity and the diversity of prey resources have been strongly linked to consumer diversity (Tews *et al.* 2004). Collectively, these three factors have been shown to shape patterns of species richness across a wide range of taxa (Tews *et al.* 2004; Jetz *et al.* 2009), although consensus on

their relative importance and potential interactions has remained elusive (Venail *et al.* 2008; Allouche *et al.* 2012; Chase & Knight 2013).

Despite nearly a century of empirical and theoretical research devoted to the study of free-living biodiversity, we know considerably less about parasite species richness and the factors underlying its variation. As written by Dunn *et al.* (2010), ‘The fact that warbler species distributions are better understood than the distribution of human pathogens is a gap that clearly deserves research attention’. This knowledge gap is even greater for parasites of wildlife, and has recently taken on added urgency in light of increased disease emergence across a range of non-human hosts (Fisher *et al.* 2012; Smith *et al.* 2014). One of the most frequently cited correlates of parasite diversity is the richness of host species (i.e. the ‘host-diversity-begets-parasite-diversity hypothesis’; Hechinger & Lafferty 2005; Thieltges *et al.* 2011; Poulin 2014). Given the intimate association between hosts and parasites as well as the expectation that many parasites are specialised to infect a small number of host species (Poulin 2014), an increase in host species richness would be expected to increase parasite diversity. For example, in a recent meta-analysis of 21 published studies, Kamiya *et al.* (2014) reported a consistent, positive correlation between host and parasite species richness, independent of parasite type or host taxon. Similarly, Dunn *et al.* (2010) reported that bird and mammal richness were strong, positive predictors of the diversity of zoonotic

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parasites at the country level. Nonetheless, the ecological mechanisms that drive this pattern have rarely been explored – what are the processes that generate and maintain the correlation between host diversity and parasite diversity? While it is often assumed to stem from niche diversification alone, this explanation overlooks alternative mechanisms, such as changes in resource availability (e.g. host abundance) and colonisation opportunities, both of which could correlate cryptically with host diversity in nature.

Building from community ecology theory, we can develop testable predictions to assess the contributions of hypothesised drivers of parasite diversity, such as colonisation opportunities, resource availability and resource heterogeneity. For instance, if parasite diversity is driven primarily by changes in resource availability (i.e. the diversity–productivity hypothesis [Poulin 2014]) – analogous with instances in which free-living diversity and productivity covary (Waide *et al.* 1999; Jetz *et al.* 2009) – host biomass or abundance should be a better predictor of parasite richness than host diversity, assuming host abundance and diversity are not overly collinear (Stevens & Carson 2002) (Fig. 1a). Decomposing community diversity (or γ diversity) into the sum of average richness per patch (α diversity) and among-patch variation (β diversity) can offer further insights into the structuring processes (Lande 1996; Gering & Crist 2002). If increased host diversity provides more opportunities for parasite colonisation (i.e. the propagule-pressure hypothesis; Levine 2000), as might occur when newly arriving hosts bring generalist parasites that spread to established hosts, then host diversity should correlate most strongly with average parasite richness per host, or *parasite α diversity*. Thus, increases in host diversity drive an overall increase in parasite richness in all host species, rather than by adding specialised parasites infectious to only one or a few host species (Fig. 1b). Finally, if the key determinant of parasite diversity is variation in niche opportunities (i.e. the habitat-heterogeneity hypothesis), host diversity should correlate most strongly with *parasite β diversity*, or the among-host diversity in parasite species. In this scenario, the overall richness of parasites within the community will increase owing to greater variation in parasite species composition among different host species, even while the average richness of parasites per host species remains constant (Fig. 1c).

Changes in the form or magnitude of the host-diversity–begets-parasite-diversity relationship across scales can offer further insights into the underlying drivers. Previous research in community ecology has highlighted the importance of both biological and spatial scale in determining the influence of putative biodiversity drivers (e.g. Levin 1992; Scheiner *et al.* 2000; Cadotte & Fukami 2005; Chase & Knight 2013). For instance, while free-living species richness often increases linearly with productivity at regional scales (i.e. for γ diversity), this relationship becomes unimodal at local scales (i.e. for α diversity) due to increased heterogeneity in species composition among sites (Chase & Leibold 2002). How the link between host and parasite diversity varies across biological levels (e.g. individual, population, community) remains uncertain, but there are many reasons to expect the factors influencing parasite richness at the individual host level differ from those affecting richness at the host community level. Similarly,

the strength or slope of the host–parasite diversity correlation could vary with spatial grain for several reasons. First, if parasite species are more spatially aggregated than their hosts, increases in spatial grain will likely yield greater numbers of parasite species per host species, increasing the slope between host and parasite richness. Alternatively, the slope may decrease with spatial grain if the local-scale interactions (i.e. host–parasite and parasite–parasite interactions) driving the relationship are swamped by processes operating at larger scales (e.g. climatic gradients, biogeographical history) (Fridley *et al.* 2007). To date, however, the challenge of quantifying host and parasite diversity across multiple scales has limited opportunities to test the mechanistic links underlying this relationship.

Here, we use a hierarchically nested dataset involving 8100 individual amphibian hosts representing 696 host populations and 424 host communities across a 758 100-ha region to systematically evaluate the mechanisms and scale dependence of the diversity–begets–diversity relationship. Specifically, we asked the following questions: (1) What drives the positive covariance between host and parasite diversity? And (2) how does this relationship change with scale, including the biological level of organisation (hosts, populations and communities) and spatial grain (ponds, parks and metacommunities)? For consistency with previous work, here we use parasite richness as our proxy for diversity, although we acknowledge the importance of additional metrics such as evenness, genetic diversity and functional diversity (e.g. Ostfeld & Keesing 2012). Adopting analytical approaches developed for free-living community ecology, we contrasted the individual and joint contributions of colonisation (propagule hypothesis), resource availability (diversity–productivity hypothesis) and resource heterogeneity (habitat-heterogeneity hypothesis) (Fig. 1). We complemented our observational data with a mesocosm experiment that tested how variation in host diversity affected parasite α , β and γ diversity while controlling host abundance and parasite colonisation. The current work also helps form a bridge between scale-dependent approaches in parasitology (e.g. Bush *et al.* 1997) and diversity-partitioning methods applied to free-living communities (e.g. Gering *et al.* 2003).

MATERIALS AND METHODS

Field sampling

To quantify the richness of host and parasites species across natural ecosystems, we sampled pond communities in the East Bay region of central California between 2009 and 2014 (see Fig. S1 in Supporting Information). We defined the community as the assemblage of larval amphibian species within a pond. Each pond was sampled two times per year using visual encounter surveys, standardised dip-net sweeps and seine hauls to ensure adequate detection of all amphibian host species (Johnson *et al.* 2013c; Richgels *et al.* 2013). We focused on larval amphibians because this developmental stage is the most reliable indicator of breeding activity and the period when many water-borne infections are acquired. Sampling amphibians as they reached metamorphosis – rather than as

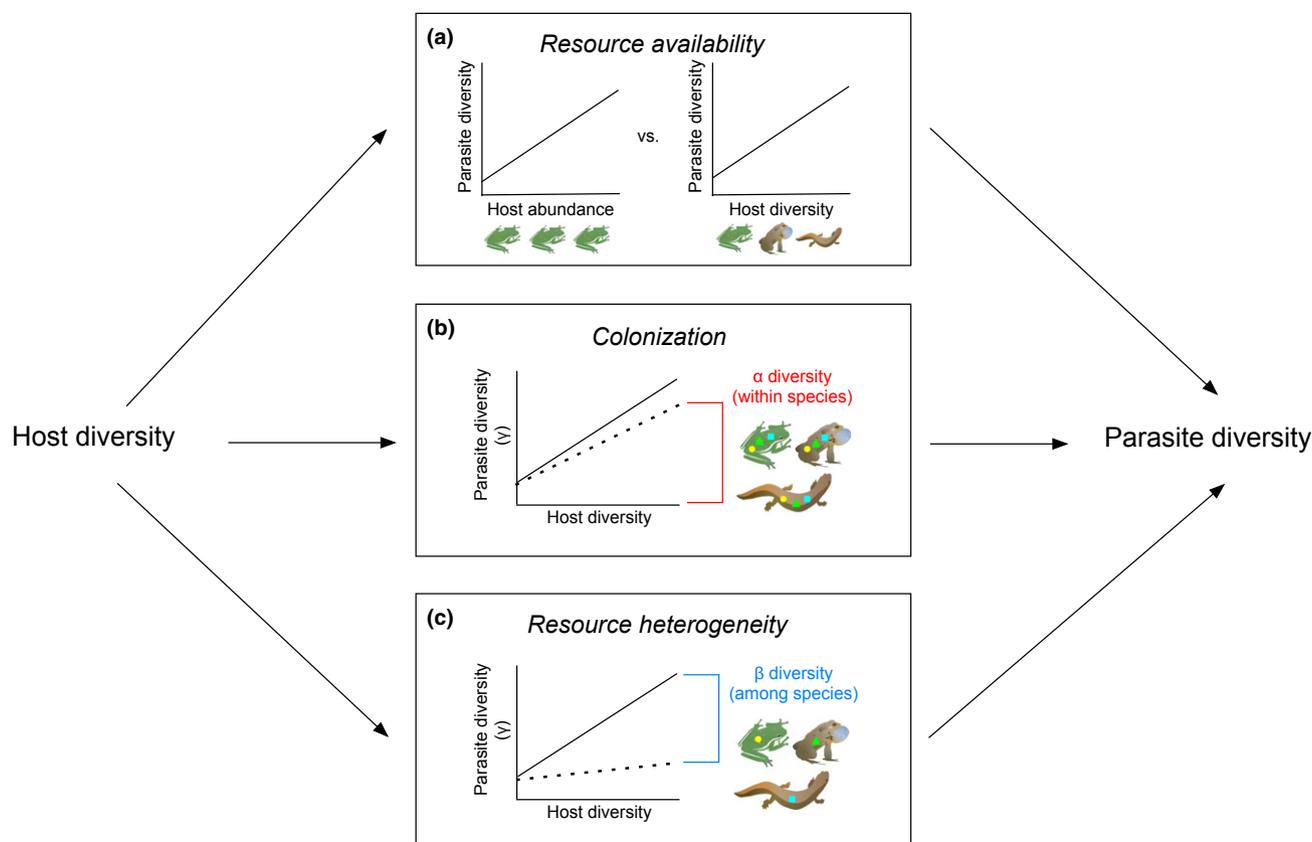


Figure 1 Host diversity can influence parasite diversity through at least three possible mechanisms. (a) First, the relationship may be driven by changes in *resource availability* (the diversity–productivity relationship), which might be expected if increased host richness correlates with an increase in total host species abundance. In this scenario, the total abundance or biomass of hosts should be a better predictor of parasite richness than is host species richness. (b) Second, variation in *parasite colonisation* (the propagule-pressure hypothesis) may underlie the correlation between host and parasite diversity. If newly arriving host species are accompanied by generalist parasites that spread to already established hosts, we would predict that increases in host diversity should correlate with an increase in the average number of parasite species per host (α diversity), regardless of the identity of that host species. (c) Alternatively, the diversity covariance between hosts and parasites may emerge from changes in *resource heterogeneity* (the habitat-heterogeneity hypothesis). Here, new host species represent novel habitats for parasites that otherwise could not establish, which would lead to a stronger relationship between host diversity and the among-host diversity in parasite species composition (β diversity). Thus, parasite diversity is increasing only at the level of the host community, rather than within individual hosts or host populations.

more mobile adults – also helped to ensure that infections reflected conditions of the system in which the animal was collected. To measure parasite richness, we collected a subsample (10–15) of each host species (excluding federally protected species) as they approached metamorphosis, and performed a systematic examination of all major tissues and organs for parasites (Hartson *et al.* 2011). These included 22 taxa of larval and adult digeneans, nematodes, cestodes, acanthocephalans and protists (Table S1). Based on previous rarefaction analyses, these techniques were effective in quantifying both amphibian and parasite species richness at the saturation of their richness–sampling effort curves (Johnson *et al.* 2013a,b). This approach allowed us to evaluate how the richness of hosts affected parasite richness at multiple, nested levels, including within individual hosts, across host populations and among amphibian communities (i.e. ponds). These scales parallel the oft-used levels from parasitology: infracommunity richness, component community richness and compound community richness (Bush *et al.* 1997).

Mesocosm experiment

To better understand the *mechanisms* linking host and parasite diversity, we performed a mesocosm experiment in which we manipulated the diversity of amphibian hosts (1 vs. 4 species) while maintaining the same total number of host individuals (40) and the same propagule input of larval trematodes (4 species). These richness values are broadly representative of the study system; among 424 host communities, the average \pm 95% CI for host and parasite richness was 2.80 ± 0.095 (range: 1–6) and 4.10 ± 0.199 (range: 0–12), respectively. Our goal was to examine how shifts in host species richness affected the average parasite richness per host (α diversity), the among-host variation in parasite richness (β diversity) and the cumulative parasite richness per mesocosm (γ diversity). Mesocosms (378 L) were filled with well water and seeded with sediment, zooplankton and algae using standard methods (Johnson *et al.* 2013b). In the low-host-diversity treatment, we added 40 larvae of *Pseudacris regilla*, the most commonly

encountered amphibian species in the region; in the high-host-diversity treatment, we added 10 individual larvae of *Pseudacris regilla*, *Taricha torosa*, *Anaxyrus americanus* and *Lithobates catesbeianus*, which collectively comprise the four most common, non-endangered amphibian species in the study region. To obtain infectious parasites, we collected snails (*Helisoma trivolvis*) infected with different larval trematodes (*Ribeiroia ondatrae*, *Echinostoma* spp., *Alaria* spp. and *Cephalogonimus* spp.) and added between 694 and 11 141 parasite cercariae of each species over a 12-day period (see Johnson *et al.* 2013b). Each of these parasites is a generalist capable of infecting a wide range of different amphibians, although their infection success is expected to vary by host (Johnson *et al.*

2013b). Two weeks after initiating the experiment, we measured each host and quantified parasite infection.

Analysis

We analysed the relationship between amphibian and parasite diversity in nature at three biological levels of organisation: the host individual, for which we used parasite richness within individual hosts as the response; the host population, for which we used the total number of parasite species within all individuals of a given host species in a given pond; and the host community, for which we used the total richness of parasites across all host species in the same pond (Fig. 2). For

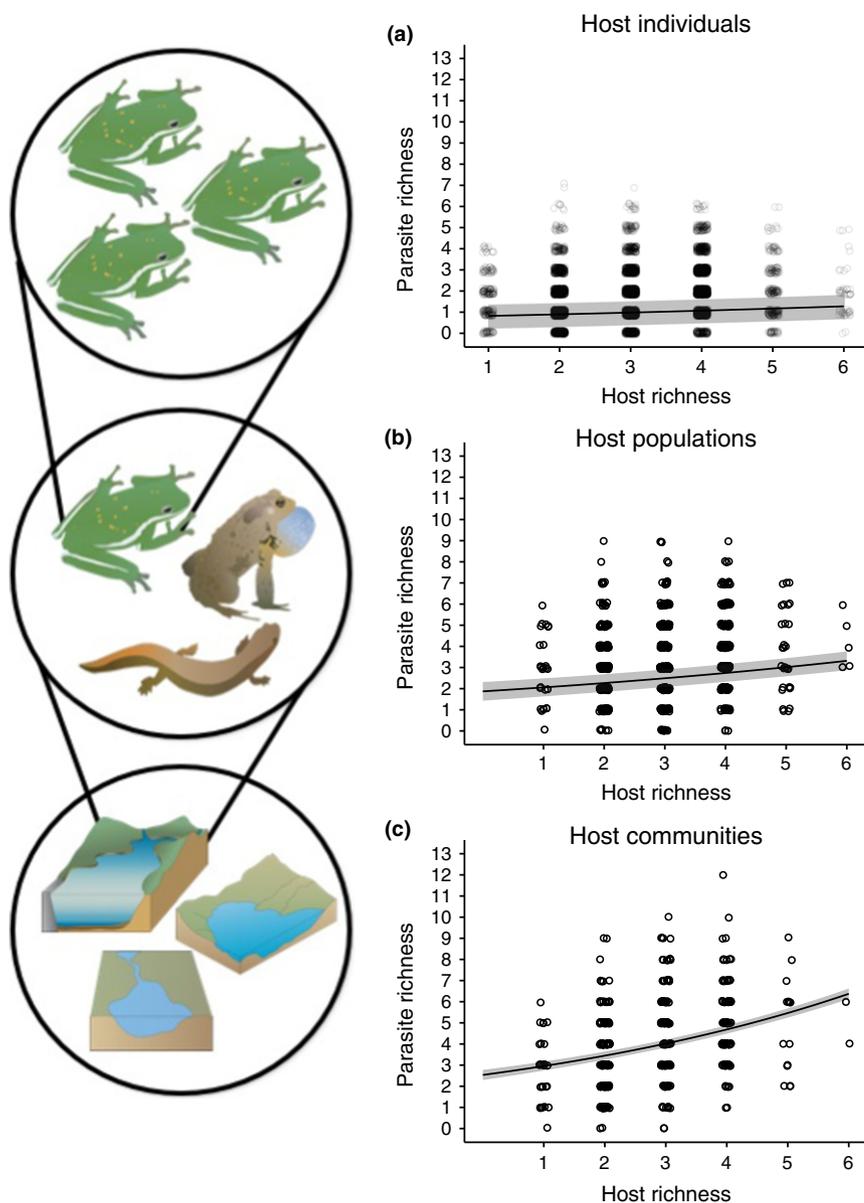


Figure 2 Effects of host diversity on parasite richness within individual hosts, host populations and host communities. For each biological scale, the richness of amphibian host species detected at the pond level was positively related to the taxonomic richness of parasites. (a) Host scale: coefficient = 0.088, conditional $R^2 = 0.47$, $P < 0.0001$, $n = 8100$; (b) Population scale: coefficient = 0.095, conditional $R^2 = 0.43$, $P = 0.0002$, $n = 696$; (c) Community scale: coefficient = 0.151, conditional $R^2 = 0.31$, $P < 0.00001$, $n = 423$.

each, we used generalised linear mixed models (GLMMs) with parasite richness as a Poisson-distributed response, host species richness in the pond as a fixed effect, and wetland, sample year and host species as random effects (depending on the level of the analysis). To subsequently evaluate the ecological drivers of detected relationships between host and parasite diversity, we ran the same GLMM described above but added a level-dependent measure of *host resource availability* as an additional fixed effect. At the individual host level, we used host body size (snout–vent length in mm) as an indicator of resource availability; at the population level, we multiplied the average number of hosts of a given species per dip-net sweep by the pond perimeter as a proxy for population abundance; at the community level, we multiplied the total number of hosts per sweep by pond perimeter as a proxy for community abundance. The population and community measures of host availability were \log_{10} transformed to help normalise their distributions. We used perimeter rather than pond area because larval amphibians tend to occur in the littoral rather than pelagic zone, although results are comparable with either variable. After verifying that host diversity and host resource availability were not collinear, we compared their coefficient estimates at each scale and evaluated their statistical significance.

To evaluate the roles of *parasite colonisation* and *niche heterogeneity* in driving covariation between host and parasite diversity, we applied diversity-decomposition methods by examining how host diversity affected the subcomponents of parasite diversity: the within patch or α diversity, the among-patch or β diversity and their sum (or product), which is γ diversity (Whittaker 1960; Lande 1996; Gering & Crist 2002; Gering *et al.* 2003). If host diversity affects parasite diversity predominantly through changes in colonisation opportunities (propagule-pressure hypothesis), the strongest effects should manifest on parasite α diversity. If, instead, the mechanism involves changes in niche heterogeneity, such that new hosts represent novel habitats for parasites, the effect of host diversity should be mediated through β diversity, or among-host species diversity.

We applied these additive partitioning methods at two biological levels: from individual hosts to populations (i.e. $\gamma_{\text{population}} = \bar{\alpha}_{\text{host}} + \beta_{\text{host}}$), and from host populations to host communities (i.e. $\gamma_{\text{community}} = \bar{\alpha}_{\text{population}} + \beta_{\text{population}}$). Thus, $\bar{\alpha}_{\text{host}}$ is the average parasite richness per host individual of species i , whereas $\bar{\alpha}_{\text{population}}$ is the average parasite richness per host species population within pond j . Following Lande (1996), we used the additive approach to estimate β (i.e. $\beta_i = \gamma_i - \alpha_i$) and complemented this by also estimating Jaccard's dissimilarity index. While Jaccard's index offers a measure of β diversity that is independent of γ , it requires exclusions of any hosts or populations with no parasites, forcing the loss of biologically meaningful data. Because $\gamma_{\text{population}} = \alpha_{\text{population}}$, we can combine these equations together to assess the cross-scale contributions of host diversity: $\gamma_{\text{community}} = \bar{\alpha}_{\text{host}} + \beta_{\text{host}} + \bar{\beta}_{\text{population}}$ (Gering & Crist 2002; Gering *et al.* 2003). To evaluate how host diversity affected parasite α and β diversity at the host and population levels (averaged values), we used linear mixed models with a fixed effect of host richness; at the host level, we included random effects

for host species, pond and year; and at the population level, we included random effects of site and year.

Finally, to evaluate the influence of spatial grain, we used a neighbourhood-based approach that combined communities together into progressively larger neighbourhoods of connected sites and assessed resultant changes in the relationship between host and parasite species richness. We considered 100 evenly spaced buffer distances ranging from 0 km, in which each pond was an independent community, to 15 km, when all wetlands fell into one of two neighbourhoods – the minimum number necessary to compare host and parasite richness. For each buffer distance, we recalculated parasite and host richness aggregated at the neighbourhood scale and fit a Poisson generalised linear model for the effect of host richness on parasite richness. We then compared how host and parasite richness changed with buffer distance (i.e. the species–area curve of each), as well as how the coefficient of the host–parasite diversity relationship varied with buffer distance.

RESULTS

Over 5 years, we sampled 198 unique ponds, many of which were sampled across multiple years (424 site-by-year combinations). We encountered six amphibian species (*Ambystoma californiense*, *Anaxyrus boreas*, *Lithobates catesbeianus*, *Pseudacris regilla*, *Rana draytonii* and *Taricha torosa*) and 22 parasite taxa, including larval trematodes ($n = 8$), adult trematodes ($n = 4$), nematodes ($n = 5$), acanthocephalans ($n = 1$), protists ($n = 3$) and cestodes ($n = 1$). Among sites, host richness varied from 1 to 6 (average ± 1 SE = 2.79 ± 0.048), whereas parasite richness ranged from 0 to 12 (average ± 1 SE = 4.08 ± 0.101). Host species richness was a consistent, positive predictor of parasite richness; however, the magnitude of this relationship varied with biological level of organisation, increasing progressively from individual hosts to host populations to host communities (Fig. 2). The coefficient of host diversity on parasite diversity at the community scale was $\sim 2\times$ that observed for individual hosts (Fig. 2). Host resource availability received marginal support as a predictor of parasite diversity at the host and population levels ($P = 0.089$ and 0.053 , respectively), but became a more important cocontributor at the community scale ($P = 0.004$) (Fig. 3).

Decomposition of parasite γ diversity into its component contributions from α and β illustrated the importance of resource heterogeneity over parasite colonisation in driving the hot-diversity-begets-parasite-diversity relationship. Although host diversity had positive effects on parasite α and β diversity at both the host and population scales (Fig. 4), the largest coefficient was for parasite $\beta_{\text{population}}$, which was $\sim 4\times$ the coefficient of host diversity on parasite $\bar{\alpha}_{\text{host}}$, β_{host} or $\bar{\alpha}_{\text{population}}$. Thus, while the increases in α diversity within individual hosts and populations suggested that changes in parasite colonisation helped explain the effects of host diversity on parasite richness, our overall results indicated that the strongest influence of host diversity was on parasite diversity at the among-host species level, consistent with predictions of the habitat-heterogeneity hypothesis. Comparable effects were observed when using Jaccard's dissimilarity index as the response

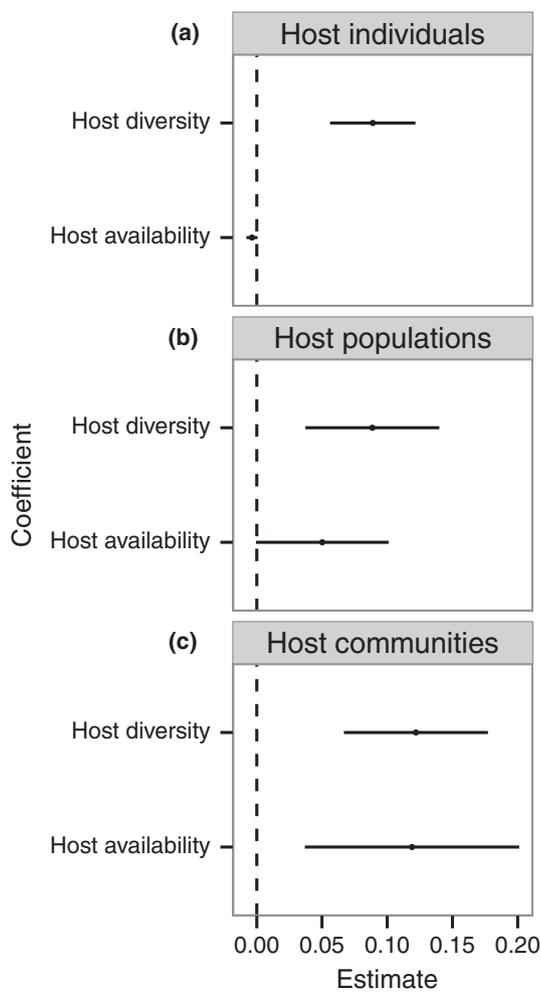


Figure 3 Assessing the roles of resource availability vs. resource heterogeneity on parasite diversity at the (a) host, (b) population and (c) community scales. Depicted are the coefficient estimates \pm 95% of host availability and host richness when included together in regression models. Host availability was defined as host body size (snout–vent length) at the individual scale, as the \log_{10} -transformed abundance of hosts of a given species at the population level and the \log_{10} -transformed total abundance of all host species at the community level. While host richness was a positive predictor of parasite richness across all three scales, host availability was a cocontributor only at the community scale.

variable. By recalculating $\gamma_{\text{community}} = \bar{\alpha}_{\text{host}} + \bar{\beta}_{\text{host}} + \bar{\beta}_{\text{population}}$, we found that the proportional contribution of $\beta_{\text{population}}$ to $\gamma_{\text{community}}$ increased monotonically with host diversity, from an average of 23.4% at low host diversity (1–3 species) to \sim 47% at high host diversity (4 or more species).

Results of the mesocosm study indicated that increases in host richness from one to four species had no effect on parasite richness at the community level (γ diversity); indeed, in all but one mesocosm, each administered parasite species established successfully, regardless of the host community ($t = 1.00$, $P = 0.331$; $n = 40$), consistent with the generalist nature of these parasites. Similarly, there was no effect of host richness on parasite richness per host individual after accounting for host species as a random effect (Poisson GLMM; coefficient \pm 1 SE = 0.117 ± 0.083 , $z = 1.418$, $P = 0.156$).

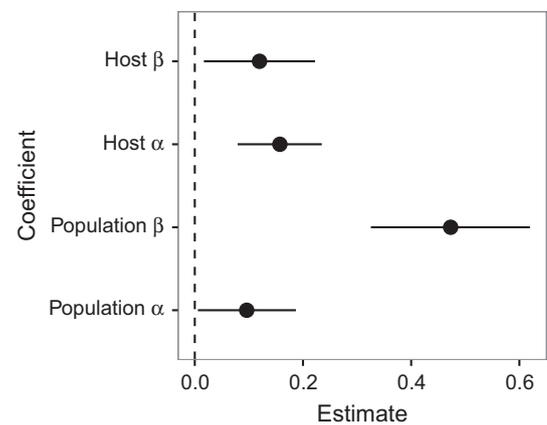


Figure 4 Diversity decomposition analysis of the host-diversity-begets-parasite-diversity relationship. Host diversity had positive but variable effects on the average richness of parasites per host ($\bar{\alpha}_{\text{host}}$; coefficient = 0.157, $P = 0.0008$), the among-host individual variation in parasite composition (β_{host} ; coefficient = 0.119, $P = 0.019$), the average richness of parasites per host population ($\bar{\alpha}_{\text{population}}$; coefficient = 0.096, $P = 0.035$), and especially the among-host population variation in parasite composition ($\beta_{\text{population}}$; coefficient = 0.423, $P < 0.0001$). Depicted is the coefficient estimate \pm 95% confidence interval of host diversity on parasite diversity as determined through linear mixed-effects models.

However, $\bar{\alpha}_{\text{host}}$ decreased from an average of 2.446, 95% CI: (2.346, 2.545) at low host diversity to 2.123, CI: (2.013, 2.233) at high host diversity. Similarly, β_{host} decreased from 1.454, CI: (1.244, 1.664) to 0.923, CI: (0.793, 1.053) between the low- and high-host-diversity treatments, respectively. Because total parasite richness was unaffected by host diversity (i.e. $\gamma_{\text{community}}$ was constant), these decreases led to a concurrent increase in $\beta_{\text{population}}$, which was fixed at 0 in the low-diversity treatment (i.e. there was only 1 host population), but increased to 0.95, CI: (0.79, 1.11) when host richness was high (\sim 24% of parasite $\gamma_{\text{community}}$). These values were similar in magnitude to field observations, for which $\beta_{\text{population}}$ increased from zero in single-host species communities to \sim 21% of gamma diversity when four host species were present. Results were comparable if we used Jaccard's dissimilarity index, which is unconstrained by gamma diversity, for which compositional dissimilarity increased from 0.22 to 0.44 between the treatments (GLM; coefficient \pm 1 SE = 0.217 ± 0.023 , $t = 9.65$, $P < 0.0001$), which compared with field observations of 0.245 and 0.433 at low (1–3) and high (4–6) host species. Within *P. regilla* specifically, which was the only host species shared between the low- and high-host-diversity treatments, there was no effect of host diversity on either parasite richness per host individual or Jaccard's dissimilarity index ($P = 0.17$), further emphasising the role of among-species variation in driving the observed results. Host survival among mesocosms averaged \sim 97% and was unrelated to treatment.

With respect to spatial scale, increasing the size of the spatial neighbourhoods in our field survey led to nonlinear increases in the estimated coefficient between host and parasite diversity (Fig. 5). Initial increases in buffer distance (e.g. 0–5 km) tended to connect ponds within the same park and led to an increase in estimated coefficient from 0.206, CI:

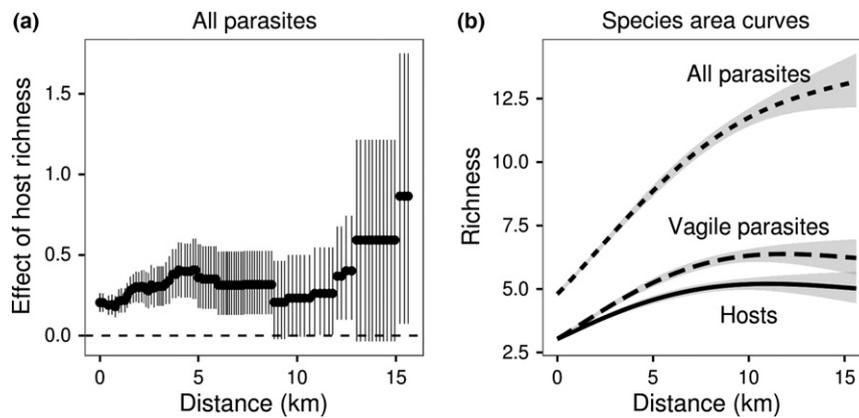


Figure 5 Influence of spatial grain and neighbourhood size on the relationship between host and parasite diversity. (a) Increases in neighbourhood size tended to nonlinearly increase the coefficient of the relationship between host and parasite richness; error bars represent the 95% CI of the estimate, for which sampling was done without replacement (i.e. sample size decreases as spatial grain is increased). (b) Species–area curves for hosts, highly vagile parasites (e.g. larval trematodes that have avian and mammalian definitive hosts, Table S1), and all parasites as a nested function of buffer distance.

(0.148, 0.263) to 0.351, CI: (0.166, 0.555). At distances between 5 and 10 km, there was little change in the number of neighbourhoods or the coefficient estimate, in large part because the minimum distance between parks averaged ~ 7 km and this increase in buffer led to few changes in connectivity. Further increases in buffer distance (> 10 km) connected parks together (i.e. into metacommunities) and led to additional increases in the coefficient (Fig. 5a), although the 95% confidence intervals also included zero due to the decrease in sample size as neighbourhoods became larger (i.e. each pond was assigned to only one neighbourhood without replacement). Importantly, the species–area curves for hosts and parasites revealed that host richness increased much more slowly than parasite richness and reached a plateau at a lower buffer distance, such that progressive increases in spatial grain disproportionately added new parasites rather than new hosts (Fig. 5b). Correspondingly, a Mantel test indicated that parasite community dissimilarity increased with geographic distance ($r = 0.06$, $P = 0.006$), whereas no such relationship was detected for hosts ($r = 0.011$, $P = 0.331$) or when selecting only parasites with highly mobile definitive hosts ($r = -0.015$, $P = 0.73$), such as birds and mammals.

DISCUSSION

By applying theory derived for free-living communities to a hierarchically nested dataset of host and parasites, our analyses revealed that host diversity is a consistent, positive predictor of parasite diversity, but that the strength of this relationship and the underlying mechanisms varied with both the biological level of organisation and the spatial scale. The magnitude of the coefficient between host and parasite diversity increased threefold as the level of biological organisation moved from individual hosts to host communities. Similarly, the coefficient increased as we connected communities into progressively larger metacommunities and recalculated their cumulative richness, emphasising both the generality of the diversity–begets–diversity relationship, as well as its scale dependence. The patterns observed are consistent with the idea that niche heterogeneity causes covariance between host

and parasite diversity (i.e. our results support the habitat–heterogeneity hypothesis), as indicated by the strong relationship between host diversity and parasite β diversity at the population scale. However, both colonisation opportunities and resource availability in the form of total host abundance also contributed to the effects of host diversity on parasite diversity, demonstrating that multiple mechanisms explain the diversity covariance between hosts and parasites.

Although host diversity has often been identified as a significant correlate of parasite diversity (e.g. Kamiya *et al.* 2014), the ecological mechanisms that drive this pattern are not readily apparent. The positive correlation between host and parasite diversity is frequently assumed to emerge from the fact that communities with higher host diversity offer additional niches in the form of more host species, especially for parasites that are highly host specific or require multiple hosts to complete their life cycles (Hechinger & Lafferty 2005; Poulin 2014). However, ecological theory developed for free-living species has shown that, alongside resource heterogeneity, species diversity is also influenced by both propagule pressure and total resource availability (Rosenzweig 1995; Levine 2000; Jetz *et al.* 2009; Stevens *et al.* 2012; Haegeman & Loreau 2014). Our analyses revealed that each of these factors contributed to the observed changes in parasite diversity, but to different degrees and at different biological scales. Comparisons of host availability and host richness as predictors suggested that host richness was the primary driver of parasite richness, particularly at the host and population levels. Host availability, which can influence parasite richness both through the amount of habitat available for parasite colonisation and by sustaining larger parasite populations, became a significant contributor only at the community scale. Use of the diversity–decomposition approach allowed us to further assess whether the effects of host diversity on parasite richness were mediated through changes in parasite colonisation or niche heterogeneity. The positive and consistent relationship between host diversity and parasite α diversity is consistent with an influential role of colonisation with progressive increases in host richness, which might stem from colonisation by generalist parasites as new host species are added or from

increased visitation by transport hosts such as bird or mammals at high-diversity sites (e.g. Hechinger & Lafferty 2005). However, the strongest effect of host diversity was on parasite $\beta_{\text{population}}$ diversity (i.e. among species), for which the coefficient of host diversity was 3–4 \times greater than those for other responses. This suggests that niche variation in the form of added host species was the dominant influence on the link between host and parasite richness, consistent with the habitat-heterogeneity hypothesis and an overall emphasis in community ecology on heterogeneity as a driver of biodiversity. Tews *et al.* (2004), for instance, reported that 85% of studies published between 1960 and 2003 found significant and positive effects of heterogeneity on the richness of taxa ranging from microbes to mammals.

Results of the mesocosm experiment provided additional support for the role of host heterogeneity in driving parasite diversity. By holding constant both host resource availability (total host abundance) and parasite colonisation (propagule pressure), we found that the only effects of increasing host richness were on parasite β diversity, or the among-host-species differences in parasite community composition. Increases in host richness from one to four species had no effect on either the average parasite richness per host individual (α diversity) or the total number of parasite species within each mesocosm (γ diversity). The levels of β diversity observed at high host richness, whether measured as the difference between γ and α or as Jaccard's dissimilarity index, were comparable to those observed in natural systems at similar diversity levels. Given the complexities inherent to manipulating host and parasite communities simultaneously, here we used a relatively small yet realistic number of generalist parasites and maintained a constant total number of hosts, but we highlight the importance of subsequent efforts to concurrently vary host evenness, host abundance and include a mixture of specialist and generalist parasites. Our results are also consistent with those of the only two previous experimental studies of the diversity-begets-diversity relationship for parasites. As part of the Jena Biodiversity Experiment, experimental increases in plant species richness and functional diversity enhanced both the richness and prevalence of colonising pathogens (Scherber *et al.* 2010; Rottstock *et al.* 2014), although whether these effects stemmed from changes in host heterogeneity alone or a combination of mechanisms was not assessed.

The strength of the relationship between host and parasite diversity also tended to increase with spatial aggregation. Thus, as ponds were connected together into parks and metacommunities, the coefficient of host diversity on parasite diversity increased in magnitude. This increase was nonlinear with respect to buffer distance, largely reflecting the uneven distributions of accessible ponds across the landscape. Based on the species–area curves for hosts and parasites, this amplification of the coefficient emerged from the fact that parasite richness increased more rapidly with increasing buffer distance and saturated more gradually than did host richness. This likely stemmed from several factors. First, parasite species richness is often greater than host species richness, as observed here, and parasites tend to be more spatially aggregated than hosts, leading to stronger increases in parasite richness as larger areas are sampled. Moreover, because dispersal

limitation is more likely to manifest at local scales (i.e. parasites cannot instantaneously colonise all suitable host habitats), the transition from local to landscape scales is expected to narrow the gap between the potential and realised niches of parasites. Depending on the mode of parasite dispersal, for instance, there will probably be temporal lags between stochastic local extinctions of parasites and their successful recolonisation, particularly at fine spatial scales. Indeed, for highly vagile parasites, such as larval trematodes with avian or mammalian definitive hosts (Table S1), parasite richness saturated much more quickly with buffer distance than for all parasites combined, highlighting the influence of dispersal limitation (Fig. 5b and Fig. S2). This result contrasts with previous findings suggesting that the slopes of species–area curves are steeper for large-bodied organisms (Drakare *et al.* 2006), which could stem from the tendency of parasites to decouple the association between body size and trophic level observed among free-living species (Lafferty & Kuris 2002).

A major priority in disease ecology is to understand the diversity and distribution of parasites, including how these patterns change with scale. Our results offer insight into the mechanisms linking host and parasite species richness, as well as how this relationship changes across spatial scales from local communities to metacommunities and biological levels from individual hosts to host communities. This complements macroecological studies aimed at characterising parasite diversity at regional to biogeographic scales (Guernier *et al.* 2004; Dunn *et al.* 2010; Smith *et al.* 2014), for which the influence of evolutionary history and diversification will also have a more prominent signal. Such findings also have immediate relevance for the ongoing debate about whether increasing free-living biodiversity reduces the transmission of infectious disease (e.g. Ostfeld & Keesing 2012; Randolph & Dobson 2012; Wood & Lafferty 2013). Resolution of this debate requires careful attention to both the scale involved (e.g. local to regional) and the epidemiological process (e.g. parasite colonisation vs. transmission) (Johnson *et al.* 2015). For instance, the richness of parasite species in an area is not equivalent to the risk of disease, which is often more closely aligned with the prevalence or load of an especially pathogenic infection. Our results indicate that increases in host species and the niches they provide allow for increases in parasite richness, particularly at higher biological scales and coarser spatial grains. Previous work has shown how changes in host richness can depress local transmission of parasites and reduce host pathology (e.g. Ostfeld & Keesing 2012; Johnson *et al.* 2013c). Thus, quantifying the concurrent and scale-dependent effects of host diversity on parasite colonisation as well as transmission represents an important area of future research for understanding the net effects of diversity on disease.

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AUTHOR CONTRIBUTIONS

PTJJ designed the study, coordinated data collection and drafted the manuscript; CLW contributed to the project design and manuscript development; MBJ assisted with data analysis and analytical framework; DLP coordinated the mesocosm experiment; SEH and YPS contributed to the project design and analytical framework. All authors contributed to editing of the manuscript.

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