REVIEW AND SYNTHESIS

Host dispersal as the driver of parasite genetic structure: a paradigm lost?

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Abstract

Understanding traits influencing the distribution of genetic diversity has major ecological and evolutionary implications for host–parasite interactions. The genetic structure of parasites is expected to conform to that of their hosts, because host dispersal is generally assumed to drive parasite dispersal. Here, we used a meta-analysis to test this paradigm and determine whether traits related to host dispersal correctly predict the spatial co-distribution of host and parasite genetic variation. We compiled data from empirical work on local adaptation and host–parasite population genetic structure from a wide range of taxonomic groups. We found that genetic differentiation was significantly lower in parasites than in hosts, suggesting that dispersal may often be higher for parasites. A significant correlation in the pairwise genetic differentiation of hosts and parasites was evident, but surprisingly weak. These results were largely explained by parasite reproductive mode, the proportion of free-living stages in the parasite life cycle and the geographical extent of the study; variables related to host dispersal were poor predictors of genetic patterns. Our results do not dispel the paradigm that parasite population genetic structure depends on host dispersal. Rather, we highlight that alternative factors are also important in driving the co-distribution of host and parasite genetic variation.

Keywords

Animals, coevolution, co-structure, gene flow, host–parasite interactions, local adaptation, meta-analyses, migration rate, plants, population genetics, trait diversity.

Ecology Letters (2016) 19: 336–347

INTRODUCTION

Host–parasite interactions are fascinating and complex because each host and parasite combination generates a unique association of species traits. These unique trait associations then dictate the ecological and evolutionary dynamics of the interacting species, including community assembly rules (Krasnov et al. 2015), disease dynamics over space and time (Barrett et al. 2008), and coevolutionary trajectories (Greischar & Koskella 2007; Hoeksema & Forde 2008). However, the task of predicting evolutionary and ecological dynamics for pairs of taxa with differing biological traits – as is the case for host–parasite interactions – remains challenging.

Parasite and host traits related to effective population size, mutation, and gene flow all have the potential to affect the distribution and degree of standing genetic variation of each interacting organism (Gandon et al. 2002). This variation is required for selection to act and permits coevolution to occur (Lively 1999; Gandon 2002; Barrett & Schluter 2008). Indeed,

experimental and theoretical studies suggest that species traits which increase standing genetic variation will determine which partner is ahead in the coevolutionary arms race between hosts and parasites (Gandon et al. 1996; Gandon & Michalakis 2002; Morgan et al. 2005). For example provided that dispersal rate is not so high as to homogenise gene pools, the interacting species with the higher dispersal rate is generally predicted to be locally adapted to the other (Gandon et al. 1996; Gandon 2002; Gandon & Michalakis 2002).

Theoretical and experimental studies that investigate the relationships between species traits, genetic variation and coevolutionary potential suggest that it is possible to predict the coevolutionary outcome of a host–parasite interaction by directly comparing the spatial distribution of genetic diversity in each species (Kawecki & Ebert 2004; Blanquart et al. 2013). Consequently, a surge of recent studies aimed at comparing patterns of genetic diversity between host and parasite populations have been performed, notably by describing the population genetic structure of each protagonist (e.g. Dybdahl

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& Lively 1996; Geist & Kuehn 2008; Prugnolle *et al.* 2008; Bruyndonckx *et al.* 2010; Brouat *et al.* 2011; Pennings *et al.* 2011). These studies usually compare population genetic structures using indices of among-population genetic differentiation such as F_{st}, G_{st}, D_{Jost} or equivalents (Jost 2008; Holsinger & Weir 2009; Meirmans & Hedrick 2011). A global level of genetic differentiation can then be calculated over all sampling locations, and in this case, a low level of genetic differentiation is often interpreted as resulting from high dispersal rates among sampling locations, and/or high effective population sizes (Criscione 2008; Holsinger & Weir 2009). Alternatively, genetic structures can be compared by estimating the correlation coefficient between pairwise indices of genetic differentiation calculated between sampling locations of the host and the parasite (McCoy *et al.* 2005). A high and significant coefficient of correlation indicates spatial congruence between host and parasite population genetic structures, suggesting that host and parasite dispersal rates are strongly related to each other (Nieberding *et al.* 2008).

A major and common assumption of co-structure analyses stipulates that parasite dispersal typically depends on host movement (Price 1980; Jarne & Theron 2001; Barrett *et al.* 2008; Criscione 2008), implying (i) that strong congruence between host and parasite genetic structures should be the norm (i.e. a high level of co-structure) and (ii) that – everything else being equal – the level of global genetic differentiation should be higher in parasite populations (Jarne & Theron 2001). However, co-structure analyses to date have led to very contrasting results. As extreme examples, the tick *Ixodes uriae* was more strongly genetically structured at large spatial scales than its seabird host species *Rissa tridactyla* (McCoy *et al.* 2005), whereas the parasitic trematode *Philophthalmus* sp. displayed weaker genetic structure than its snail host *Zeacumantus subcarinatus*. These two examples generate opposite predictions regarding the adaptive potential of each partner and, in line with the high variability of empirical results from other systems, make it difficult to draw clear-cut predictions about co-evolutionary outcomes. These contrasting results are likely due to the great diversity of host and parasite life cycles and traits found in nature (Barrett *et al.* 2008) and call for a better understanding of how these traits interact to shape genetic co-structure.

Several biological traits may determine parasite population structure in addition to dispersal associated with a particular host. For example many parasites have complex life cycles such that opportunities for dispersal may increase with the number of hosts in the life cycle and lead to incongruence in genetic co-structure with one particular host type (Lively *et al.* 2004; Prugnolle *et al.* 2005; Louhi *et al.* 2010). Likewise, parasites with one or more free-living stages may use several dispersal pathways, unrelated to their hosts (e.g. water or wind), which can result in incongruence between host and parasite population structures (Biek & Real 2010). Shorter generation times are also predicted to lead to faster evolutionary rates (Price 1980; Ebert & Hamilton 1996), which should also influence spatial patterns of genetic differentiation (Huyse *et al.* 2005). Genetic differentiation among pairwise populations will also be affected by respective effective population sizes, and thus genetic drift (Wright 1950, 1965). This process can be particularly important for parasites when dispersal rates are low to moderate (Box 1). Finally, non-biological factors, such as the geographical extent of the study area and the historical context, can also affect spatial patterns of genetic co-structure (Bowler & Benton 2005). The analysis of co-structure should therefore be considered on the same spatial and temporal scale as that of local adaptation. To make robust predictions on the outcome of coevolutionary interactions, it is necessary to understand the relative role of these different factors on the degree of genetic co-structuring in host–parasite interactions (see Table 1 for an extended list of factors and specific predictions derived from Huyse *et al.* 2005; Barrett *et al.* 2008). However, to our knowledge, no study has yet quantitatively synthesised comparisons between host and parasite genetic structures to: (i) test whether parasite genetic structure is indeed constrained by host movements, and (ii) disentangle the relative contributions of different species traits in explaining the differences or similarities in co-structure.

In this study, we performed a meta-analysis of published data sets across a large spectrum of host–parasite interactions (Table S1) to gain insights into general patterns of genetic co-structure, and to explore potential factors affecting this co-structure. More specifically, we first tested whether or not parasites and hosts display similar global levels of genetic differentiation. Assuming that parasites frequently disperse with their hosts (Price 1980; Jarne & Theron 2001), and all else is equal, we would expect the global level of genetic differentiation to be similar for both species. However, we predict this will not often be the case as many biological traits related to both parasite and host species may drive the genetic structures of these interacting species (see above and Table 1). We next assessed the congruence (i.e. correlation) between pairwise indices of genetic differentiation of host and parasite populations; congruence should be weak and non-significant if parasite dispersal is related to species traits other than host dispersal ability, whereas the reverse is expected if parasites strongly rely on hosts to disperse. Finally, for both components of genetic structure (global indices of genetic differentiation and correlations in pairwise genetic differentiation), we quantified the relative contributions of several biological and non-biological factors (see Table 1 for details on these factors and associated predictions) that may explain differences or similarities among different biological systems, to draw general conclusions on the factors influencing the distribution of genetic variation in host–parasite systems.

METHODS

Data compilation

Published studies on genetic co-structure in host–parasite interactions were found on the ISI Web of Knowledge platform® combining the following key words: 'host, parasite, parasitoid, population, genetic, structure, co-structure'. The platform was last accessed 31 January 2015, and we retained studies that provided values for (i) the global level of population genetic differentiation for both the host and the parasite species and/or (ii) the correlation coefficient (from Mantel tests in most cases) between the pairwise genetic differentia-

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Box 1 Some methodological issues of biological importance for future studies of host–parasite genetic co-structure

Here, we highlight three methodological issues that we feel are important to consider for future studies; the importance of accounting for drift, the choice of genetic marker and the use of appropriate metrics of genetic differentiation (GD).

First, GD is the by-product of drift and dispersal, although GD is often assumed to largely reflect dispersal. However, the effect of drift on GD cannot be ruled out, notably when dispersal rates are low to moderate. We therefore urge evolutionary biologists to more systematically decompose GD into its drift and dispersal components. We notably emphasize the use of metrics that can be used to account for drift when measuring GD (Relethford 1991) or even quantify the relative influence of drift on GD (Serrouya *et al.* 2012). This second option could be insightful since both dispersal rate and population size (which alters drift effects) have been shown to be influential in predicting the outcome of co-evolutionary dynamics (Gandon & Michalakis 2002).

Second, we found that GD tended to be higher in hosts than in parasites for mitochondrial markers (mtDNA) than for nuclear markers (nucDNA), and that pairwise correlations between GDs were stronger for mtDNA than for nucDNA. mtDNA is largely non-recombinant, subject to relatively low mutation rates, and maternally inherited, contrary to nucDNA (Foitzik *et al.* 2009). These characteristics result in mtDNA having lower effective population sizes than nucDNA (1 Ne vs. 4 Ne), rendering it more sensitive to demographic changes (such as those observed in parasites, (Nyakaana & Arctander 1999; Pennings *et al.* 2011). This means that differences in the genetic structure of hosts and parasites may better reflect the effects of demographic processes when mtDNA is used, whereas it is likely that these effects are partially blurred (to the profit of dispersal) when nucDNA are used. In other words, each marker gives more or less weight to demographic and dispersal processes. We cannot recommend one or the other of the markers, but rather suggest care when interpreting results, notably when using the two marker types simultaneously (which is probably the best route).

Third, several indices of GD can be used for nucDNA and mtDNA (e.g. Nei-Fst, Gst, Jost-D), each of them having unique properties. There has been much debate on which index is best under which circumstances and what conclusions can be robustly drawn from each of them (e.g. Jost 2008; Meirmans & Hedrick 2011). Because Fst and Gst mathematically decrease with increasing polymorphism (Jost 2008), they might be poorly suited to compare the genetic structures of species that vary intrinsically in their levels of genetic diversity, which is often the case in hosts and parasites. Hence, the mathematical differences in the estimators of GD may lead to biased interpretations when comparing GD between hosts and parasites (for more details, see Jost 2008; Meirmans & Hedrick 2011). As recommended elsewhere (Huyse *et al.* 2005), we therefore strongly suggest using unbiased indices of GD such as Jost-D, F'st or G'st (see Jost 2008; Meirmans & Hedrick 2011) when analysing host–parasite genetic co-structure.

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tion of host and parasite (i.e. congruence between estimates of pairwise genetic differentiation). We also retained studies providing a biplot of the pairwise genetic differentiation of host and parasite, from which the congruence between the host and parasite could be calculated (using Mantel tests) by extracting raw data using the freeware PlotDigitizer 2.6.3© (SourceForge, DHI Group Inc, New York, New York, USA). Furthermore, the reference lists of all relevant studies were scrutinised to expand the database.

We gathered 39 scientific papers leading to 52 unique case studies (i.e. host–parasite combinations) from which an effect size on the global level of genetic differentiation between host and parasite species could be computed, and thirty unique case studies reporting the correlation coefficient between host–parasite pairwise genetic differentiation, which was the statistic used to calculate an effect size (see Table S1). The database covered parasites and hosts from various taxonomic groups including plants, protozoa, nematodes, trematodes, molluscs, acarina, insects, birds and mammals (see Table S1 for details). For several papers, data from more than one molecular marker was used to estimate genetic differentiation, and/or more than one index of genetic differentiation was computed for a same set of markers. In these cases, we reported the estimates of each comparison independently (i.e. several estimates were sometimes available for a single host–parasite combination). With respect to molecular markers,

mitochondrial and/or nuclear markers (in general microsatellites or allozymes) were typically used. This has implications for interpreting patterns of genetic differentiation as the inheritance mode and the mutation rates differ among marker types (e.g. mitochondrial markers are maternally inherited whereas nuclear markers are bi-parentally inherited, Box 1). Likewise, many studies discuss the underlying assumptions, disadvantages and advantages of the various indices that have been proposed to measure genetic differentiation (e.g. Jost 2008; Meirmans & Hedrick 2011). Some indices, such as Wright's F_{st}, are dependent on the genetic diversity within populations, whereas others are mathematically independent of genetic diversity (e.g. the D_{Jost}, Jost 2008); such differences need to be accounted for when comparing genetic structures among species. For these reasons, we reported the estimates for each marker type and/or each index of genetic differentiation independently (Box 1). Overall, this lead to a database of 97 entries (i.e. lines of raw data) for the global level of genetic differentiation of host and parasite populations, and 36 entries for the pairwise genetic differentiation of host and parasite populations (Table S1).

To provide a general description of the database, we first examined two variables associated with each entry: (i) the marker type used to calculate genetic differentiation (nuclear or mitochondrial), and (ii) the parasitic mode (parasitoid, nest-, social-, endo- or ecto-parasite). Classification into a par-

Table 1 Description of explanatory variables associated with host and parasite traits that might influence the global level of genetic differentiation of hosts and parasites (lnRR) and the congruence in pairwise genetic differentiation of hosts and parasites (Zr). For discrete variables, the different categories are described. For each variable, we provide verbal ('General predictions') and statistical ('Model predictions') expectations for lnRR and Zr. Predictions were drawn from the null assumption that parasites do not rely on their host to disperse.

Explanatory variables	Type of variable	Definition and predicted effects on co-structure
Geographical extent (log)	Continuous	General predictions: At large spatial scales, overall genetic differentiation should be weaker for hosts than for parasites because hosts are expected to disperse over greater distances than parasites. In the same way, weaker congruence in genetic structure is expected with increasing spatial scales Model predictions for increasing geographical extent: • lnRR*: $\beta\ddagger > 0$ • Zr†: $\beta < 0$
Percentage of free-living stages	Continuous	General predictions: Following our null assumption that parasites do not rely on their hosts to disperse, parasites should be more structured than their hosts (ln$RR > 0$) when the percentage of free-living stages is high (close to 100%) because hosts disperse without their parasites. The null assumption should be violated when parasites have no free-living stages, and therefore the global level of genetic differentiation of hosts and parasites should be more similar (ln$RR \approx 0$). lnRR should hence increase with the percentage of free-living stages in the life cycle. Conversely, congruence between the pairwise genetic differentiation of host and parasite species should decrease with an increasing percentage of free-living stages Model predictions for increasing percentage of free-living stages: • lnRR: $\beta > 0$ • Zr: $\beta < 0$
Parasite sexual mode	Categorical (strictly gonochoric sexual, gonochoric sexual & asexual, hermaphrodite sexual & asexual)	General predictions: Hermaphroditic parasites and/or those capable of asexual reproduction should display low genetic diversity within demes and high genetic structure between demes. The global level of genetic differentiation for these parasites should hence be relatively large compared to their hosts (ln$RR > 0$). In contrast, strictly gonochoric parasites should display lower levels of global genetic differentiation, but should still be more structured than their hosts. As a result, congruence between the pairwise genetic differentiation of host and parasite species should be higher in strictly gonochoric parasites than for any other category Model predictions: • lnRR: ln$RR_{\text{herma}+\text{asex}} >$ ln$RR_{\text{gono}+\text{asex}} >$ lnRR_{gono} • Zr: $Zr_{\text{gono}} > Zr_{\text{gono}+\text{asex}} > Zr_{\text{herma}+\text{asex}}$
Host spectrum	Categorical (small, medium, large)	General predictions: Following our null assumption, neither lnRR nor Zr should be affected by the ability of parasites to use hosts with different dispersal abilities; the two statistics should be similar across all categories. The categories 'small, medium and large' correspond to (i) parasites with a direct life cycle infecting a single host species, (ii) parasites with a direct life cycle but able to infect several host species from the same genus, and (iii) parasites with a direct life cycle but able to infect several host species from different families or parasites with a complex life cycle (see the main text for details) Model predictions: • lnRR: ln$RR_{\text{small}} =$ ln$RR_{\text{medium}} =$ lnRR_{large} • Zr: $Zr_{\text{small}} = Zr_{\text{medium}} = Zr_{\text{large}}$
Host mobility mode	Categorical (aerial, aquatic, terrestrial or sessile)	General predictions: The global level of genetic differentiation should be lower for hosts with high mobility ability; the level of parasite genetic differentiation relative to that of the host should thus be higher for highly mobile hosts (hosts using aerial pathways) than for weakly mobile (using terrestrial or aquatic pathways) and sessile hosts (i.e. plants). Congruence in pairwise genetic differentiation should be lower for hosts using aerial, aquatic and terrestrial dispersal modes than for sessile organisms Model predictions: • lnRR: ln$RR_{\text{aerial}} >$ ln$RR_{\text{terrestrial}} >$ ln$RR_{\text{aquatic}} >$ lnRR_{sessile} • Zr: $Zr_{\text{aerial}} < Zr_{\text{terrestrial}} < Zr_{\text{aquatic}} < Zr_{\text{sessile}}$
Relative host dispersal	Categorical (higher or lower)	General predictions: For multi-host parasites, the global level of genetic differentiation should be weaker for hosts than for parasites when the parasite has been sampled on the most dispersive host, but does not rely on this host for dispersal. Under the same null hypothesis, congruence in pairwise genetic differentiation should be lower when the parasite has been sampled on the most dispersive host Model predictions: • lnRR: ln$RR_{\text{lower}} <$ lnRR_{higher} • Zr: $Zr_{\text{lower}} > Zr_{\text{higher}}$

(continued)

Table 1 (continued)

Explanatory variables	Type of variable	Definition and predicted effects on co-structure
Ratio of generation times	Continuous (generation time parasite/generation time host)	General predictions: The higher the ratio in generation times, the higher the generation times of the parasite relative to its host and the higher the global level of genetic differentiation of the parasite should be compared to the host. Congruence in pairwise genetic differentiation should be higher when the ratio is close to 1 Model predictions for an increasing ratio in generation times: • $\ln RR$: $\beta > 0$ • Zr: $\beta > 0$

*$\ln RR$; negative values indicate that global genetic differentiation parasites are lower than that of the hosts (and *vice versa*); null values indicate similar global genetic differentiation for the two species.

†Zr; the stronger Zr, the stronger the congruence between pairwise genetic differentiation of hosts and parasites.

‡β is the expected slope of the relationship between one of the two dependent variables ($\ln RR$ or Zr) and one of the continuous explanatory variables.

ticular parasitic mode was subjective and categories were not necessarily mutually exclusive; for instance some parasitoids are also endo-parasitic, etc. Nonetheless, we still consider this classification useful because it reflects the general nature of the study systems included in the analyses.

For each entry, we then included information on the seven explanatory variables listed in Table 1 and detailed hereafter. When possible, this information was taken directly from the publications. Any missing information was gathered from additional sources in scientific and non-academic literature (e.g. online generalist and biodiversity encyclopedias, naturalist books, etc.). These explanatory variables included:

Parasite-related variables

(1) The 'percentage of free-living stages' in the parasite life cycle was considered by attributing a score based on the number of free-living stages (eggs, larvae and/or adults) divided by the total number of life stages. For example a score of 66% was given if two out of three stages were free-living, 33% if one out of three stages were free-living and 0% if no stages were free-living. It certain cases (e.g. nest or social parasites) all stages were free-living (reaching a score of 100%);

(2) The 'parasite sexual mode', that is whether the parasite species can (i) reproduce sexually with unisexual individuals only (i.e. gonochorous or dioecious), (ii) reproduce both sexually with unisexual individuals and asexually (e.g. parthenogenetic) or (iii) reproduce both sexually with bisexual individuals (i.e. hermaphroditism) and asexually. We assumed that the ability of species to generate and maintain genetic diversity decreases from category (i) to category (iii);

(3) The 'host spectrum' describes the ability of parasites to use host species with different dispersal abilities. We considered three categories of parasites: (i) parasites with a direct life cycle infecting a single host species (i.e. specific), (ii) parasites with a direct life cycle, but able to infect several host species from the same genus (i.e. weak generalist) and (iii) parasites with a direct life cycle, but able to infect several host species from different families (i.e. strong generalist) or parasites with a complex life cycle (i.e. several successive host species that generally include species from different families/phyla). We assumed that, in most cases, host species

from different families are more likely to display different dispersal abilities (category (iii)), meaning that the ability of parasites to use host species with different dispersal abilities increases from category (i) to category (iii);

Host-related variables

(4) The 'host mobility mode' describes whether the host species on which the parasite was sampled uses aerial, aquatic or terrestrial habitats to move, or whether there is no active movement (i.e. sessile organisms such as plants). We assumed that the most mobile hosts are those using aerial pathways (i.e. flying hosts), followed by those using terrestrial and aquatic pathways respectively. We assumed that sessile organisms are the least mobile hosts;

(5) The 'relative host dispersal', that is whether or not the host species on which the parasite was sampled is the most dispersive host in the succession of hosts used by the parasite (based on the categories described in (4));

General variables

(6) The 'geographical extent' (km²) of the studied populations (the area of the sampled populations) was either gathered from the publication directly or calculated as the area of a polygon drawn from the four farthest sampling locations using Google Earth©.

(7) The 'ratio of generation times' synthesises the information about the generation time of the host and the parasite. This relative generation time index was calculated by dividing the generation time of the parasite by that of its host (in days). In a few cases, we could not find direct information on the focal species; in these cases, we inferred the generation time from closely related species by assuming phylogenetic conservatism.

Statistical analyses

Calculation of effect sizes

To compare the global level of genetic differentiation (irrespective of the index used) between host and parasite species, we used the log-transformed response ratio ($\ln RR$) as an effect size. We could not use more classical effect sizes such as the Hedge's *d*, because within-study variance is rarely associated with indices of genetic differentiation. In our case, $\ln RR$ was

computed for each entry as the natural logarithm of the ratio of the global level of genetic differentiation of the parasite species over the global level of genetic differentiation of the host species. In all but two cases, the same marker type was used to calculate and compare host and parasite genetic differentiation. lnRR varies from $-\infty$ to $+\infty$, with negative values indicating that the global genetic differentiation of the parasite species is lower than that of the host species (and *vice versa* for positive values); null values indicate similar global genetic differentiation for the two species. Variance associated with each lnRR value was estimated based on the number of sampled demes (K) and the average number of individuals genotyped or sequenced (N) within each deme as follows: $w_i = [(K - 2)N]^{1/2}$. We used this variance estimate because it was generally impossible to retrieve within-study variance from measures of genetic differentiation (notably for mitochondrial markers, Honnay *et al.* 2010).

To compare pairwise genetic differentiation between host and parasite species, we calculated a standardised effect size by applying Fisher's Z-transformation to the Mantel correlation coefficient of each entry (Zr, Nakagawa & Cuthill 2007). For each transformed effect size, we calculated the asymptotic variance (v_z) using the following formula: $v_z = (n - 3)$ where n is the number of sampled populations (Nakagawa & Cuthill 2007).

Meta-analyses
We first wanted to estimate the mean effect size (MES) over all case studies for the global level of genetic differentiation (lnRR) and the pairwise genetic differentiation of host and parasite species (Zr) respectively. To do so, we used a meta-regression approach based on Bayesian mixed-effects meta-analyses (BMMs; Hadfield 2010; Nakagawa & Santos 2012). We ran two independent models (one for each measure of genetic co-structure, lnRR and Zr respectively) in which effect sizes were the response variables, and we estimated the MES (with its 95% credible intervals) as the intercept of the null models (i.e. no fixed effect). In these models, we included 'paper identity', 'host–parasite combination' and the inverse of the respective variance estimates (w_i or v_z) as random factors (Koricheva *et al.* 2013). Including 'paper identity' and 'host–parasite combination' as random terms allowed us to account for the possibility that multiple effect sizes were available for a single host–parasite combination, even within a single article (e.g. when several molecular marker types were used), and thus avoid problems related to pseudo-replication (Horváthová *et al.* 2011). We included the reciprocal of variance estimates as a random weighting parameter to give more weight to studies with larger sample sizes, which should provide more precise effect size estimates (Koricheva *et al.* 2013). In mixed-effects meta-regressions, the inverse of the variance is typically used to weight the effect of each study (Koricheva *et al.* 2013).

To provide a general description of the database, we then ran BMMs to test if MES varied among (i) the types of molecular markers used for estimating genetic differentiation (mitochondrial or nuclear) and (ii) the parasitic mode. For each descriptive variable of genetic structure, we ran two independent models that were structurally similar to the null models described above, except that they included one of the

two above-mentioned variables (marker type or parasitic mode) as a categorical fixed effect. The deviance information criteria (DIC) of each of these two models was compared to the DIC of the null model to test which of the models were best supported by the data and hence to infer the influence of each descriptive variable; we considered that a model was more supported by the data when its DIC was lower than the DIC of the null model by at least four units (i.e. ΔDIC < 4, Burnham & Anderson 2002).

We then ran BMMs to explicitly test which of the host- and parasite-related variables listed above and in Table 1 best explained variance in effect sizes. For each measure of genetic co-structure, we built a full model in which the respective effect size was the dependent variable and the seven explanatory variables listed in Table 1 were fixed effects. The random factors included in these models were similar to those of the models described above (paper identity, host–parasite combination and the weighting parameter). We also included information on the marker type as a random factor as we found that MES tended to vary between the two main marker types (see Results). For the analysis of pairwise genetic differentiation, we did not include the variable 'relative host dispersal' because of low sample size in one of the two categories. Similarly, categories (ii) and (iii) of the variable 'parasite sexual mode' were grouped into a single category as category (iii) was represented by only two case studies in this data set. From the full model, we ran all possible models (i.e. all combinations of variables, excluding interaction terms which were not considered due to low sample sizes) and calculated the DIC for each model. We retained all models that fell within a ΔDIC < 4 (Burnham & Anderson 2002). This restricted set of models was used to calculate the relative influence (RI) of each variable (i.e. the sum of the DIC weights of each variable across all the models in which each independent variable occurs, expressed as a percentage), and weighted parameters were estimated for the most influential variables (Burnham & Anderson 2002).

We finally assessed potential publication biases in our data set by combining funnel plots and Egger's regressions (Egger *et al.* 1997). A funnel plot is a scatterplot of effect sizes (lnRR and Zr respectively) against a measure of study size (we here chose the inverse of the weighting parameters, w_i and v_z respectively). An unbiased data set is expected to generate a funnel plot in which large studies will be near the average whereas small studies will be spread on both size of the average. In addition, we ran Egger's regressions based on the null models (no fixed effects) described above (Egger *et al.* 1997; Horváthová *et al.* 2011). The slope of the regression is not expected to be significantly different from zero if the data set is unbiased towards significant results.

All models were implemented using the *MCMCglmm* function in the R package 'MCMCglmm' (Hadfield 2010), and the set of all possible models were tested using the *dredge* function in the R package 'MuMIn'. For each model, two parallel MCMC chains with different starting values were ran. For each chain, the number of iterations was set to 15.10^5, the thinning interval was 500 and the burn-in interval was 5.10^5. We used non-informative priors to run the models. The convergence of MCMC outputs was tested calculating the 'potential scale reduction factor' (*psrf*) proposed

by Gelman & Rubin (1992). This diagnostic is based on a comparison of within-chain and between-chain variances; a *psrf* value close to 1 indicates that each chain converged correctly. The Gelman & Rubin's diagnostic indicated that all models converged as all *psrf* were equal to 1. We further used the autocorrelation statistic provided in the MCMCglmm package to ensure that each successive value in the output did no depend strongly on the previous ones, which was obviously not the case as autocorrelation values were < 0.10 for all models.

RESULTS

Global level of genetic differentiation of host and parasite species

Mean effect size and general description of the data
Overall, the MES comparing the genetic differentiation between host and parasite was negative and its 95% CI did not include 0 (Fig. 1, MES = −0.609, 95% CI = −1.051 to
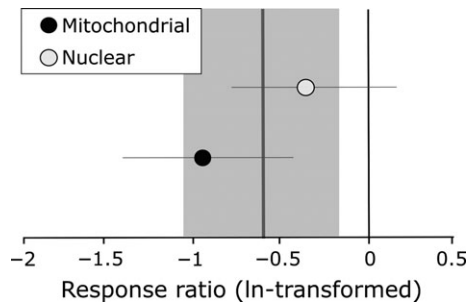

Figure 1 Forest plot showing the mean effect size (log-transformed response ratio, ln*RR*) of the global level of genetic differentiation between host and parasite species. The mean effect size calculated over all case studies is shown (grey line) together with the 95% credible interval (grey area). We also show the mean effect size calculated from the case studies that used either nuclear (white dot) or mitochondrial (black dot) markers to estimate genetic differentiation. Horizontal thin lines are associated 95% credible intervals.

−0.165, DIC_{null} = 283.28), indicating that genetic differentiation is higher in hosts than in parasites. The model including marker type as a fixed effect did not fit the data better (DIC_{marker} = 287.03), although MES tended to be higher for studies using mitochondrial markers, than those using nuclear markers (Fig. 1). There were no differences (and no tendencies) in the MES among parasitic modes (DIC_{mode} = 284.89).

The influence of host- and parasite-related variables
Seventeen models reached a ΔDIC < 4, and within this restricted set of models, the three most influential variables were the parasite sexual mode, the percentage of free-living stages and the geographical extent of the study. The relative influence (RI) of each of these three variables was > 0.912, whereas the RI of all other variables was < 0.550 (not shown). The response ratio was strikingly lower for hermaphroditic parasites than for parasites with gonochoric reproduction (associated or not with asexual reproduction, Fig. 2a). The response ratio increased as the percentage of free-living stages increased (β = 0.020, 95% CI = 0.006–0.034, Fig. 2b); more specifically the response ratio tended to be negative when the percentage of free-living stages in the parasite is low, but reached values close to zero for parasites having all their life stages being free in the environment (Fig. 2b). Finally, the response ratio decreased with increasing geographical scale (β = −0.085, 95% CI = −0.173 to −0.009) (Fig. 2c).

The correlation between the values predicted by a model including these three terms as fixed effects and the observed values was high and significant ($r_{Pearson}$ = 0.439, d.f. = 94, *t* = 4.738, *P* < 0.001). The predicted values from the model show greater deviance from observed values at extremely low values of the response ratio (not shown).

Pairwise genetic differentiation of host and parasite species

Mean effect size and general description of the data
Overall, we found a positive Fisher's *Z* transformed correlation between pairwise genetic differentiation measured for
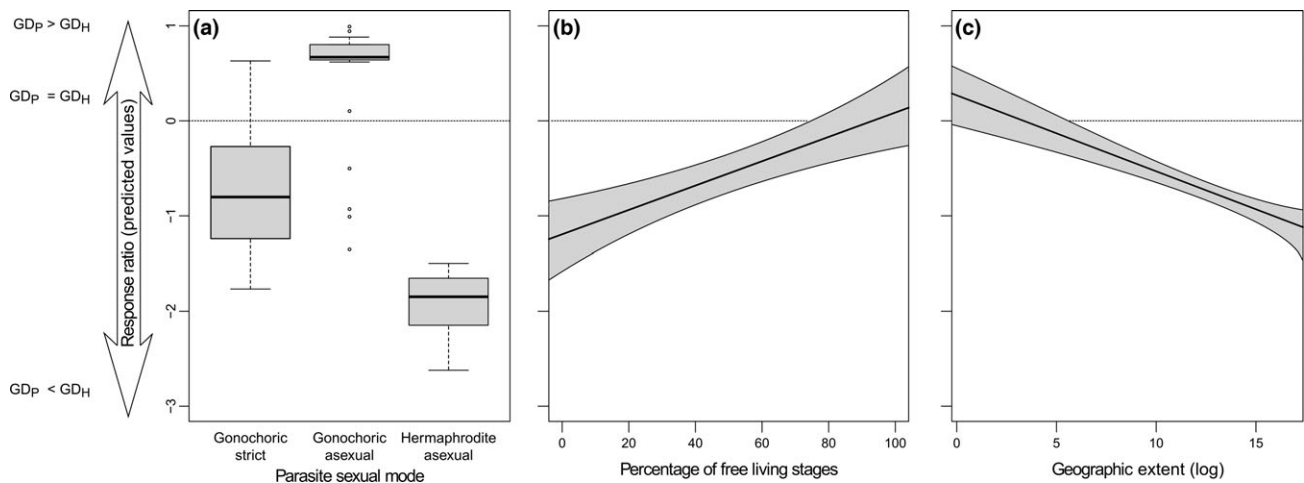

Figure 2 Illustration of the effects of two parasite-related traits and an ecological variable on the mean effect size (log-transformed response ratio, ln*RR*). (a) Decomposition of ln*RR* owing to parasite sexual mode. Relationships between ln*RR* and (b) the percentage of free-living stages in the parasite life cycle and (c) the geographical extent of the study (km²). Regression lines and 95% CI (grey area) are represented. GD_P means "genetic differentiation of the parasite species", whereas GD_H means "genetic differentiation of the host species".

host and parasite ($Zr = 0.351$, 95% CI = 0.149–0.558, $DIC_{null} = -43.08$). The correlation was stronger when mitochondrial markers ($Zr = 0.450$, 95% CI = 0.145–0.770), rather than nuclear markers ($Zr = 0.291$, 95% CI = 0.035–0.545), were used to assess pairwise genetic differentiation ($DIC_{markers} = -49.88$). There were no differences among parasitic modes ($DIC_{mode} = -29.83$).

The influence of host- and parasite-related variables

Six models reached a $\Delta DIC < 4$, from which we identified the parasite sexual mode (RI = 0.787) and to a lesser extent the geographical extent (RI = 0.528) and the ratio of generation times (RI = 0.330) as the most influential variables. The correlation between the values predicted by a model including these three variables as fixed effects and the observed values was weak and non-significant ($r_{pearson} = 0.221$, d.f. = 30, $t = 1.285$, $P = 0.208$), indicating that these variables were poor predictors of Z_r. The effect size tended to decrease with increasing geographical extent ($\beta = -0.011$, 95% CI = -0.068 to 0.050) and the ratio of generation times ($\beta = -0.006$, 95% CI = -0.031 to 0.018), although the 95% CI of both parameters included 0. In addition, Z_r tended to be lower for parasites with both sexual and asexual reproduction (i.e. gonochoric sexual/asexual parasites and hermaphrodite asexual parasites) than for strictly gonochoric sexual parasites (Fig. 3).

Publication biases

There were no signs of publication biases, for either the global level of genetic differentiation or for the pairwise genetic differentiation of host and parasite species. This is seen visually by
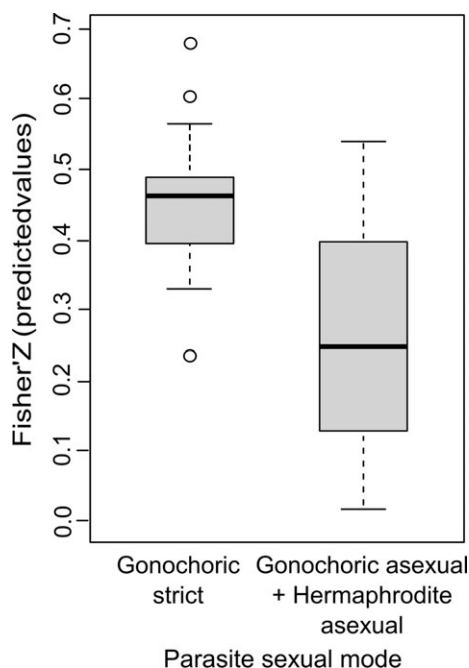

Figure 4 Funnel plots representing effect sizes across studies plotted against corresponding precision (i.e. the inverse of weighting parameters, see the main text); (a) for the comparison of the global level of genetic differentiation between host and parasite species, and (b) for the comparison between the pairwise genetic differentiation of host and parasite species. The dashed lines represent a null effect size, and the solid lines represent the overall mean effect size.

the funnel plots (Fig. 4) and statistically by the Egger's regressions (regression for the global level of genetic differentiation: $\beta = 0.512$, 95% CI: -3.065 to 4.060; regression for the pairwise genetic differentiation: $\beta = 0.118$, 95% CI: -1.010 to 1.211).

DISCUSSION

It is often assumed that parasites mainly rely on their hosts to disperse, and hence that the genetic structure of parasites is primarily shaped by the most dispersive host species in the life cycle (Barrett *et al.* 2008). This hypothesis implies that parasite dispersal rates should not be higher than those of the host and thus that parasite population genetic differentiation should typically be higher than host population genetic differentiation (but see Nadler 1995). The results of our meta-analysis call these notions into question. Indeed, we show that the global level of genetic differentiation, evaluated across a large taxonomic range of species, is typically higher in hosts than in parasites, suggesting that dispersal rates of parasites might be higher on average than those of hosts. Moreover, the correlation between pairwise genetic differentiation of hosts and parasites was significant, but weak. This result corroborates the idea that host dispersal is an influential factor shaping parasite population structure, but the weak support suggests that host–parasite genetic co-structures are also shaped by other species traits that may counter-balance the supposedly strong influence of host dispersal. These traits may also explain why we observed that the global level of genetic differentiation was higher in hosts than in parasites.

In our meta-analysis, we evaluated a series of species traits (both related and unrelated to dispersal) that may influence the spatial distribution of genetic diversity in host and parasite populations. Interestingly, our results show that traits related to host dispersal are not the most important in shaping host and parasite genetic co-structures, and hence the spatial distribution of genetic diversity. Hereafter, we discuss how several factors related to the interaction can affect and explain the spatial distribution of genetic diversity in host and parasite populations.

Figure 3 Boxplots showing the relationship between the predicted mean effect sizes (of Fisher's Z transformed correlation, Z_r) and the parasite sexual mode. In this figure, the category 'gonochoric asexual + hermaphrodite asexual' groups gonochoric sexual/asexual parasites and hermaphrodite asexual parasites because of low sample sizes.
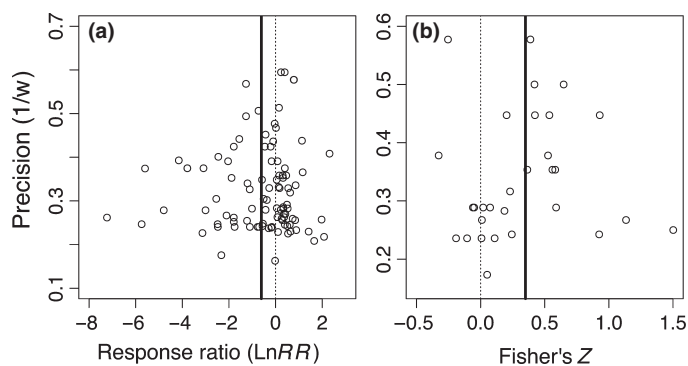
Global level of genetic differentiation in host and parasite populations

Our analysis suggests that the global level of population genetic differentiation was lower in parasites than in hosts. Using meta-regressions, we demonstrated that three variables (out of seven related to the host, the parasite or geography, see Table 1) significantly explained this pattern and its exceptions. In particular, the parasite reproductive mode, the percentage of free-living stages in the parasite life cycle and the geographical extent of the study were all significant predictors, whereas – contrary to common expectations (Huyse *et al.* 2005; Barrett *et al.* 2008) and to a previous meta-analysis on the genetic structure of trematodes (Blasco-Costa & Poulin 2013) – host-related variables (host mobility mode and relative host dispersal) were weak predictors.

Regarding the parasite reproductive mode, our meta-analysis suggested that gonochoric parasites display global levels of genetic differentiation similar to or higher than those of their hosts, whereas hermaphroditic parasites are significantly less structured than the host. Self-fertilisation is predicted to generate little neutral genetic diversity (Ingvarsson 2002; Huyse *et al.* 2005), which should lead to low within-population genetic variation, but high between-population genetic differentiation (Hamrick & Godt 1996; Silvertown & Antonovics 2001), provided that the dispersal rate is low (Ingvarsson 2002). However, when dispersal rate in self-fertilising organisms is high, this may lead to the rapid genetic homogenisation of populations at the meta-population scale. This may explain what we observed; the global level of genetic differentiation in hermaphroditic parasites was extremely low in our data set (mean differentiation = 0.062, $n = 14$, see also Blasco-Costa & Poulin 2013) compared to gonochoric (strictly sexual or partly asexual) parasites (mean differentiation = 0.24, $n = 82$). Most of these hermaphroditic parasites are trematode species that use birds as definitive hosts, which may favour frequent long distance dispersal events in this group of parasites and hence quickly homogenise gene pools (Blasco-Costa & Poulin 2013; Feis *et al.* 2015). The probability that each dispersal event is effective (i.e. results in gene flow) may also be higher for hermaphroditic parasites because, by definition, both sexes are *de facto* present in the population (i.e. there is no need for multiple dispersal events to colonise efficiently). This could also contribute to high gene flow and hence low levels of genetic differentiation. The interaction between parasite reproductive mode and host dispersal ability may therefore explain why the global genetic differentiation of parasites is unexpectedly lower than that of the hosts in certain host–parasite interactions. On the contrary, in gonochoric parasites, constraints on dispersal rates may be more similar to the host because, in most cases, the two sexes have to successfully disperse and meet for gene flow to occur, resulting in similar degrees of population genetic structure. Given these results, we may predict that parasite local adaptation should be more frequent in hermaphroditic parasites than in other types of parasites.

We further found that the percentage of free-living stages in the parasite life cycle significantly influences the genetic differentiation of parasites. More precisely, parasites with no free-living stages (e.g. lice, protozoans) exhibit a global level of genetic differentiation that tended to be lower than that of their hosts. As a greater proportion of free-living stages are added to the life cycle, parasite population structure became higher and similar to that of the host. Hence, as expected (see Table 1), the ratio between the global level of differentiation of the parasite and that of the host increases with the percentage of free-living stages. We further expected that, because parasites with no free-living stages are thought to move only with their hosts (Criscione & Blouin 2004; Bruyndonckx *et al.* 2010), the intercept of the regression slope should be close to zero (see Table 1). Intriguingly, we observed a negative intercept, meaning that even parasites that are always attached to their hosts are less genetically differentiated than their hosts. This result strengthens the conclusion that other ecological or evolutionary forces act in interaction with host dispersal to shape the genetic co-structure of hosts and parasites. For instance parasites are more prone to frequent bottlenecks than their hosts (Huyse *et al.* 2005), a demographic process decreasing genetic diversity. Parasite population dynamics are therefore expected to be characterised by frequent extinction-recolonisations, contrary to host populations that may be closer to an equilibrium state (Price 1980; Barrett *et al.* 2008; Morand *et al.* 2014). Bruyndonckx *et al.* (2009) found, for example that parasitic mites are affected by recurrent bottlenecks, leading to a high between-year genetic turnover and weak large-scale population genetic structure. This stochastic demography of parasite populations could explain why parasites have global levels of genetic differentiation that are consistently lower than what is expected when we only consider host dispersal as the factor determining host–parasite genetic co-structure. The possibility for highly stochastic demography in parasite populations may hinder our ability to make clear predictions about host and parasite local adaptation, as it implies that demographic shifts over time will strongly affect the distribution of standing genetic variation in parasites; this aspect has rarely been considered in experimental and theoretical work to date (but see Morand *et al.* 1996; Gandon & Nuismer 2009). Alternatively, but not exclusively, our finding may suggest that parasites weakly associated to their hosts (i.e. with all or a large percentage of free-living stages) have a dispersal potential similar to their hosts, either through active dispersal (e.g. bird parasites) or through passive dispersal via alternative vectors (e.g. wind or water currents) (Figuerola & Green 2004; Witsenburg *et al.* 2015). An important future research avenue will be to better identify the free-living *dispersive* stages of parasite species, and to test to what extent they contribute to congruence (and incongruence) in host and parasite patterns of genetic differentiation.

Finally, the geographical extent of the studied area influenced the ratio of global genetic differentiation between parasites and hosts. Contrary to our expectation (Table 1), the global levels of genetic differentiation of parasites tended to be similar to that of their hosts at small spatial scales, and lower at large spatial scales. This is surprising given that it can suggest that parasites have longer and/or more frequent dispersal events than their hosts. However, when a single host disperses, it often transports several parasite individuals (sometimes tens of thousands of individuals in the case of

super-spreader hosts; Paull *et al.* 2012). This may decrease the parasite's genetic differentiation more quickly and more efficiently than the host's, and particularly so at large spatial scales when host dispersal events are generally scarce. In addition, hosts may efficiently disperse parasites (i.e. movement leading to gene flow) over great distances without dispersing themselves; seasonal migrations or prospecting movements in birds (i.e. when birds visit an area, but do not reproduce there) are excellent examples of this phenomenon (Gómez-Díaz *et al.* 2012). Thus, although our findings are not in complete accord with the notion that parasites rely on the more dispersive host and match its population structure, they do not refute the general paradigm that parasite dispersal is related to host movement.

Correlation between pairwise host–parasite genetic differentiations

The correlation between pairwise genetic differentiation measured for host and parasite was positive and weakly significant, indicating that the spatial genetic structure of parasites is related to that of their hosts, and hence that dispersal rates in parasites might partially reflect dispersal rates of their hosts. Given the low sample size (and hence statistical power), identifying the variables underlying this correlation was not straightforward and, accordingly, the best explanatory variables explained only a low percentage of the observed variance in the correlation. There was a tendency for pairwise correlations to be weaker for parasites with both sexual and asexual reproduction, compared to strictly sexual parasites. Sexual reproduction generally maintains genetic polymorphism, whereas asexual reproduction reduces it (Fox *et al.* 1996; Liu *et al.* 1996; Huyse *et al.* 2005). Low genotypic diversity in asexual populations could decrease the strength of the correlation in genetic spatial structures of hosts and parasites, simply because the level of polymorphism (i.e. within-population diversity) can mathematically bias some indices of genetic differentiation such as F_{st} (see Box 1 for details, Jost 2008; Meirmans & Hedrick 2011). In contrast, parasites that reproduce only sexually may have effective population sizes and levels of polymorphism approaching those of their hosts, favouring stronger pairwise correlations. No other trait, including those related to host dispersal ability, could explain a reasonable amount of the variance observed in the correlation. Further empirical and/or theoretical studies are therefore clearly needed to better appraise the interactions among biological traits and environmental constraints in shaping the spatial genetic structures and dispersal rates of hosts and parasites.

PERSPECTIVES

The spatial distribution of genetic diversity on which selection can act is the by-product of biological factors affecting dispersal, genetic drift and mutation (Hutchison & Templeton 1999). Host and parasite populations are no exception to this rule. For instance our observation that genetic differentiation is lower in parasites than in hosts might be partly explained by higher effective population sizes in parasites than in hosts. Higher effective population sizes have also been shown to

facilitate local adaptation in host and parasite populations (Gandon & Michalakis 2002), which means that low levels of genetic differentiation that are due to large population sizes could also explain why parasite populations are generally adapted locally to their hosts. It would be fascinating to empirically test to what extent the low level of genetic differentiation observed in some parasite populations is actually due to effective population size, relative to dispersal rates (see Box 1). More generally, we propose that future studies should consider the genetic co-structure of host and parasite populations within the framework of interactions affecting the migration, mutation and drift equilibrium. A powerful way to examine these interactive effects is via theoretical modelling. For instance the influence of demographic processes, such as the frequency of extinction-recolonisation events, on current genetic differentiation, under low or high dispersal abilities might be explored with simulations or mathematical models (Lion & Gandon 2015). In addition, a fascinating perspective would be to directly confront results of genetic co-structure studies to those testing experimentally for parasite (or host) local adaptation (Greischar & Koskella 2007; Hoeksema & Forde 2008), to test how well co-evolution can be predicted from data synthesising the spatial distribution of genetic diversity in host–parasite systems. In the meantime, we advocate for more case studies (see Box 1 for some technical guidelines), and a better integration of landscape features (Biek & Real 2010) that could simultaneously explain genetic differentiation of the two protagonists. Greater diversity in the biological systems studied and in the methods used to study them would enable us to better identify the fundamental processes acting on host and parasite genetic structures and evolution.

CONCLUDING REMARKS

Of the case studies we reviewed here, most assumed *a priori* (and tested the working hypothesis) that parasite dispersal strongly depends on host movement, and hence that this relationship should be reflected in a strong congruence between host and parasite genetic structures. This hypothesis was proposed fourteen years ago by Jarne & Theron (2001), and rapidly spread in the host–parasite research community until becoming a paradigm. Our study does not dispel this paradigm and actually supports the logical view that parasite dispersal rates depend on the dispersal of their hosts. However, interpretations of host–parasite genetic co-structure – and hence of relative dispersal rates – are not so straightforward, and evolutionary and ecological forces other than host dispersal *per se* are also major drivers of the spatial distribution of genetic diversity in hosts and parasites. We notably identified two life-history traits of parasites (parasite sexual mode and percentage of free-living stages in the parasite life cycle) that are reasonable predictors of the relative spatial distribution of genetic diversity of host and parasite populations. Importantly, we found that certain combinations of parasite traits are associated with lower levels of genetic differentiation in parasites compared to hosts, suggesting that the arrival and maintenance of novel genetic diversity at local scales may favour local adaptation of parasites to their hosts.

AUTHORSHIP

EMG, SB and GL designed the work and collected the data. EMG and SB performed the meta-analysis. EMG, SB, KDM and GL interpreted the results and wrote the manuscript.

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