

Host genetic diversity limits parasite success beyond agricultural systems: a meta-analysis

Authors

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Authorship

A.K.E.E. and K.C.K. conceived and designed the study. A.K.E.E. gathered the data and performed the statistical analysis with C.R-M. A.K.E.E. and K.C.K. wrote the paper.

Key words: Genetic diversity, host-parasite interactions, monoculture effect, meta-analysis, microparasite

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Abstract

There is evidence that human activities are reducing the population genetic diversity of species worldwide. Given the prediction that parasites better exploit genetically homogeneous host populations, many species could be vulnerable to disease outbreaks. Whilst agricultural studies have shown the devastating effects of infectious disease in crop monocultures, the widespread nature of this diversity-disease relationship remains unclear in natural systems. Here, we provide broad support that high population genetic diversity can protect against infectious disease by conducting a meta-analysis of 23 studies, with a total of 67 effect sizes. We found that parasite functional group (micro- or macroparasite) affects the presence of the effect and study setting (field or lab-based environment) influences the magnitude. Our study also suggests that host genetic diversity is overall a robust defence against infection regardless of host reproduction, parasite's host range, parasite diversity, virulence, and the method by which parasite success was recorded. Combined, these results highlight the importance of monitoring declines of host population genetic diversity as shifts in parasite distributions could have devastating effects on at-risk populations in nature.

Introduction

Most natural populations are genetically diverse (1). Given there is often specificity between hosts and parasites (2), host population genetic diversity is thought to increase the chance that one or more individuals is resistant to infection. The likelihood of a parasite encountering a susceptible host is thus reduced (3). Genetically homogenous host populations are conversely predicted to be more vulnerable to infection given the uniformity of host susceptibility. This negative relationship between host genetic diversity and parasite success is often referred to as the 'monoculture effect' (4).

The study of the monoculture effect in agricultural settings is extensive (5–7). A recent meta-analysis showed that with increased diversity in intraspecific cultivar mixtures, disease presence is reduced and crop yields increased (7). However, crop plants are under artificial selection for high yield, and may therefore exhibit less genetic polymorphism than hosts in the wild. We consequently know little of the extent to which low genetic diversity influences parasite success across species and environments beyond agricultural contexts.

Threats to within-species genetic diversity are on the rise. There is evidence that habitat alterations, pollution, and global temperature changes, as well as the restriction of species geographical ranges may lead to increased genetic drift and reduced population genetic diversity (8,9). Impacts of humans on local species biodiversity, however, remain controversial (10,11). Populations with reduced genetic diversity might suffer diminished evolutionary potential (12) and increased inbreeding depression (13,14). Knowing whether

there is an additional threat of outbreaks in these populations is crucial for disease management and species conservation approaches.

Theory has illuminated the dynamics of parasite spread (4,15–18) in diverse host populations as well as examined the level of diversity required to stop transmission (19,20). However, whether population genetic diversity can impact parasite success in nature more broadly remains unclear for several reasons. Firstly, given that parasite transmission can be determined by host density (3), the relative effects of density versus host genetic diversity need to be elucidated (20). Shrinking habitats, for example, can result in higher population densities (and lower resource availability) where parasites can transmit better due to more contact between hosts (21,22). Secondly, even when focusing on host genetic diversity alone, there is great variation across systems in the conditions under which infection and diversity are measured. In comparison to diverse populations, genetically homogenous bumble bee (*Bombus terrestris* L.) populations, the microsporidian *Nosema bombi* has higher success, but the trypanosomelid *Crithidia bombi* does not (23). In other cases, we see an increase in parasite success on the homogenous host populations when multiple parasite species infect (23–26) but not always between one host-parasite species pair (27,28). Thirdly, because parasite success is measured differently across studies, and even within systems, there is the potential that the relevant measure of parasite success is not used. For example, in honeybee (*Apis mellifera*) host populations, genetic diversity has a negative impact on parasite success when infection prevalence or parasite load is measured, but not always when host survival is calculated (29). Host survival might be less informative because of the interplay of virulence, force of infection, and the timing of infection might determine the overall spread of pathogens in host populations (30). It is therefore unclear whether the

effect of low host genetic diversity on parasite success is relevant to host-parasite interactions in non-agricultural systems across the tree of life.

We tested the effect of host population genetic diversity on parasite success with a formal meta-analysis across a range of host-parasite systems. We searched the published literature for all publicly available data sources and compared the effects of low and high host genetic diversity on parasite success using Hedges's effect size g (with positive values indicating an effect of low host genetic diversity on parasite success) with a nested random mixed effects meta-analysis model. We also tested whether biological traits, associated with the species in the interaction, as well as study settings and measures could explain variation in the effects of genetic diversity on parasite success.

Materials and methods

Literature search

In July 2019, the literature was searched using keyword searches on Web of Knowledge, Google Scholar and PubMed, with a subset of the terms 'host genetic diversity', 'low versus/and high host genetic diversity', 'heterogeneous versus/and homogenous host populations', 'monoculture effect', 'disease spread', and 'parasite prevalence' to investigate the effect of low versus high host population diversity on parasite disease impact (see Supp. Fig. 1 for PRISMA flowchart (31) summarising study collection process). We gathered data of parasite success in host populations of varying genetic diversity. We define 'parasite success' as any measure of a parasite's ability to proliferate within a host population

reported in a given study. As parasite presence within a host population is measured differently across studies, the following terms were included as measurements of parasite success; parasite load, parasite virulence, parasite abundance, host mortality rate, viral concentrations, viral load, infection rate, and infection intensity. We also checked reference lists for other potential papers. Studies were also searched for and extracted from review papers.

Papers were included in this study if they met the following inclusion criteria:

- i. The study was published in a peer reviewed academic journal.
- ii. The study collected parasite success data from two distinct comparable host population groups with any measured difference in diversity, such as low versus high genetic diversity, inbred versus outbred, and monoculture versus polyculture.
- iii. In the study, both host population groups contained the same species.
- iv. The study measured genetic diversity at the host population level and not community diversity or individual-level genetic heterozygosity.
- v. The study was not conducted in an agricultural system.

vi. The study did not interfere with parasite or host lifecycle, as in passaging manipulations.

We excluded agricultural studies as a recent meta-analysis had already demonstrated the benefits of intraspecific diversity to crop yields (and thus host fitness) in the presence of infectious disease (7).

Statistical analysis

We calculated Hedges's g , from studies using the method described in Hedges 1981 (32) . This is a standard and widely used method of calculating effect sizes in meta-analyses which takes into account small sample sizes (33,34). To calculate effect size g , mean parasite measurements and their standard deviation for each treatment were extracted in the order of low host population diversity and high host population diversity. We extracted data from either paper figures, reported statistics in the text, or raw data received from authors. Where means and standard deviations in each group were not available (2 out of 23 studies), t-values and degrees of freedom were extracted.

We calculated the standard mean differences using the *escalc* function in the package *metafor* in R version 3.6.0 (R core development team) before performing a nested random mixed effects meta-analysis model using the *rma.mv* function. We chose this model to account for the fact that we collected several effect sizes per study, where some studies shared the same host species, which has the potential for pseudo-replication and

phylogenetic non-independence. Estimates of effect size g were extracted from the model.

We first tested for an overall relationship between host population genetic diversity and parasite success using the entire dataset. We then tested whether the magnitude of the relationship was dependent on the following moderator variables: study setting, parasite success measure, host reproduction, parasite functional group, parasite's host range, parasite diversity, and ability of parasite to cause host death (See Supp. Table 1 for variable definitions). The measure of heterogeneity of moderator variables was reported as Cochran's Q test, where Q is the weighted sum of squares about the fixed effect estimate between subgroups (35).

We tested for an effect of both study setting (field or lab-based environments) and parasite success measure on the relationship between host genetic diversity and parasite success. For the latter, we separated measures into three groups based on those used in studies included in the meta-analysis: parasite prevalence, parasite load, and host mortality (Supp. Table 1). Studies looking at overall parasite presence in a host population were placed under the category 'parasite prevalence'. Where measures of parasite propagules per host were taken, studies were placed under 'parasite load'. Measures of mortality within a population were placed under 'host mortality'. In order to incorporate studies publishing survival data, measures of host mortality were taken as the inverse of published survival measures.

We then focused on the impact of host and parasite biological traits on variation in the magnitude and direction of effect sizes. We firstly considered host reproductive mode, given sexual and asexual strategies can generate disparate levels of population genetic diversity. However, one study was placed under a separate reproduction group as the host (*Daphnia*

magna) had undergone both sexual and asexual reproduction in the study. Secondly, we looked at infection by parasite functional group (micro- or macroparasites) as the former tends to be associated with higher mortality (36), and thirdly the parasite's host range (1 host species or >1 host species) as this factor has been shown to have an impact in crop studies (37,38) due to the reduced genetic specificity between hosts and multi-host parasites. Fourthly, we separated studies into three categories – one genotype of one parasite species (1 Genotype), multiple parasite genotypes of one parasite species (>1 Genotypes), and multiple parasite species (>1 Species) – to determine whether the diversity-disease relationship was dependent on parasite diversity. Higher levels of parasite diversity might increase the pool of susceptible hosts in a diverse population. Lastly, we tested whether effect sizes were dependent on the parasite's ability to cause host death. Compared to less harmful parasites, virulent parasites could select for greater levels and variation of resistance in the host population.

Assessing for potential publication bias

Studies that report larger effects are more likely to get published in comparison to studies reporting smaller effects (39). To check for publication bias, we visualised the spread of our effect sizes by creating a funnel plot (Supp. Fig. 2). We then performed a Fail-Safe N analysis to calculate the number of additional studies needed to reduce the significance level of the weighted average effect size (40).

Results

We found 32 unique host-parasite interactions in 23 papers containing data that followed the inclusion criteria. Papers often included results from multiple experiments or exposures to multiple parasite species. A total of 67 effect sizes were retrieved from this data set, covering a diverse range of host and parasite species (Table 1).

After the construction of a funnel plot, we find no indication of a publication bias in this meta-analysis data set, with the majority of points falling symmetrically within the plot (Supp. Fig. 1). The unusual shape of the plot can be explained by the fact that small sample sizes were predominantly found in laboratory studies, whereas large sample sizes were predominantly found in field studies. Consequently, studies with large sample sizes had higher errors than those with small sample sizes explaining the shape of the plot (we highlight this by colourising the plot by study setting). Rosenberg's Fail-safe N analysis showed that an additional 604 studies would need to be added to reduce the significance level of this meta-analysis.

Our results are consistent with the hypothesis that low host genetic diversity results in higher parasite success ($g = 0.3527$, $p < 0.0001$, Fig. 1A). We found that the effect size is influenced by study setting ($Q = 9.2111$, $d.f. = 1$, $p = 0.0024$, Fig. 1B), where the magnitude of the effect size is significantly greater for field studies ($g = 0.7003$) in comparison to lab studies ($g = -0.5249$). Parasite success measures used in the studies do not significantly influence the effect size ($Q = 2.6526$, $d.f. = 2$, $p = 0.2655$, Fig. 1C).

We found no evidence of an effect of host reproduction on the direction or magnitude of the effect size ($Q = 4.0711$, $d.f. = 2$, $p = 0.1306$, Fig. 2A), even when we excluded the *Daphnia*

study by Altermatt & Ebert (2008) ($Q = 0.9147$, d.f. = 1, $p = 0.3389$). Conversely, we found that the effect size was dependent on parasite functional group ($Q = 8.3621$, d.f. = 1, $p = 0.0038$, Fig. 2B). The success of microparasites ($g = 0.6277$), and not macroparasites ($g = -0.1725$) was limited by high host population genetic diversity. Neither the direction nor magnitude of the effect size was influenced by host range ($Q = 0.2864$, d.f. = 1, $p = 0.5925$, Fig. 2C), parasite diversity ($Q = 3.1047$, d.f. = 2, $p = 0.2118$, Fig. 2D), or whether parasites caused host mortality ($Q = 3.5504$, d.f. = 1, $p = 0.0595$, Fig. 2E).

Discussion

Our meta-analysis shows that host population genetic diversity reduces parasite success across multiple natural systems. In particular, we find that host population genetic diversity is effective at limiting microparasite infection success, with little to no effect on the macroparasites tested, and the protection is stronger when measured in the field. Our findings additionally highlight the potential damage that emerging infectious diseases may have on genetically homogenous host populations.

The parasites included in our meta-analysis were highly variable in terms of their host range. However, we found no evidence that a parasite's host range affected its success in host populations of low or high genetic diversity. Indeed, we see evidence of resistance in more diverse populations involving highly specialised interactions (41–43), in broad spectrum interactions at the genotypic level (44), and in those that cross host-species boundaries (25,26,45). That host range is not a factor here is in contrast to those results found in crop studies. For example, in rusts and powdery mildews, disease severity is driven by a

pathogen's host specificity (6). The mirroring of parasite virulence genes to host resistance genes means that crop mixtures need to contain both susceptible and resistant cultivars to avoid a monoculture effect. When there is a lack of host specificity, mixed cultivar populations are just as susceptible as monocultures. For example, mixed cultivar populations have been observed to be slightly more susceptible to infection (37) or completely susceptible (38) in comparison to monocultures to the fungal pathogen *Mycosphaerella graminicola*. These findings suggest that the threat to crops from generalist parasites is greater than specialist parasites.

Given that host range did not influence whether parasite success was reduced by host genetic diversity, it is possible that novel parasites, just as well-adapted parasites, could have high success in host populations with low genetic diversity. Essentially, homogenous populations could be vulnerable to outbreaks with spill-over or emerging infectious diseases which are less likely to be host specific (46), but for which there is clearly genetic variation for resistance. The resistance to emerging parasites in these cases could be due to historical contact or similar mechanisms of infection applied by parasites with an evolutionary history to the host (8). Nevertheless, this result is concerning from a conservation perspective as global climate change has the potential to reduce within-species genetic diversity (47) and alter host population ranges (9,48). Natural movement of individuals between populations has always served to bolster host diversity (9), and introducing new genotypes is an approach applied by conservation biologists to improve population viability (14). Whilst adding individuals to a population could increase diversity and reduce inbreeding (49), a risk may be that new individuals, new species, and changes in ecological opportunities bring in new parasites to the population (50,51). There is potential here for an increased overlap

between host populations with low genetic diversity and novel infections. Given that we found a stronger effect in field studies, these consequences are of real concern.

The difference in parasite success between diverse and homogeneous host populations was more pronounced in field studies, compared to lab studies, despite the additional environmental noise data collection in nature might involve. One reason could be that less diverse populations in the wild are more susceptible to infection than they are in the lab for reasons unrelated to genetic diversity. Hosts on islands as well as social insects, such as bees (52), ants (53), and termites (54), live in tight proximities to each other making parasite transmission easier in homogenous populations. The stronger effect in field studies highlights the importance of the maintenance of diversity in natural populations.

In our meta-analysis, the success of macroparasites was not impeded by genetic heterogeneity in host populations. The macroparasites in the studies included herein were all ectoparasites, and their biology may explain our result. Ectoparasite transmission is often dependent on host-to-host contact (55,56) and thus host density is likely a critical factor in parasite success (55). Host density may play a more important role than host genetic diversity here such that similarly aggregated populations varying in diversity might be equally susceptible to infection. It has been shown that clustering of captive animal populations restricted by movement or wild animal populations restricted by ranges are highly vulnerable to ectoparasites (50,57). Moreover, host social behaviours, such as grooming (29) or preening (26) can reduce ectoparasite success. In fact, in populations where social grooming is correlated with relatedness, ectoparasite load is dramatically

reduced in highly related individuals (58). Taken together, host diversity on its own does not always explain a reduction in parasite success, particularly in the case of ectoparasites.

Understanding the impact of host population genetic diversity on parasite infection outside of agricultural systems is crucial because of anthropogenic threats to the diversity of wild populations. This meta-analysis reveals that the susceptibility conferred by low host genetic diversity is a widespread phenomenon in nature, with microparasites most likely to encounter resistance in diverse host populations. Indeed, these broad patterns show that genetic diversity is a robust weapon against infection, similar to the effects of species biodiversity (59). Our findings suggest that further erosion of within-species genetic diversity could drive outbreaks of both coevolving and emerging infectious diseases. Conservation efforts should focus on preserving population genetic diversity in vulnerable populations to improve their ability to fight off infections.

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Table 1: Summary of literature on the effect of host population genetic diversity on measures of parasite success across host-parasite systems.

Source paper	Paper number	Host	Measure of host diversity	Parasite	Host range (ref)	Parasite type	Infection measure	Data source	Data extracted	n Effect sizes
Altermatt and Ebert (2008)	1	<i>Daphnia magna</i>	1 vs 10 genotypes	<i>Octosporea bayeri</i>	1 Host species (41)	Fungus	Parasite load	Figure 2, Raw data	Mean \pm SD	2
Baer and Schmid-Hempel (1999)	2	Bumblebee (<i>Bombus terrestris</i>)	Queens inseminated with sperm from 1 vs 2 or 4 males	<i>Crithidia bombi</i> , <i>Nosema bombi</i>	>1 Host species (60)	Protozoa, Fungus	Parasite load	Figure 1, Raw data	Mean \pm SE	4
Baer and Schmid-Hempel (2001)	3	Bumblebee (<i>Bombus terrestris</i>)	Queens inseminated with sperm from 1 vs 2 or 4 males	<i>Crithidia bombi</i>	>1 Host species (60)	Protozoa	Parasite load, Parasite prevalence	Figure 1, Raw data	Mean \pm SD	4
Baer and Schmid-Hempel (2003)	4	Bumblebee (<i>Bombus terrestris</i>)	Queens inseminated with sperm from 1 vs 2 or 4 males	<i>Crithidia bombi</i>	>1 Host species (60)	Protozoa	Parasite load, Parasite prevalence	Raw data	Mean \pm SD	4

Calleri <i>et al.</i> (2006)	5	Termite <i>(Zootermopsis angusticollis)</i>	Inbred vs outbred	<i>Metarhizium anisopliae</i>	>1 Host species (61)	Fungus	Parasite load	In text	Mean \pm SD	1
Desai and Currie (2015)	6	Honeybee (<i>Apis mellifera</i> L.)	Queens inseminated with sperm from 1 vs 12 drones	<i>Varroa destructor</i> , Deformed Wing Virus, Black Queen Cell Virus, Israeli Acute Paralysis Virus	>1 Host species (62-64)	Mite, Virus, Virus	Parasite load, Host mortality, Parasite prevalence	Figure 1, 2, 4, 5, 7, 8	Mean \pm SE	11
Ganz & Ebert (2010)	7	<i>Daphnia magna</i>	1 vs 10 genotypes	<i>Glugoides intestinalis</i> , <i>Ordospora colligate</i> , <i>Microsporidium sp.</i> <i>(undescribed species)</i>	1 Host species (65) and >1 Host species (65)	Fungus, Fungus, Fungus	Parasite prevalence	Figure 2	Mean \pm SE	3

Hale & Briskie (2007)	8	New Zealand Robin (<i>Petroica australis</i>)	Bottleneck vs source population	Hippoboscid flies (<i>Ornithomya spp.</i> and <i>Ornithoica spp.</i>), Feather mite	>1 Host species (66)	Fly, Mite	Parasite load	Figure 1	Mean \pm SD	2
Hughes & Boomsma (2004)	9	Ant (<i>Acromyrmex echinator</i>)	1 patriline vs 3 patrilines	<i>Metarhizium anisopliae</i> (strain KVL 02-73)	>1 Host species (61)	Fungus	Host mortality	Figure 4	Mean \pm SE	2
Liersch and Schmid-Hempel (1998)	10	Bumblebee (<i>Bombus terrestris</i>)	Full sister workers vs mixed workers	<i>Crithidia bombi</i> , <i>Nosema bombi</i> , <i>Apicystis (Mattesia) bombi</i>	>1 Host species (60, 67)	Protozoa, Fungus, Protozoa	Parasite prevalence, Parasite load	Figure 1	Mean + CI	2
Manlik et al. (2017)	11	Bumblebee (<i>Bombus terrestris</i>)	Island vs land population	<i>Nosema bombi</i>	>1 Host species (60)	Fungus	Parasite prevalence	In text	Mean \pm SE	1
Pearman & Garner (2005)	12	Italian agile frog (<i>Rana latastei</i>)	Low vs high population genetic variability	<i>Ranavirus</i> (frog virus 3)	>1 Host species (44)	Virus	Host mortality	Figure 2, Raw data	Mean \pm SD	3

Reber <i>et al.</i> (2008)	13	Ant (<i>Formica selysi</i>)	Monogynous vs polygynous colonies	<i>Metarhizium</i> <i>anisopliae</i>	>1 Host species (61)	Fungus	Host mortality	Figure 1, 2	Mean \pm SE	3
Schmidt <i>et al.</i> (2011)	14	Ant (<i>Monomorium</i> <i>pharaonis</i>)	Inbred vs mixed colonies	<i>Beauveria bassiana</i>	>1 Host species (61)	Fungus	Host mortality	Figure 3	Mean + CI	3
Seeley and Tarpy (2007)	15	Honeybee (<i>Apis</i> <i>mellifera</i> L.)	Queens inseminated with sperm from 1 vs 10 drones	American foulbrood (<i>Paenibacillus larvae</i>)	>1 Host species (71)	Bacteria	Parasite prevalence	Figure 2, Raw data	Mean \pm SD	2
Shykoff and Schmid-Hempel (1991)	16	Bumblebee (<i>Bombus</i> <i>terrestris</i>)	1 genotype vs 3 genotypes	<i>Crithidia bombi</i>	>1 Host species (60)	Protozoa	Parasite prevalence	Figure 2	t - value	2
Smallbone <i>et al.</i> (2016)	17	Guppy (<i>Poecilia</i> <i>reticulata</i>)	Inbred vs outbred	<i>Gyrodactylus</i> <i>turnbulli</i> (strain Gt3)	>1 Host species (68)	Worm	Parasite load	Figure 2	Mean \pm SE	1
Strauss <i>et al.</i> (2017)	18	<i>Daphnia dentifera</i>	1 genotype vs 3 genotypes	<i>Metschnikowia</i> <i>bicuspidata</i>	>1 Host species (70)	Fungus	Parasite prevalence	Figure S1B	Mean \pm SE	1

Tarpy (2003)	19	Honeybee (<i>Apis mellifera</i> L.)	Queens inseminated with sperm from 1 vs multiple drones	Chalkbrood disease (<i>Acosphaera apis</i>)	>1 Host species (74)	Fungus	Parasite prevalence	Figure 2	Mean \pm SD	1
Tarpy and Seeley (2006)	20	Honeybee (<i>Apis mellifera</i> L.)	Queens inseminated with sperm from 1 vs 10 drones	Sacbrood (Iflavirus genus), Chalkbrood disease (<i>Acosphaera apis</i>), European foulbrood (<i>Melissococcus plutonius</i>), American foulbrood (<i>Paenibacillus larvae</i>)	>1 Host species (69,71,74,75)	Virus, Fungus, Bacteria, Bacteria	Parasite prevalence	In text	t - value	4
van Houte <i>et al.</i> (2016)	21	<i>Pseudomonas aeruginosa</i> , <i>Streptococcus thermophilus</i>	1 genotype vs 6, 8, 12, 24, and 48 genotypes and 1 genotype vs 44 genotypes	Bacteriophage (DMS3), Bacteriophage (2972)	1 Host species (72,73)	Virus, Virus	Parasite prevalence	Figure 2, Raw data	Mean \pm SD	5

Wargo et al.	22	Rainbow trout	Inbred vs outbred	Infectious	1 Host species	Virus	Parasite	Figure 2,	Mean \pm SE	4
(2012)		(<i>Oncorhynchus mykiss</i>)		hematopoietic necrosis virus (IHNV) isolates: 220:90 (HV), WRAC 039-82 (LV), FF020-91 (B), FF030-91(C)	(42)		prevalence	Raw data		
Whiteman et al.	23	Galapagos Hawk	Inbred vs outbred	<i>Colpocephalum</i>	>1 Host	Louse,	Parasite load	Figure 2,	Mean \pm SD	2
(2006)		(<i>Buteo galapagoensis</i>)		<i>turbinatum</i> , <i>Degeeriella regalis</i>	species (56)	Louse		Raw data		

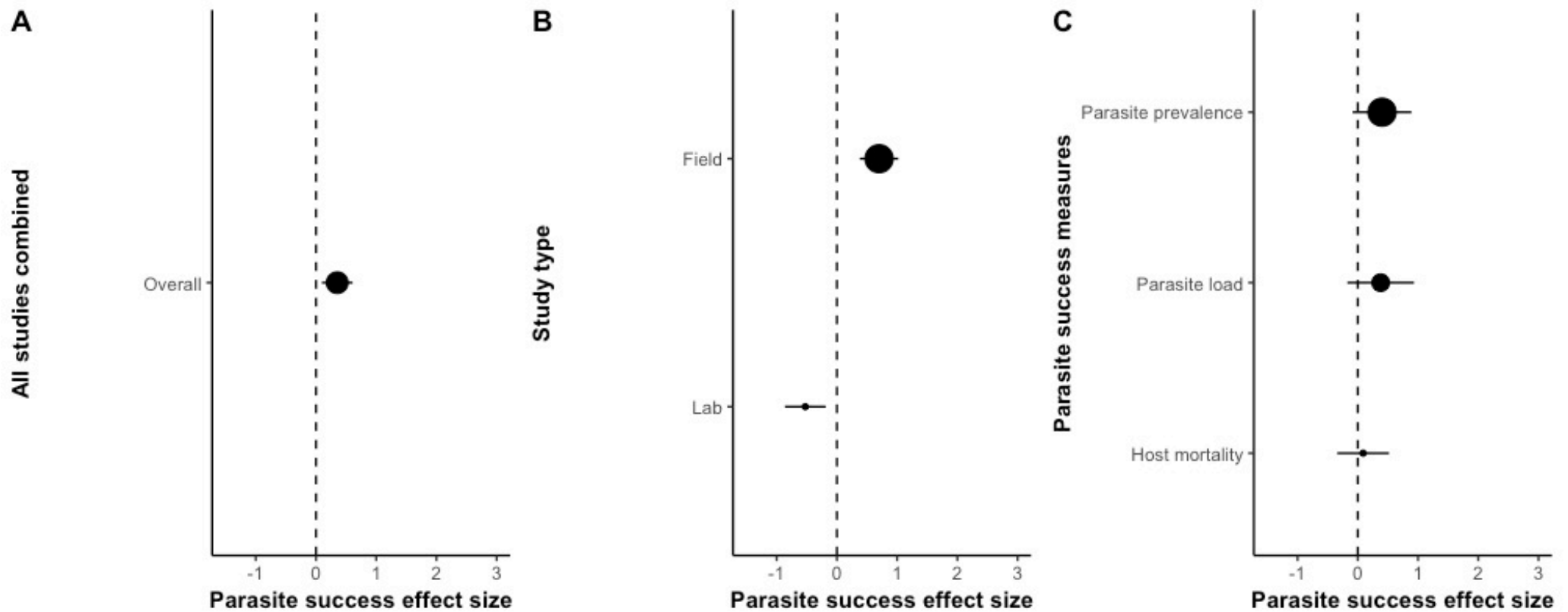


Figure 1: Impact of study setting on the effect of host genetic diversity on parasite success. Positive values indicate that low host genetic diversity has an impact on parasite success (i.e., a negative association between genetic diversity and parasite success). Negative values represent the opposite relationship. At an effect size of zero (dashed line), there is no relationship between host genetic diversity and parasite success. (A) Overall effect size (n = 67). (B) Moderator analysis of study type between field (n = 36) and lab (n = 31) studies. (C) Moderator analysis of parasite success measures between parasite load (n = 19), parasite prevalence (n = 35), and host mortality (n = 13). The size of the dot corresponds to the sample size. Effect sizes are shown with 95% confidence intervals.

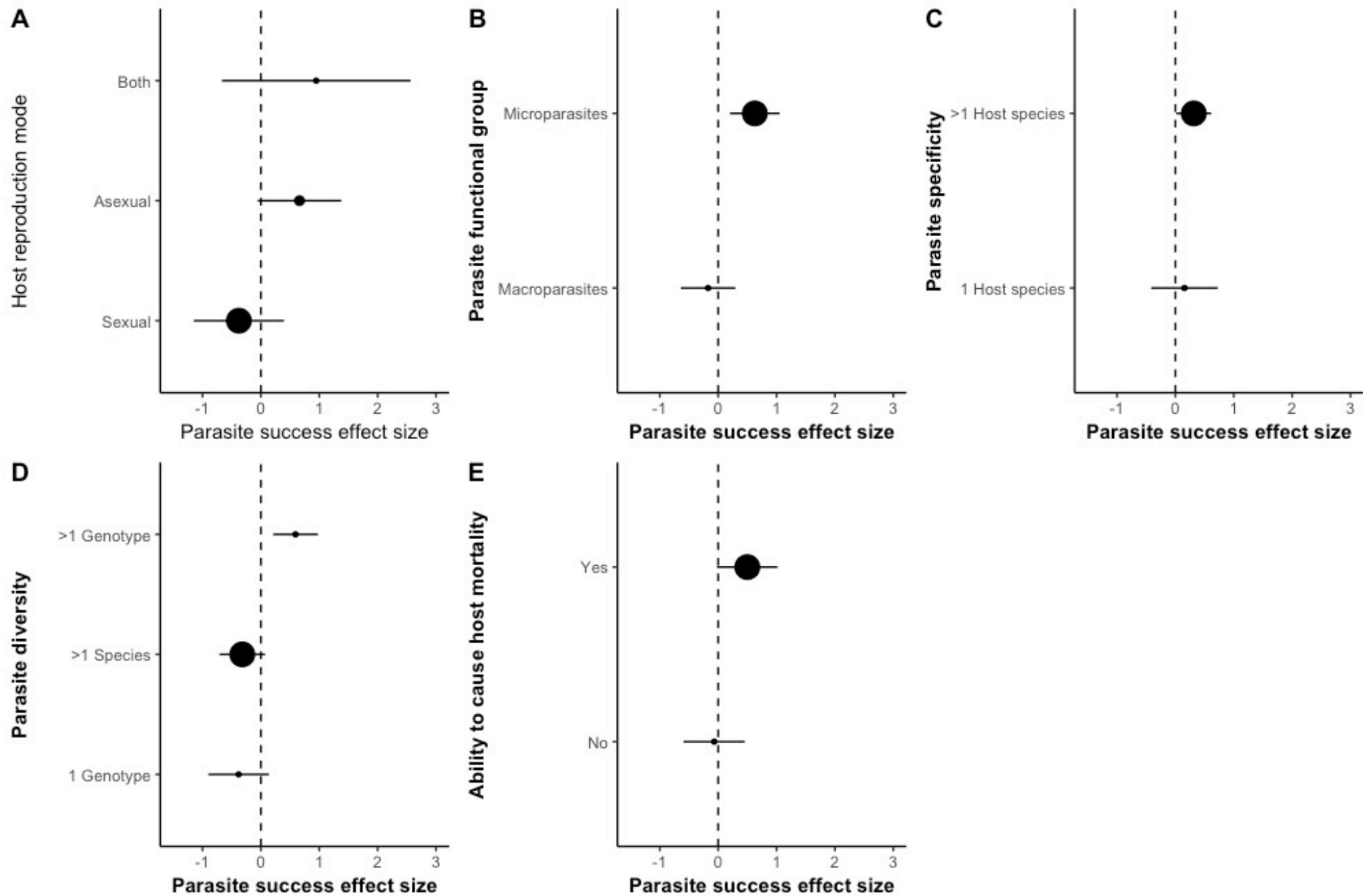
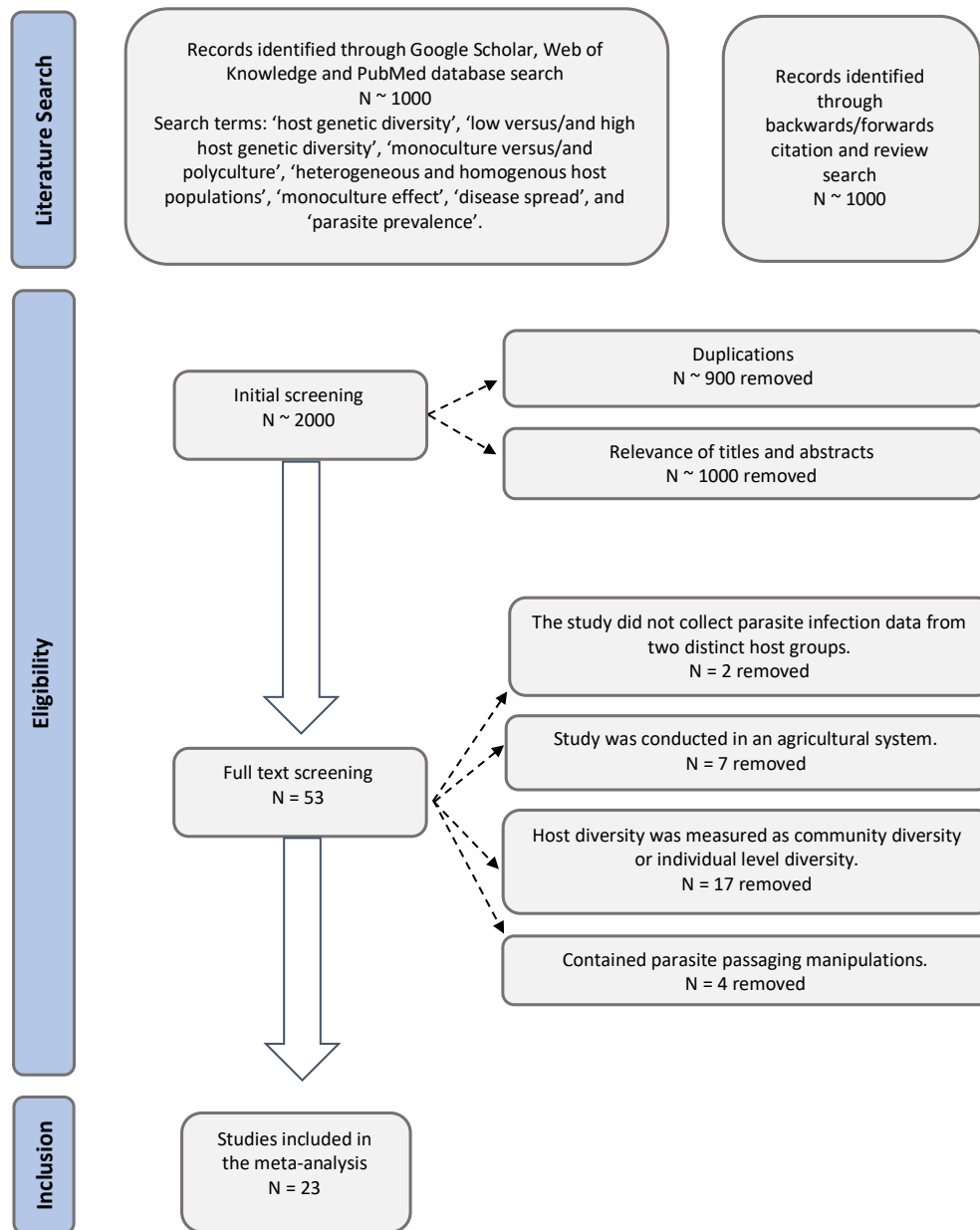
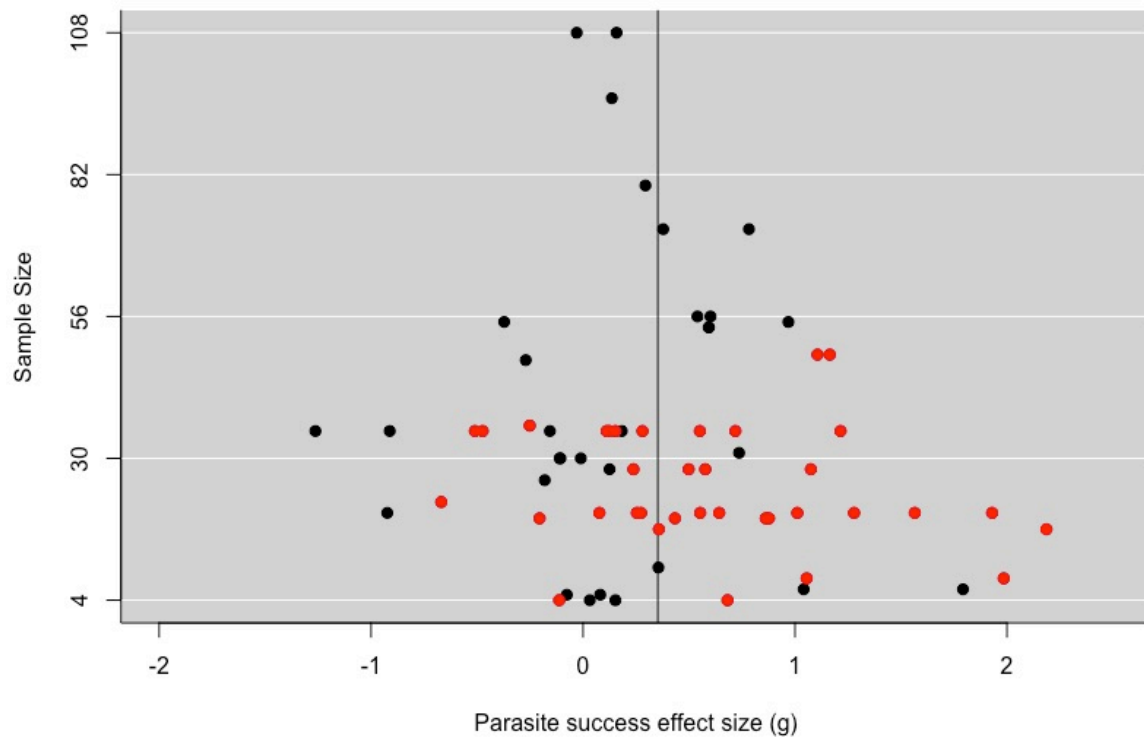


Figure 2: Impact of host and parasite characteristics on the effect of host genetic diversity on parasite success. Positive values indicate that low host genetic diversity has an impact on parasite success (i.e., a negative association between genetic diversity and parasite success). Negative values represent the opposite relationship. The dashed line (effect size of zero) represents no relationship between host genetic diversity and disease spread. Moderator analysis of (A) host reproduction mode: asexual (n = 5), both (n = 2), and sexual (n = 60) effect sizes, (B) of parasite functional group between microparasite (n = 57) and macroparasite (n = 10) effect sizes, (C) host range between 1 host (n = 13) and >1 host (n = 54) parasite effect sizes, (D) parasite diversity between >1 genotype (n = 15), 1 genotype (n = 15), and >1 species (n = 37) effect sizes, and (E) of the ability of a parasite to cause host death, displayed as yes (n = 57) and no (n = 10) effect sizes. The size of the dot corresponds to the sample size. Effect sizes are shown with 95% confidence intervals.

Supplementary Information



Supplementary Figure 1: PRISMA flow chart of literature search and study selection process. Studies excluded from the analysis at the full text screening stage are listed in Supplementary Table 2.



Supplementary Figure 2: Funnel plot of the meta-analysis data set. Points on the graph represent the relationship between effect size and experiment sample size for host genetic diversity and parasite success for each study. Points in red represent lab studies and points in black represent field studies. The vertical line shows the effect size predicted by the meta-analysis model.

Supplementary Table 1: Definition of variable terms used in the meta-analysis

Moderator variable	Definition
Study setting	Lab – An experimental study that occurred in a controlled lab environment Field – An experimental or observational study that occurred in the field
Parasite infection measure	Parasite prevalence – Measures of proportion of individuals infected within a given population (e.g., parasite prevalence, virus concentration, % infection of queens, and brood disease infection intensity) Parasite load – Measures of parasite propagules per host (e.g., parasite load, infection intensity, viral load, and parasite abundance) Host mortality – Measures of host mortality and transformed measures of host survival (e.g., mortality, mortality rate, proportion dead, and proportion survived)
Host reproduction	Sexual – A host species within a study that only undergoes sexual reproduction Asexual – A host species within a study only undergoes asexual reproduction Both – A host population within a study undergoing both sexual and asexual reproduction during an experiment
Parasite functional group	Microparasite – A parasite that has short generation times and high rates of reproduction within its host (e.g., fungi, protozoa, viruses, and bacteria) Macroparasite – A parasite that has long generation times and does not necessarily reproduce within its host (e.g., lice, worms, mites, and flies)
Host range	1 Host species – A parasite found to only infect one host species >1 Host species – A parasite found to infect multiple host species
Parasite diversity	1 Genotype – Host populations were infected by 1 parasite genotype >1 Genotype – Host populations were infected by >1 parasite genotype >1 Species – Host populations were infected by >1 parasite species
Ability to cause host death	Yes – A parasite that can cause host mortality No – A parasite that does not cause host mortality

Supplementary Table 2: Studies excluded from meta-analysis at full text screen stage

Study	Reason for exclusion
Abdala-Roberts , L., Gonzalez-Moreno, A., Mooney, K.A., Moreira, X., Gonzalez-Hernandez, A. & Parra-Tabla, V. (2016) Effects of tree species diversity and genotypic diversity on leafminers and parasitoids in a tropical forest plantation. <i>Agricultural and Forest Entomology</i> , 18, 43-51.	Host diversity was measured as community diversity.
Acevedo-Whitehouse , K., Gulland, F., Greig, D. & Amos, W. (2003) Disease susceptibility in California sea lions. <i>Nature</i> , 422, 35.	Host diversity was measured as individual level diversity.
Agha , R., Gross, A., Rohrlack, T. & Wolinska, J. (2018) Adaptation of a Chytrid Parasite to Its Cyanobacterial Host Is Hampered by Host Intraspecific Diversity. <i>Frontiers in Microbiology</i> , 9:921.	Contained parasite passing manipulations.
Alexander , H.M., Roelfs, A.P. & Cobbs, G. (1986) Effects of disease and plant competition on yield in monocultures and mixtures of two wheat cultivars. <i>Plant Pathology</i> , 35, 457-465.	Study was conducted in an agricultural system.
Andras , J.P. (2017) Genetic variation of the Caribbean sea fan coral, <i>Gorgonia ventalina</i> , correlates with survival of a fungal epizootic. <i>Marine Biology</i> , 164:130.	Host diversity was measured as individual level diversity.
Browning , J.A. & Frey, K.J. (1969) Multiline cultivars as a means of disease control. <i>Annual Review of Phytopathology</i> , 7, 355-382.	Study was conducted in an agricultural system.
Campbell , G., Noble, L.R., Rollinson, D., Southgate, V.R., Webster, J.P. & Jones, C.S. (2010) Low genetic diversity in a snail intermediate host (<i>Biomphalaria pfeifferi</i> Krass, 1848) and schistosomiasis transmission in the Senegal River Basin. <i>Molecular Ecology</i> , 19, 241-256.	Host diversity was measured as individual level diversity.

Ellison, A., Cable, J. & Consuegra, S. (2011) Best of both worlds? Association between outcrossing and parasite loads in a selfing fish. <i>Evolution</i> , 65, 3021-3026.	Host diversity was measured as individual level diversity.
Hendrick, P.W., Kim, T.J & Parker, K.M. (2001) Parasite resistance and genetic variation in the endangered <i>Gila topminnow</i> . <i>Animal Conservation Forum</i> , 4, 103-109.	Host diversity was measured as individual level diversity.
Hughes, W.O.H. & Boomsma, J.J. (2006) Does genetic diversity hinder parasite evolution in social insect colonies? <i>Journal of Evolution Biology</i> , 19, 132-143.	Contained parasite passaging manipulations.
Jinks, J.L. & Grindle, M. (1963) Changes induced by training in <i>Phytophthora infestans</i> . <i>Heredity</i> , 18, 245-264.	Contained parasite passaging manipulations.
Kerstes, N.A.G. & Wegner, K.M. (2011) The effect of inbreeding and outcrossing of <i>Tribolium castaneum</i> on resistance to the parasite <i>Nosema whitei</i> . <i>Evolutionary Ecology Research</i> , 13, 681-696.	Host diversity was measured as individual level diversity.
Knott, E.A. & Mundt, C.C. (1990) Mixing ability analysis of wheat cultivar mixtures under diseased and nondiseased conditions. <i>Theoretical and Applied Genetics</i> , 80, 313-320.	Study was conducted in an agricultural system.
Kubinak, J.L., Cornwall, D.H., Hasenkrug, K.J., Adler, F.R. & Potts, W.K. (2014) Serial infection of diverse host (<i>Mus</i>) genotypes rapidly impedes pathogen fitness and virulence. <i>Proceedings of the Royal Society B: Biological Sciences</i> , 282: 20141568.	Contained parasite passaging manipulations.
Lai, R., You, M., Zhu, C., Gu, G., Lin, Z., Liao, L., Lin, L. & Zhong, X. (2017) <i>Myzus persicae</i> and aphid-transmitted viral disease control via variety intercropping in flue-cured tobacco. <i>Crop protection</i> , 100, 157-162.	Study was conducted in an agricultural system.
Liao, H., Luo, W., Pal, R., Peng, S. & Callaway, R.M. (2016) Context-dependency	

and the effects of species diversity on ecosystem function. <i>Biological Invasions</i> , 18, 3063-3079.	Host diversity was measured as community diversity.
Lopez-Urbe , M.M., Appler, R.H., Youngsteadt, E., Dunn, R.R., Frank, S.D. & Tarpy, D.R. (2017) Higher immunocompetence is associated with higher genetic diversity in feral honey bee colonies (<i>Apis mellifera</i>). <i>Conservation Genetics</i> , 18, 659-666.	Host diversity was measured as individual level diversity.
Meagher , S. (1999) Genetic diversity and <i>Capillaria hepatica</i> (Nematoda) prevalence in Michigan deer mouse populations. <i>Evolution</i> , 53, 1318-1324.	Host diversity was measured as individual level diversity.
Meyer-Lucht , Y., Otten, C., Puttker, T., Pardini, R., Metzger, J.P. & Sommer, S. (2010) Variety matters: adaptive genetic diversity and parasite load in two mouse opossums from the Brazilian Atlantic forest. <i>Conservation Genetics</i> , 11, 2001-2013.	Host diversity was measured as individual level diversity.
Mitchell , C.E., Tilman, D. & Groth, J.V. (2002) Effects of grassland plant species diversity, abundance, and composition on foliar fungal disease. <i>Ecological Society of America</i> , 83, 1713-1726.	Host diversity was measured as community diversity.
O'Brien , S.J., Roelke, M.E., Marker, L., Newman, A., Winkler, C.A., Meltzer, D., Colly, L., Evermann, J.F., Bush, M. & Wildt, D.E. (1985) Genetic basis for species vulnerability in the cheetah. <i>Science</i> , 227, 1428-1434.	Host diversity was measured as individual level diversity.
Palmer , K.A. & Oldroyd, B.P. (2003) Evidence for intra-colonial genetic variance in resistance to American foulbrood of honey bees (<i>Apis mellifera</i>): further support for the parasite/pathogen hypothesis for the evolution of polyandry. <i>Naturwissenschaften</i> , 90, 265-268.	Host diversity was measured as individual level diversity.
Pilet , F., Chacon, G., Forbes, G.A., Andrivon, D. (2006) Protection of susceptible potato	

cultivars against late blight in mixtures increases with decreasing disease pressure. <i>Phytopathology</i> , 96, 777-783.	Study was conducted in an agricultural system.
Rottstock, T., Joshi, J., Kummer, V. & Fischer, M. (2014) Higher plant diversity promotes higher diversity of fungal pathogens, while it decreases pathogen infection per plant. <i>Ecological Society of America</i> , 95, 1907-1917.	Host diversity was measured as community diversity.
Schmid-Hempel, P. & Crozier, R.H. (1999) Polygyny versus polyandry versus parasites. <i>Philosophical Transactions of the Royal Society Biological Sciences</i> , 354, 507-515.	Host diversity was measured as individual level diversity.
Spielman, D., Brook, B.W., Briscoe, D.A. & Frankham, R. (2004) Does inbreeding and loss of genetic diversity decrease disease resistance? <i>Conservation Genetics</i> , 5, 439-448.	Host diversity was measured as individual level diversity.
Velo-Anton, G., Rodriguez, D., Savage, A.E., Parra-Olea, G., Lips, K.R. & Zamudio, K.R. (2012) Amphibian-killing fungus loses genetic diversity as it spread across the New World. <i>Biological Conservation</i> , 146, 213-218.	The study did not collect parasite infection data from two distinct host groups.
Whitehorn, P.R., Tinsley, M.C., Brown, M.J.F., Darvill, B. & Goulson, D. (2011) Genetic diversity, parasite prevalence and immunity in wild bumblebees. <i>Proceedings of the Royal Society B: Biological Sciences</i> , 278, 1195-1202.	The study did not collect parasite infection data from two distinct host groups.
Yang, B., Ge, F., Ouyang, F. & Parajulee, M. (2012) Intra-species Mixture Alters Pest and Disease Severity in Cotton. <i>Environmental Entomology</i> , 41, 1029-1036.	Study was conducted in an agricultural system.
Zhu, Y., Chen, H., Fan, J., Wang, Y., Li, Y., et al. (2000) Genetic diversity and disease control in rice. <i>Nature</i> , 406, 718-722.	Study was conducted in an agricultural system.