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Evolutionary biology

Parasitism shapes selection by drastically reducing host fitness and increasing host fitness variation

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Determining the effects of parasites on host reproduction is key to understanding how parasites affect the underpinnings of selection on hosts. Although infection is expected to be costly, reducing mean fitness, infection could also increase variation in fitness costs among hosts, both of which determine the potential for selection on hosts. To test these ideas, we used a phylogenetically informed meta-analysis of 118 studies to examine how changes in the mean and variance in the outcome of reproduction differed between parasitized and non-parasitized hosts. We found that parasites had severe negative effects on mean fitness, with parasitized hosts suffering reductions in fecundity, viability and mating success. Parasite infection also increased variance in reproduction, particularly fecundity and offspring viability. Surprisingly, parasites had similar effects on viability when either the male or female was parasitized. These results not only provide the first synthetic, comparative, and quantitative summary of the strong deleterious effects of parasites on host reproductive fitness, but also reveal a consistent role for parasites in shaping the opportunity for selection.

1. Introduction

Determining host fitness costs of parasite infection is necessary for understanding the ecology and evolution of host–parasite interactions. Infection by parasites could have two key effects underlying the potential for adaptive evolution through selection. First, infection can reduce mean fitness, which generally strengthens selection when driven by antagonistic interactions [1,2]. Second, infection could also increase variation in fitness components (e.g. premating effects on mating success or postmating effects on viability). Such effects could arise if infection responses vary among individuals, differentially affecting fitness. Such differences may also depend on host sex because males often suffer greater parasite-mediated costs [3,4].

Despite considerable interest in parasites as selective agents generating fitness costs for hosts [3,5–9], we have no general, quantitative understanding of the extent to which parasitism may differentially affect host reproductive fitness components and their variance. Identifying which fitness components are most strongly affected by parasites will allow for predicting when parasite-mediated selection is likely to be strong or weak. Moreover, evaluating these central ideas in a robust comparative framework will allow for a better understanding of how parasitism affects selection and the potential for adaptive evolution [1,10,11].

To evaluate these core ideas and quantitatively summarize how parasitism affects the potential for selection through reproduction, we used a phylogenetically informed meta-analysis of 118 studies across a broad array of host species. First, we asked what the overall effects of parasites are on host reproductive fitness. Second, we asked if the effects of parasites on premating effects differ

from those of postmating effects. Third, we asked how parasites affect individual host fitness components. Lastly, we asked if effects of parasites on host fitness vary with host sex. For each question, we assessed how parasitism affected the mean and variation of host fitness responses.

2. Methods

Here we present a short description of our approach; details can be found in the electronic supplementary material. We performed a systematic literature search for studies investigating how parasitism affects host reproduction on ISI Web of Science, applying the following criteria. First, studies had to measure the impact of parasitism on host reproduction. Because we were interested in the effects of parasitism on reproduction, not how the effects vary with the intensity of parasitism, studies had to measure differences in a metric of reproduction between non-parasitized and parasitized groups. Second, we included studies of parasitic organisms as both parasites and parasitoids (excluding brood or social parasites). We included experimental, observational, field, and laboratory studies.

For each study, we extracted several moderators: (i) infection timing, (ii) fitness component, and (iii) host sex. Infection timing represented the stage of reproduction affected by parasitism: premating or postmating; the fitness component represented aspects of reproduction impacted by parasites: offspring viability, mating success, fertility, fecundity, and competitiveness (i.e. competition for mates); and host sex was the sex of the parasitized host.

To compare differences in mean responses (i.e. effects of parasites on mean fitness) we used Hedge's g, or the standardized mean difference (SMD)—the difference between two groups (parasitized and non-parasitized hosts) in units of standard deviations [12]. This effect size estimate corrects for bias associated with small sample sizes [12]. Negative values of Hedge's g represent detrimental effects of parasites and positive values represent advantageous effects. To compare differences in variances between parasitized and non-parasitized groups (a measure akin to, but not equivalent to the opportunity for selection) we used the natural logarithm of the ratio between the coefficients of variation from two groups (lnCVR, [13]), correcting for bias per the recommendation of Senior *et al*. [14]. lnCVR is a useful metric for comparing how parasitism affects fitness variance, as fitness variance is the raw material that selection acts upon (see also [15]) and lnCVR is a standardized measure amenable to meta-analyses that is not biased by differences in the mean [14]. Though this is not a standard metric of the opportunity for selection, which is variance in relative fitness [16,17], our use of lnCVR is informative because it provides a standardized metric for comparing variances among studies [18]. Positive lnCVR values indicate parasites increase variance in host responses, negative values indicate decreased variance. We include back-transformed values to ease interpretation.

For each of our four questions, we constructed separate multi-level mixed-effect models with categorical moderators as fixed effects [19,20]. Because most studies ($n = 81$) had multiple effect sizes, we nested each effect size within study, including both terms as random effects [20]. To take into account shared evolutionary histories of hosts and parasites [21], we constructed phylogenies of host and parasite taxa using the *rotl* package [22] to trim the Open Tree of Life [23] to include only host ($n = 102$) or parasite taxa ($n = 63$) and included phylogeny as a random effect [21,24].

We used a restricted maximum-likelihood estimator to calculate residual heterogeneity (τ^2) among effect sizes [25], providing a measure of heterogeneity due to differences in parameters measured, studies included, and taxa investigated [26,27]. We built all models with the rma.mv function in *metafor* [19], weighting each effect size by the inverse of its sampling variance [28]. Significance of effect sizes was determined by lack of overlap of the 95% CI with zero; for comparisons among effect sizes we considered them significantly different if their 95% CIs did not overlap.

Multiple effect sizes in our dataset were extracted from studies investigating viruses, which do not have a resolved position on the Tree of Life; thus, we could not include all studies in our complete dataset while also controlling for phylogeny. We therefore conducted three sets of analyses on two separate datasets. The first dataset included only studies for which we could control for both host and parasite phylogenies ($n = 352$ effect sizes from $n = 116$ studies), while the second dataset included viruses ($n = 357$ effect sizes from $n = 118$ studies). The first and second analyses analysed the first dataset with and without accounting for the effects of phylogeny, respectively, while the third analysis on the second dataset did not account for effects of phylogeny.

In our dataset, most studies examined effects of parasites on invertebrate ($n = 84$) and vertebrate ($n = 25$) animal hosts, while only nine studies investigated plant hosts (electronic supplementary material, figure S1a). Most parasites were either fungi, arthropods or platyhelminths ($n = 46$, 18 and 14 studies respectively, electronic supplementary material, figure S1b). Because there were no qualitative differences controlling for phylogeny and excluding viruses (electronic supplementary material, figure S2), not controlling for phylogeny and excluding viruses (electronic supplementary material, figure S3), or removing outliers and including viruses (electronic supplementary material, figure S4), we focus our analyses on the most comprehensive dataset—including viruses and not controlling for phylogeny. Importantly, although the imposed intensity of infection is likely more severe and less variable among individuals on average for some groups (e.g. arthropod and helminth parasites) in laboratory-based studies, which may lead to artificially exaggerated effect sizes when compared to natural populations, we found no difference in the effects of parasites on reproduction between field- and laboratory-based studies, nor between experimental and observational studies (electronic supplementary material).

We found some evidence for publication bias toward large, negative effects of parasites on mean responses (electronic supplementary material, figure S5c), as well as bias toward large, positive effects of parasites on response variance (electronic supplementary material, figure S5f). Importantly, despite biases toward large effects in both analyses, these effect sizes also had a larger sampling variance and so were down-weighted in our analyses.

3. Results and discussion

Our analysis of 357 effect sizes from 118 studies indicates that parasites have a large, detrimental effect on host reproductive fitness ($\theta_{mean} = -0.87$ [−1.07, −0.68], $p < 0.0001$, figure 1*a*). This result, although not previously quantified in a broad comparative fashion as done here, is expected given that parasites usurp host's resources that could otherwise go towards reproduction. While fully expected, the magnitude of this overall reduction (considered a 'large' effect size in meta-analyses, [29]) highlights the importance of parasitism in understanding host reproduction. Notably, all three analyses had a high degree of heterogeneity (I^2 all > 95%; electronic supplementary material, table S1), indicating that there are substantial among study and/or species differences in responses of hosts to parasite infection that may have important ecological and evolutionary implications [28]. Indeed, although the contribution of parasitism in population ecology is increasingly recognized, many studies have not traditionally considered the effects of parasitism, despite many calls to do so [30–33]. Infection by

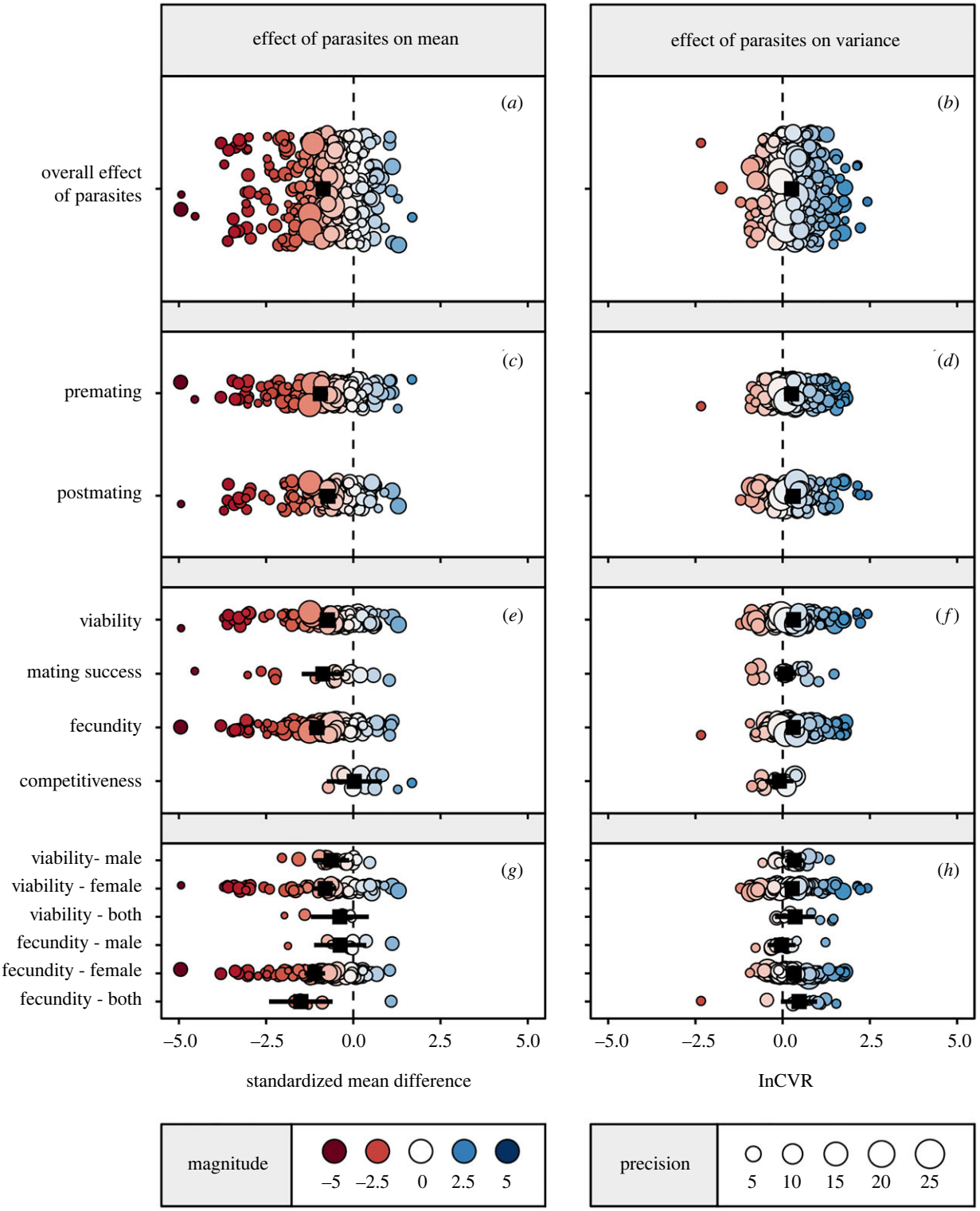

Figure 1. Effects of parasite infection on the underpinnings of selection—mean fitness and fitness variation—experienced by hosts: (*a*) and (*b*) denote overall effects of parasites on host fitness mean responses and response variance, respectively; (*c*) and (*d*) denote effect of parasites on host pre- and postmating mean responses and response variance, respectively; (*e*) and (*f*) denote effects of parasites on host fitness component mean responses and response variance, respectively; and (*g*) and (*h*) denote effects of parasites on host viability and fecundity mean responses and response variance, respectively, broken up by the sex parasitized. Each panel of mean responses only shows SMD effect sizes from −5 to 5 (electronic supplementary material, figure S7 shows the full range). Black squares in each panel denote estimated effects of parasites on mean fitness and fitness variation, respectively. Smaller points in all panels are individual effect sizes, colour denotes magnitude and sign of effect sizes, size denotes precision of estimates (1/SE), and error bars represent 95% CIs.

parasites may be subtle or affect only a few individuals in a population [34,35], but as shown here those effects can be quite substantial.

We also found parasites increased variance in host reproduction by 29.7%, though this effect was slightly weaker than the effect on mean fitness ($\theta_{\text{variance}} = 0.26$ [0.17, 0.35], $p < 0.0001$, figure 1*b*). As with fitness, there was a high degree of heterogeneity in effect sizes (I^2 all > 67%; electronic supplementary material, table S1). That parasitism increases variance in host reproductive fitness highlights the

substantial role of parasites in shaping this second key facet of selection. This, in concert with our finding that parasites drastically reduce host reproductive fitness, illustrates the strong potential for parasites to act as agents of selection. Importantly, these facets of selection are often related. Namely, as mean fitness declines, the variance in fitness should increase, generating a concomitant increase in the opportunity for selection [2,36,37].

While our analysis did not explicitly include phenotypic traits underlying fitness variation, nor standardized selection coefficients, lower fitness and higher variance in fitness are often associated with stronger selection on traits mediating antagonistic interactions [1,2]. Yet, by focusing on how variation in reproductive fitness is affected by parasitism, and not focusing on traits mediating selection *per se*, this approach provides an integrative measure of all organismal traits shaping potential selection [15]. Regardless, our broad comparative analysis has revealed a consistent role for parasitism in shaping the potential for selection. These results both confirm and expand upon the findings of previous meta-analyses that demonstrated a role for parasites in shaping selection experienced by their hosts [38,39], but add to them by revealing the specific underpinnings of potential selection operating.

We further explored which host and parasite groups experienced and drove, respectively, fitness consequences by analysing the effects of parasites on mean fitness and fitness variation among host and parasite groups. The limited sample sizes replicated among host and parasite groups prohibited us from breaking up all other analyses by host and parasite group. Among hosts, we found that arthropods, fish, molluscs, and plants experienced severe fitness reductions, with arthropod and fish hosts experiencing concomitant increases in fitness variation (electronic supplementary material, figure S6a–b, table S2). Among parasites, we found that arthropods, fungi, and platyhelminths strongly reduced host fitness, with fungi and platyhelminths also increasing host fitness variation (electronic supplementary material, figure S6c–d, table S3). These results suggest that host groups experiencing strong fitness consequences of parasitism (e.g. fish, plants, and molluscs) should typically favour the evolution of resistance strategies, while those experiencing no net effect (e.g. birds, mammals) should instead favour tolerance. Likewise, hosts infected with arthropods, fungi, and platyhelminths should generally favour resistance, yet hosts infected with apicomplexans, bacteria, nematodes, and protozoans may instead be expected to favour the evolution of tolerance strategies.

To determine the potential underpinnings of these broader findings, we investigated how mean reproductive fitness and fitness variation differed between pre- and postmating components. We found parasites had large detrimental effects on both components of host reproduction mean responses, though premating effects were 30% greater than postmating on average ($\theta_{premating} = -0.95$ [−1.17, −0.72], $\theta_{postmating} = -0.73$ [−0.99, −0.46], figure 1*c*). Parasites also increased variance in both premating ($\theta_{premating} = 0.25$ [0.15, 0.36], $p < 0.0001$, increasing 28.4%) and postmating response variance ($\theta_{postmating} = 0.30$ [0.17, 0.43], $p < 0.0001$, increasing 35%, figure 1*d*). The disproportionate strength of premating effects imply that they may be critical aspects underlying host fitness and potential selection. However, the roughly equivalent increases in response variance for pre- and postmating reproductive measures suggests they both play similar roles in shaping the opportunity for selection.

To better understand which pre- and postmating measures were associated with the above patterns, we next compared individual fitness components. We found that parasitized hosts had less viable offspring, were less successful in mating, and suffered severely reduced fecundity (figure 1*e*). Surprisingly, parasitism did not reduce mate competitiveness. Parasites also increased variance in both host offspring viability ($\theta_{viability} = 0.31$ [0.18, 0.44], increasing 36.3%) and fecundity ($\theta_{fecundity} = 0.30$ [0.19, 0.41], increasing 35%), though they did not significantly increase variance in mating success, nor significantly reduce variance in competitiveness (figure 1*f*). With the exception of mate competitiveness, parasites reduced mean responses for all pre- and postmating fitness components, with concomitant increases in fitness variance for fecundity and viability. This implies the impact of parasites on selection through both fitness and variances in fitness can occur before or after mating. The lack of an effect on competitiveness, despite an effect on mating success, suggests that other factors beyond competition for mates are affected by parasitism (e.g. female choice [40]), subsequently driving variation in reproductive success. Moreover, no parasite-mediated effect on mating success variation may speak to a general (and consistently negative) impact of parasites on this measure of host fitness.

Finally, we asked if the effects of parasites on offspring viability and fecundity varied with host sex. We focus on these fitness components because there were insufficient sample sizes for the remaining components. We found parasites strongly decreased viability when either males ($\theta_{male} = -0.63$ [−1.15, −0.12]) or females ($\theta_{female} = -0.81$ [−1.13, −0.48]) were parasitized, yet there was no effect if both sexes were parasitized ($\theta_{both} = -0.39$ [−1.22, 0.44], figure 1*g*). By contrast, we found parasites were exceedingly costly to fecundity when females were parasitized ($\theta_{female} = -1.10$ [−1.40, −0.81]), with no effect when males were parasitized ($\theta_{male} = -0.38$ [−1.12, 0.37], figure 1*g*). Parasites had similar effects on variance of offspring viability for males ($\theta_{male} = 0.33$ [0.07, 0.60], increasing 39.1%) and females ($\theta_{female} = 0.27$ [0.12, 0.43], increasing 31%), though parasites only increased variance in fecundity of females ($\theta_{female} = 0.33$ [0.20, 0.47], increasing 39.1%, figure 1*g*). Our finding that both sexes experienced similar fitness costs to offspring viability contrasts with previous evidence whereby either males [3,4] or females [41,42] experienced greater reductions in fitness. We cannot rule out the observed pattern stemming from parasitized males experiencing reduced fertility, though there seems to be an indirect effect of parasites on offspring through males. Indeed, in studies where non-parasitized females mated with parasitized males both hatching success [43,44] and the number of offspring sired by the male [45,46] were reduced, which suggests parasite-mediated effects on male quality may manifest through reproduction, though whether this effect is independent of or related to females is unknown.

4. Conclusion

Parasitism is a ubiquitous species interaction affecting organisms across the tree of life [47,48]. Quantifying effects of parasitism on the underpinnings of selection experienced by host organisms is critical for understanding host (and parasite) adaptive evolutionary dynamics and how

organisms persist in environments subject to parasitic infection. Our meta-analysis of the effects of parasites on host reproduction revealed severe parasite-mediated reductions in mean fitness, with concomitant increases in fitness variation. Because the strength of selection in antagonistic species interactions tends to increase as mean fitness declines, and fitness variance is the raw material that selection acts upon, our results show that parasites are a key agent driving the underlying components of reproductive selection hosts experience.

Data accessibility. Data are available from the Dryad Digital Repositoy: https://doi.org/10.5061/dryad.9zw3r22hj [49].

The data are provided in electronic supplementary material [50].

Authors' contributions. A.Z.H.: conceptualization, data curation, formal analysis, writing—original draft; A.M.S.: conceptualization, formal analysis, writing—review and editing.

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

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

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