Limits on populations of herbivorous insects

by

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Abstract

Herbivorous insect populations are necessarily limited by biotic and abiotic factors. This dissertation seeks to uncover some of those limiting factors using using synthetic comparative statistical analyses including meta-analysis and path modeling. I conduct a meta-analysis on interspecific competition between herbivorous insects, a meta-analysis on stressor interactions on the health of honey bees, and a series of path models on the spatial diversity of aphids. I report a number of novel findings, including that competition limits the populations of herbivorous insects, that anthropogenic honey bee stressors interact antagonistically, and that abotic factors play an important role in the spatial diversity of aphids.

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List of Abbreviations

MCMC	Markov Chain Monte Carlo
SE	Standard error
CI	Confidence interval
d-sep	Direct separation
CIC	Fisher's C
PC	Principal component
SMD	Standard mean difference

Introduction

In this dissertation I explore what limits herbivorous insect populations, both in terms of the performance and size of individual populations and the richness of reproductively-isolated populations across space and clades. Throughout, I try to see the big picture by using synthetic comparative statistical analyses. In other words, I pull together information produced by many previous researchers to get a broad overall view of what ecological features limit herbivorous insect populations.

The first two chapters are formal meta-analyses. The first investigates the extent to which herbivorous insects compete for host-plant resources and tries to identify specific predictors of when competition will occur. I find that although the previous research had left us in doubt, competition is, in fact, a pervasive limiter of herbivorous insect populations, both in the form of direct exploitative competition and in the form of indirect, or apparent, competition mediated through both host-plants and natural enemies.

The second chapter investigates how multiple anthropogenic stressors combine to limit the fitness of domesticated honey bee populations. Using a novel meta-analytic approach that combines data from a number of fully-factorial experiments, I find that, contrary to what has been assumed by most researchers, stressors appear to interact antagonistically, that is, their combined adverse effects on honey bee health are less than would be predicted if the stressors combined additively. This follows a general trend in the ecological research on stressors, namely that antagonistic interactions between stressors are more common than synergistic ones.

The third chapter uses structural equation models to investigate how plant diversity and abiotic environmental diversity limit the species richness of aphid communities. I find that although both plant diversity and abiotic features are influential, which is more important depends on the spatial scale of analysis. This spatial dependency may help explain inconsistencies in previous investigations in this field of research.

In summary, the chapters in this dissertation address a disparate set of research questions. But they are united by a common research approach entailing comprehensive and comparative statistical analysis, and all relate to the general theme of trying to understand the ecological factors that limit populations of herbivorous insects.

Chapter 1. When do herbivorous insects compete? A phylogenetic meta-analysis.

Gwendolyn Bird, Chloe Kaczvinsky, Alan Wilson, and Nate Hardy

Abstract

When herbivorous insects interact, they can increase or decrease each other's fitness. As it stands, we know little of what causes this variation. Classic competition theory predicts that competition will increase with niche overlap and population density. And classic hypotheses of herbivorous insect diversification predict that diet specialists will be superior competitors to generalists. Here, we test these predictions using phylogenetic meta-analysis. We estimate the effects of diet breadth, population density, and proxies of niche overlap: phylogenetic relatedness, physical proximity, and feeding-guild membership. As predicted, we find that competition between herbivorous insects increases with population density and phylogenetic and physical proximity. Contrary to predictions, competition tends to be stronger between than within feeding guilds, and affects specialists as much as generalists. This is the first statistical evidence that niche overlap increases competition between herbivorous insects. However, niche overlap is not everything; complex feeding guild effects indicate important indirect interactions.

Introduction

Herbivorous insects form a major component of terrestrial communities and account for ~ 1/3 of all described species (Grimaldi & Engel 2005). Community assembly and species diversification are thought to depend on how interacting species affect one another's fitness (Levins 1968). For herbivorous insects such effects can be variable, ranging from fierce competition to facilitation. We currently know little of what causes this variation. Knowing more could bring us to a deeper

understanding of how speciation happens (Alatalo *et al.* 2018), and of how communities are structured.

When should we expect herbivorous insects to compete? According to classic competition theory, competition should increase with niche overlap and population density, that is, when more individuals vie for the same resources (Levins 1968). But it is not clear how useful this theory is for herbivorous insects. One problem is that most herbivorous insects tend to occur at low density. This rarity inspired the Green World Hypothesis (Hairston *et al.* 1960): Herbivorous insect populations are under such strong natural enemy pressure that they do not compete for host plant resources. Thus, most plant parts go uneaten and the world is green. Of course, this takes a simplistic view of how plants interact with their insect herbivores. For herbivorous insects, plants are more than food; they can provide refuge from natural enemies, and a place to interact with mutualists. Moreover, not all plant tissues are equally nutritious, and they can vary in their state of defensiveness against herbivory. This means that competition between herbivorous insects can be indirect, as they induce host-plant defenses (Price *et al.* 2011), attract natural enemies (Faeth 1986; Mooney *et al.* 2012), and distract mutualists (Styrsky & Eubanks 2007). If the interactions between herbivorous insects are mostly indirect, classic competition theory predictions might not apply.

Decades of research (Denno et al. 1995; Kaplan & Denno 2007; Radville et al. 2014) have shown us that – at least in experiments – competition between herbivorous insects is common, even if two individuals are never in physical contact, or even on the same host at the same time (e.g., Ammunét *et al.* 2010; Anderson *et al.* 2011; Van Dam & Heil 2011). In the current view, the fitness effects of herbivorous insect interactions do not appear to depend much on niche

overlap (Kaplan and Denno 2007). Thus, classic competition theory seems of little use. However, this view could be warped by two major biases. First, until recently, researchers have had little ability to account for the phylogenetic non-independence of their fitness measures: similarity in competitive ability between two species could be due to similar values for a predictor variable such as trophic mode, or it could be due to shared ancestry. Second, researchers have tended not to control for density when assaying the fitness effects of herbivorous insect interactions. Both biases, phylogeny and density, can be addressed with current meta-analytic approaches.

Predictions about when herbivorous insects compete can also be drawn from classic hypotheses about what drives their speciation. Most herbivorous insects are host-use specialists, and this specificity is commonly assumed to be part of why they are so species rich. But the adaptiveness of specificity is anything but clear (see reviews by Futuyma & Moreno 1988; Forister et al. 2012). Few host-use trade-offs have been identified that would select against broad diets (e.g., Agosta & Klemens 2008). But trade-offs in competitive ability *per se* have yet to be tested; perhaps specialists predominate because they are better indirect competitors.

Here we use phylogeny-informed meta-analysis to address three main questions. (1) On average, do herbivorous insects compete? (2) Does classic competition theory predict the fitness effects of an interaction? (3) Can we predict the outcome of interspecific interactions on the basis of a species' diet breadth? Answers to these questions will advance our understanding of herbivorous insect community assembly and species diversification.

Methods

1. Data

To assemble a set of relevant and comparable studies, we began with those used by Kaplan and Denno (2007). We then extended that set to include another decade of empirical research, first looking at studies that cited Kaplan and Denno (2007). We conducted Google Scholar searches for the following terms: “interspecific competition insects”, “insect interaction”, “plant-mediated interactions insects”, “enemy-mediated interactions insects”, and “apparent competition insects”. These searches yielded >3,000,000 studies, but we only reviewed the first thousand results for each search term. As we approached that point, studies were largely repeats of previous hits, not peer-reviewed, or did not examine the effects of herbivorous insect interactions on their fitness.

Literature searches were conducted from 5-15 December 2017. Most studies were not suitable for inclusion in our meta-analysis. To be included, a study needed to satisfy five criteria: it must (1) have been published in a peer-reviewed journal; (2) examine the interaction between two or more species of herbivorous insects; (3) unambiguously identify the focal species, (4) measure proxies of fitness of the focal species, and (5) provide effect means, variances, and sample sizes. Exceptions to the last criterion were made for studies that reported results as population proportions. Sixty-four studies met these criteria and were added to the core set of studies examined by Kaplan and Denno (2007). However, the same criteria also required us to remove 46 of the studies used by Kaplan and Denno (2007), most often because they did not unambiguously identify the focal species or did not provide treatment means, sample sizes and variances. In the end, we examined a total of 167 studies that included 1,641 effect sizes. A total of five insect orders and 179 species were represented, after standardizing scientific names using

the Global Names Resolver (Accessed 12 July 2018, https://resolver.globalnames.org/). A list of the studies we used in our analysis and a more detailed view of how the studies were chosen is included in the appendix.

Whenever possible, treatment means, variances, and sample sizes were taken directly from tables provided in publications. Otherwise we extracted these values from figures using WebPlotDigitizer (Rohatgi 2018). To make effect sizes comparable across studies, we converted effect size means into Hedge's *g*, which scales mean differences across experimental treatments to unit variance, and weights variances by sample size (Hedges 1981). Note that for Hedge's *g*, the rule of thumb is that effects ~ 0.3 are considered weak, ~ 0.5 are moderate, and > 0.7 are strong (Cohen 1988). These calculations were performed using the Metafor package (Viechtbauer 2010) in R (v.3.2.1 R core team 2013). As mentioned above, most of the analyzed effects were expressed as group means and variances, but some were expressed as the proportion of each of two possible outcomes in an experiment (for example, the proportion of insects choosing to settle on one of two hosts). In these cases, we needed to first calculate an effect size as a log odds ratio, and then convert the log odds ratios to Hedge's *g*.

2. Model parameterization

Response variables – measures of fitness changes

Studies of herbivorous insect interactions have measured fitness changes with a variety of proxies, that is, fitness components. (Definitions of these proxies are given in Table 1.) We analyzed each of these fitness components individually and in composite, in which case effect sizes from all component types were pooled and weighted equally. Below, we focus on models

of composite fitness and models of abundance, which is the most commonly measured fitness component, and the one that probably best encapsulates fitness overall. Negative effect sizes indicate competition, and positive effect sizes indicate facilitation.

Fitness component	**Units**	**Effect sizes**
Abundance	Number of individuals, per treatment, at end of experiment	430
Body size	Total body mass, body length, or length of specific body part (e.g., femur)	314
Development time	Time to develop from one life stage to another, (e.g., from hatching to pupation)	148
Emigration	Proportion of dispersive individuals in population (e.g., number of alates in a population)	48
Fecundity	Number of eggs or egg batches laid	269
Feeding preference	Proportion of insects that feed on particular host resource in two-choice trial	19
Oviposition	Proportion of insects that oviposit on particular host resource in two-choice trial	38
Relative growth rate (RGR)	Change in body size over time	79
Survival	Proportion of population surviving	271
Others	Fitness components that did not fit into any of these categories	30

Table 1: Analyzed fitness components. Across published studies, changes in fitness were measured with nine variables, referred to here as fitness components. The last column gives the number of effect size for each component type.

Fixed effects – predictors of fitness changes

We first estimated the average impact of insect interaction on each fitness component by fitting linear models in which the intercept was the only fixed effect. We then attempted to explain the variation in how herbivorous insect interactions affected their fitness by considering models with several potential explanatory variables: (1) *Within vs. between species* was a binary variable, distinguishing between intraspecific and interspecific interactions. (2) *Population density* was a binary variable, distinguishing between experiments in which population densities were the same in control and treatment groups from experiments in which they were higher in treatments. It would have been preferable to parameterize the absolute density of insect populations in each experiment, and to directly compare experimental densities to those that are typical in nature. But we lacked sufficient information. (Note that previous authors have expressed concern that much of the published research comparing intraspecific and interspecific interactions has been biased by ignoring these kinds of density disparities (Connell 1983; Inouye 2001). (3) *Diet breadth* was a continuous variable: a count of known host plant families for an insect species. This information was taken from (Hardy et al. 2018). We also examined models in which diet breadth was a binary variable, distinguishing between specialists (one host plant family) and generalists (two or more host plant families). It made no qualitative difference. Summaries of these analyses are provided in appendix; below we discuss only analyses with the continuous diet-breadth predictor. (4) *Phylogenetic distance* was a continuous variable, quantifying the evolutionary divergence between a focal and competitor species. It was calculated with the cophenetic function from the R package Ape (Paradis *et al.* 2004), using a phylogeny estimated from published DNA sequence data. (5) *Spatial separation* was a binary variable. Studies were classified as either permitting or forbidding physical contact between the focal and competitor

species. We considered physical contact permissible if insects fed on the same host and tissue (e.g., leaves, roots, or stems) unless the experimental design took measures to isolate individuals on the same tissue type. (6) *Temporal separation* was coded as a three-level factor. In the first level, the competitor was introduced at the same time as the focal species. In the second level, the competitor was introduced before and remained after the introduction of the focal species. In the third level, the competitor was introduced and removed before the introduction of the focal species. Significant fitness effects when herbivores are temporally separated indicate important indirect interactions. (7) *Feeding guild* was coded as a factor with three levels: chewers (including leaf and root feeders), sap-feeders, and internal feeders (including gallers, leaf miners, and stem borers). Because the majority of herbivorous insects are chewers, this category was used as the reference in comparison to other guilds. Although it could have helped us understand indirect interactions, we were unable to include a variable indicating whether tri-trophic indirect interactions (via mutualists or natural enemies) could have occurred in an experiment, as too few studies were unambiguously tri-trophic.

Random effects

The co-variates in our models do not vary independently. Specific causes of non-independence include the phylogenetic relatedness of focal species, experimental design differences between research groups, and the year a study was published. We sought to account for these sources of non-independence by including them as random variables in our regression models. To account for non-independence caused by researchers using similar methods, we grouped researchers who had written papers together and included those groups as a random term in all models. (Alternative models with un-grouped studies as random effects yielded similar results.)

We used two approaches to account for phylogenetic relatedness. One set of models specified explicit co-variance structures based on estimated phylogenetic relationships among species (see appendix for details). We refer to these as phylogenetic models. A second set of models approximated phylogenetic relatedness with nested random effects corresponding to three levels of hierarchical classification: genus, family and order. We refer to these as taxonomic models. The phylogenetic models more accurately express evolutionary relationships, but since phylogenetic data were not available for all species, the taxonomic models were more inclusive. Moreover, the phylogenetic and taxonomic models imply distinct evolutionary processes; the phylogenetic models imply that traits evolve gradually, via Brownian Motion over phylogenetic branches, whereas the taxonomic models imply a more saltatory process. If fixed effect estimates are consistent across phylogenetic and taxonomic models, it suggests that the results are robust to our assumptions about macro-evolutionary processes.

3. Analysis

We fit mixed effect meta-regression models using the Bayesian approach implemented in the R package MCMCglmm (Hadfield 2010). Each MCMC chain was run for 1,000,000 iterations, with a 500,000 iteration burn-in and a 100-iteration thinning interval. The Geweke diagnostic (Plummer *et al.* 2006) was used to test for convergence; all models were sampled adequately from the stationary distribution. To avoid problems with repeatedly testing the same data for different effects, and to gain a more comprehensive view of how potential predictor variables interact, all fixed effects were included in each model, with two exceptions: diet breadth and pairwise phylogenetic distance. Data for these variables were available for only a subset of species. Specifically, inclusion of diet breadth as a co-variate would have required us to drop 183

of 1429 effect sizes from the composite fitness taxonomic model and 112 of 1219 effect sizes from the composite fitness phylogenetic model. And inclusion of the phylogenetic distance between focal and competitor species would have required dropping 437 and 227 effect sizes from the taxonomic and phylogenetic models, respectively. Therefore, we excluded these co-variates from our main models, and independently estimated their effects on fitness.

To test for publication bias, we used three functions in the R package Metafor. We first checked a funnel plot for strong asymmetries (appendix), which showed a small positive skew, but appeared close to symmetrical. We then performed Egger's regression test (Nakagawa & Santos 2012; Egger *et al.* 2015) , and the trim-and-fill test (Duval & Tweedie 2000) . Statistical tests for bias were inconsistent; Eggar's test indicated that publication bias was significant ($z = 9.17$, $p < 0.0001$), which contradicted the trim-and-fill test, which indicated no missing studies (SE = 23).

Results

1. Average fitness effects

Do herbivorous insects tend to compete? It depends on how fitness and evolutionary ancestry are parameterized (Fig. 1). In the composite fitness taxonomic model (all fitness components combined, nested random effects from taxonomic classification, no fixed effects), the mean fitness effect was significantly less than zero (intercept: -0.30, p-value: 0.013). By contrast, in the composite fitness phylogenetic model (with an explicit phylogeny-derived co-variance structure), the interaction between herbivorous insects had a non-significant impact on fitness, although the mean effect was similar in sign and magnitude (intercept: -0.24, p-value: 0.29). Results were more consistent when fitness changes were measured via changes in abundance;

interactions between herbivorous insects were significantly negative in both taxonomic (intercept: -0.58, p-value: 0.0022) and phylogenetic models (intercept: -0.46, p-value: 0.016). For other fitness components (body size, fecundity, and survival), competition was not significant (Fig. 1).

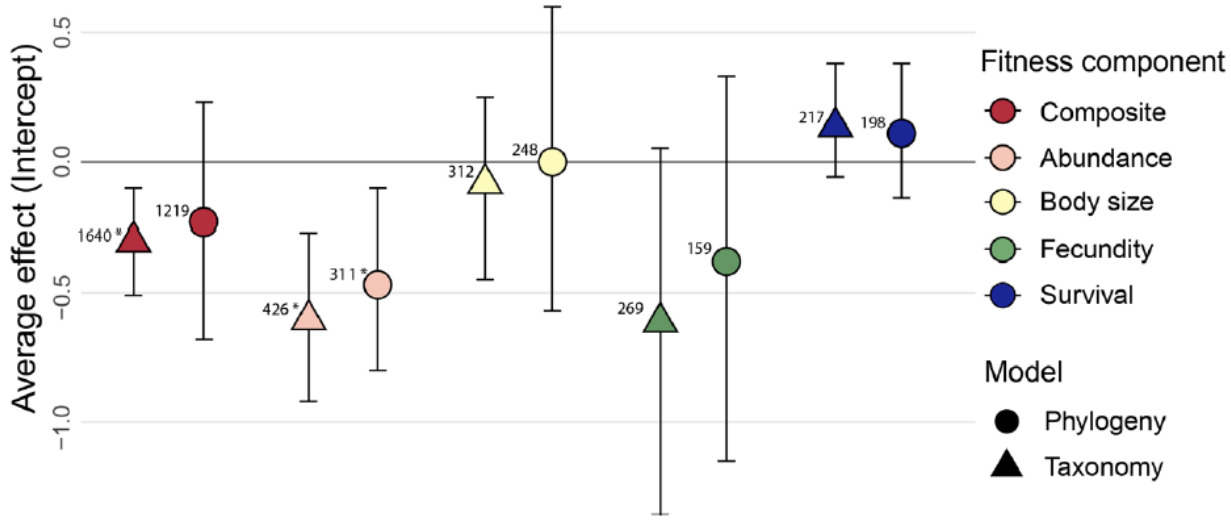

Figure 1. Average effect of interaction between herbivorous insects for each fitness metric. Units are in measurements of Hedges' *g* with units of standard deviation weighted by sample size. Vertical bars give ± 95% high posterior probabilities for effect means, asterisks denote significance to the 0.05 level, the number near each mean effect denotes sample size.

2. Variation in fitness effects

Estimated effects of model predictors are given in the appendix. For the most part, effects were qualitatively similar across response variables. Here we mention only the significant and near significant effects. Significant effects for all fitness components are given in Figs 2 and 3.

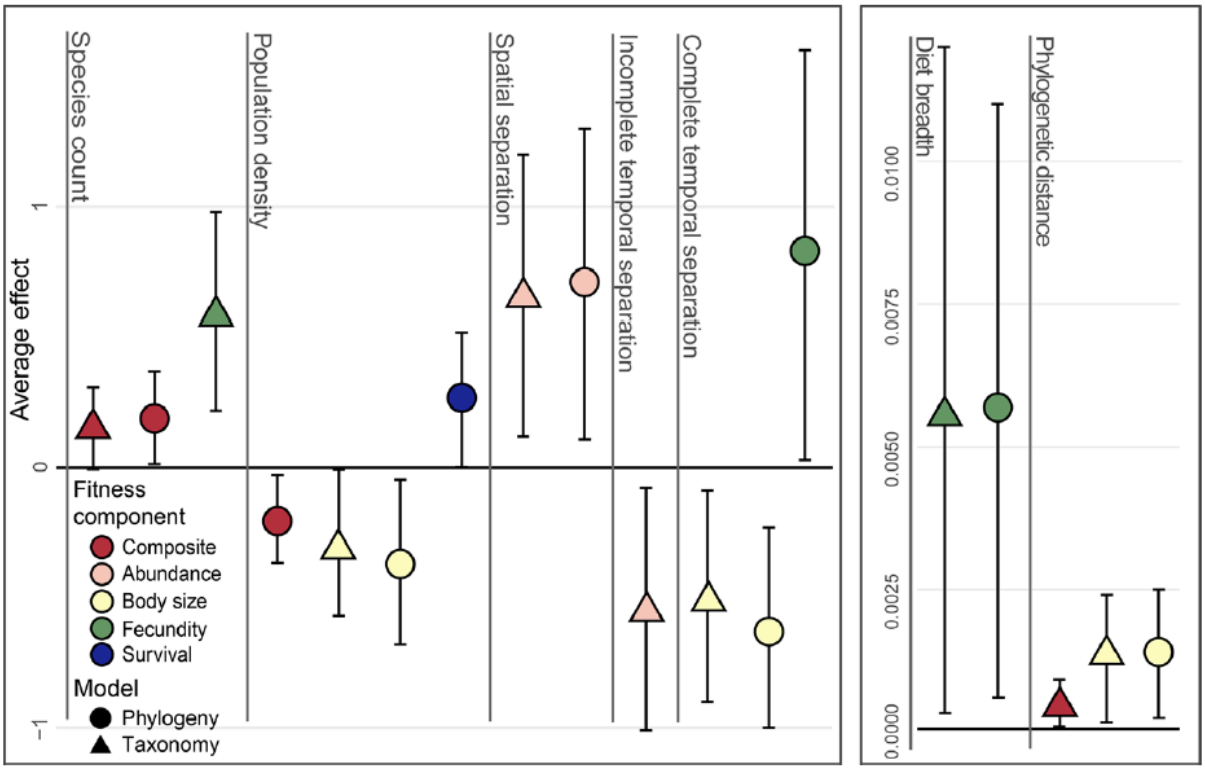

Figure 2. Significant fixed effects for all fitness components. Units are in measurements of Hedges' *g* with units of standard deviation weighted by sample size. Vertical bars give ± 95% high posterior probabilities for effect means. Note that diet breadth and phylogenetic distance are

given on a different scale than the other components. Vertical bars give ± 95% high posterior probability densities for effect means.

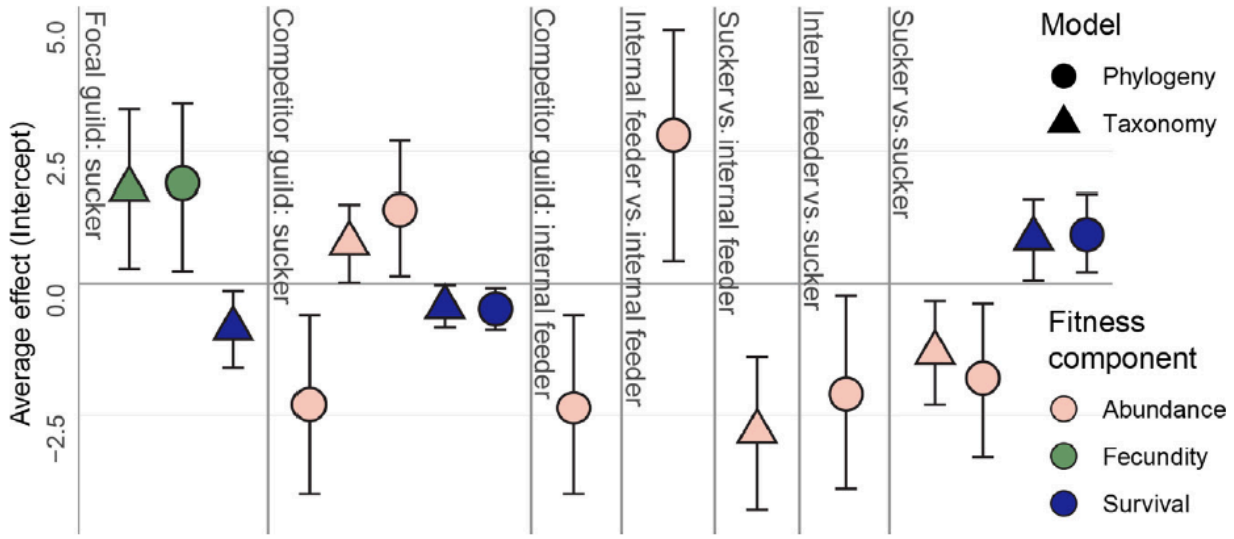

Figure 3. **Significant effects from feeding guild on all fitness components.** Units are in measurements of Hedges' *g* with units of standard deviation weighted by sample size. Vertical bars give ± 95% high posterior probabilities for effect means. For statistical interaction effects, the focal insect's feeding guild is given first, followed by the competitor insect's guild. Vertical bars give ± 95% high posterior probability densities for effect means.

Composite fitness

First of all, competition was stronger within than between species (taxonomic model; coefficient: 0.16, p-value: 0.047, phylogenetic model; coefficient: 0.20, p-value: 0.027). Population density negatively affected fitness in the phylogenetic model (coefficient: -0.20, p-value: 0.026) and taxonomic model (coefficient: -0.13, p-value: 0.067). Phylogenetic distance between focal and

competitor species decreased competition in the taxonomic model (coefficient: 0.00048, p-value 0.025). Note that small coefficient of this effect is partly due to the broad range of phylogenetic distances between species; the greatest phylogenetic distances have an effect of -0.34 *g* on fitness. In the taxonomic model, competition increased when the focal insect was a sucker and the competitor species was an internal feeder (coefficient: 1.05, p-value: 0.018).

Abundance

Spatial separation had a positive effect on fitness (taxonomic model coefficient: 0.66, p-value 0.014; phylogenetic model coefficient: 0.71, p-value 0.023), while incomplete temporal separation had a negative effect on fitness in the taxonomic model (coefficient: -0.54, p-value: 0.027). In contrast to the paucity of significant guild effects in the composite fitness models, we found several for abundance (appendix). For brevity, we discuss only the phylogenetic model. Competition was significantly reduced when the competitor was a sucker (coefficient: 1.4, p-value: 0.025), or when both insects were internal feeders (coefficient: 2.7, p-value: 0.014). By contrast, competition was significantly increased when the competitor was an internal feeder (coefficient: -2.3, p-value: 0.0096), and when both insects were sap-feeders (phylogenetic model; coefficient: -1.8, p-value: 0.014).

Other fitness components

Summaries of models on the other fitness components (body size, fecundity and survival) are provided in the appendix. Here, we note only that although diet breadth did not have a significant effect on abundance or composite fitness, it did have significant positive effect on fecundity

(taxonomic model coefficient: 0.0056, p-value: 0.038; phylogenetic model coefficient: 0.0057, p-value 0.027).

Discussion

If we ignore the differences between experiments and between the species that were part of those experiments, it appears that herbivorous insects tend to compete. But when we take those differences into account things appear more complicated. How one herbivorous insect affects the fitness of another can depend strongly on many factors, including phylogenetic relatedness, population density, spatial-temporal separation, and feeding guild. Some of these effects are predicted nicely by classic competition theory; competition is more intense at higher population densities and when insect herbivores are more closely related and overlap more in time and space. But classic competition theory fails to predict the effects of feeding guild; by and large, competition tends to be fiercer between than within guilds. Although this mixture of effects is currently difficult to explain, by estimating them we have made progress. In fact, this is the first study to successfully explain any of the variation in fitness outcomes from herbivorous insect interactions.

Why do our estimates differ from those of previous meta-analyses, in particular Kaplan and Denno (2007)? It could be due to differences between statistical models, or differences in the data analyzed. To find out, we used our statistical models to analyze only the data from Kaplan and Denno (2007). We found some but not all of the same significant effects that we recovered from our main analyses (see appendix for details). Thus, it seems that both the new data and new models have made a difference. Previous meta-analyses have not accounted for phylogenetic

non-independence. To get a sense for the influence of phylogeny, we also looked at models of abundance and composite fitness without phylogenetic random effects. For the most part, the results of these models were qualitatively similar to those of the taxonomic model, except that phylogenetic distance and the distinction between within vs between species were not significant effects in the ancestry-free model (see the appendix). Thus, random phylogenetic effects were influential.

Exactly how we accounted for phylogenetic relatedness also made a difference. These differences could be due to different underlying models of phylogenetic trait evolution, or due to the fact that the phylogenetic models analyzed only a subset of the data in the taxonomic models. To rule out the latter, we fit taxonomic models with only the data used by phylogenetic models. We found that the taxonomic model inferences were robust to this sub-setting; the critical difference between models seems to be in the assumptions they make about evolutionary process. To be clear, the only differences we see are in which effects are significant, specifically, the significance of effects from population density, temporal separation, phylogenetic distance, and feeding guild. By contrast, the effects of spatial separation, diet breadth, and the distinction between within and between species interactions were consistent across taxonomic and phylogenetic models., Let us now consider in greater depth some of these effects.

Almost all of the significant negative effects on fitness were between rather than within feeding guilds. But classic competition theory would predict the opposite, as niche overlap should be greater within than between feeding guilds. The interactions between sap-feeders and internal feeders appear to be especially antagonistic. Across models, the fitness of sap-feeders declines when they interact with internal feeders. And in the phylogenetic models of abundance, the

fitness of internal feeders decreases in the presence of sap-feeders. Since interactions between internal feeders actually tend to improve fitness, it would seem that direct competition for food is insufficient to explain how internal feeders negatively affect the fitness of sap-feeders (Nyman & Julkunen-Tiitto 2000; Giron *et al.* 2016). But sap-feeders and internal feeders might induce plant defenses that are especially harmful to each other. Researchers have shown that the plant defenses induced by sap-feeders are distinct from those induced by chewers (Ali & Agrawal 2012). Given that internal feeders are known to induce dramatic changes in the physiology of their hosts, the same could also be true of sap-feeders and internal feeders, but more research is needed in this area (Oliveira *et al.* 2016).

The only cases in which within feeding-guild interactions were significantly negative were the effects on abundance and survival when both of the interacting species were sap-feeders. Why would these interactions be especially negative? It could be because sap-feeders tend to reproduce rapidly and form dense and persistent aggregations on their hosts. Internal feeders also have persistent relationships with their hosts, but seldom reproduce as rapidly or form such dense aggregations (Ibbotson & Kennedy 1951; Hardy et *al*. 2018). It could also be because the relatively poor diet of sap-feeders leaves them especially vulnerable to changes in host plant physiology (Hardy 2018) .

In accord with classic competition theory, we found that competition tends to be fiercer at higher population density. Nevertheless, we also found that on average herbivorous insects compete even at low densities; in the phylogenetic model of composite fitness, the model intercept remained significantly negative even after accounting for the effects of changing density. In other models, population density did not have a significant effect on fitness. Thus, competition

could be important at the low densities typical in nature. However, our parameterization of population density was quite coarse; we were only able to distinguish between experiments in density increased in treatments, from those in which it was held constant. Densities were likely artificially high even in experiments that did match control and treatment densities. In fact, density is only one of several differences between experimental and natural conditions that could affect the outcome of an interaction between herbivorous insects. For example, in the field, in contrast to most experimental designs, insects might disperse away from poor conditions. Such dispersal would have a cost, but one that could be cheaper than the cost of poor performance on a crowded host. Published experiments have also tended to greatly simplify communities. In most studies only two herbivores interact on one host. And even when designs are more complicated these are nowhere near as complex as what might happen in the field. In particular, more experiments with tri-trophic designs could allow us to address fascinating questions about associational susceptibility and resistance. We look forward to future studies like this.

When assaying fitness effects of herbivorous insect interactions, researchers have tended to look at agricultural pests, which tend to be generalists. Hence the frequency distributions of these species' diet breadth do not match what is typically found in nature (Forister et al. 2015). Nevertheless, the estimated effects of diet breadth on competition were robust to the manner in which diet breadth was parameterized (as a continuous trait, or as binary factor distinguishing between one-host-family specialists and multiple-host-family generalists). Hence, it seems unlikely that our results have been biased by a data set enriched for generalists.

In most models of abundance and composite fitness, competition is exacerbated by spatial contact. This is consistent with predictions from classic competition theory. The phylogenetic

model of abundance also indicated that incomplete temporal separation (where a competitor species has a head start on a common host) tends to increase competition. This could reflect the gradual accumulation of induced plant defenses and natural enemies; an early window of time for feeding before these changes could have lasting impacts on fitness.

The estimated effects of diet breadth on competition were not what would be expected under the traditional view of herbivorous insect diversification, in which speciation is driven by adaptive diet specialization (Ehrlich and Raven 1964; Futuyma and Moreno 1988). In models of composite fitness and abundance, we found no evidence that competition depends on diet breadth. Up to this point, little evidence has been found of the adaptiveness of diet specificity, whereas considerable evidence supports that diet generalism can be relatively cheap and advantageous (e.g., Agosta & Klemens 2008; Peterson et al. 2015, 2016). The adaptiveness of diet specificity might have been in the boost it gave to an herbivore competing for host resources. But instead, we found a weak but significant positive effect of the focal species' diet breadth on fecundity. If anything, it looks as though more polyphagous species are superior competitors. This can be added to the list of challenges for any explanation of herbivorous insect diversification via host use specialization.

We found evidence of extensive facilitation. Nearly a quarter of our effect sizes were greater than $g = 0.30$, indicating that the interaction substantially increased the fitness of the focal species. One herbivore can increase the fitness of another by helping to overwhelm host defenses, distract natural enemies, or improve habitats, for example by creating leaf mines and rolls (Bronstein 2009; Karban *et al.* 2012; Soler *et al.* 2012). Although it was not our main focus, we also used our data to try and predict facilitation by estimating the same fixed effects on

categorical transformations of our response variables (appendix). Specifically, we classified each effect size as either facilitation (Hedges $g \geq 0.30$), competition (Hedges $g \leq -0.30$) or no effect ($-0.29 <$ Hedges $g < 0.29$). We found that facilitation was more likely between than within species (composite phylogeny model, coefficient: 6654.6, p-value: 0.022), and more likely among distantly related species than among closely related species (composite taxonomy model, coefficient: 0.18, p-value: 0.047). This suggests that facilitation is more likely when direct competition is less likely.

Conclusions

What causes competition between herbivorous insects? The last meta-analyses of herbivorous insect interactions (Kaplan and Denno 2007; Radville et al. 2014) provided little resolution. Competition was frequent but could not be predicted by indicators of niche overlap such as phylogenetic relatedness and physical proximity. This was taken to mean that competition among herbivorous insect was largely indirect, mediated via adjacent trophic levels. But note that this interpretation was based largely on a lack of evidence for strong direct interactions. Here, we go further. We identify ways in which the classic theory of direct competition does successfully explain the fitness effects of herbivorous insect interactions: herbivorous insects are more likely to compete when they occur at higher densities, are more closely related, and come into direct contact. Moreover, we find significant statistical evidence *against* certain predictions of the classic theory. Specifically, it seems that in many cases competition tends to be more intense between than within feeding guilds. These latter results underscore the need of accounting for indirect interactions that remain poorly understood. From our perspective, a priority for improving that understanding should be experimental studies on herbivorous insect

interactions that are more explicitly tri-trophic, along with further study of the molecular mechanisms of induced host plant defense.

Chapter 2: Parasites and pesticides act antagonistically on honey bee health

Gwendolyn Bird, Alan E. Wilson, Geoffrey R. Williams, and Nate B. Hardy

Abstract

1. Pesticides and parasites have each been linked to increased mortality in western honey bees (Apis mellifera). Currently, it is uncertain if one makes the other worse; several studies have tested for potential synergistic stressor effects, but results have been mixed.

2. Here, we use a hierarchical meta-analysis of 63 experiments from 26 studies to gain a clearer view of the combined effects of parasites and pesticides on honey bee health.

3. We found that combined pesticide-parasite treatments do tend to be deadlier than uncombined treatments but are significantly less deadly than predicted additive or multiplicative effects. In other words, combined treatment effects are not synergistic, but antagonistic.

4. Much of the previous uncertainty about the combined effects of pesticides and parasites on honey bee health can be attributed to a bias in the previous research against stressor antagonism; many researchers have excluded the possibility of antagonism a priori.

5. Synthesis and applications: When honey bees are stressed by combinations of pesticides and parasites, they fare better than predicted under models of additive or multiplicative stressor interaction. A better understanding of the mechanisms underlying this antagonism could prove critical for effective management of honey bee health.

Introduction

Few species are more vital to modern agriculture than western honey bees (Apis mellifera). They produce several valuable commodities – such as honey, beeswax, and propolis – and more importantly, they provide the lion's share of worldwide crop pollination services, which have been valued at €153 billion (Gallai et al., 2009). Recent overwintering colony losses in the US have been alarming. Since 2010, mortality rates have consistently been above 20%, reaching 37.7% in the winter of 2018-2019 (Bruckner et al., 2018). Increased mortality has also been reported in Europe (Brodschneider et al., 2018; Gray et al., 2019). This has reduced the profitability of bee keeping, and threatens the sustainability of agricultural systems that rely on honey bee pollination.

Declines in honey bee populations appear to have multiple causes (Potts et al., 2010; Ratnieks and Carreck, 2010), the most important of which are increased exposure to parasites and pesticides. The most troublesome parasites have been microsporidian species in the genus Nosema, and the mite species Varroa destructor. Infections by Nosema spp. degrade honey bee midgut integrity and immune response (Paris et al., 2018), while Varroa mites feed upon immature bees and vector several debilitating viruses (Ramsey et al., 2019). As the prevalence of these parasites has increased, so too has honey bee exposure to pesticides (Goulson et al., 2015; Wintermantel et al., 2020). Managed honey bee colonies are often chronically exposed to sublethal doses of pesticides, so much so that pesticide residues are frequently detected in bee products (Mitchell et al., 2017; Mullin et al., 2010). Chronic pesticide exposure can impede development (Friedli et al., 2020; Tomé et al., 2020), impair behaviors such as learning, foraging, and homing (Aliouane et al., 2009; Yang et al., 2008), and increase overall mortality

rates (Rondeau et al., 2015). In sum, much of the recent increase in honey bee mortality can be traced to increases in parasite and pesticide prevalence.

Of course, in the field, honey bees can face multiple stressors simultaneously (Little et al., 2015; Shutler et al., 2014), which raises the possibility of a variety of interactions between stressors. In the simple additive case, the combined effect of parasites and pesticides would be the sum of their individual effects, for example on instantaneous mortality rates. If mortality is measured as the proportion of dead bees in a finite sample of individuals, it makes more sense to express additive effects on a logarithmic scale; otherwise, the predicted additive mortality effect could be greater than 100% (Sih et al., 1998). This logarithmic additive effect is commonly referred to as the predicted multiplicative effect (Côté et al., 2016). Two other possibilities are that stressors combine synergistically, in which case their combined effects are greater than the expected additive or multiplicative effect, or the stressors combine antagonistically, in which case their combined effects are less than the expected additive or multiplicative effect. This classification of stressor interactions could have management implications. Synergistic interactions are thought to reduce the resiliency of a system, and thus could motivate more aggressive and expensive interventions (Côté et al., 2016), whereas antagonistic interactions raise the possibility of mitigating effects between stressors, in which case reducing one stressor could actually be harmful overall (Brown et al., 2013).

Although several types of stressor interaction are possible, the research on how pesticides and parasites affect honey bee heath has focused almost exclusively on potential stressor synergies (e.g. Collison et al., 2016; Sánchez-Bayo et al., 2016). This bias is not without a basis. In addition to the adverse health effects mentioned previously, exposure to sublethal doses of

pesticides can impair honey bee immune function by reducing antimicrobial capacity, delaying wound healing, and lowering the number of circulating hemocytes (Brandt et al., 2017; James and Xu, 2012). Moreover, pesticides can disrupt health-promoting behaviors such as grooming, hive cleaning, and foraging (de Mattos et al., 2017; Henry et al., 2012; Yang et al., 2008). But there is also a basis for expecting antagonism between stressors. Broad surveys of how multiple stressors affect the health of animal populations show that antagonism is as least as common as synergy (Brown et al., 2013; Côté et al., 2016; Darling and Côté, 2008). And for honey bees, there is evidence of mitigating effects between pesticides and parasites: pesticide exposure can reduce the density of Nosema spp. in the midgut epithelium (Aufauvre et al., 2012; Gregorc et al., 2016. Thus, the bias against stressor antagonism in the honey bee health research may be unwarranted.

Studies of the interactions between stressors on honey bee health have had mixed results, with synergism detected in some studies but not in others, but to repeat, previous studies have mostly ignored the possibility of stressor antagonism, and have inconsistently tested for significant non-additive interactions. Here, to improve our view of the effects of pesticide-parasite interactions on honey bee health, we use meta-analysis. Our primary objective is to estimate, across studies, if the effects of combined pesticide-parasite treatments are greater, less than, or indistinguishable from predicted additive or multiplicative effects. We also quantify the relative harm of single and combined stressors, and account for how measures of stress on honey bee health could depend on variations in experimental design.

Methods

To assemble a set of relevant studies we conducted a literature search using the search parameters (bee* or honeybee* or bumblebee*) and (pesticide* or neonicotinoid* or neo-nicotinoid* or pesticide* or acaricide*) and (parasite* or nosema* or virus* or viral* or varroa* or mite*). Literature searches were conducted from 31 May to 17 June 2019 using Google Scholar and 12–13 August 2020 using Web of Science. Studies were also identified from the citations of three recent review articles and one meta-analysis on the interactions between pesticides and parasites on the health of Apis mellifera (Collison et al., 2016; Havard et al., 2020; O'Neal et al., 2017; Sánchez-Bayo et al., 2016).

To be included in our analysis, the study had to use a factorial experimental design where bees were exposed to a) a control treatment without parasites or pesticides, b) parasite treatments, c) pesticide treatments, and d) combined pesticide-parasite treatments. In addition to chemicals that honey bees might encounter while foraging, we included pesticides that are typically used by beekeepers to manage Varroa mite infestations. From the abstracts of studies returned by the literature searches, we identified 102 candidate studies. After more careful review, 75 of these studies were excluded because they did not meet the criteria for inclusion, leaving 27 suitable studies. One more study was excluded because stressor effects on mortality were so high that detecting synergy would have been problematic. For a list of studies used see the appendix. Further details on the search are presented as a PRISMA flow diagram (Moher et al., 2009) in the appendix.

The included studies examine a wide variety of health-related variables, including honey bee mortality, gene expression, behavior, body size, and fecundity. We focused on mortality, as it

was the most commonly used variable and varied least in how it was measured and reported. We only included studies on the worker cast, as there has been little done with queens or drones. Since most of the 26 studies included multiple experimental observations, and since each factorial experiment measured three effect sizes (pesticide-only, parasites-only, and combined pesticide-parasite treatments), the total number of measured effect sizes was 189. Where possible, experimental effects were taken from the original text and tables. Otherwise, data were extracted from graphs with the R (R Core Team, 2019) package metaDigitise (Pick et al., 2019).

To account for variation in experimental design, we also recorded (1) the identity of focal pesticides and parasites, (2) the number of days from the onset of stress to the time of mortality measurement, (3) the life stage (immature or adult) of bees at the beginning of the treatment, and (4) whether bees were housed in hives or in cages. There were insufficient replicates of specific pesticides and parasites to include each as a level in a predictive factor, so pesticides were classified as either neonicotinoid pesticides or non-neonicotinoid pesticides. We were not able to model the effect of pesticide dose, as it was inconsistently reported and is difficult to quantify when mixed with sucrose and provided ad libitum, as was true of many experiments.

Stressor synergism and antagonism is determined relative to a predicted additive effect. As mentioned previously, for a mortality response expressed as the proportion of dead individuals in a finite sample, the use of a predicted multiplicative effect (appendix) as the threshold between synergy and antagonism avoids the problem of predicted mortalities greater than 100% (Côté et al., 2016). But choosing between predicted additive and multiplicative effects can depend more generally on which null model is a better match to the biological dynamics at hand. Predicted additive effects are better suited for stressors with non-overlapping modes of action and for

systems without strong density-dependence, whereas the opposite applies to predicted multiplicative effects (Hay, 1996). The choice of predictive effect can also depend on the particular hypothesis being tested; since predictive additive effects are invariably greater than predictive multiplicative effects, the use of a predicted additive threshold results in a more conservative test for synergy (and a more liberal test for antagonism). Given these considerations, along with gaps in our understanding of how pesticides and parasites might interact to affect honey bee health, we repeated our analyses with each predicted effect type.

Predicted effects were calculated from the proportion of mortality in pesticide-only and parasite-only treatments. The predicted additive is the sum of these proportions, while the predicted multiplicative is the sum of the proportions minus the product of the proportions. To avoid nonsensical predicted additive effects, we examined only experimental observations taken before the summed mortality proportions of individual stressors reached 100%. As mentioned above, this forced us to drop one study from the analysis (Grassl et al., 2018), since only data from the end of the experiment were reported. For consistency, these same data were analyzed in models with predicted multiplicative effects. All effect sizes, both real and predicted, were converted into log risk ratios and variances using the R package metafor (Viechtbauer, 2010; appendix).

Fixed-effect predictor variables, referred to as moderators in a meta-analysis, were selected based on an exploratory analysis using the R package MuMin (Bartoń, 2019). These were (1) treatment type, a binary variable that distinguished between observed combined treatment effects and predicted additive or multiplicative effects; (2) trial duration, measured in days and mean-centered; (3) parasite type, a factor with levels for Nosema spp., viruses, bacteria, or Varroa mites; (4) pesticide type, a binary variable that distinguished between neonicotinoid and non-

neonicotinoid pesticides; (5) accommodation type, that is, whether bees were housed in cages or in hives, and (6) life stage, a binary variable the distinguished between adult and immature bees. Variance inflation tests did not indicate significant multicollinearity between moderators. In addition to these fixed effects, study and trial were included as nested random effects, as the majority of studies included multiple trials and multiple effect observations from each trial.

The two models, additive and multiplicative, were fit using the rma.mv function in metafor (Viechtbauer, 2010), with test statistics of the individual coefficients based on a t-distribution, similar to the Knapp and Hartung method (Hartung and Knapp, 2003; Viechtbauer, 2010; Viechtbauer et al., 2015; appendix). Each of the models had a total of 126 effect sizes, the sum of 63 observed combined treatment effects and 63 predicted additive or 63 predicted multiplicative effects (appendix). To test for significant differences in mortality effects from different parasite classes – the only multi-state discrete fixed effect predictor variable – we used the R package multcomp (Hothorn et al., 2008), with the Holm adjustment to correct for multiple-testing p-value inflation (Holm, 1979).

We also fit a model to get a sense of the relative magnitudes of the main effects of the single and combined stressors. The main fixed and random effects in this model were the same as in the main analyses, except that the pesticide and parasite type variables were excluded (since some treatments lacked one or the other), and the treatment type was a factor with three levels: pesticide-only treatments, parasite-only treatments, and combined-stressor treatments. No multicollinearity was detected between moderators. This model had a total of 189 effect size observations, 63 from each single-stressor treatment and 63 from the combined treatment.

Pairwise comparisons between treatments were conducted using the R package multcomp with the Holm adjustment.

We ran several tests of model fit and bias. To test for publication bias, which occurs when significant results are more likely to be published than non-significant results, we used a version of Egger's test (Egger et al., 1997), implemented in the R package metafor and modified for hierarchical multivariate analyses. We performed this test on the combined-vs-single-stressors model, as it did not contain predicted – that is, unobserved – additive or multiplicative effects. We failed to reject the null hypothesis of no bias (p-value = 0.14). We tested for outliers using Cook's distance (Cook, 1977), as implemented in the R package metafor. These tests only suggested disproportionate influence in our single vs multiple stressors model, and so we carried out a leave-one-out analysis for that model (appendix). We also quantified effect heterogeneity across studies. Rather than using Cochran's Q, which has low power to detect heterogeneity in hierarchical mixed models when the number of studies is small (Gavaghan et al., 2000), we used an I2 test, following Nakagawa and Santos (2012), with code provided by (Viechtbauer, 2019). This estimates the relative proportions of between-study to within-study effect size variance (appendix).

For ease of interpretation, after model coefficients and confidence intervals were estimated, they were transformed back to linear-scale risk ratios. Hence, reported confidence intervals are asymmetric. Risk ratios express the multiplicative increase or decrease of risk of events between treatments (Higgins et al., 2019). A risk ratio of one indicates no effect; a risk ratio of less than one indicates a decrease in mortality; and a risk ratio of greater than one indicates an increase in mortality.

Results

Comparison of combined and predicted additive effects

We found a significant difference between the mean combined treatment effect and the predicted additive effect of parasites and pesticides on honey bee mortality: the combined treatment was 1.44 fold (1.37-1.44±95% CI; p-value < 0.001) less likely to cause mortality than the predicted additive effect (Fig. 4). The interaction between pesticides and parasites was antagonistic.

Trial duration also had a significant effect, with the risk of mortality decreasing by 1.04 fold per day (1.02-1.07 ±95% CI; p-value = 0.002). As trial duration ranged from 3-25 days, our findings suggest that the risk of mortality was 2.40 fold less likely at 25 days than at three days. We also found a significant interaction between treatment and accommodation types: keeping bees in hives, rather than in cages, decreased the risk of mortality by 3.51 fold (1.30 -9.49 ±95% CI; p-value = 0.014). In the vast majority of experiments bees were in cages (114 cage effects vs 12 hive effects). We found no significant differences in the effects of different parasite kinds, between neonicotinoid and non-neonicotinoid pesticides (p-value = 0.59), or between immature and adult worker bees (p-value = 0.57) (Fig. 4, appendix).

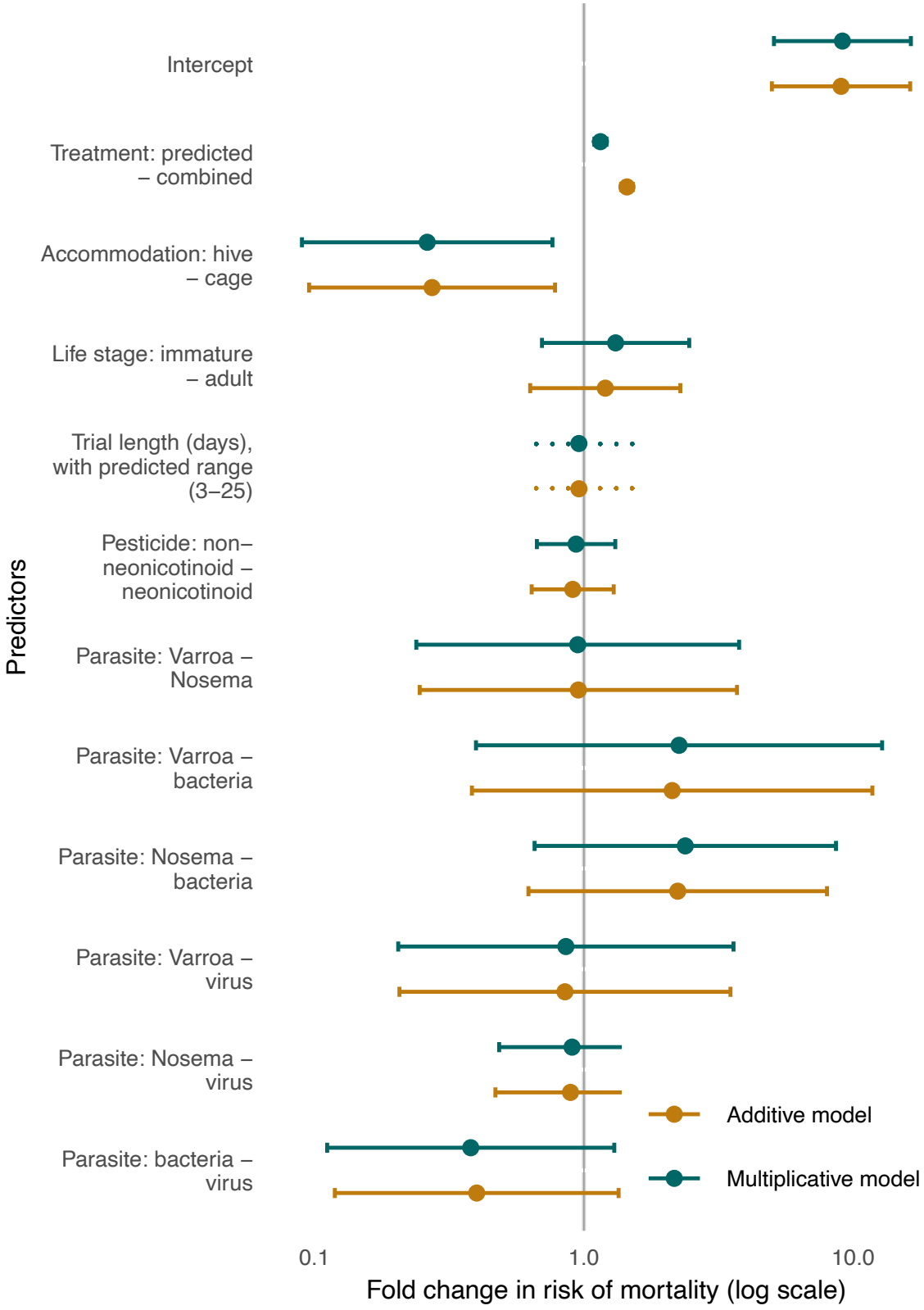
Intercept
Treatment: predicted – combined
Accommodation: hive – cage
Life stage: immature – adult
Trial length (days), with predicted range (3–25)
Pesticide: non–neonicotinoid – neonicotinoid
Parasite: Varroa – Nosema
Parasite: Varroa – bacteria
Parasite: Nosema – bacteria
Parasite: Varroa – virus
Parasite: Nosema – virus
Parasite: bacteria – virus
Predictors
Additive model
Multiplicative model
0.1
1.0
10.0
Fold change in risk of mortality (log scale)

Figure 4. Effects of predictor variables on the relative risk of mortality of honey bees in additive and multiplicative models. The x-axis represents the relative risk of predictors on mortality and is on a log scale. Horizontal bars represent ± 95% CI. Effects greater than one indicate an increased risk of mortality; effects less than one indicate a decreased risk. For binary variables the first state listed is the basis for comparison. For example, for the treatment variable, the predicted combined effects are the basis for comparison; thus effects greater than one indicate the observed combined effects were more harmful, that is, that the interaction was antagonistic. Pesticide category 'mixed' is not shown. The effect of trial length was estimated on a per-day basis; dotted horizontal lines show how such effects accumulate from the third (right side of line) to twenty-fifth (left side) days of a trial.

Comparison of combined and predicted multiplicative effects

Results with predicted multiplicative effects were much the same as they were with predicted additive effects; combined treatments were 1.15 fold (1.09 – 1.21 ±95% CI; p-value < 0.001) less likely to cause mortality than the predicted multiplicative effect (Fig. 4). The effects of trial duration, accommodation, and pesticide and parasite type were also similar to what we found with predicted additive thresholds. Trial length had a 1.04 fold (1.02 – 1.07 ±95% CI; p-value = 0.001) decrease in mortality per day, and bees treated in hives had a 3.66 fold (1.33 – 10.1 ±95% CI; p-value = 0.012) decrease in risk of mortality compared to bees treated in cages. No other results were significant (Fig. 4, appendix).

Comparing single and multiple stressor effects

Although combined pesticide-parasite effects tended to be antagonistic, they were nonetheless significantly more deadly than single-stressor treatments (appendix). On average, combined treatments were 1.29 fold (1.21 – 1.37 ±95% CI; p-value < 0.001) more likely to cause mortality than parasite treatments, and 1.54 fold (1.44 – 1.65 ±95% CI; p-value < 0.001) more likely to cause mortality than pesticide treatments. Also, parasite treatments were 1.20 fold (1.19 – 1.28 ±95% CI; p-value < 0.001) more likely to cause mortality than pesticide treatments. All treatments significantly increased the risk of mortality when compared to controls: parasite treatments increased the likelihood of mortality by 5.44 fold (3.49 – 8.48 ±95% CI; p-value < 0.0001), pesticide treatments increased the likelihood of mortality by 4.54 fold (2.91 – 7.08 ±95% CI; p-value < 0.0001), and the combined treatment increased the likelihood of mortality by 7.00 fold (4.50 – 10.91 ±95% CI; p-value < 0.0001, Fig. 5).

As for the other predictors, we found a significant effect for trial duration – the risk of mortality decreased by 1.05 fold (1.02-1.07 ±95% CI; p-value = 0.0004) per day of the experiment. We also found that bees housed in hives had a 3.02 fold (1.37 – 6.66 ±95% CI; p-value < 0.007) decreased likelihood of mortality when compared to bees housed in cages. There was no significant result for life stage (p-value = 0.40) (Fig. 5, appendix).

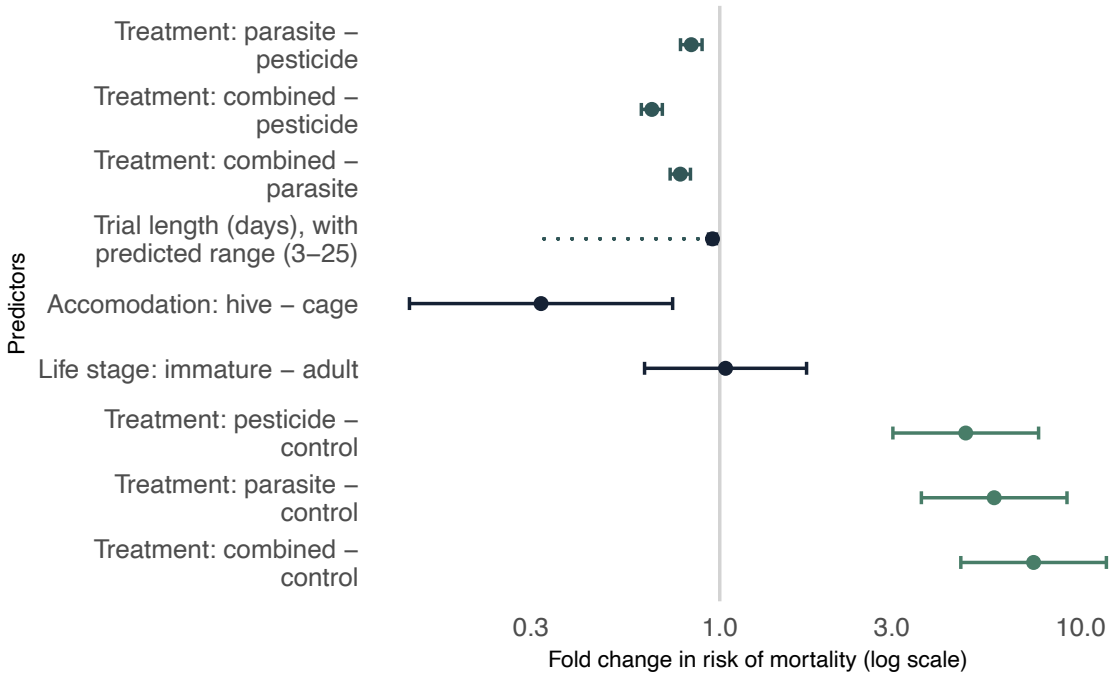

Figure 5. Effects of pesticides, parasites, and combined treatments on the relative risk of mortality of honey bees, along with the effect of other predictor variables. The x-axis shows the relative risk of predictors on mortality and is on a log scale. Horizontal bars represent ± 95% CI. To improve readability, the model was fit without an intercept. Values greater than one indicate an increased risk of mortality and less than one indicate a decreased risk. Shown are pairwise comparisons between treatments, and between treatments and controls. The effect of trial length was estimated on a per-day basis; dotted horizontal lines show how such effects accumulate from the third (right side of line) to twenty-fifth (left side) days of a trial. There were 26 studies and 189 effect sizes included in this model.

Discussion

This meta-analysis shows that pesticides and parasites tend to act antagonistically on the health of honey bees. This antagonism is significant and robust to variation in experimental approaches. It is also robust to whether the predicted combined linear effect is additive or multiplicative. And yet none of the studies that we meta-analyzed had reported pesticide-parasite antagonism. One explanation for this is that researchers have tended to exclude antagonism a priori. Indeed, in more than 75% (20/26) of the analyzed studies, authors make no mention of the possibility of stressor antagonism. Moreover, in ten studies there was no explicit statistical test of non-additive stressor interactions. It could also be that single studies have lacked statistical power, as non-additive interactions take more statistical power to detect than main effects (Slinker, 1998).

To get a sense for how variation in statistical power might have skewed the view of how combinations of parasites and pesticides affect honey bee health, we re-analyzed individual studies. For each study, we compared the difference between the observed combined effect and the predicted additive or multiplicative effects to a null distribution of such differences generated through non-parametric bootstrapping. Using both predicted additive and multiplicative effects, we found significant antagonism in studies which did not report it. With the multiplicative model, which is more conservative for antagonism, we found pronounced antagonism in 19 out of 63 trials, in ten studies. With the additive model, which is more conservative for synergism, we found antagonism in 28 out of 63 trials, in 15 studies (Fig. 6, appendix). Thus, the previous lack of evidence in support of pesticide-parasite antagonism on honey bee health cannot be attributed solely to a lack of single-study statistical power.

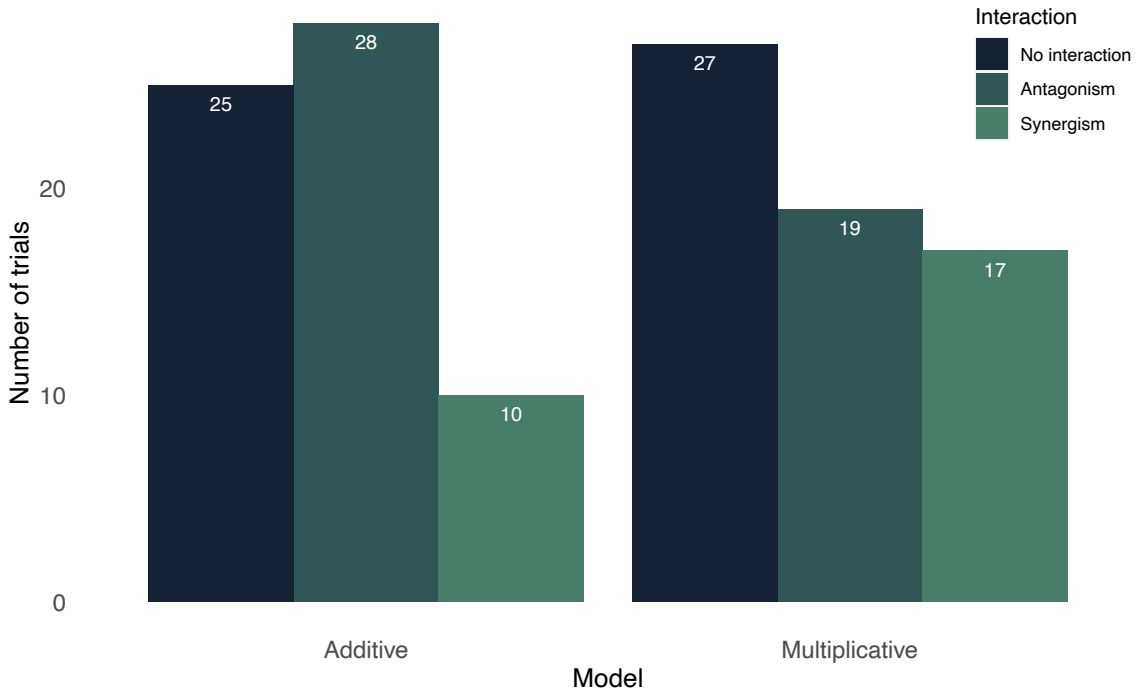

Figure 6. Reanalysis of individual studies of the interactions between parasites and pesticides on honey bee health. We used non-parametric bootstrapping to assess the significance of the observed difference between combined treatment effects and predicted additive and multiplicative effects. An interactions were considered antagonistic when a combined effect was significantly less than a predicted effect, and synergistic when a combined effect was significantly greater than a predicted effect. Sixty three trials from 26 studies were included, detailed results in the appendix.

One counter-intuitive effect estimate from our meta-analysis warrants a brief consideration: increasing trial duration reduced mortality. As a reminder, the trial duration variable was the number of days from the start of an experiment until the experiment ended or the sum of individual-stressor effects exceeded 100%. The most likely explanation of this effect is that

causal relationships run in the opposite direction, and that more lethal parasite and pesticide treatments resulted in shorter trial durations.

It is important to point out that most of the published research has been on small groups of bees kept in laboratory cages and isolated from the rest of their colony; relatively few studies have been of intact hives. Given the degree of interdependence within a honey bee colony and the potential of intact colonies to buffer against stress (Henry et al., 2015; Osterman et al., 2019; Straub et al., 2015), this strains the mapping of experimental effects to what may happen in field conditions. The cages used in experiments on individual bees are likely to be stressors themselves (Williams et al., 2013, 2012), as caged bees are prevented from performing many normal behaviors that could exacerbate the effect of other stressors. Caged bees could also be prevented from excreting toxins during cleaning flights – as may happen in field situations (Coulon et al., 2018). In our analysis, bees kept in cages had more than three-fold the risk of mortality when compared to bees kept in hives. But we had many more effect sizes for experiments on bees treated in cages (114 in our predictor analyses, 171 in our single vs multiple stressor analysis) than for bees treated in hives (12 in our main analysis, 18 in our single vs multiple stressor analysis).

In the meta-analyzed studies, 39 experiments tested the effects of neonicotinoid pesticides, 22 tested the effects of non-neonicotinoid pesticides, and two tested a combination of neonicotinoid and non-neonicotinoid pesticides. We found no significant difference between the two pesticide classes. We cannot rule out that this stems from consistent between-class differences in experimental doses, as information about doses were insufficient. But since most studies attempted to expose honey bees to field-realistic levels, this seems unlikely.

What is the mechanistic basis of the antagonism between pesticides and parasites on honey bee health? We see two main possibilities. The first possibility is mitigation, whereby one stressor ameliorates the effects of another. An example mentioned previously is pesticides reducing the intensity of infections by Nosema spp. (Aufauvre et al., 2012; Gregorc et al., 2016). If this is the case, then reducing pesticide exposure could actually be detrimental to honey bee health, although we found no evidence of this. The second possibility is tolerance induction, whereby one stressor activates a plastic stress-compensation phenotype that confers resistance to a broad array of stressors (Vinebrooke et al., 2004). For example, both pesticide exposure and parasite infection can increase oxidative stress and induce generalized physiological mechanisms for restoring redox homeostasis (Kodrík et al., 2015)). Of course, other mechanisms are possible, but none that occur to us seem as likely.

In conclusion, on average, when honey bees are exposed to parasites and pesticides in concert, their combined effects are antagonistic, and a clear view of this has heretofore been hindered by a systematic bias in the research community against multi-stressor antagonism. More research is needed to evaluate how living in hives can ameliorate stress, and more routine and consistent quantification of pesticide dose would also be useful. As it stands, the physiological mechanisms underlying this antagonism are unclear, but different possibilities would have different management implications. Thus, sound interventions to diminish honey bee mortality may hinge on an improved understanding of the ecology and physiology of the interactions between honey bees, pesticides and parasites. At the very least, now we know that antagonism is what we need to understand.

Chapter 3. Causes of spatial variation in aphid species richness

Gwendolyn Bird, Bob Foottit, Eric Maw, Nate B Hardy

Abstract

What causes spatial variation in the species richness of herbivorous insects? Herbivorous insect ecology is closely intertwined with that of their host plants, and herbivore communities tend to be more diverse when plant communities are more diverse. But this does not necessarily mean that local variation in herbivore species richness is caused by differences in host plant species richness. Alternatively, plant and herbivore communities could be responding in parallel to underlying variation in the abiotic environment. Here, we infer the causal relationships between herbivore species richness, plant diversity, and abiotic niche factors, using North American aphids as a model and using spatial structural equation models at five levels of spatial resolution. We find that both plant diversity and abiotic factors affect aphid species richness, but the relative importance of plant and abiotic factors depends on scale. At small spatial scales, aphid species richness is determined more by abiotic niche factors, whereas at large spatial scales host plant diversity is prime. We consider possible mechanisms for this scale-dependency, and note the parallels between what we show here, to what we have recently found in investigations of the causes of variation in aphid species richness across clades.

Introduction

The diversities of plant and herbivore communities tend to positively covary (Siemann et al 1998). This could be because plant and herbivore diversities are each respond independently to the same underlying abiotic environmental variation. Alternatively, herbivore diversity might

respond to the abiotic environment only indirectly, via its effects on plant diversity. These two alternatives can be viewed as extremes of a continuum of possible explanations for what causes spacial variation in herbivore species richness. Between those extremes are explanations entailing some mix of direct and indirect abiotic effects.

Among herbivores, herbivorous insects have particularly intense and specific relationships with their host-plants (Castagneyrol & Jactel 2012). Thus, we might expect local patterns of herbivorous insect diversity to be especially sensitive to local plant diversity. Indeed, the high species richness of herbivorous insect clades has been explained as the result of co-evolutionary antagonism with host plants, and many of the most convincing examples of sympatric speciation entail divergence selection on host-use in a herbivorous insect population (Agrawal et al. 2006)

Nevertheless, abiotic conditions, such as solar radiation, precipitation, and soil composition, could also restrict were herbivorous insects occur. In fact, several studies have found that abiotic conditions can explain more of the variation in animal species richness than plant diversity (Grill et al 2005, Hawkins & Porter 2003a, Hawkins & Porter 2003b, Stefanescue 2011). This work has also indicated that the causes of spatial variation in species richness can depend on the spatial scale. For example, Stein et al (2014) found that environmental heterogeneity had a greater impact on species richness at higher resolutions, while Wolters et al (2006) found the greatest correlations between the species richness of different taxa at larger spatial scales.

Here, we examine spatial patterns of species richness in the North American fauna of a diverse group of herbivorous insects: aphids (Insecta: Hemiptera: Strenorrhyncha: Aphidomorpha). Aphidoids, which feed directly on plant sap and have relatively limited mobility, represent a group of organisms particularly dependent on their host plants. We test explicit causal

hypotheses by fitting spatial-autocorrelation-corrected path models at five different spatial scales. The hypotheses are: 1. Abiotic conditions effect both aphid diversity and plant diversity, but plant diversity does not affect aphid diversity, 2. abiotic conditions affect plant diversity and plant diversity affects aphid diversity, but abiotic conditions do not affect aphid diversity directly, a 3. both abiotic diversity and plant diversity affect aphid diversity.

Methods

Specimen records for aphidoids and for terrestrial plants were obtained from the specimen record databases GBIF and iDigBio ("GBIF.org," 2020; "iDigBio Portal," 2020). Aphid specimen records were also obtained from collaborators at the Canadian National Collection. Scientific names were standardized using the R (R Core Team, 2019) package *rgbif* (Chamberlain et al., 2020; Chamberlain and Boettiger, 2017). Aphidoid occurrence data were restricted to the US and Canada, as this region had the most throughout coverage. We had a total of 396,912 aphid occurrence records, and 10,534,622 plant occurrence records.

Specimen coordinate data were used to make rasters of diversity values at resolutions of 20, 40, 80, 160, and 320 arc minutes using the R package *raster* (Hijmans, 2019). Aphid species richness was calculated for each raster square using Menhinick's index (total number of species)/√(total number of aphids) to reduce the influence of sampling bias (Menhinick, 1964). To further mitigate against sampling bias, we also considered only raster squares that contained 10 or more aphid observations, an observation level at which predictive spatial models have been shown to have 90% accuracy (Stockwell & Peterson, 2002). Host-plant use information for aphids were obtained from published catalogs (Blackman and Eastop 1994, 2007; Holman, 2009; Hardy *et al.*, 2017) as well as directly from specimen data. Both all-plant and only-host-plant

diversity for each raster square was calculated as Shannon's H using the R package *bipartite* (Dormann et al., 2009, 2009).

Abiotic environmental variable data were downloaded as raster maps from several sources. Climatic (including 19 bioclimatic variables, water vapor, windspeed, and solar radiation data) and elevation data at each resolution were taken from worldclim.org (Fick and Hijmans, 2017). From MODIS, we took data on elevation, net solar radiation, net primary production (as measured by carbon dioxide output), vegetation and leaf area indices, and land cover types. We used soil data from the Unified North American Soil Map (Liu *et al.*, 2013). And we took evapotranspiration data from the Consortium for Spatial Information (Trabucco and Zomer, no date; Zomer *et al.*, 2008). Ranges and means were calculated for each variable.

Before analysis, each niche variable was centered and scaled to units of standard deviations. Data that were non-normal were normalized according to the ordered quantile method using the R package bestNormalize (Peterson and Cavanaugh, 2019). Data dimensionality was reduced with Principle Components Analysis. At each spatial resolution, the first four PCs were used; depending on the spatial resolution those PCs accounted for between 73% and 81% of the total environmental variation (appendix).

We used regression analysis to fit the data to wavelet-revised spatial models using the R package *spind* (Carl and Kuehn, 2016, Carl, Levin and Kuehn, 2018). We then combined regression models into structural equation models using the d-sep method as described in Shipley (2016). As mentioned above, we compared the fit of three explicit causal hypotheses (Figure 7). In the first, abiotic factors only affected aphid diversity indirectly, via their effects on plant diversity. In the second, abiotic and host-related niche components each directly affected aphid diversity, and plant diversity was independent of abiotic diversity. In the third, abiotic factors directly affected

both plant and aphid diversity, and indirectly affected aphid diversity via effects on plant diversity. Model fit was assessed using CIC (Fisher's C) (Shipley, 2016). CIC was calculated by taking the log of the product of the p-values of the independence test and multiplying by negative two. Significance of a CIC value was calculated by taking the reciprocal of the chi-square at the CIC value at two times the degrees of freedom. Models where the p-values were greater than alpha (0.05) were considered significant, as a p-value below alpha would indicate that correlations between some or all of the predictors not connected by edges. For each model, we found the overall direct effects of abiotic variables on aphid and plant diversity by summing the absolute values of standardized coefficients of the relationships between abiotic PCs and aphid species richness, and between abiotic PCs and plant diversity. The indirect effect of the abiotic environment on aphid diversity was calculated as the product of the summed effects of the abiotic environment on plant diversity and the direct effects of plant diversity on aphid diversity.

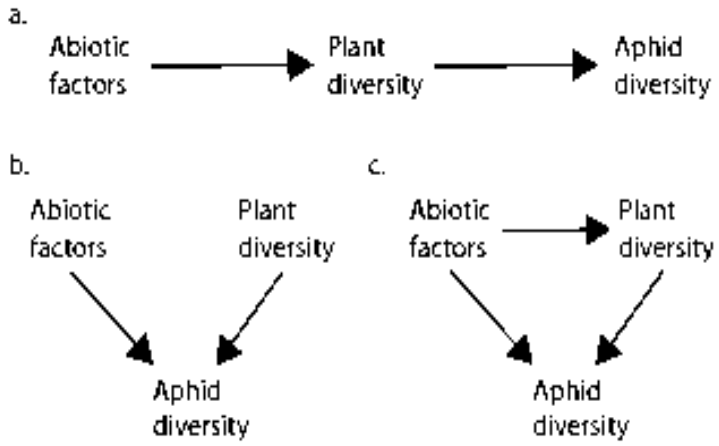

Figure 7: Three path models used in hypothesis testing. Note that 'abiotic factors' is incorporated into the model as four principal components.

Results

At all spatial resolutions, models in which aphid species richness is caused by a mix of abiotic and plant factors had the best fit. With the exception of the lowest spatial scale, host plant diversity had the greater affect on aphid diversity when compared to total plant diversity. The relative weights of abiotic and plant effects depended on spatial scale. At the smallest examined spatial scale (20 arc minutes), the direct effect of abiotic factors was greater than that of plant diversity, regardless of whether we looked at all of the plants in an area, or only known aphid host plants; at 20 arc minutes, the effect of plant diversity on aphid diversity was 0.15 and 0.13 SMD for all plants and host plants, while the direct effect of abiotic factors on aphid diversity was 0.28 and 0.19 SMD. The direct effects of abiotic factors where greater than the direct plant diversity effects also at the 80 arc minute scale, but the opposite was true at the 40 arc minute scale.

Table 2: Main results of path models at all resolutions.

Model	Effect of plant diversity on aphid diversity	Effect of environment on plant diversity	Direct effect of environment on aphid diversity	Indirect effect of environment on aphid diversity	Total effect of environment on aphid diversity	Model P-value	Model cCIC
20 arc min							
All plants							
Mixed model	0.15	0.68	0.28	0.10	0.39	0.68	5.68
H only model	0.15	0.75	0	0.11	0.11	0	32.11
E only model	0.15	0	0.68	0	0.68	0	INF
Host plants							
Mixed model	0.13	0.72	0.19	0.10	0.29	0.60	6.4
H only model	0.15	0.64	0	0.10	0.10	0	28.8
E only model	0.14	0	0.62	0	0.62	0	163.86
40 arc min							
All plants							
Mixed model	0.24	1.01	0.22	0.24	0.46	0.53	5.12
H only model	0.26	1.01	0	0.26	0.26	0	29.44
E only model	0.24	0	0.74	0	0.74	0	INF
Host plants							
Mixed model	0.28	1.01	0.19	0.29	0.47	0.63	4.33
H only model	0.31	0.89	0	0.27	0.27	0	23.28
E only model	0.28	0	0.63	0	0.63	0	INF
80 arc min							
All plants							
Mixed model	0.24	0.97	0.61	0.23	0.85	0.47	1.52
H only model	0.34	1.00	0	0.34	0.34	0	68.39
E only model	0.24	0	0.88	0	0.88	0	INF
Host plants							
Mixed model	0.33	1.00	0.44	0.33	0.77	0	10.7
H only model	0.39	0.92	0	0.36	0.36	0	60.65
E only model	0.29	0	0.73	0	0.73	0	INF
160 arc min							
All plants							
Mixed model	0.32	0.91	0.46	0.30	0.78	0.46	3.62
H only model	0.46	0.97	0	0.44	0.44	0	30.8

E only model	0.32	0	0.44	0	0.44	0	160.67
Host plants							
Mixed model	0.32	0.82	0.26	0.27	0.52	0.39	6.34
H only model	0.58	0.91	0	0.53	0.53	0.01	21.43
E only model	0.52	0	0.27	0	0.27	0	INF
320 arc min							
All plants							
Mixed model	0.4	1.21	0.35	0.48	0.83	0.12	10.2
H only model	0.52	1.33	0	0.69	0.69	0.09	13.86
E only model	0.33	0	0.85	0	0.85	0	87.83
Host plants							
Mixed model	0.74	0.81	0.12	0.60	0.72	0.40	6.22
H only model	0.70	1.56	0	1.09	1.09	0.23	10.56
E only model	0.77	0	0.2	0	0.20	0	131.4

At the larger spatial scales, 160 and 320 arc minutes), we found that the direct effect of host plant diversity on aphid species richness outweighed that from the abiotic environment and at the largest scale (320 arc minutes) this was also true for all-plant diversity. At this resolution, the effect of plant diversity on aphid diversity was 0.40 and 0.74 SMD for all plants and host plants, while the direct effect of abiotic factors on aphid diversity was 0.35 and 0.12 SMD.

A similar picture emerges from comparisons of the direct effects of plant diversity on aphid species richness to the combined direct and indirect effects from the abiotic environment. At all spatial resolutions the direct and indirect abiotic effects are close in magnitude, and in most of our models, the combined direct and indirect effects from the abiotic environment are greater than the direct effects of plant diversity on aphid species richness. But at the largest spatial scale (320 arc minutes) we found that the direct effects of host plant diversity is greater than the combined direct and indirect effects from the abiotic environment. (Table 2, Figure 8).

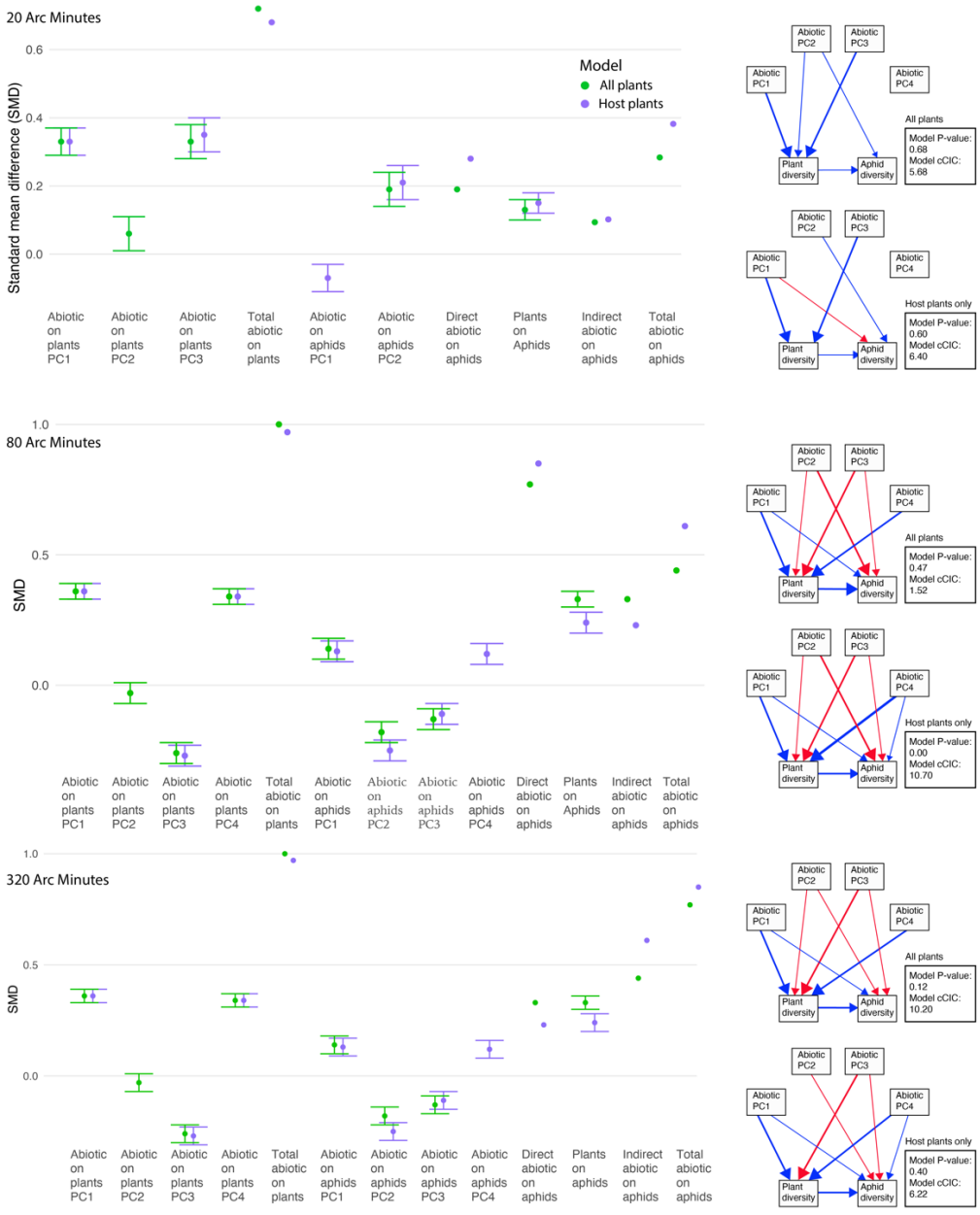

Figure 8: Effect sizes and path models for models at resolutions of 20, 140 and 200 arc minutes. Effect sizes are given in units of standard mean difference, with significant error. No error is given when the result is a combination of effect sizes. Edges shown in blue are positive, edges shown in red are negative and arrow weight denotes the strength of the effect.

The strength of the effect of both plant diversity and environmental abiotic factors increased with resolution. At a scale of 20 arc minutes, host plant diversity affected aphid species richness by 0.13 standard mean difference (SMD), and environmental abiotic factors affected aphid diversity by 0.29 SMD, while, at a resolution of 320 arc minutes, host plant diversity affected aphid diversity by 0.74 SMD, and environmental abiotic factors affected aphid diversity by 0.72 SMD.

Discussion

Our path model analyses show that spatial variation in the species richness of aphid communities is caused by a mix of abiotic and host-plant factors that depends on scale. More specifically, we found a general trend of increasing influence of host-plant diversity as we move from the local to regional scale. This accords with previous studies that have also found that the positive correlations between plant diversity and animal diversity are greater across than within habitats (Castagneyrol & Jactel, 2012). What could explain this scale dependency?

We see three main explanations. First, it could be a matter of statistical power. The sampling intensity of aphid occurrences across space has not been uniform. As we zoom out and consider larger regions, this could reduce the effects of sampling bias and improve the signal to noise ratio. In this case, host-plant diversity might be the prime factor determining aphid species richness, but we are only able to see that causal relationship clearly at the largest scales. A second possible explanation is that the frequency distribution of aphid host-use breadth changes with spatial scale; specifically, the proportion of host-use specialists increases with increasing spatial scale (as suggested by Castagneyrol and Jactel 2021). Simply put, generalists tend to be more broadly distributed than specialists, so as we expand the geographic scope of analysis, we mainly capture additional specialists and thus are dealing with increasingly specialized aphid communities, which are increasingly sensitive to regional plant diversity. A third explanation is

that at different spatial scales, our abiotic variables are telling us different things about the environment. We are not sure what exactly those differences are, but it strikes us that at the largest scale we are comparing broad classes of habitat types, whereas at the smallest scales we are comparing microhabitats. In that case, the scale dependence that we observed could be construed as evidence of nested factors structuring aphid communities, with microhabitat playing the primary role, followed by host plant diversity, and then non-host biome-level environmental features.

One of the big challenges facing researchers seeking to understand the causes of spatial variation in species richness is spatial autocorrelation. In this study, the relatively large size of the data set made it infeasible to use some of the more conventional methods of correcting for spatial autocorrelation (Hijmans, 2019) The d-sep method of path modeling used here has some inherent limitations. One is that it cannot fit models in which all predictors are correlated. Another is that it can only assess the statistical significance of missing paths in a restricted version of a general model; it can not assess the significance of the estimated effect coefficients along the causal paths that are present in a model. We look forward to methodological advancements that will permit researchers to better integrate a spatial auto-correlation correction into a maximum likelihood structural equation model.

As touched on above, another caveat of our study was the unevenness of the sampling data. In comparison to most groups of herbivorous insects, our characterization of the geographic distributions and host associations of North American aphid species are exceptionally rich. But sampling of occurrence data is likely to be more even for some groups, in particular butterflies (Lepidoptera: Papilionoidea), which we plan to study next.

This study has many parallels with our recent comparative phylogenetic work examining the ecological causes of the variation in aphid species richness and speciation rates across clades (Hardy et al. in review). In both cases aphid species richness is positively correlated to the diversity of their host plants. And in both cases, it is not clear if it is plant diversity per se or some underlying abiotic environmental features that cause the variation in aphid species richness. Earlier, we alluded to the fact that popular explanations of herbivorous insect species diversification have put host-plant interactions front and center; herbivorous insect diversification has been hypothesized to have been driven by co-evolutionary antagonism with their host plants. But our comparative phylogenetic work suggests that divergence along abiotic niche axes has tended to play a more important role in the speciation of aphids, with subsequent divergence in host use raising the ecological limits on local aphid communities and clades. Our analysis of the spatial variation in aphid species richness also underscores the importance of the abiotic environmental variables as components of aphid niches. Especially at local scales, host plant diversity is of secondary importance.

In conclusion, both host-use and abiotic factors are important causes of the spatial variation in aphid species richness. But which is more forceful depends on the spatial scale. As it stands, the mechanisms of these scale effects are unclear. Considerable progress in our understanding of the ecology and evolution of herbivorous insects could be made by revealing those mechanisms.

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Appendix 1: Supplementary information from chapter 1

1. Studies included

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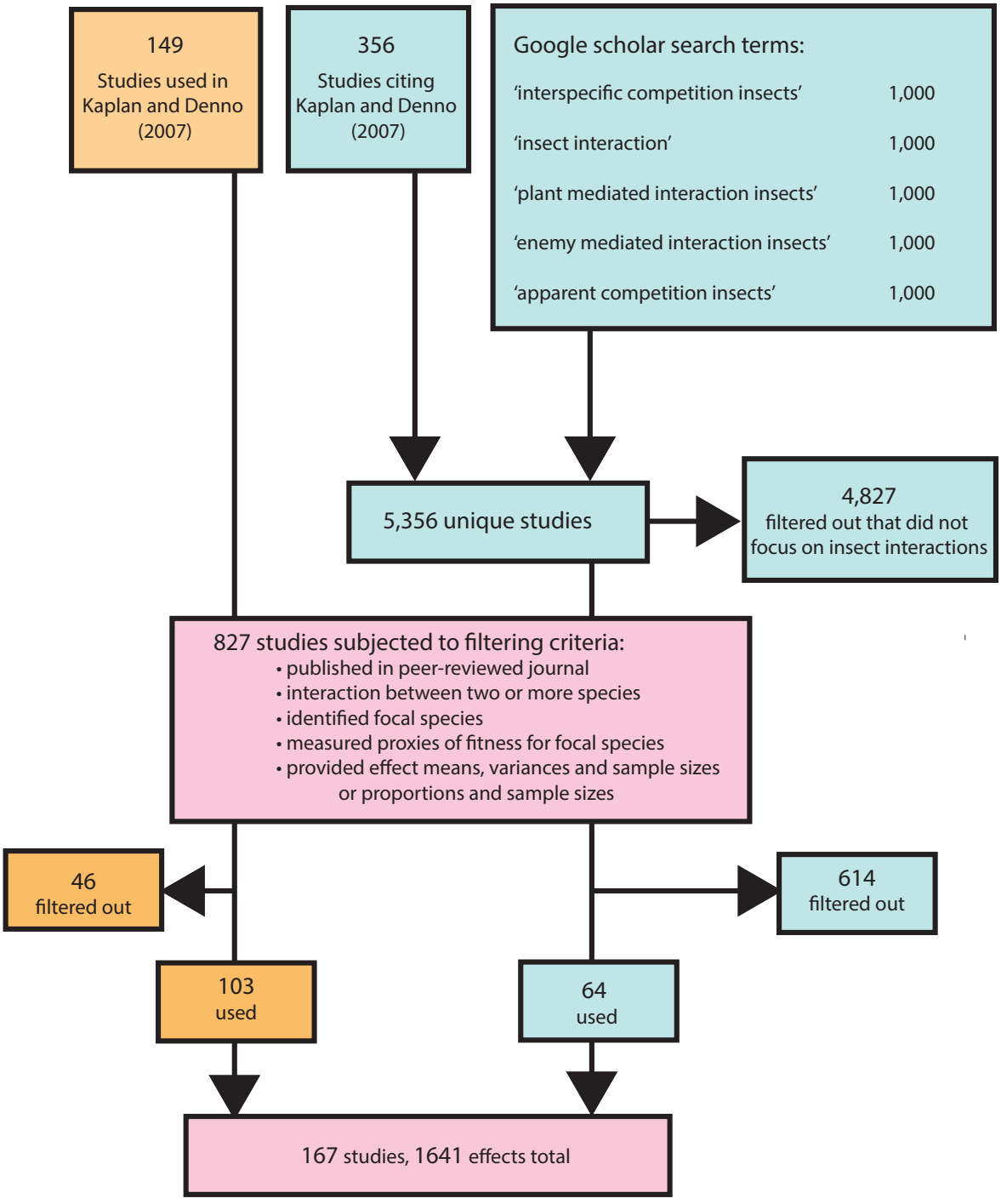
2. PRISMA flow diagram



3. **Complete results for all fitness components, according to model and fixed effect.**

Models with continuous effect sizes

Table 1: Intercept only models. Effects significant at or below the 0.05 level are given in bold.

Fitness component	Taxonomy model				Phylogeny model			
	Intercept	Upper CI	Lower CI	p-value	Intercept	Upper CI	Lower CI	p-value
Composite	**-0.30**	**-0.10**	**-0.51**	**0.0076**	-0.23	0.23	-0.68	0.31
Composite without abundance	-0.22	0.048	-0.52	0.075	-0.13	0.25	-0.48	0.43
Abundance	**-0.60**	**-0.27**	**-0.92**	**0.0033**	**-0.47**	**-0.10**	**-0.80**	**0.013**
Body size	-0.079	0.25	-0.45	0.62	-.000060	.60	-.57	0.98
Fecundity	-0.61	0.053	-1.36	0.055	-0.38	0.33	-1.15	0.24
Survival	0.14	0.38	-0.058	0.156	0.11	0.38	-0.14	0.36

The following are from multiple fixed effect models

Table 2: Intercepts for multiple fixed effect models

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	-0.45	0.075	-1.1	0.078	0.33	0.55	-1.2	0.42
Abundance	-0.72	0.14	-1.6	0.095	**-1.1**	**-0.024**	**-2.3**	**0.047**
Body size	0.021	0.81	-0.83	0.99	0.20	1.3	-0.68	0.69
Fecundity	**-1.4**	**-0.35**	**-2.7**	**0.014**	-1.2	0.042	-2.4	0.053
Survival	**0.45**	**0.89**	**-0.00050**	**0.046**	0.30	0.76	-0.18	0.21

Table 3: Species count (intraspecific/intraspecific interactions as the intercept, intraspecific/interspecific interactions as the coefficient

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	**0.16**	**0.31**	**0.0010**	**0.047**	**0.19**	**0.37**	**0.014**	**0.047**
Abundance	-0.42	0.072	-0.88	0.089	-0.26	0.33	-0.82	0.38
Body size	0.20	0.53	-0.12	0.23	0.27	0.62	-0.093	0.16
Fecundity	**0.59**	**0.98**	**0.22**	**0.00013**	0.30	0.76	-0.23	0.24
Survival	-0.14	0.11	-0.41	0.31	0.044	0.34	-0.24	0.77

Table 4: Density (density is held constant between control and experimental group as the intercept, density is lower in control group and higher in experimental group as the coefficient)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	-0.13	0.0098	-0.27	0.067	**-0.20**	**-0.027**	**-0.37**	**0.026**
Abundance	0.29	0.71	-0.093	0.14	0.39	0.85	-0.11	0.12
Body size	**-0.30**	**-0.0047**	**-0.57**	**0.039**	**-0.37**	**-0.046**	**-0.68**	**0.019**
Fecundity	-0.15	0.24	-0.53	0.44	-0.31	0.23	-0.90	0.28
Survival	0.21	0.43	-0.035	0.084	**0.27**	**0.52**	**0.001**	**0.048**

Table 5: Spatial separation (experiments where insects were not separated as the intercept, experiments where insects were separated as the coefficient)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	0.076	0.30	-0.13	0.49	0.0022	0.25	-0.24	0.99
Abundance	**0.66**	**1.2**	**0.12**	**0.014**	**0.71**	**1.3**	**0.11**	**0.023**
Body size	0.11	0.60	-0.36	0.64	-0.010	0.46	-0.47	0.95
Fecundity	-0.27	0.30	-0.79	0.33	-0.44	0.14	-1.0	0.13

Survival	-0.11	0.17	-0.36	0.41	-0.12	0.34	-0.39	0.37

Table 6: Temporal separation (experiments where insects were contemporaneous as the intercept, experiments where insects were introduced first and then not removed before the introduction of the focal insect (incomplete), or introduced first and then removed before the introduction of the focal insect (complete) as the coefficients)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite, incomplete	0.019	0.26	-0.22	0.86	-0.055	0.24	-0.32	0.69
complete	*-0.0046*	*0.19*	*-0.20*	*0.98*	*-0.035*	*0.20*	*-0.27*	*0.77*
Abundance incomplete	**-0.54**	**-0.078**	**-1.01**	**0.027**	-0.60	0.029	-1.2	0.058
complete	*0.0078*	*0.42*	*-0.45*	*0.97*	*0.080*	*0.65*	*-0.49*	*0.79*
Body size, incomplete	0.033	0.49	-0.46	0.88	-0.025	0.44	-0.53	0.92
complete	***-0.50***	***-0.088***	***-0.90***	***0.017***	***-0.63***	***-0.23***	***-1.0***	***0.0028***
Fecundity, incomplete	0.34	1.0	-0.38	0.34	0.69	1.4	-0.17	0.099
complete	*0.51*	*1.2*	*-0.17*	*0.14*	***0.83***	***1.6***	***0.030***	***0.041***
Survival, incomplete	0.0081	0.33	-0.29	0.96	0.025	0.34	-0.29	0.89
complete	*0.16*	*0.42*	*-0.091*	*0.22*	*0.14*	*0.42*	*-0.11*	*0.28*

Table 7: Focal insect feeding guild (chewer as the intercept, internal feeder (IF) and sucker as coefficients)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite, IF	0.0098	0.64	-0.68	0.96	0.31	1.1	-0.56	0.46
sucker	*0.22*	*0.88*	*-0.41*	*0.47*	*0.16*	*1.0*	*-0.68*	*0.72*

Abundance IF	0.35	1.3	-0.63	0.47	0.59	2.0	-0.70	0.39
sucker	*0.49*	*1.3*	*-0.38*	*0.25*	*0.55*	*1.7*	*-0.42*	*0.28*
Body size, IF	0.060	0.91	-0.84	0.88	-0.032	0.76	-0.85	0.96
sucker	*0.96*	*2.9*	*-0.97*	*0.30*	*1.0*	*2.9*	*-0.77*	*0.24*
Fecundity, IF	0.61	2.3	-0.95	0.47	0.65	2.2	-0.97	0.40
sucker	***1.8***	***3.3***	***0.28***	***0.018***	***1.9***	***3.4***	***0.23***	***0.020***
Survival, IF	-0.53	0.32	-1.4	0.22	-0.61	0.19	-1.4	0.13
sucker	***-0.84***	***-0.14***	***-1.6***	***0.015***	*-0.48*	*-0.090*	*-0.88*	*0.021*

Table 8: Competing insect feeding guild (chewer as the intercept, internal feeder (IF) and sucker as coefficients)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite, IF	0.32	0.87	-0.25	0.26	0.38	1.2	-0.36	0.31
sucker	*-0.021*	*0.26*	*-0.31*	*0.89*	*0.0041*	*0.40*	*-0.34*	*0.98*
Abundance IF	1.0	2.0	-0.060	0.058	**-2.3**	**-0.60**	**-4.0**	**0.0096**
sucker	***0.78***	***1.5***	***0.018***	***0.040***	***1.4***	***2.7***	***0.14***	***0.025***
Body size, IF	0.84	1.9	-0.27	0.13	0.81	1.8	-0.27	0.13
sucker	*0.29*	*0.88*	*-0.31*	*0.33*	*0.37*	*0.96*	*-0.24*	*0.22*
Fecundity, IF	0.89	2.8	-0.92	0.32	-0.11	1.8	-2.0	0.89
sucker	*-0.66*	*0.31*	*-1.6*	*0.18*	*-1.1*	*0.022*	*-2.4*	*0.072*
Survival, IF	-0.17	0.39	-0.72	0.55	-0.16	0.42	-0.72	0.57

sucker	***-0.43***	***-0.033***	***-0.83***	***0.042***	***-0.48***	***-0.090***	***-0.88***	***0.021***

Table 9: Feeding guild, internal feeder: internal feeder (as coefficient. Intercept is chewer: chewer). Some effects were not estimable and so are not given.

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	-0.25	0.61	-1.2	0.59	-0.43	0.81	-1.5	0.47
Abundance	-0.61	0.84	-2.0	0.39	**2.8**	**4.8**	**0.42**	**0.014**
Body size	-1.1	0.29	-1.7	0.14	-1.2	0.13	-1.7	0.088
Fecundity	-1.3	1.2	-3.7	0.30	NA	NA	NA	NA
Survival	0.27	1.4	-0.80	0.61	0.33	1.3	-0.68	0.52

Table 10: Feeding guild, sucker: internal feeder (as coefficient. Intercept is chewer: chewer). Some effects were not estimable and so are not given.

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	-0.25	0.61	-1.2	0.59	-0.94	0.36	-2.3	0.16
Abundance	**-2.8**	**-1.4**	**-4.3**	**0.00022**	NA	NA	NA	NA
Body size	-1.4	1.0	-3.7	0.27	NA	NA	NA	NA
Fecundity	-2.2	0.36	-4.6	0.073	NA	NA	NA	NA
Survival	0.37	1.4	-0.52	0.43	0.34	1.3	-0.67	0.47

Table 11: Feeding guild, internal feeder: sucker (as coefficient. Intercept is chewer: chewer). Some effects were not estimable and so are not given.

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	-0.091	0.081	-1.0	0.85	0.067	1.3	-1.0	0.92
Abundance	-1.2	0.13	-2.7	0.077	**-2.1**	**-0.23**	**-3.9**	**0.023**

Body size	-1.3	1.2	-3.8	0.30	-1.3	1.2	-3.7	0.28
Fecundity	NA	NA	NA	NA	NA	NA	NA	NA
Survival	0.67	1.7	-0.43	0.21	0.74	1.7	0.26	1.5

Table 12: Feeding guild, sucker: sucker (as coefficient. Intercept is chewer: chewer). Some effects were not estimable and so are not given.

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	-0.26	0.22	-0.75	0.31	-0.27	0.31	-0.87	0.39
Abundance	**-1.3**	**-0.33**	**-2.3**	**0.011**	**-1.8**	**-0.38**	**-3.3**	**0.014**
Body size	-1.4	0.38	-3.2	0.14	-1.6	0.14	-3.2	0.070
Fecundity	-0.43	1.0	-2.1	0.58	-0.21	1.6	-2.1	0.85
Survival	**0.87**	**1.6**	**0.065**	**0.025**				

Table 13: Diet breadth (a continuous variable, with a range of 1-171. The coefficient represents an increase of 1).

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	0.0011	0.0043	-0.0022	0.93	1.7	0.21	0.050	0.37
Abundance	-0.0010	0.0024	-0.0043	0.56	-0.00097	0.0022	-0.0039	0.52
Body size	0.00096	0.020	-0.000012	0.060	0.0098	0.020	-0.00039	0.052
Fecundity	**0.0056**	**0.012**	**0.00033**	**0.038**	**0.0057**	**0.011**	**0.00059**	**0.027**
Survival	-0.0023	0.0014	-0.0058	0.21	-0.0026	0.00083	-0.0063	0.14

Table 14: Phylogeny distance (a continuous variable, with a range of 1-715.52. The coefficient represents an increase of 1).

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value

Composite	**0.00049**	**0.00091**	**0.000077**	**0.025**	0.00040	0.00082	-0.000027	0.068
Abundance	0.00039	0.0010	-0.00028	0.25	0.00037	0.0010	-0.00022	0.26
Body size	**0.0014**	**0.0024**	**0.00016**	**0.016**	**0.0014**	**0.0025**	**0.00024**	**0.016**
Fecundity	0.00079	0.0026	-0.0011	0.40	0.00018	0.0020	-0.0018	0.84
Survival	-0.0011	0.00048	-0.00069	0.72	0.000026	0.000060	-0.00050	0.92

Models with categorical effect sizes

Table 15: Intercept only models

Fitness component	Taxonomy model				Phylogeny model			
	Intercept	Upper CI	Lower CI	p-value	Intercept	Upper CI	Lower CI	p-value
Composite	**-0.38**	**-0.27**	**-0.50**	**0.0002**	**-0.40**	**-0.28**	**-0.52**	**0.0002**
Abundance	**-0.38**	**-0.26**	**-0.49**	**0.0002**	**-0.40**	**-0.29**	**-0.51**	**0.0002**

The following are from the multiple fixed effect models

Table 16: Intercepts for multiple fixed effect models

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	**-0.32**	**-0.11**	**-0.53**	**0.0068**	**-0.32**	**-0.11**	**0.53**	**0.0072**
Abundance	**-0.32**	**-0.10**	**-0.53**	**0.0064**	**-0.32**	**-0.10**	**-0.52**	**0.0032**

Table 17: Species count (intraspecific/intraspecific interactions as the intercept, intraspecific/interspecific interactions as the coefficient

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	**-0.12**	**-0.013**	**-0.22**	**0.022**	**-0.12**	**-0.010**	**-0.22**	**0.019**

Abundance	**-0.12**	**-0.018**	**-0.22**	**0.027**	**0.012**	**0.020**	**0.22**	**0.024**

Table 18: Density (density is held constant between control and experimental group as the intercept, density is lower in control group and higher in experimental group as the coefficient)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	0.086	0.18	0.00017	0.055	0.086	0.18	0.0013	0.056
Abundance	0.085	0.17	-0.0087	0.064	0.085	0.17	-0.0032	0.057

Table 19: Spatial separation (experiments where insects were not separated as the intercept, experiments where insects were separated as the coefficient)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	0.090	0.019	-0.016	0.083	0.091	0.19	-0.012	0.080
Abundance	0.092	0.19	-0.0039	0.061	0.091	0.19	-0.0045	0.071

Table 20: Temporal separation (experiments where insects were contemporaneous as the intercept, experiments where insects were introduced first and then not removed before the introduction of the focal insect (incomplete), or introduced first and then removed before the introduction of the focal insect (complete) as the coefficients)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite, incomplete	-0.95	0.29	-0.23	0.15	-0.097	0.028	-0.22	0.14
complete	*-0.079*	*0.016*	*-0.18*	*0.11*	*-0.082*	*0.017*	*-0.18*	*0.099*
Abundance incomplete	-0.095	0.033	-0.22	0.15	-0.096	0.032	-0.21	0.14
complete	*-0.081*	*0.015*	*-0.17*	*0.094*	*-0.081*	*0.016*	*-0.18*	*0.10*

Table 21: Focal insect feeding guild (chewer as the intercept, internal feeder (IF) and sucker as coefficients)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite, IF	0.016	0.22	-0.20	0.90	0.019	0.22	-0.19	0.86
sucker	*-0.065*	*0.14*	*-0.28*	*0.55*	*-0.066*	*0.15*	*-0.27*	*0.53*
Abundance IF	0.016	0.23	-0.19	0.88	0.017	0.22	-0.19	0.88
sucker	*-0.065*	*0.15*	*-0.28*	*0.56*	*-0.064*	*0.15*	*-0.28*	*0.55*

Table 22: Competing insect feeding guild (chewer as the intercept, internal feeder (IF) and sucker as coefficients)

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite, IF	**-0.35**	**-0.040**	**-0.68**	**0.037**	**-0.35**	**-0.040**	**-0.68**	**0.037**
sucker	*-0.080*	*0.075*	*-0.25*	*0.33*	*-0.081*	*0.078*	*-0.24*	*0.34*
Abundance IF	**-0.35**	**-0.020**	**0.66**	**0.034**	**-0.35**	**-0.22**	**-0.64**	**0.023**
sucker	*-0.80*	*0.084*	*-0.24*	*0.33*	*-0.082*	*0.089*	*-0.23*	*0.31*

Table 23: Feeding guild, internal feeder: internal feeder (as coefficient. Intercept is chewer: chewer). Some effects were not estimable and so are not given.

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	0.27	0.70	-0.12	0.18	0.27	0.67	-0.12	0.19
Abundance	0.27	0.66	-0.12	0.18	0.27	0.67	-0.11	0.18

Table 24: Feeding guild, sucker: internal feeder (as coefficient. Intercept is chewer: chewer). Some effects were not estimable and so are not given.

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	0.13	0.62	-0.34	0.61	0.12	0.63	-0.34	0.63
Abundance	0.13	0.66	-0.34	0.62	0.13	0.61	-0.37	0.61

Table 25: Feeding guild, internal feeder: sucker (as coefficient. Intercept is chewer: chewer). Some effects were not estimable and so are not given.

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	0.22	0.58	-0.12	0.21	0.22	0.38	-0.14	0.22
Abundance	0.22	0.56	-0.13	0.21	0.23	0.59	-0.10	0.21

Table 26: Feeding guild, sucker: sucker (as coefficient. Intercept is chewer: chewer). Some effects were not estimable and so are not given.

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	0.15	0.39	-0.078	0.21	0.15	0.38	-0.091	0.22
Abundance	0.15	0.39	-0.076	0.20	0.15	0.38	-0.092	0.20

Table 27: Diet breadth (a continuous variable, with a range of 1-171. The coefficient represents an increase of 1).

Fitness component	Taxonomy model				Phylogeny model			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	-0.00011	0.0011	-0.0014	0.85	-0.00012	0.00098	-0.0013	0.82
Abundance	-0.00012	0.0011	-0.0014	0.86	-0.00011	0.0010	-0.0011	0.84

Table 28: Phylogeny distance (a continuous variable, with a range of 1-715.52. The coefficient represents an increase of 1).

Fitness component	Taxonomy model	Phylogeny model

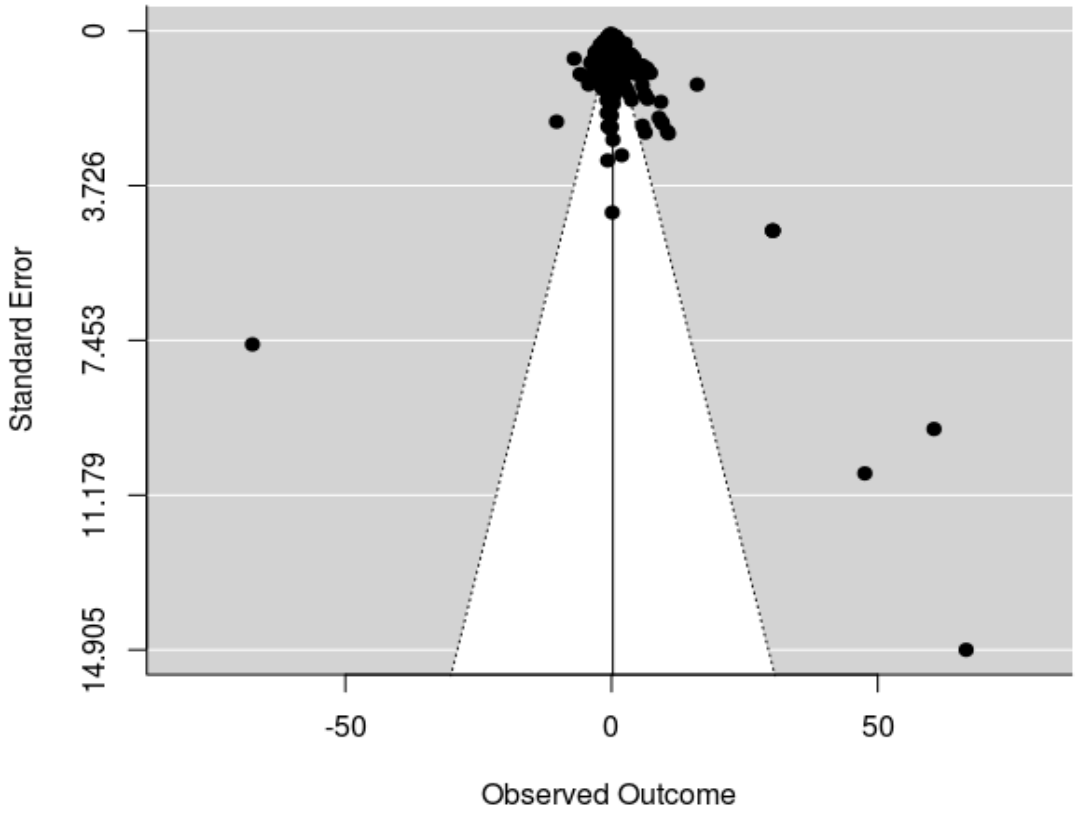
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Composite	**-0.00024**	**-0.000033**	**-0.00043**	**0.016**	**-0.00022**	**-0.000029**	**-0.00042**	**0.031**
Abundance	**-0.00024**	**-0.000047**	**-0.00044**	**0.021**	**-0.00021**	**-0.000021**	**-0.00040**	**0.024**

Table 29: Models without accounting for phylogeny

Fixed effect	Composite				Abundance			
	coefficient	Upper CI	Lower CI	p-value	coefficient	Upper CI	Lower CI	p-value
Intercept only	**-0.28**	**-0.15**	**-0.40**	**0.0002**	**0.52**	**0.34**	**0.73**	**0.0002**
Intercept of mixed-effect model	**-0.30**	**-0.034**	**-0.58**	**0.032**	**-0.65**	**-0.022**	**-1.3**	**0.041**
Species count	0.16	0.30	0.00	0.038	-0.25	0.13	-0.65	0.20
Density	**-0.14**	**-0.0037**	**-0.27**	**0.041**	**0.47**	**0.82**	**0.13**	**0.0072**
Spatial separation	-0.020	0.16	-0.20	0.82	**0.40**	**0.76**	**0.046**	**0.025**
Temporal separation	0.048	0.25	-0.17	0.65	**-0.43**	**-0.039**	**-0.80**	**0.24**
Complete temporal separation	0.057	0.23	-0.11	0.52	0.069	0.42	-0.29	0.69
F_Guild sucker	0.18	0.52	-0.14	0.28	0.16	0.60	-0.25	0.45
F_Guild IF	0.053	0.42	-0.31	0.78	0.21	0.69	-0.25	0.40
C_Guild sucker	-0.73	0.19	-0.33	0.58	0.27	0.79	-0.22	0.32
C_Guild IF	0.29	0.78	-0.22	0.26	0.53	1.2	-0.20	0.14
F_Guild IF, C_Guild IF	-0.32	0.32	-1.0	0.35	-0.51	0.36	-1.4	0.27
F_Guild Sucker, C_Guild IF	-0.70	0.040	-1.37	0.055	**-1.7**	**-0.66**	**-2.7**	**0.0002**
F_Guild IF, C_Guild sucker	-0.35	0.30	-0.98	0.29	**-0.99**	**-0.079**	**-1.9**	**0.035**

F_Guild sucker, C_Guild sucker	-0.19	0.23	-0.61	0.38	**-0.67**	**0.0092**	**-1.4**	**0.054.**

4. **Funnel plot.** Asymmetry represents possible bias. Mean effect sizes are on the x-axis. Measures of variance are on the y-axis.



Appendix 2: Chapter 2 Supplimentary information

1. Studies included

Alaux, C., Brunet, J.-L., Dussaubat, C., Mondet, F., Tchamitchan, S., Cousin, M., Brillard, J., Baldy, A., Belzunces, L.P., Le Conte, Y., 2010. Interactions between Nosema microspores and a neonicotinoid weaken honeybees (Apis mellifera). Environmental Microbiology 12, 774–782. https://doi.org/10.1111/j.1462-2920.2009.02123.x

Aufauvre, J., Biron, D.G., Vidau, C., Fontbonne, R., Roudel, M., Diogon, M., Viguès, B., Belzunces, L.P., Delbac, F., Blot, N., 2012. Parasite-insecticide interactions: a case study of Nosema ceranae and fipronil synergy on honeybee. Scientific Reports 2, 326. https://doi.org/10.1038/srep00326

Aufauvre, J., Misme-Aucouturier, B., Viguès, B., Texier, C., Delbac, F., Blot, N., 2014. Transcriptome analyses of the honeybee response to Nosema ceranae and insecticides. PLoS ONE 9, e91686. https://doi.org/10.1371/journal.pone.0091686

Coulon, M., Dalmon, A., Di Prisco, G., Prado, A., Arban, F., Dubois, E., Ribière-Chabert, M., Alaux, C., Thiéry, R., Le Conte, Y., 2020. Interactions between thiamethoxam and deformed

wing virus can drastically impair flight behavior of honey bees. Frontiers in Microbiology 11. https://doi.org/10.3389/fmicb.2020.00766

Coulon, M., Schurr, F., Martel, A.-C., Cougoule, N., Bégaud, A., Mangoni, P., Dalmon, A., Alaux, C., Le Conte, Y., Thiéry, R., Ribière-Chabert, M., Dubois, E., 2018. Metabolisation of thiamethoxam (a neonicotinoid pesticide) and interaction with the Chronic bee paralysis virus in honeybees. Pesticide Biochemistry and Physiology 144, 10–18. https://doi.org/10.1016/j.pestbp.2017.10.009

Coulon, M., Schurr, F., Martel, A.-C., Cougoule, N., Bégaud, A., Mangoni, P., Prisco, G.D., Dalmon, A., Alaux, C., Ribière-Chabert, M., Conte, Y.L., Thiéry, R., Dubois, E., 2019. Influence of chronic exposure to thiamethoxam and chronic bee paralysis virus on winter honey bees. PLOS ONE 14, e0220703. https://doi.org/10.1371/journal.pone.0220703

Diao, Q., Li, B., Zhao, H., Wu, Y., Guo, R., Dai, P., Chen, D., Wang, Q., Hou, C., 2018. Enhancement of chronic bee paralysis virus levels in honeybees acute exposed to imidacloprid: A Chinese case study. Science of The Total Environment 630, 487–494. https://doi.org/10.1016/j.scitotenv.2018.02.258

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Faita, M.R., Cardozo, M.M., Amandio, D.T.T., Orth, A.I., Nodari, R.O., 2020. Glyphosate-based herbicides and Nosema sp. microsporidia reduce honey bee (Apis mellifera L.) survivability

under laboratory conditions. Journal of Apicultural Research 59, 332–342. https://doi.org/10.1080/00218839.2020.1736782

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O'Neal, S.T., Brewster, C.C., Bloomquist, J.R., Anderson, T.D., 2017. Amitraz and its metabolite modulate honey bee cardiac function and tolerance to viral infection. Journal of Invertebrate Pathology 149, 119–126. https://doi.org/10.1016/j.jip.2017.08.005

O'Neal, S.T., Reeves, A.M., Fell, R.D., Brewster, C.C., Anderson, T.D., 2019. Chlorothalonil exposure alters virus susceptibility and markers of immunity, nutrition, and development in honey bees. Journal of Insect Science 19. https://doi.org/10.1093/jisesa/iez051

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Paris, L., Peghaire, E., Moné, A., Diogon, M., Debroas, D., Delbac, F., El Alaoui, H., 2020. Honeybee gut microbiota dysbiosis in pesticide/parasite co-exposures is mainly induced by Nosema ceranae. Journal of Invertebrate Pathology 172, 107348. https://doi.org/10.1016/j.jip.2020.107348

Paris, L., Roussel, M., Pereira, B., Delbac, F., Diogon, M., 2017. Disruption of oxidative balance in the gut of the western honeybee Apis mellifera exposed to the intracellular parasite Nosema ceranae and to the insecticide fipronil. Microbial Biotechnology 10, 1702–1717. https://doi.org/10.1111/1751-7915.12772

Retschnig, G., Neumann, P., Williams, G.R., 2014. Thiacloprid–Nosema ceranae interactions in honey bees: Host survivorship but not parasite reproduction is dependent on pesticide dose. Journal of Invertebrate Pathology 118, 18–19. https://doi.org/10.1016/j.jip.2014.02.008

Retschnig, G., Williams, G.R., Odemer, R., Boltin, J., Di Poto, C., Mehmann, M.M., Retschnig, P., Winiger, P., Rosenkranz, P., Neumann, P., 2015. Effects, but no interactions, of ubiquitous pesticide and parasite stressors on honey bee (Apis mellifera) lifespan and behaviour in a colony environment: Stressor effects in honey bee colonies. Environmental Microbiology 17, 4322–4331. https://doi.org/10.1111/1462-2920.12825

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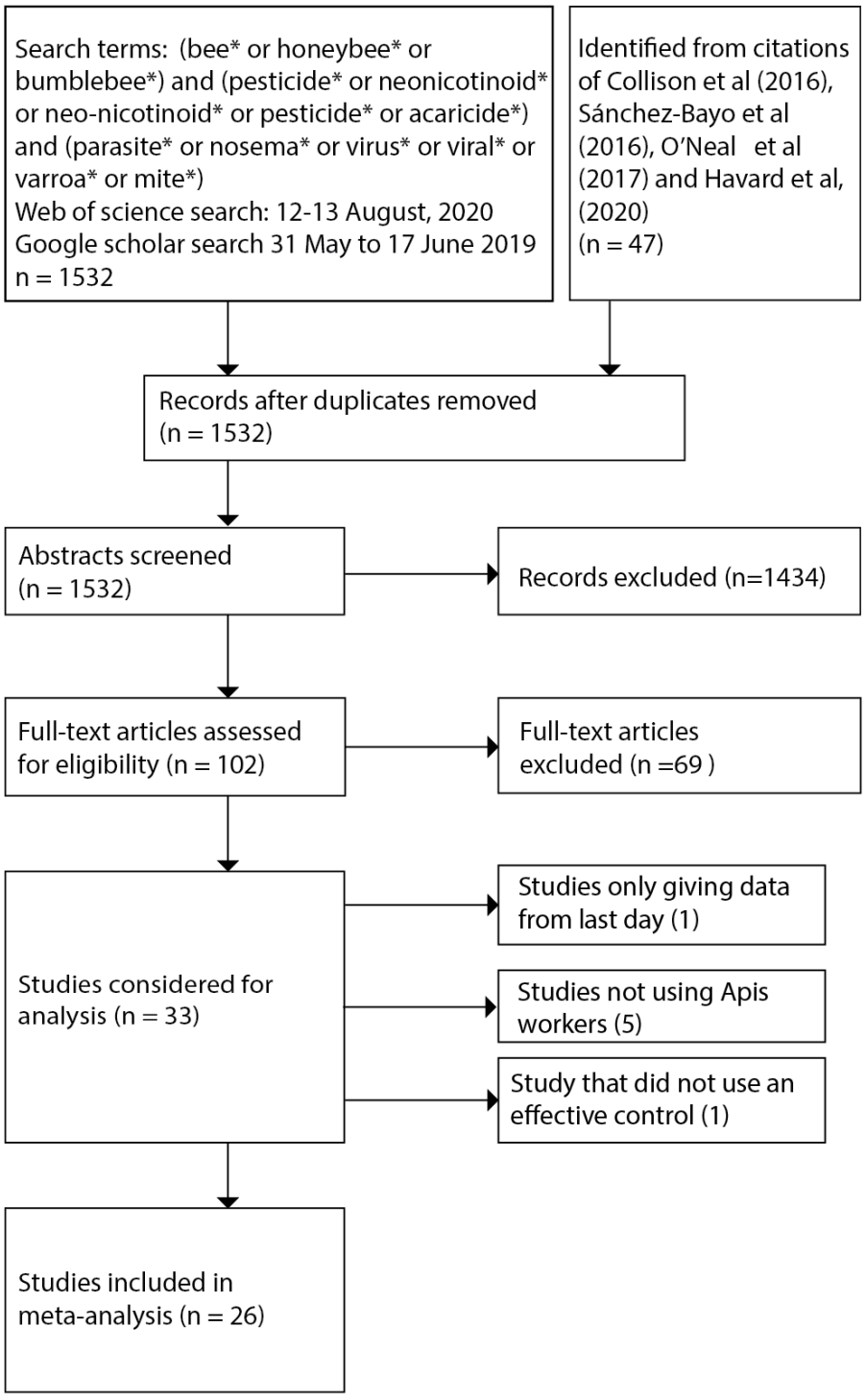
Vidau, C., Diogon, M., Aufauvre, J., Fontbonne, R., Viguès, B., Brunet, J.-L., Texier, C., Biron, D.G., Blot, N., El Alaoui, H., Belzunces, L.P., Delbac, F., 2011. Exposure to sublethal doses of fipronil and thiacloprid highly increases mortality of honeybees previously infected by Nosema ceranae. PLoS ONE 6, e21550. https://doi.org/10.1371/journal.pone.0021550

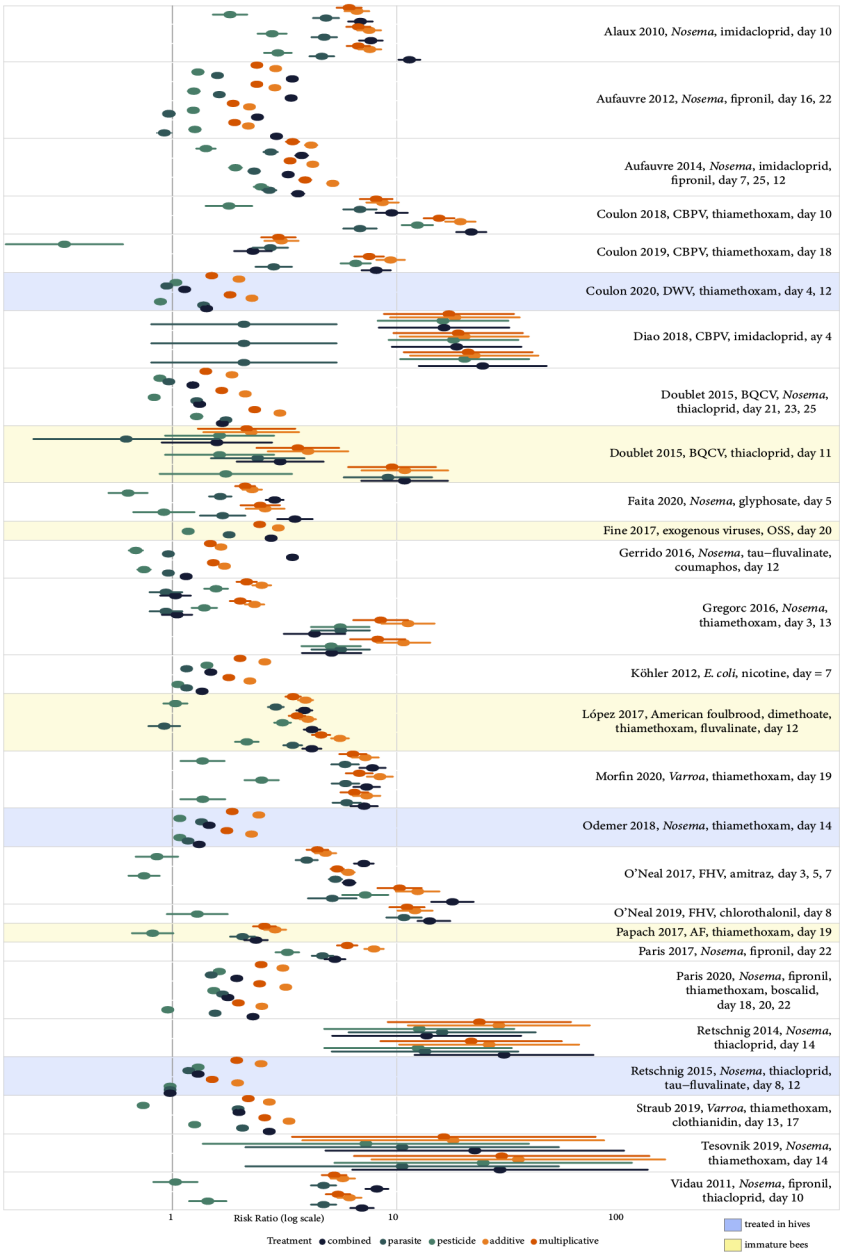
2. Supplementary figures

Supplementary figure 1. PRISMA diagram for literature search.

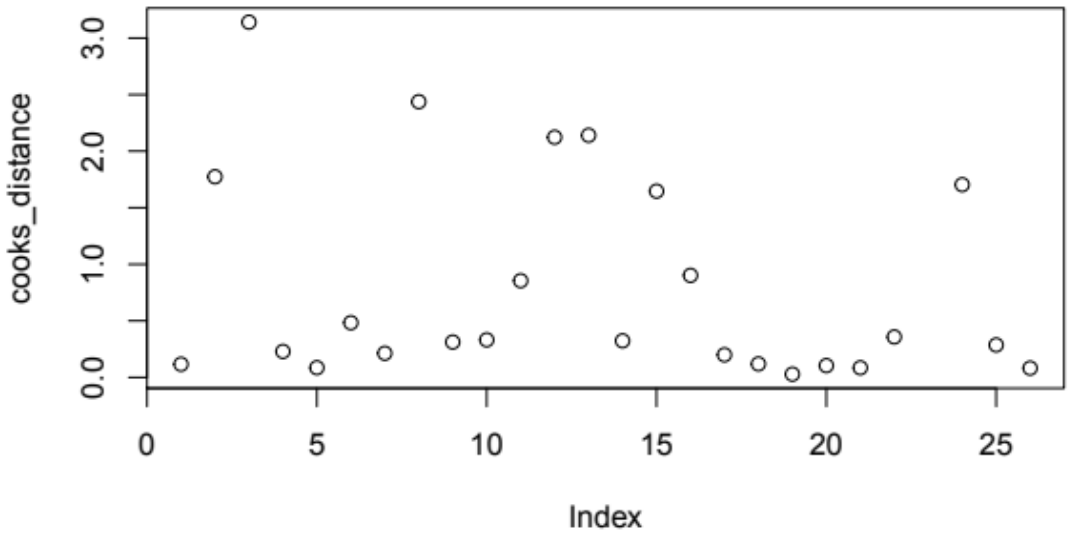


(Moher et al 2009)

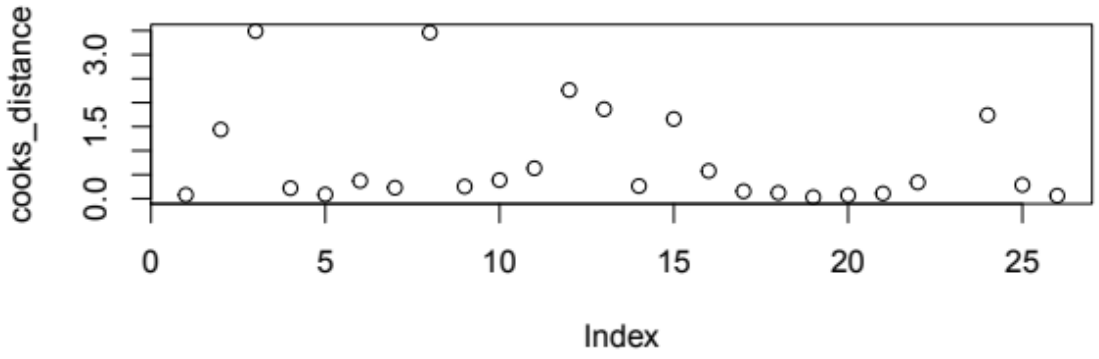
Supplementary figure 2. Forest plot showing published effects of pesticides and parasites, individually and in concert, on the mortality of honey bees. Also shown are predicted additive and multiplicative effects for each trial. Closed circles show risk ratios, and horizontal lines show 95% confidence intervals. The vertical dashed line shows a risk ratio of 1.0, which corresponds to no change in mortality relative to the control. Study citations are on the right, along with data on trial duration and parasite and pesticide identities. Shaded boxes indicate experiments where bees were immature or were treated in hives. Abbreviations: CPVB = chronic bee paralysis virus, AF = American foulbrood, BQCV = black queen cell virus, DVW = deformed wing virus, OSS = organic sulfate surfactants.


Alaux 2010, *Nosema*, imidacloprid, day 10
Aufauvre 2012, *Nosema*, fipronil, day 16, 22
Aufauvre 2014, *Nosema*, imidacloprid, fipronil, day 7, 25, 12
Coulon 2018, CBPV, thiamethoxam, day 10
Coulon 2019, CBPV, thiamethoxam, day 18
Coulon 2020, DWV, thiamethoxam, day 4, 12
Diao 2018, CBPV, imidacloprid, ay 4
Doublet 2015, BQCV, *Nosema*, thiacloprid, day 21, 23, 25
Doublet 2015, BQCV, thiacloprid, day 11
Faita 2020, *Nosema*, glyphosate, day 5
Fine 2017, exogenous viruses, OSS, day 20
Gerrido 2016, *Nosema*, tau−fluvalinate, coumaphos, day 12
Gregorc 2016, *Nosema*, thiamethoxam, day 3, 13
Köhler 2012, *E. coli*, nicotine, day = 7
López 2017, American foulbrood, dimethoate, thiamethoxam, fluvalinate, day 12
Morfin 2020, *Varroa*, thiamethoxam, day 19
Odemer 2018, *Nosema*, thiamethoxam, day 14
O'Neal 2017, FHV, amitraz, day 3, 5, 7
O'Neal 2019, FHV, chlorothalonil, day 8
Papach 2017, AF, thiamethoxam, day 19
Paris 2017, *Nosema*, fipronil, day 22
Paris 2020, *Nosema*, fipronil, thiamethoxam, boscalid, day 18, 20, 22
Retschnig 2014, *Nosema*, thiacloprid, day 14
Retschnig 2015, *Nosema*, thiacloprid, tau−fluvalinate, day 8, 12
Straub 2019, *Varroa*, thiamethoxam, clothianidin, day 13, 17
Tesovnik 2019, *Nosema*, thiamethoxam, day 14
Vidau 2011, *Nosema*, fipronil, thiacloprid, day 10
1
10
100
Risk Ratio (log scale)
Treatment
combined
parasite
pesticide
additive
multiplicative
treated in hives
immature bees

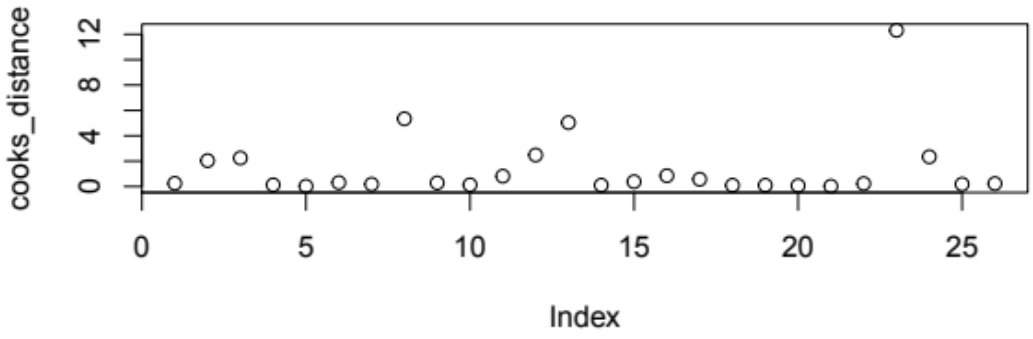
Supplementary figure 3. Cook's distance test for outliers for the predicted additive model



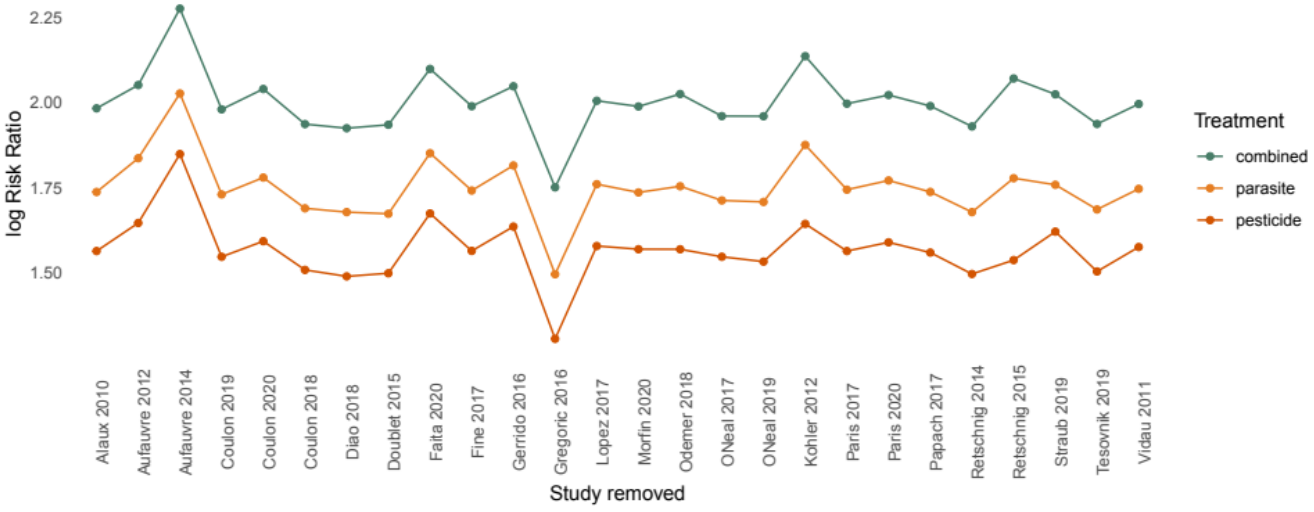
Supplementary figure 4. Cook's distance test for outliers for the predicted multiplicative model



Supplementary figure 5. Cook's distance test for outliers for the single and multiple effects model



Supplementary Figure 6. Leave one out analysis for single and multiple effects model



3. Supplementary tables

Supplementary Table 1. I^2 scores

Model	Total I^2	Between study I^2	Between trial I^2
Predicted additive	95.93	80.58	15.34
Predicted multiplicative	95.76	83.18	12.57
Treatment comparison	95.03	79.59	15.44

Supplementary Table 2. Comparison of combined and predicted additive effects of pesticide and parasite treatments on honey bee mortality, along with estimated moderator effects. The intercept term provides a basis for comparison and corresponds to the observed combined treatment effect, with bees treated in cages, with viruses, and with neonicotinoid pesticides. Trial duration is mean-centered. The I^2 estimates for this model were 95.78% total heterogeneity, 79.56% between-study heterogeneity, and 16.22% within-study heterogeneity.

Moderators	Log Risk Ratio estimate (raw)	Risk Ratio estimate (anti-logged)	t-value	Confidence intervals (raw)	P-value
Intercept	**2.2**	**9.01**	**7.37**	**1.61 – 2.79**	**0**
Treatment (combined additive)	**0.37**	**1.44**	**4.61**	**0.32 – 0.42**	**0**
Life stage (immatures)	0.18	1.2	0.56	-0.46 – 0.82	0.57
Accommodation (hive)	-1.3	0.27	-2.45	-2.35 – -0.25	0.02
Pesticide family (non-neonicotinoid)	0	0.91	-0.54	-0.45 – 0.25	0.59
Pesticide family (mixed)	0	0.95	0	-1.76 – 1.66	0.95
Trial duration (per day, 3-25)	**0**	**0.96**	**-3.17**	**-0.07 – -0.016**	**0**
Parasite pair-wise tests					
Bacteria – virus	-0.92	0	-1.91	-2.13 – 0.3 0	0.21
Nosema – virus	-0.11	0.89	-0.45	-0.76 – 0.53	0.97
Varroa - virus	-0.16	0.85	-0.29	-1.58 – 1.25	0.99
Bacteria – *Nosema*	0	2.23	1.59	-0.48 – 2.08	0.36
Bacteria – *Varroa*	0.75	2.13	1.11	-0.96 – 2.47	0.66
Nosema – *Varroa*	-0.05	0.95	-0.09	-1.41 – 1.31	1

Supplementary Table 3. Comparison of combined and predicted multiplicative effects of pesticide and parasite treatments on honey bee mortality, along with estimated moderator effects. The intercept term provides a basis for comparison and corresponds to the combined treatment effect, with bees treated in cages, infected with *Nosema* spp, and exposed to neonicotinoids, at trial day = 0. The I^2 estimates for this model were 95.76% total heterogeneity, 83.18% between-study heterogeneity, and 12.57% within-study heterogeneity.

Moderators	Log Risk Ratio estimate (raw)	Risk Ratio estimate (anti-logged)	t-value	Confidence intervals (raw)	P-value
Intercept	**2.21**	**9.12**	**7.48**	**1.62 – 2.80**	**0**
Treatment (multiplicative)	**0.14**	**1.15**	**5.44**	**0.09 0 – 0.19**	**0**
Life stage (immatures)	0.27	1.31	0.85	-0.36 – 0.9 0	0.4
Accommodation (hive)	**-1.34**	**0.26**	**-2.48**	**-2.41 – -0.27**	**0.02**
Pesticide family (non-neonicotinoid)	-0.07	0.94	-0.39	-0.4 0 – 1.68	0.69
Pesticide family (mixed)	0	0.94	0	-1.81 – 0.27	0.94
Trial duration (per day, 3-25)	**0**	**0.96**	**-3.31**	**-0.068 – -0.02**	**0**
Parasite pair-wise tests					
Nosema – bacteria	-0.97	0.38	-1.99	-2.2 0 – 0.26	0.18
Varroa – bacteria	0	0	-0.41	-0.73 – 0.52	0.97
Virus – bacteria	-0.15	0.86	-0.27	-1.59 – 1.28	0.99
Varroa – Nosema	0.87	2.38	0	-0.42 – 2.16	0.3
Virus – Nosema	0.81	2.26	1.18	-0.92 – 2.55	0.62
Virus – Varroa	-0.05	0.95	-0.1	-1.43 – 1.33	1

Supplementary Table 4. Effects of single and combined stressors on honey bee mortality, along with estimated moderator effects. The first three rows show the main effects of combined, only-parasite, and only-pesticide treatments relative to a predicted null effect when bees are kept in cages at day = 0. (This model was fit without an intercept for ease of interpretation). The next three rows show the effect of other moderator variables. The final three rows each show a pairwise comparison, where the p-values and confidence intervals were corrected for multiple testing. I^2 statistics show 95.03% of the heterogeneity were explained by random effects, with 79.59% explained by between-study heterogeneity and 15.44% explained by within-study heterogeneity.

Moderators	Log Risk Ratio estimate (raw)	Risk Ratio estimate (anti-logged)	t-value	Confidence intervals (raw)	P-value
Treatments compared to control:					
Combined treatment	**2**	**7.41**	**8.51**	**1.54 – 2.47**	**0**
Parasite treatment	**1.75**	**5.76**	**7.44**	**1.29 – 2.22**	**0**
Pesticide treatment	**1.57**	**4.81**	**6.66**	**1.1 0 – 2.03**	**0**
Other predictor variables:					
Accommodation (hive)	**-0.05**	**0.32**	**-2.68**	**-1.98 – -0.3 0**	**0.01**
Trial duration (per day, 3-40)	**-0.05**	**0.95**	**-3.62**	**-0.07 1 – -0.02 1**	**0**
Life stage (immatures)	0.04	1.04	0.14	-0.48 – 0.55	0.89
Pairwise comparisons:					
Parasite – combined	**-0.25**	**0.78**	**-9.1**	**-0.32 – -0.19**	**0**
Pesticide – combined	**-0.43**	**0.65**	**-15.15**	**-0.5 0 – -0.37**	**0**
Pesticide – parasite	**-0.18**	**0.83**	**-6.17**	**-0.25 – -0.11**	**0**

Supplementary Table 5. Re-analysis of individual published studies, with the significance of non-additive or non-multiplicative effects assessed with non-parametric bootstrapping. Predicted effects were calculated as in the main meta-analysis, and then subtracted from the observed combined effect ('Difference' in the table). Interactions were considered synergistic if the predicted treatment effect was less than the observed combined treatment, and antagonistic if the predicted treatment was greater than the combined treatment, with an alpha level of 0.05. More details are given in the code provided below in Supp. Code 2.

		Additive			Multiplicative		
Trial	Study	Interaction	Difference	P-value	Interaction	Difference	P-value
Al1	Alaux 2010	No interaction	-0.01	0.74	No interaction	-0.04	0.29
Al2	Alaux 2010	No interaction	-0.01	0.87	No interaction	-0.06	0.19
Al3	Alaux 2010	Synergism	-0.23	0	Synergism	-0.29	0
Au1	Aufauvre 2012	Synergism	-0.13	0.01	Synergism	-0.25	0
Au2	Aufauvre 2012	Synergism	-0.13	0.01	Synergism	-0.24	0
Au3	Aufauvre 2012	No interaction	-0.05	0.37	Synergism	-0.15	0.01
Au4	Aufauvre 2012	Synergism	-0.18	0	Synergism	-0.25	0
Auf1	Aufauvre 2014	No interaction	0.07	0.16	No interaction	-0.06	0.29
Auf2	Aufauvre 2014	Antagonism	0.18	0	No interaction	0.01	0.79
Auf3	Aufauvre 2014	Antagonism	0.29	0	No interaction	0.05	0.32
Co1	Coulon 2018	No interaction	-0.04	0.43	No interaction	-0.06	0.15
Co2	Coulon 2018	Synergism	-0.1	0	Synergism	-0.27	0
Cou1	Coulon 2019	No interaction	0.08	0.14	No interaction	0.08	0.17
Cou2	Coulon 2019	Antagonism	0.13	0	No interaction	-0.06	0.31
Coul1	Coulon 2020	Antagonism	0.42	0	Antagonism	0.18	0
Coul2	Coulon 2020	Antagonism	0.31	0	Antagonism	0.15	0
Di1	Diao 2018	No interaction	0.07	0.36	No interaction	0.03	0.61
Di2	Diao 2018	No interaction	0.04	0.48	No interaction	0.01	0.85

		Additive			Multiplicative		
Trial	Study	Interaction	Difference	P-value	Interaction	Difference	P-value
Di3	Diao 2018	No interaction	-0.06	0.43	No interaction	-0.1	0.16
Do1	Doublet 2015	Antagonism	0.31	0	No interaction	0.09	0.12
Do2	Doublet 2015	Antagonism	0.33	0	Antagonism	0.14	0.02
Do3	Doublet 2015	Antagonism	0.42	0	Antagonism	0.2	0
Do4	Doublet 2015	No interaction	0.06	0.37	No interaction	0.06	0.38
Do5	Doublet 2015	No interaction	0.1	0.25	No interaction	0.06	0.46
Do6	Doublet 2015	No interaction	0	0.85	No interaction	-0.1	0.18
Fa1	Faita 2020	No interaction	-0.09	0.12	Synergism	-0.12	0.04
Fa2	Faita 2020	No interaction	-0.08	0.14	No interaction	-0.09	0.08
Fi1	Fine 2017	No interaction	0.05	0.23	No interaction	-0.07	0.12
Ge1	Gerrido 2016	Synergism	-0.46	0	Synergism	-0.5	0
Ge2	Gerrido 2016	Antagonism	0.15	0	Antagonism	0.1	0.03
Gre1	Gregoric 2016	Antagonism	0.36	0	Antagonism	0.27	0
Gre2	Gregoric 2016	Antagonism	0.32	0	Antagonism	0.24	0
Gre3	Gregoric 2016	Antagonism	0.6	0	Antagonism	0.36	0
Gre4	Gregoric 2016	Antagonism	0.49	0	Antagonism	0.28	0
Ko1	Kohler 2012	Antagonism	0.39	0	Antagonism	0.18	0
Ko2	Kohler 2012	Antagonism	0.3	0	Antagonism	0.15	0
Lo1	Lopez 2017	No interaction	0.01	0.86	No interaction	-0.06	0.28
Lo2	Lopez 2017	No interaction	-0.03	0.61	No interaction	-0.09	0.09
Lo3	Lopez 2017	Antagonism	0.19	0	No interaction	0.06	0.31
Mo1	Morfin 2020	No interaction	-0.06	0.26	Synergism	-0.16	0
Mo2	Morfin 2020	Antagonism	0.12	0.01	No interaction	-0.06	0.32
Mo3	Morfin 2020	No interaction	0.02	0.75	No interaction	-0.08	0.19
Od1	Odemer 2018	Antagonism	0.39	0	Antagonism	0.16	0
Od2	Odemer 2018	Antagonism	0.37	0	Antagonism	0.17	0

		Additive			Multiplicative		
Trial	Study	Interaction	Difference	P-value	Interaction	Difference	P-value
On1	ONeal 2017	Synergism	-0.27	0	Synergism	-0.31	0
On2	ONeal 2017	No interaction	-0.01	0.25	Synergism	-0.11	0
On3	ONeal 2017	Synergism	-0.3	0	Synergism	-0.42	0
One1	ONeal 2019	Synergism	-0.17	0	Synergism	-0.24	0
Pa1	Papach 2017	No interaction	0.09	0.21	No interaction	0.04	0.58
Par1	Paris 2017	Antagonism	0.33	0	No interaction	0.09	0.1
Pari1	Paris 2020	Antagonism	0.3	0	Antagonism	0.14	0.01
Pari2	Paris 2020	Antagonism	0.43	0	Antagonism	0.2	0
Pari3	Paris 2020	Antagonism	0.08	0.04	Synergism	-0.11	0.02
Re1	Retschnig 2014	Antagonism	0.39	0	Antagonism	0.25	0
Re2	Retschnig 2014	No interaction	-0.11	0.13	Synergism	-0.22	0
Ret1	Retschnig 2015	Antagonism	0.42	0	Antagonism	0.23	0
Ret2	Retschnig 2015	Antagonism	0.46	0	Antagonism	0.25	0
St1	Straub 2019	Antagonism	0.26	0	Antagonism	0.07	0.04
St2	Straub 2019	Antagonism	0.17	0	No interaction	-0.04	0.28
Te1	Tesovnik 2019	No interaction	-0.1	0.32	No interaction	-0.13	0.16
Te2	Tesovnik 2019	No interaction	0.13	0.1	No interaction	0.02	0.86
Vi1	Vidau 2011	Synergism	-0.25	0	Synergism	-0.29	0
Vi2	Vidau 2011	No interaction	-0.09	0.13	Synergism	-0.15	0.01