

Research



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Evolutionary biology

Interactions between insect vectors and plant pathogens span the parasitism–mutualism continuum

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Agricultural crops infected with vector-borne pathogens can suffer severe negative consequences, but the extent to which phytopathogens affect the fitness of their vector hosts remains unclear. Evolutionary theory predicts that selection on vector-borne pathogens will favour low virulence or mutualistic phenotypes in the vector, traits facilitating effective transmission between plant hosts. Here, we use a multivariate meta-analytic approach on 115 effect sizes across 34 unique plant–vector–pathogen systems to quantify the overall effect of phytopathogens on vector host fitness. In support of theoretical models, we report that phytopathogens overall have a neutral fitness effect on vector hosts. However, the range of fitness outcomes is diverse and span the parasitism–mutualism continuum. We found no evidence that various transmission strategies, or direct effects and indirect (plant-mediated) effects, of phytopathogens have divergent fitness outcomes for the vector. Our finding emphasizes diversity in tripartite interactions and the necessity for pathosystem-specific approaches to vector control.

1. Introduction

Many viral and bacterial pathogens that cause plant disease epidemics rely on herbivorous insect vectors for transmission [1,2]. Vector-borne pathogens should be selected to enhance their transmission to plant hosts, via direct effects in the vector host or indirectly by manipulating the host plant [3]. However, high virulence to the vector can negatively impact transmission as phytopathogens rely on the mobility of the vector for transmission and dispersal to non-motile plant hosts [4–6]. Consequently, evolutionary theory predicts that vector-borne agents will be relatively less virulent to the vector or even have beneficial phenotypes in the vector host [7–10]. Despite predictions, a wide range of fitness effects have been reported across vector species. Squash vein yellowing virus (SqVYV) improves the longevity and fecundity of its whitefly vector [11], whereas Watermelon bud necrosis virus (WBNV) reduces these fitness-associated parameters in its vector *Thrips palmi* [12]. Contradictory results have also been reported within the same taxa: *Candidatus Liberibacter asiaticus* positively affects the citrus psyllid's fecundity [13,14], but negatively affects survival and longevity [14,15], underscoring the complexity of vector–pathogen interactions. The extent to which phytopathogens affect the fitness of vector hosts remains unquantified (figure 1*a*), despite the importance for the ecology and evolution of these pathosystems (a plant–pathogen–vector association) [3].

The majority of attention has focused on the nature of pathogen tropism, retention and replication in the vector (referred to as transmission mode) [3]. Non-circulative pathogens are restricted to the vector's stylet or foregut for short periods. Circulative viruses, and all bacteria, enter the haemocoel and

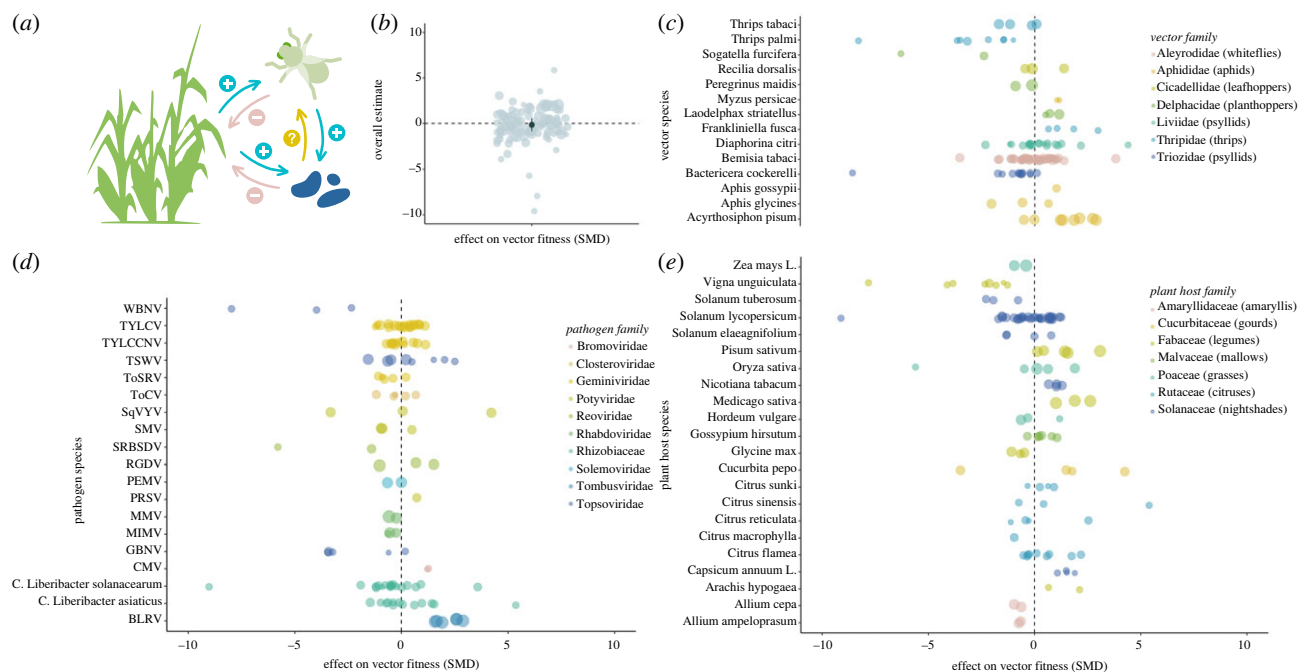


Figure 1. Crops infected with vector-borne phytopathogens suffer negative consequences, but the extent to which phytopathogens affect the fitness of their vector hosts is unclear (a). Vector–phytopathogen interactions are diverse and range from parasitic to mutualistic (SMD = -0.1354 , 95% CI: -0.8088 – 0.5381) (b). Mean effect and 95% confidence intervals are plotted in black. Effect sizes reported according to vector species (c), pathogen species (d) and plant host species (e). Individual effect sizes are jittered and coloured by family. Point size represents study sample size.

render the vector infectious for longer periods [16], with some also propagating within the vector [17]. Consequently, it has been suggested that non-circulative, non-persistent pathogens will predominantly have indirect effects on vector fitness, for example by affecting the volatile profile of infected plants [18] or altering plant defence against herbivory [19]. By contrast, circulative, propagative viruses and bacteria are more likely to affect vector fitness directly, for example by hijacking the vector's cellular machinery, in addition to plant-mediated indirect effects. The outcome of vector–pathogen interactions may be affected by additional factors such as pathogen and vector taxonomy [20,21], differences in study methods [22], and whether vertical transmission occurs in the vector (see electronic supplementary material, table S1 for hypotheses).

Previous vote-counting studies highlight a positive effect of phytopathogens on their vectors [3,23,24]; these have focused either exclusively on viruses or included effects on vector behaviour. Here, we use a meta-analytic approach to test the hypothesis that vector-borne bacterial and viral phytopathogens benefit their insect vectors. We include only fitness-associated metrics to assess the scope for mutualism in these interactions. Further analysis tests for ecological drivers of variation in vector–pathogen interactions. Our synthesis of 34 pathosystems suggests the mean effect of infection on vector fitness is neutral, but the range of outcomes is considerable and spans the parasitism–mutualism continuum. As fitness costs affect the abundance and population dynamics of insect vectors, an improved understanding of vector–pathogen interactions will be critical for preventing plant disease epidemics.

2. Methods

(a) Literature review

We searched Web of Science for relevant studies and retrieved 888 papers published up to July 2021 (electronic supplementary

material, information and figure S3). Studies on plant pathosystems were included when each experiment had a treatment and control group with independent samples; an infection challenge, where greater than or equal to 70% of individuals were infected; a standardized way to quantify fitness; and greater than or equal to five observations. Studies that evaluated the effects of temperature, co-infections, or starvation, and studies testing the effect of infection on host preference and feeding behaviours, were excluded. Overall, 26 studies were included covering 34 unique pathosystems.

(b) Calculation of effect sizes

Of the 115 effect sizes extracted, 85 were in count form and 30 were dichotomous. To combine the datasets, dichotomous data were first expressed as odds ratios and accompanying s.e. as described in [25], then converted into standardized mean differences (SMDs) [26]. Where count data were provided, the SMD was calculated as the difference in mean outcome between the infected and control groups, divided by the standard deviation of the outcome among participants. Where mean outcomes were not reported, data were obtained from plots by extracting data points using web-plotdigitiser [27]. Missing s.e. and sample sizes were extrapolated using methods described in [28].

(c) Meta-analytical model

Most studies reported multiple effect sizes. To account for any dependencies within studies, multi-level models were fitted using restricted maximum-likelihood estimation using the *metafor* package (v3.0-2) [29] in R v. 4.0.5 [30,31]. Nesting the effects reported within the study ID allowed for differentiation of the effect sizes due to sampling variation within and between studies [32]. Pathogen species, host species and vector species were also included as random effects, allowing multiple representations of the same species to be accounted for [33]. Taxonomic subgroup analysis was performed by calculating mean effect sizes for each pathogen genus and vector genus. The contribution of ecological and methodological predictors to the overall effect was then assessed using univariate models (electronic supplementary

material, table S1). Omnibus tests were used to assess differences in mean effect size between groups, and likelihood ratio tests using maximum-likelihood estimation were used to assess the significance of each predictor.

(d) Assessing bias

Publication bias was visually assessed using funnel plots (electronic supplementary material, figure S4). The weighted Rosenberg method [34] was used to calculate a fail-safe number, which estimates the number of studies averaging a null result that would have to be added to the dataset to reduce the significance level to a target alpha level (e.g. 0.05). The fail-safe number was 581, which is not greater than the threshold considered for a robust analysis ($nfs > 5N + 10$ where N = no. effect sizes). These results suggest that the dataset could be affected by bias towards positive results. However, as noted by others [23], negative results would be biologically interesting in this case and less likely to remain unpublished.

3. Results

All 34 pathosystems included domesticated crop hosts and insect vectors, 29% of the vector-borne agents were bacteria and 71% were viruses (table 1).

(a) Neutral effect of infection on vector host

The mean effect of phytopathogen infection on vector hosts was neutral (figure 1b), but highly variable (full model: $SMD = -0.1354$, $s.e. = 0.3436$, 95% CI: -0.8088 , 0.5381 , $p = 0.6936$) with interactions spanning from parasitic to mutualistic. Heterogeneity across studies was high ($Q(d.f. = 114) = 1090.6284$, $p < 0.0001$).

(b) Subgroup analysis according to phylogeny

Mean effects were calculated for taxonomic subgroups at the family level and most showed neutral effects overall. However, associations in the vector family Triozidae (jumping plant lice) were associated with a negative impact on vector fitness, ($SMD = -1.2066$, $s.e. = 0.5329$, 95% CI = -2.2511 , -0.1621 , $p = 0.0236$) and in the pathogen family Tombusviridae (single-stranded RNA viruses) a positive effect was observed ($SMD = 2.0543$, $s.e. = 0.2404$, 95% CI = 1.5831 , 2.5255 , $p = < 0.0001$).

(c) Biological predictors

We tested multiple ecological factors to assess if they accounted for variation in the effect of infection on vector fitness (figure 2). Fitness effects did not vary between bacterial and viral pathogens ($QM(d.f. = 1) = 0.2041$, $p = 0.6514$), and transmission mode did not significantly affect the fitness of the vector host ($QM(d.f. = 3) = 0.7933$, $p = 0.8511$). Estimates for vector fitness were not impacted by pathogens that are also transmitted vertically in the vector (horizontal/mixed-mode transmission, $QM(d.f. = 1) = 0.0090$, $p = 0.9244$). We did not detect significant differences between the measures used to estimate fitness (e.g. longevity, fecundity, fertility and survival ($QM(d.f. = 3) = 1.0689$, $p = 0.7846$)).

Pathosystems were classified by whether the infections were expected to directly affect fitness as they infect the vector host (Direct effects); indirectly affect fitness by altering plant host defences to herbivory and/or facilitating predation

(Indirect effects); a certain combination of direct and indirect effects (Both) or a likely combination of the two (Likely both). For classification criteria see electronic supplementary material, figure S2. We were unable to detect significant differences in the fitness outcomes conferred by infections across these categories ($QM(d.f. = 3) = 0.6531$, $p = 0.8842$).

Overall, sex was not a significant driver of variation in vector-phytopathogen outcomes ($QM(d.f. = 2) = 5.5433$, $p = 0.0626$). However, infected males were generally more negatively affected by infection ($SMD = -0.9706$, 95% CI = -1.9570 , 0.0159) than females ($SMD = -0.2159$, 95% CI = -0.9220 , 0.4902) or mixed groups ($SMD = 0.1208$, 95% CI = -0.6425 , 0.8842).

(d) Methodological predictors

We found limited evidence that methodological factors influenced the effect of infection on vector host fitness. The method of pathogen inoculation to the vector host did not significantly affect fitness outcomes ($QM(d.f. = 2) = 0.2068$, $p = 0.9018$), and for studies that inoculated the plant host no effect of inoculation method was detected ($QM(d.f. = 3) = 2.2931$, $p = 0.5138$). The parties experimentally infected during the study also did not have a significant impact on the infection outcome ($QM(d.f. = 2) = 0.6016$, $p = 0.7402$). However, vector fitness was more negatively affected in studies where both plant and vector hosts, or only the vector host was infected ($SMD = -0.1619$, 95% CI = -1.276 , 0.9522 ; $SMD = -0.3198$, 95% CI = -1.1064 , 0.4668), whereas vectors showed neutral fitness effects in studies where only the plant host was infected ($SMD = -0.0189$, 95% CI = -0.7388 , 0.7011).

Finally, the amount of time allotted for the vector to acquire the pathogen (acquisition time) did not significantly affect the outcome of infection ($QM(d.f. = 2) = 3.7372$, $p = 0.1543$). That said, vectors that had shorter acquisition periods of a week or less generally had reduced fitness ($SMD = -1.0049$, 95% CI = -2.1278 , 0.118) but those with longer acquisition periods (adult to death/lifetime) showed neutral fitness effects ($SMD = 0.0542$, 95% CI = -0.8832 , 0.9917 ; $SMD = 0.2564$, 95% CI = -0.5433 , 1.0561).

4. Discussion

Evolutionary theory suggests that vector-borne pathogens will be selected for low virulence or even beneficial phenotypes in the vector host, as mobility of this host is crucial for transmission [5,6,56]. Across 34 pathosystems, we show the average effect of infection on vector fitness is neutral, in line with theoretical models [7–10]. However, our analysis shows vector-pathogen interactions span the symbiosis continuum, like many other host-microbe interactions [57], with infections ranging from beneficial to highly detrimental for vectors.

Phylogeny is frequently an important predictor of host-microbe interactions [20,21]. Our analysis revealed some differences in infection outcomes among pathogen and vector families; however, the number of species represented was limited. Sex-specific differences also drive variation in host-parasite interactions [58], and in our study male vectors appeared to suffer more from associations. The negative trend for males may be influenced by differences in immunocompetence [59]; alternatively, less harm may be favoured in females who are generally the dominant transmitters [60].

Table 1. Summary of pathosystems included.

plant host	vector	pathogen	fitness effects ^a	effect ^b	references
<i>Viral pathosystems</i>					
<i>Allium ampeloprasum</i> (wild leek)	<i>Thrips tabaci</i> (onion thrips)	tomato spotted wilt virus (TSWV)	adult longevity, fecundity	both	[35]
<i>Allium cepa</i> (onion)	<i>Thrips tabaci</i>	tomato spotted wilt virus (TSWV)	adult longevity, survival	both	[35]
<i>Arachis hypogaea</i> (groundnut)	<i>Frankliniella fusca</i> (tobacco thrips)	tomato spotted wilt virus (TSWV)	fecundity	likely both	[36]
<i>Capsicum annuum</i> L. (red pepper)	<i>Frankliniella fusca</i> (tobacco thrips)	cucumber mosaic virus (CMV)	fecundity	indirect	[37]
<i>Capsicum annuum</i> L.	<i>Frankliniella fusca</i>	tomato spotted wilt virus (TSWV)	fecundity	likely both	[37]
<i>Capsicum annuum</i> L.	<i>Myzus persicae</i> (green peach aphid)	cucumber mosaic virus (CMV)	fecundity	indirect	[37]
<i>Capsicum annuum</i> L.	<i>Myzus persicae</i>	tomato spotted wilt virus (TSWV)	fecundity	likely both	[37]
<i>Cucurbita pepo</i> (pumpkin)	<i>Aphis gossypii</i> (melon aphid)	papaya ring spot virus (PRSV)	fecundity	indirect	[38]
<i>Cucurbita pepo</i>	<i>Bemisia tabaci</i> (whitefly)	squash vein yellowing virus (SqVYV)	adult longevity, fecundity	indirect	[11]
<i>Glycine max</i> (soya bean)	<i>Aphis glycines</i> (soya bean aphid)	soya bean mosaic virus (SMV)	fecundity	indirect	[39,40]
<i>Gossypium hirsutum</i> (cotton)	<i>Bemisia tabaci</i> (whitefly)	tomato yellow leaf curl China virus (TYLCCNV)	fecundity	direct	[41]
<i>Gossypium hirsutum</i>	<i>Bemisia tabaci</i>	tomato yellow leaf curl virus (TYLCV)	adult longevity, fecundity	likely both	[42]
<i>Hordeum vulgare</i> (barley)	<i>Laodelphax striatellus</i> (small brown planthopper)	maize Iranian mosaic virus (MIMV)	adult longevity, fecundity	direct	[43]
<i>Medicago sativa</i> (alfalfa)	<i>Acyrtosiphon pisum</i> (pea aphid)	bean leafroll virus (BLRV)	survival	likely both	[44]
<i>Nicotiana tabacum</i> (tobacco)	<i>Bemisia tabaci</i> (whitefly)	tomato yellow leaf curl China virus (TYLCCNV)	adult longevity, fecundity	likely both	[41]
<i>Oryza sativa</i> (rice)	<i>Recilia dorsalis</i> (zigzag leafhopper)	rice gall dwarf virus (RGDV)	adult longevity, fecundity, survival	direct	[45]
<i>Oryza sativa</i> (rice)	<i>Sogatella furcifera</i> (white- backed planthopper)	southern rice black-streaked dwarf virus (SRBSDV)	fecundity, fertility	direct	[46]
<i>Pisum sativum</i> (pea plant)	<i>Acyrtosiphon pisum</i> (pea aphid)	bean leafroll virus	survival	both	[44]
<i>Pisum sativum</i>	<i>Acyrtosiphon pisum</i>	pea enation mosaic virus (PEMV)	fecundity, survival	likely both	[47]
<i>Solanum lycopersicum</i> (tomato)	<i>Bemisia tabaci</i> (whitefly)	tomato chlorosis virus (ToCV)	adult longevity, fecundity, fertility, survival	indirect	[48]
<i>Solanum lycopersicum</i>	<i>Bemisia tabaci</i>	tomato severe rugose virus (ToSRV)	adult longevity, fecundity, fertility, survival	indirect	[48]

(Continued.)

Table 1. (Continued.)

plant host	vector	pathogen	fitness effects ^a	effect ^b	references
<i>Solanum lycopersicum</i>	<i>Bemisia tabaci</i>	tomato yellow leaf curl China virus (TYLCCNV)	adult longevity, fecundity, survival	likely both	[49]
<i>Solanum lycopersicum</i>	<i>Bemisia tabaci</i>	tomato yellow leaf curl virus (TYLCV)	adult longevity, fecundity, survival	likely both	[42,49]
<i>Vigna unguiculata</i> (cowpea)	<i>Thrips palmi</i> (melon thrips)	groundnut bud necrosis virus (GBNV)	adult longevity, fecundity	direct, likely both	[50]
<i>Vigna unguiculata</i>	<i>Thrips palmi</i>	WBNV	adult longevity, fecundity	likely both	[12]
<i>Zea mays</i> L. (corn)	<i>Peregrinus maidis</i> (corn planthopper)	maize mosaic virus (MMV)	adult longevity, fecundity	both	[51]
<i>Bacterial pathosystems</i>					
<i>Citrus flamea</i> (shatangju)	<i>Diaphorina citri</i> (Asian citrus psyllid)	<i>Candidatus</i> Liberibacter asiaticus	adult longevity, fecundity, survival	both	[14]
<i>Citrus macrophylla</i> (alemow)	<i>Diaphorina citri</i>	<i>Candidatus</i> Liberibacter asiaticus	adult longevity	likely both	[15]
<i>Citrus reticulata</i> (tangerine)	<i>Diaphorina citri</i>	<i>Candidatus</i> Liberibacter solanacearum	adult longevity, fecundity, fertility, survival	likely both	[52]
<i>Citrus sinensis</i> (sweet orange)	<i>Diaphorina citri</i>	<i>Candidatus</i> Liberibacter asiaticus	fecundity, fertility, survival	both	[13]
<i>Citrus sunki</i> (sour mandarin)	<i>Diaphorina citri</i>	<i>Candidatus</i> Liberibacter solanacearum	adult longevity, fecundity, fertility, survival	likely both	[52]
<i>Solanum elaeagnifolium</i> (silverleaf nightshade)	<i>Bactericera cockerelli</i> (potato psyllid)	<i>Candidatus</i> Liberibacter solanacearum	adult longevity, fecundity, survival	direct	[53]
<i>Solanum lycopersicum</i> (tomato)	<i>Bactericera cockerelli</i>	<i>Candidatus</i> Liberibacter solanacearum	fecundity, fertility	direct	[54,55]
<i>Solanum tuberosum</i> (potato)	<i>Bactericera cockerelli</i>	<i>Candidatus</i> Liberibacter solanacearum	adult longevity, fecundity	direct	[53]

^aMeasure/proxy of vector fitness.

^bWhether the study tested direct effects of phytopathogen association with vector (Direct); indirect effects driven by altered plant host state (Indirect); a combination of direct and indirect effects when both vector and plant were confirmed infected (Both); or a likely combination of the two in cases where the plant was infected but vector may have gained infection (but not confirmed) from feeding (Likely both). See electronic supplementary material, figure S2 for detail.

For the systems studied herein, circulative pathogens had similar fitness effects on vectors to those that do not circulate within the host. Similarly, we found no evidence that direct effects of infection differ significantly from indirect effects. These findings, contrary to our initial hypothesis, come with two caveats: the number of studies remains relatively small, and vectors typically acquire pathogens by feeding on infected plants, making it challenging to disentangle the direct and indirect effects of the pathogen on the insect vector. Some studies limited the contribution of indirect effects by infecting vectors *in vitro* [14,41,43,45,46,50,53–55] or transferring them regularly onto unexposed plants [50]. For most other studies, whether effects were direct, indirect, likely both, or certainly both were inferred based on pathogen

transmission mode and the experimentally infected party (electronic supplementary material, figure S2). More studies are needed that distinguish the direct and indirect effects of plant pathogens on vectors, and establish whether mutualistic phenotypes evolve more readily within a given effect class.

Methodological differences frequently drive variation in study outcomes. For example, plant hosts inoculated mechanically versus vector inoculated will not induce processes such as herbivore effector-triggered immunity or other changes to plant chemical composition [61–63]. However, inoculation method explained little of the variation in vector fitness, suggesting methods may be relatively comparable. Notably, negative fitness outcomes were slightly more prevalent if the vector was the only party infected in

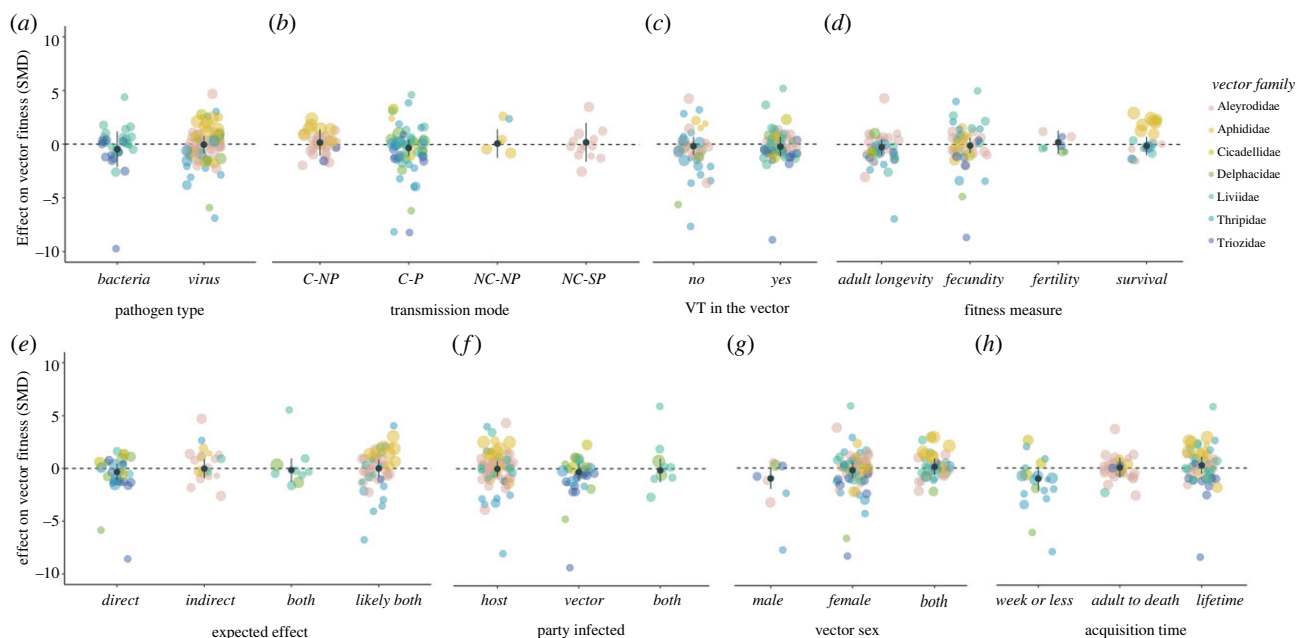


Figure 2. Variation in the effect of infection on vector fitness was not significantly affected by pathogen type (a), transmission mode (circulative non-propagative; circulative-propagative; non-circulative, non-persistent; non-circulative, semi-persistent) (b), vertical transmission in vector (c), fitness measure (d), direct or indirect effects (e), vector sex (f), party infected (g) or acquisition time (h). Individual effect sizes are jittered and coloured by vector family. Point size represents study sample size. Mean effect sizes and 95% confidence intervals are plotted in black.

the experiment. This suggests that beneficial phenotypes in the vector may depend upon the interaction of both direct and plant-mediated (indirect) effects of a pathogen. To disentangle the contribution of direct and indirect effects, studies must infect each party individually and in combination.

Domesticated crop hosts that are closely related, and grown in dense, low diversity, cultures are overrepresented here. These characteristics likely shape the interaction between vector and phytopathogen. Non-domesticated pathosystems differ greatly in these characteristics [64], and would be a valuable point of comparison for the evolution of tripartite interactions. Such systems are underrepresented in the literature. Given the controlled conditions of experiments, it is also likely that context-dependent cost/benefits for vector hosts are unaccounted for here.

Our study formally quantifies the effect of agriculturally important phytopathogens on insect vectors. We report an overall neutral fitness effect, but one that is underpinned by considerable diversity in both parasitic and mutualistic phenotypes. This finding highlights the value of

vector–phytopathogen systems for exploring the evolution of tripartite symbiotic interactions and emphasizes the necessity for pathosystem-specific approaches to vector control.

Data accessibility. All data, code and supplementary files associated with the manuscript are publicly available at: <https://doi.org/10.6084/m9.figshare.c.6215267.v2> [65].

Authors' contributions. M.F.M.S.: data curation, formal analysis, investigation, methodology, visualization, writing—original draft; K.C.K.: conceptualization, funding acquisition, supervision and writing—review and editing; G.C.D.: conceptualization, supervision, validation, writing—original draft and writing—review and editing.

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

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

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