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Tri-trophic interactions mediate the spread of a vector-borne plant pathogen

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Abstract. Many insect herbivores are vectors that transmit plant pathogens as they forage. Within food webs, vectors interact with a range of host plants, other herbivores, and predators. Yet, few studies have examined how tri-trophic interactions involving vectors affect the spread of pathogens. Here we assessed effects of food web structure on aphid vectors and the prevalence of an aphid-borne persistent pathogen (Pea enation mosaic virus, PEMV) in pea plants. We experimentally manipulated ladybird predators, alternative host plants, and non-vector herbivores and assessed responses of pea aphids and PEMV using structural equation models. We show that variation in bottom-up, top-down, and horizontal interactions mediated PEMV prevalence. Predators reduced PEMV prevalence by consuming aphids, but an alternative host plant (vetch) had the opposite effect by promoting aphid movement and abundance. Non-vector herbivores (pea leaf weevil) increased PEMV susceptibility in peas. In turn, weevils offset the positive effects of predators on PEMV, but increased the negative effects of vetch. Our results show that tri-trophic interactions within insect and plant food webs can mediate vector biology with synergistic and opposing effects on pathogens. Continuing to assess how community-wide interactions affect vectors will aid in our understanding of vector-borne pathosystems.

Key words: agroecology; aphids; direct effects; food webs; indirect effects; plant–insect–pathogen interactions; structural equation modeling; vector biology.

Introduction

Insect herbivores are plant pathogen vectors in many ecosystems (Gray and Banerjee 1999). Vectors forage in heterogenous environments and interact with other herbivores and predators as they move within and among host plants. Interactions between vectors and predators, hosts, and other herbivores can in turn affect the transmission of plant viruses by altering vector abundance (density-mediated effects) and behavior (trait-mediated effects; Power 1991, Smyrnioudis et al. 2001, Malmstrom et al. 2005, Hodge and Powell 2008, Thaler et al. 2010, Dáder et al. 2012, Finke 2012, Ostfeld and Keesing 2012, Long and Finke 2015, Chisholm et al. 2018).

Empirical studies show predators often reduce vector abundance, but the threat of predation may promote vector movement (Preisser et al. 2005, Hodge and Powell 2008, Long and Finke 2015). Increased vector movement due to predators can promote pathogen spread, even though predators reduce vector abundance, by increasing the rate at which vectors encounter new hosts (Jeger et al. 2012). However, greater vector movement due to predators may decrease pathogen spread if moving vectors spend less time feeding (Long and Finke 2015). Non-vector herbivores may similarly affect vector abundance and behavior by altering host plant quality (Thaler et al. 2010, Chisholm et al. 2018). For example, herbivory by non-vectors can increase expression of anti-herbivore plant defenses, which can decrease vector performance and alter vector movement behavior in ways that affect pathogen transmission (Thaler et al. 2010, Chisholm et al. 2018). Moreover, vectors often forage in heterogenous plant communities, where vector fitness and host choice are affected by variation in plant traits across host species (Kim and Underwood 2015).

Despite evidence that interactions between vectors and other species might affect pathogens, few empirical or modeling studies have examined such interactions in

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a food web framework. This is problematic because effects of trophic interactions may vary based on food web structure (Agrawal 2000), as effects of predators and non-vector herbivores on vectors can be contingent on host plant traits (Mooney and Singer 2012, Chisholm et al. 2018). For example, when plants express resistance to vectors, vector abundance decreases, which can in turn limit the ability of predators to find and consume vector prey (Kersch-Becker and Thaler 2015). This suggests that a better assessment of how tri-trophic interactions involving vectors affect pathogens in complex food webs could greatly increase our understanding of disease ecology.

Here, we assessed how tri-trophic interactions affected a vector-borne plant pathogen. We manipulated interactions between *Acyrthosiphon pisum* (pea aphid) vectors and *Hippodamia convergens* (convergent ladybeetle) predators, *Pisum sativum* (pea) and *Vicia sativa* (common vetch) hosts, and *Sitona lineatus* (pea leaf weevil) herbivores. We measured prevalence of an aphid-borne persistent pathogen (*Pea-enation mosaic virus*, PEMV) in pea plants in response to this trophic network. Data were analyzed with path analysis to assess how ecological interactions involving aphid vectors affected PEMV through density- and trait-mediated pathways. Our study shows that interactions involving vectors within complex tri-trophic food webs can exert strong direct and indirect effects on the spread of persistent pathogens through plant communities.

Methods

Study system

Pea aphids are generalist herbivores that transmit pathogens while feeding on phloem (Ng and Perry 2004). PEMV is a persistent circulative RNA virus that is obligately transmitted by aphids, with pea aphids the main vector (Hodge and Powell 2008). PEMV infection occurs when a pair of mutualistic RNA viruses (an *Enamovirus* and *Umbravirus*) are transmitted during aphid feeding, with host infection occurring within hours (Chisholm et al. 2018). In landscapes that produce legumes, such as the Palouse region (eastern Washington and northern Idaho), outbreaks of PEMV are economically devastating (Paudel et al. 2018). In the Palouse, pea aphids co-occur with *S. lineatus* and *H. convergens* on *P. sativum* plants (see Appendix S1 for more details).

Interactions within food webs may affect PEMV by altering aphid fitness and behavior. For example, predators like *H. convergens* eat aphids, but predators induce aphid movement, which can increase infection (Hodge and Powell 2008). Pea aphids also feed on crop (*P. sativum*) and weedy (*V. sativa*) legumes (McVean and Dixon 2002). Weedy hosts acting as sources of aphids to crops, but aphid fitness differs across host species (Davis et al. 2015). Aphids also co-occur on crops with herbivores like *S. lineatus*, whose feeding increases plant susceptibility to PEMV by decreasing expression of salicylic acid hormone (Chisholm et al. 2018). Such interactions may thus mediate the spread of PEMV, but they have not been examined in a food web framework.

We constructed an a priori network to test predictions about effects of food web structure on aphids and PEMV (Fig. 1). First, we expected predators (*H. convergens*) would decrease PEMV prevalence by reducing aphid abundance; low-quality alternative hosts (*V. sativa*) were expected to have similar effects. Second, we predicted predators may affect PEMV prevalence by altering aphid movement; we also predicted that aphids would move away from lower-quality vetch hosts to peas. Increased vector movement could increase PEMV incidence if aphids encounter more susceptible hosts (Smyrnioudis et al. 2001, Jeger et al. 2012, Crowder et al. 2019) but may decrease PEMV incidence if movement decreases the time aphids spend feeding (Long and Finke 2015, Crowder et al. 2019). Third, we expected non-vector herbivores (*S. lineatus*) might affect PEMV by increasing susceptibility of hosts to aphids and PEMV (Chisholm et al. 2018).

Effects of ecological interactions on aphids and PEMV

We conducted greenhouse experiments to test our predictions (see Appendix S1 for more details). We selected 16 2-week-old pea plants and 16 2-week-old vetch plants that were cultivated in the greenhouse and placed 25 adult maternal viruliferous aphids on the stem of each plant. Aphids came from viruliferous colonies that originated from dry pea fields in Whitman County, Washington, USA, and Latah County, Idaho, USA (Chisholm et al. 2018). In July 2017, we collected adult *S. lineatus* and *H. convergens* from pea fields in Colton, Washington with sweep netting. Field collected *S. lineatus* and *H. convergens* were stored in a low-temperature humid incubator at 4°C to induce brumation and then used in both temporal blocks of the experiment.

After 24 h for aphids to establish, plants were placed in mesh dorms (35 × 35 × 60 cm). Each dorm was randomly assigned to three treatments in a factorial 2 × 2 × 2 experiment: (1) source plant (pea or vetch); (2) non-vector herbivore (*S. lineatus* present or absent); and (3) predator (*H. convergens* present or absent). The plant on which aphids were established was the "source" in each dorm. We then added 10 two-week-old "recipient" pea plants in five rows (two per row); each dorm thus had two columns of five recipient plants adjacent to a source. Pots and tendrils of pea plants touched to allow aphid movement (Appendix S1: Fig. S1). For non-vector treatments, we added five *S. lineatus* adults 24 h before source and recipient plants were combined (this herbivore to plant ratio was based on Chisholm et al. 2018). For predator treatments, we added five adult *H. convergens* at the same time recipient plants were placed into dorms (this predator to plant ratio was based on Northfield et al. [2012]). Each of the eight unique

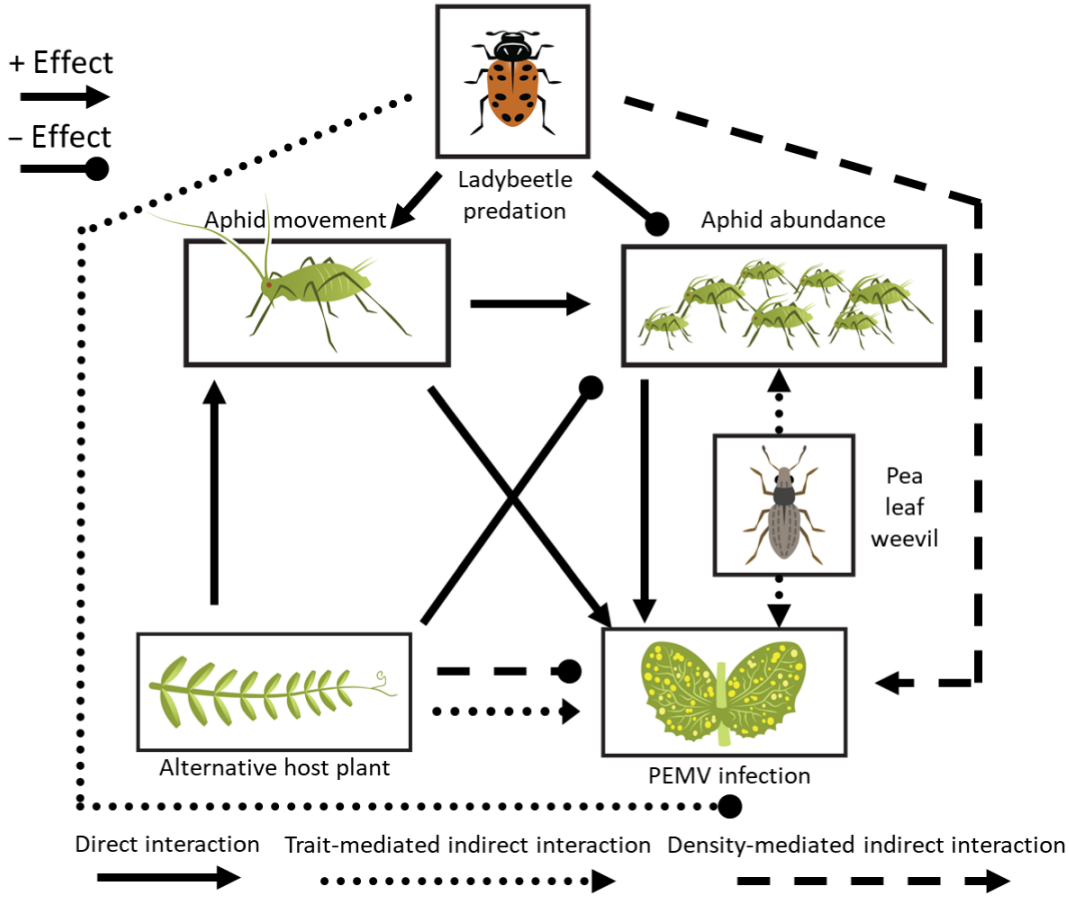

FIG. 1. Interaction network with a priori predictions about indirect effects and directionality of responses. Boxes show treatments (predators, source hosts, or herbivores) and response variables (movement, abundance, and prevalence). We predicted that predators and hosts may directly affect vector movement and abundance, while herbivores may indirectly affect vector abundance and plant susceptibility via plant trait-mediated mechanisms. PEMV, pea enation mosaic virus.

treatments in the 2 × 2 × 2 design was replicated four times per temporal block, for 32 total replicates per block. The experiment was conducted in two blocks (the second began 30 August 2017), with 64 replicates in total.

After mesocosms were set up, we tracked aphid abundance and adult movement (Appendix S1: Fig. S2) on each plant every 24 h for 8 d using visual scans with a dental mirror to minimize disturbance. In block one, we removed aphids by hand after 9 d, and after an additional 7 d we collected plant tissue for PEMV quantification (Chisholm et al. 2018). Block two used the same procedures except we treated pots after 9 d with a granular formulation of imidacloprid (Bayer Crop Science, Montvale, New Jersey, USA) to kill all aphids; as in block one, we then collected plant tissue after an additional 7 d. The two aphid removal methods (hand vs. insecticide) did not significantly affect the final prevalence of PEMV in recipient pea plants (GLMM, $\chi^2 = 0.44$, $P = 0.51$).

PEMV infection in recipient pea plants was determined using rtPCR (Chisholm et al. 2018). We froze entire plants in liquid nitrogen and stored samples at −80°C at the end of each temporal block. Plant tissue was ground in sterile conditions with a mortar and pestle and liquid nitrogen. We completed RNA extraction, cDNA synthesis, and PCR using PEMV primers for coat protein (see Appendix S1). For this assay, we pooled columns of plants (Appendix S1: Fig. S3), testing for PEMV infection in plant samples in electrophoresis gels. To demonstrate the reliability of this pooling approach, we further tested whether PEMV prevalence was correlated with direct quantitative estimate of PEMV titer using q-rtPCR (see Appendix S1 for more details) and found that the two metrics were significantly associated (GLMM, $\chi^2 = 13.95$, $P < 0.001$).

Data analysis

Analyses were conducted in R v. 3.5.2 (R Development Core Team 2018) using the packages *lme4* for generalized linear mixed models (GLMMs; Bates et al. 2015) and *piecewiseSEM* for structural equation models

(SEMS; Lefcheck 2016). Parameter estimates (predicted marginal means and standard errors) and posthoc tests were calculated using the *emmeans* package (Lenth 2016). Significance tests were based on analysis of deviance χ^2 tests using the *car* package (Fox and Weisberg 2018). All models used experimental replicate as a random effect unless indicated.

We used SEMs to test a priori predictions (Fig. 1), with predator, weevil, and host treatments as predictors, and aphid abundance, aphid movement, and PEMV prevalence as responses (Fig. 1). PEMV prevalence was the log-odds ratio of infected columns for each separate treatment (32 observations per treatment). For analysis, nonsignificant paths that reduced AIC were dropped, and paths were added if models without them were rejected via directed separation tests (Lefcheck 2016). Parameters reflect numerical (aphid counts or average distance moved) or proportional (virus prevalence) responses (Fig. 2). We partitioned aphid counts into two response variables. Vector movement was the average distance of adult aphids in each column (e.g., source plant = distance of 0, row 5 = distance of 5; $n = 128$). Vector abundance was the log-transformed number of aphid nymphs produced in an entire plant column. The primary treatment effects used to construct the model (predators, alternative hosts, and weevils), and their effects on nymph production, movement, and PEMV infection are plotted in Appendix S1: Fig. S4.

We next ran a series of GLMMs to examine mechanisms driving patterns seen in SEMS, but focused on total aphid abundance (adult and nymphs). We modeled abundance on all recipient plants over 7 d as a response, with predator, weevil, and source host treatments as fixed effects. In this repeated measures design, replicate × day was treated as a random effect ($n = 448$). We ran an additional GLMM on the average abundance of aphids on each row of plants (rows 1–5, with 1 closest to source) to assess relative distribution of all aphids at the end of the 7-d assay.

Results

Food web path model

Aphid abundance was directly and positively correlated with PEMV prevalence, while adult aphid movement was indirectly correlated with PEMV prevalence. Predators suppressed aphids (Fig. 2; Appendix S1: Table S1), which reduced PEMV prevalence. Predators also increased aphid movement, but this effect's magnitude was lower than predator effects on abundance, and movement did not directly affect PEMV (Fig. 2; Appendix S1: Table S1). Vetch source plants increased adult aphid movement and aphid abundance (Appendix S1: Table S1). Consequently, both the direct and indirect effects of vetch increased PEMV prevalence (Fig. 2; Appendix S1: Table S1). Weevils affected relationships between aphid abundance and PEMV prevalence (Fig. 3). At low aphid abundance (<100), PEMV prevalence increased when weevils were present, while prevalence approached zero at low aphid abundance when weevils were absent (Fig. 3; >0.25 log odds ratio vs. <0.25 log odds ratio, respectively). However, when aphid abundance was high (>100), weevils did not have a significant effect on PEMV prevalence (Fig. 3).

Mechanisms driving variation in vector abundance and vector movement

Aphid abundance was reduced by predators, and the magnitude of this effect increased as aphid abundance increased (Appendix S1: Table S2). Predators reduced aphid abundance across all rows of recipient plants regardless of distance from the source (Appendix S1: Fig. S5, Table S3). In contrast, vetch increased aphid abundance on recipient pea plants (Appendix S1: Table S2). We observed more aphids on pea source plants throughout the experiment compared to vetch source plants (Appendix S1: Fig. S6). Conversely, aphid abundance was higher on distal plants when aphids started on vetch than pea by the end of 7 d (Appendix S1: Fig. S6, Table S3). Weevils did not affect aphid abundance or movement (Appendix S1: Fig. S7, Tables S2, S3).

Discussion

Our study shows that interactions between a host, a vector, and a vector-borne pathogen were mediated by predators, non-vector herbivores, and alternative host plants. Notably, the effects of pairwise-interactions among vectors and other species for pathogens were context dependent in the broader food web. For example, weevils make host plants more susceptible to PEMV (Fig. 1; Chisholm et al. 2018) and decrease the density-mediated aphid threshold needed to infect plants (Fig. 1). When weevils were present with predators, in terms of PEMV prevalence, weevils thus acted in opposition to predators (Fig. 1). However, in terms of PEMV prevalence, weevils acted in synergy with vetch because both promoted PEMV. This suggests that non-vector herbivores strongly affect plant pathosystems through plant-mediated indirect mechanisms that may impact multiple species (Thaler et al. 2010, Kersch-Becker and Thaler 2015, Chisholm et al. 2018).

Our results demonstrating density- and trait-mediated indirect effects are in line with studies showing that top-down effects of predators can affects pathogens through consumptive and non-consumptive pathways (Finke 2012, Long and Finke 2015). We found that predators decreased aphid abundance but increased aphid movement, which has been observed for other predators (Long and Finke 2015). Disease ecology models suggest that when predators both reduce vector abundance and increase vector movement, effects on pathogens depend on the magnitude and direction of effects (Finke 2012,

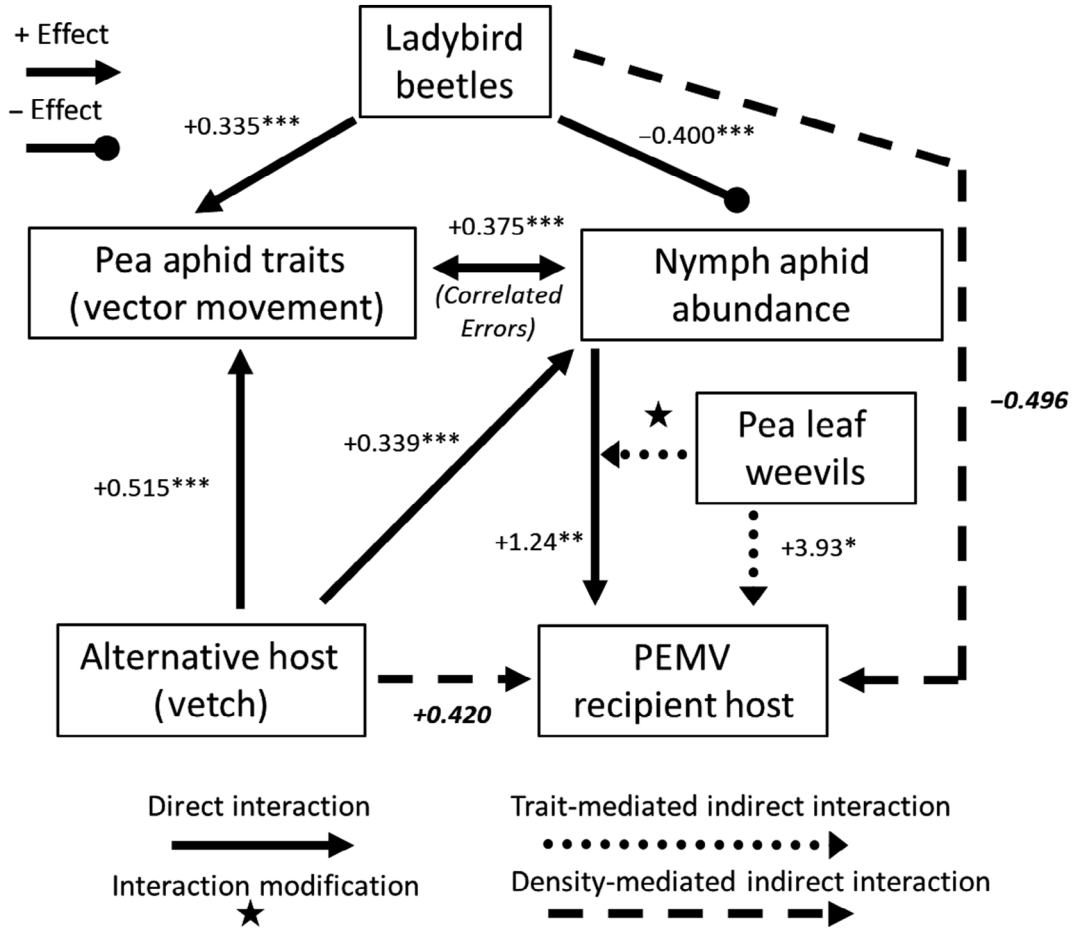

FIG. 2. Supported model from confirmatory path analysis ($P = 0.75$, Fischer's $C = 6.77$, and the number of independence claims $k = 12$). Bidirectional arrow shows correlated errors between vector abundance and movement. Density-mediated indirect effects of predators and vetch are indicated with dashed lines, with estimates coefficients derived from intermediate paths. Weevils altered the prevalence of PEMV at lower aphid densities (aphid × weevil interaction, $P = 0.034$, Fig. 3; Appendix S1: Table S1). Number of asterisks indicate levels of significance (*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$).

Jeger et al. 2012, Crowder et al. 2019). In this system, we found that the magnitude of predator effects on aphid abundance, which reduced PEMV prevalence, were considerably stronger than predator effects on aphid movement. Moreover, while aphid abundance was directly linked with PEMV prevalence, aphid movement was not. Thus, we show that predators reduced PEMV prevalence primarily through direct consumptive effects (Fig. 2).

Reduced pathogen prevalence in response to predators has also been observed for aphid-borne viruses of wheat (Long and Finke 2015). In this system, effects of predators on virus were attributed to reduced aphid feeding time, where aphids escaping predators spent less time feeding than aphids not exposed to predation threat. This matches other studies where non-consumptive effects of predators on reducing wheat dwarf virus prevalence was mediated by reduced aphid feeding in response to predators (Tholt et al. 2018). Our study shows predators have the strongest effects on pathogens when they suppress vector abundance even if vector movement increases. However, it is possible that the increased aphid movement we observed in response to predators was also associated with decreased feeding time, which could have resulted in the overall lack of direct effects of vector movement on PEMV prevalence (Crowder et al. 2019). More broadly, these results show that considering both density- and trait-mediated effects of pairwise interactions can reveal which effects predominate in driving the spread of pathogens.

Our study also shows that alternative hosts may strongly affect vector-borne pathosystems. Herbivores can be affected by traits of nearby plants (Kim and Underwood 2015), but empirical work on such plant-associational effects in pathosystems is lacking. Our results showed vetch source plants increased aphid movement to recipient pea plants. Models shows that

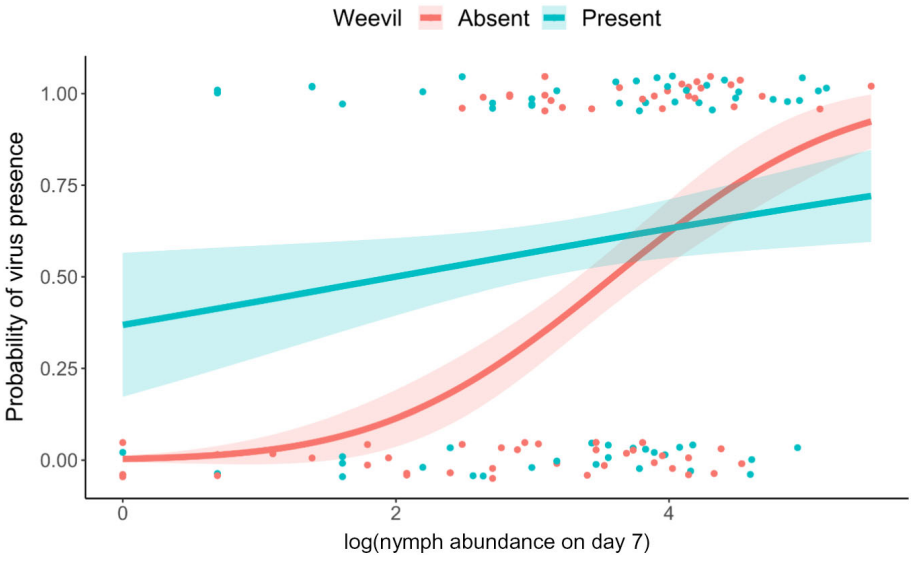

FIG. 3. Effects of weevils on PEMV across levels of aphid abundance. Weevils increased plant susceptibility to PEMV by lowering the aphid threshold required for infection: infection occurred in plants even at relatively low aphid densities. Lines indicate predicted slope from binomial GLMM for weevil × nymph interaction term, with 95% CI shaded. Dots indicate experimental observations for each replicate.

prevalence of persistent pathogens can increase in response to density-dependent movement (Shaw et al. 2017). Given our design, we could not directly test if this mechanism affected PEMV. However, our results are consistent with this idea, as aphids quickly reached a movement threshold on vetch, which had a lower carrying capacity than pea, resulting in increased aphid movement. Moreover, mesocosms with vetch plants also had higher aphid abundance than those with pea source plants.

These results suggest that low-quality vetch plants may relax intraspecific density-dependent competition among aphids by promoting colonization of more recipient pea plants early in the experiments, but the specific response of aphids to alternative host-plant quality requires further investigation. These mechanisms fit similar models in herbivore movement between plant species where the traits of adjacent plants strongly determine herbivory (Hambäck et al. 2014). Pea-associated host races of aphids in the Palouse commonly use both vetch and pea as hosts, and vetch is a common weed found throughout disturbed areas surrounding crop fields (R. E. Clark, *personal observations*). To our knowledge this study provides some of the first evidence that associational effects among hosts may play an important role in dynamics of pathogens.

While our study revealed strong effects of pairwise interactions involving aphid vectors on PEMV, it also shows why such interactions need to be assessed within a multi-trophic food web context. Weevils, predators, aphids, and PEMV often co-occur on vetch and pea plants of the Palouse (Chisholm et al. 2018). While predators may reduce PEMV prevalence by consuming aphids, if weevils co-occur on the same hosts the effects of weevils may counteract the effects of predators. Similarly, predators and vetch may act in opposition in terms of affecting PEMV, as predators decrease aphid abundance while vetch has the opposite effects. Vetch and weevils would be expected to act synergistically, as both promote PEMV through effects on aphids and host plant susceptibility. From an applied perspective, management of pea aphids and PEMV may benefit from tactics that suppress weevils and vetch or those that promote predators.

Our study supports recent reviews and models that posit food web theory can make important predictions about the potential direct and indirect effects of ecological interactions involving vectors on pathogen transmission (Seabloom et al. 2015, Eigenbrode et al. 2017, Crowder et al. 2019). Notably, at food web scales, effects of predators, herbivores, and hosts on vectors and pathogens can be synergistic or act in opposition. In these cases, the strength and magnitude of positive and negative effects on vector fitness and movement mediate the transmission of pathogens. More broadly, we show that a priori food web models can be a useful tool in predicting the local spread of vector-borne pathogens. Manipulative studies that integrate multitrophic frameworks to assess plant–arthropod–pathogen communities will aid in our understanding of vector-borne pathogens and promote management of harmful plant diseases.

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Literature Cited

Agrawal, A. A. 2000. Mechanisms, ecological consequences and agricultural implications of tri-trophic interactions. Current Opinion in Plant Biology 3:329–335.

Bates, D., M. Mächler, B. Bolker, and S. Walker. 2015. Fitting linear mixed-effects models using lme4. Journal of Statistical Software 67:1–48.

Chisholm, P. J., N. Sertsuvalkul, C. L. Casteel, and D. W. Crowder. 2018. Reciprocal plant-mediated interactions between a virus and a non-vector herbivore. Ecology 99:2139–2144.

Crowder, D. W., J. Li, E. T. Borer, D. L. Finke, R. Sharon, D. E. Pattemore, and J. Medlock. 2019. Species interactions affect the spread of vector-borne plant pathogens independent of transmission mode. Ecology 100:e02782. https://doi.org/10.1002/ecy.2782

Dáder, B., A. Moreno, E. Viñuela, and A. Fereres. 2012. Spatio-temporal dynamics of viruses are differentially affected by parasitoids depending on the mode of transmission. Viruses 4:3069–3089.

Davis, T. S., Y. Wu, and S. D. Eigenbrode. 2015. Chickpea variety and phenology affect acquisition of pea enation mosaic virus, subsequent plant injury and aphid vector performance: Virus acquisition in chickpea. Annals of Applied Biology 167:420–425.

Eigenbrode, S. D., N. Bosque-Pérez, and T. S. Davis. 2017. Insect-borne plant pathogens and their vectors: ecology, evolution, and complex interactions. Annual Review of Entomology 63:169–191.

Finke, D. L. 2012. Contrasting the consumptive and non-consumptive cascading effects of natural enemies on vector-borne pathogens. Entomologia Experimentalis et Applicata 144:45–55.

Fox, J., and S. Weisberg. 2018. Visualizing fit and lack of fit in complex regression models with predictor effect plots and partial residuals. Journal of Statistical Software 87:1–27.

Gray, S. M., and N. Banerjee. 1999. Mechanisms of arthropod transmission of plant and animal viruses. Microbiology and Molecular Biology Reviews 63:128–148.

Hambäck, P. A., B. D. Inouye, P. Andersson, and N. Underwood. 2014. Effects of plant neighborhoods on plant–herbivore interactions: resource dilution and associational effects. Ecology 95:1370–1383.

Hodge, S., and G. Powell. 2008. Complex interactions between a plant pathogen and insect parasitoid via the shared vector-host: consequences for host plant infection. Oecologia 157:387–397.

Jeger, M., Z. Chen, E. Cunningham, G. Martin, and G. Powell. 2012. Population biology and epidemiology of plant virus epidemics: from tripartite to tritrophic interactions. European Journal of Plant Pathology 133:3–23.

Kersch-Becker, M. F., and J. S. Thaler. 2015. Plant resistance reduces the strength of consumptive and non-consumptive effects of predators on aphids. Journal of Animal Ecology 84:1222–1232.

Kim, T. N., and N. Underwood. 2015. Plant neighborhood effects on herbivory: damage is both density and frequency dependent. Ecology 96:1431–1437.

Lefcheck, J. S. 2016. piecewiseSEM: Piecewise structural equation modelling in r for ecology, evolution, and systematics. Methods in Ecology and Evolution 7: 573–579.

Lenth, R. V. 2016. Least-squares means: The R package lsmeans. Journal of Statistical Software 69:1–33.

Long, E. Y., and D. L. Finke. 2015. Predators indirectly reduce the prevalence of an insect-vectored plant pathogen independent of predator diversity. Oecologia 177:1067–1074.

Malmstrom, C. M., A. J. McCullough, H. A. Johnson, L. A. Newton, and E. T. Borer. 2005. Invasive annual grasses indirectly increase virus incidence in California native perennial bunchgrasses. Oecologia 145:153–164.

McVean, R. I. K., and A. F. G. Dixon. 2002. The host plant range of the pea aphid subspecies *Acyrthosiphon pisum* ssp. destructor (Johnson) (Hom., Aphididae). Journal of Applied Entomology 126:281–286.

Mooney, K. A., and M. S. Singer. 2012. Plant effects on herbivore-enemy interactions in natural systems. Pages 107–130 *in* T. Ohgushi, O. Schmitz, and R. D. Holt, editors. Ecology and evolution of trait-mediated indirect interactions. Cambridge University Press, Cambridge, UK.

Ng, J. C. K., and K. L. Perry. 2004. Transmission of plant viruses by aphid vectors. Molecular Plant Pathology 5:505–511.

Northfield, T. D., W. E. Snyder, G. B. Snyder, and S. D. Eigenbrode. 2012. A simple plant mutation abets a predator-diversity cascade. Ecology 93:411–420.

Ostfeld, R. S., and F. Keesing. 2012. Effects of host diversity on infectious disease. Annual Review of Ecology, Evolution, and Systematics 43:157–182.

Paudel, S., E. J. Bechinski, B. S. Stokes, H. R. Pappu, and S. D. Eigenbrode. 2018. Deriving economic models for pea aphid (Hemiptera: Aphididae) as a direct-pest and a virus-vector on commercial lentils. Journal of Economic Entomology 111:2225–2232.

Power, A. G. 1991. Virus spread and vector dynamics in genetically diverse plant populations. Ecology 72:232–241.

Preisser, E. L., D. I. Bolnick, and M. F. Benard. 2005. Scared to death? The effects of intimidation and consumption in predator–prey interactions. Ecology 86:501–509.

R Development Core Team. 2018. R version 3.5.2. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria.

Seabloom, E. W., E. T. Borer, K. Gross, A. E. Kendig, C. Lacroizx, C. E. Mitchell, E. A. Mordecai, and A. G. Power. 2015. The community ecology of pathogens: coinfection, coexistence, and community composition. Ecology Letters 18:401–415.

Shaw, A. K., A. Peace, A. G. Power, and N. A. Bosque-Pérez. 2017. Vector population growth and condition-dependent movement drive the spread of plant pathogens. Ecology 98:2145–2157.

Smyrnioudis, I. N., R. Harrington, S. J. Clark, and S. J. Katis. 2001. The effect of natural enemies on the spread of barley yellow dwarf virus (BYDV) by *Rhopalosiphum padi* (Hemiptera: Aphididae). Bulletin of Entomological Research 91:301–306.

Thaler, J. S., A. A. Agrawal, and R. Halitschke. 2010. Salicylate-mediated interactions between pathogens and herbivores. Ecology 91:1075–1082.

Tholt, G., A. Kis, A. Medzihradszky, É. Szita, Z. Tóth, Z. Havelda, and F. Samu. 2018. Could vectors' fear of predators reduce the spread of plant diseases? Scientific Reports 8:8705.

Supporting Information

Additional supporting information may be found in the online version of this article at http://onlinelibrary.wiley.com/doi/10.1002/ecy.2879/suppinfo

Data Availability

Data are available from Figshare: https://doi.org/10.6084/m9.figshare.9729638.v1