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Predators affect a plant virus through density and trait-mediated indirect effects on vectors

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ABSTRACT

Biological control programs frequently rely on insect predators to control pests that vector plant pathogens in agroecosystems. Predators affect vectors by eating them (consumptive effects) and by inducing antipredator behaviors (non-consumptive effects), and these interactions may affect transmission of vector-borne pathogens. However, it has proven difficult to experimentally tease apart the effects of predators on vector fitness and behavior as they are often correlated. We addressed this problem by assessing how both aphids and an aphid-borne pathogen were affected by variable predation risk. Specifically, we experimentally manipulated lady-beetle predators' mouthparts to isolate consumptive, and non-consumptive, effects of predators on aphid fitness, movement, and virus transmission. We show that although lethal predators decreased aphid vector abundance, they increased pathogen transmission by increasing aphid movement among hosts. Moreover, aphids responded to risk of predation by moving to younger plant tissue that was more susceptible to the pathogen. Aphids also responded to predator risk through compensatory reproduction, which offset direct consumptive effects. Our results support predictions of disease models showing alterations of vector movement due to predators can have greater effects on transmission of pathogens than vector consumption, which should be considered when examining natural enemies' role in pathogen dynamics. Broadly, our study shows isolating direct and indirect predation effects can reveal novel pathways by which predators affect vector-borne pathogens.

1. Introduction

Within food webs, arthropod vectors that transmit plant pathogens engage in direct and indirect trophic interactions with individuals of other species such as competitors, mutualists, and predators (Clark et al., 2019; Crowder et al., 2019). Models and empirical studies show that species such as predators and competitors can indirectly affect the spread of vector-borne pathogens through both density and trait-mediated effects on vectors (Finke, 2012; Chisholm et al., 2019; Clark et al., 2019). For example, predators may interfere with pathogen transmission by killing vectors (consumptive effects) but may increase pathogen spread if vectors increase their movement when predators are present (non-consumptive effects) (Crowder et al., 2019). However, experimentally untangling the direct and indirect effects of predators on vectors and vector-borne pathogens has proven difficult, given that consumptive and non-consumptive effects are not independent and interactions between vector densities and behaviors are likely to influence pathogen transmission.

Most insect vector species are attacked by predators; if predators only killed vectors, they should decrease vector-borne pathogen transmission by reducing vector abundance (Moore et al., 2010). However, in response to predators, insect vectors exhibit a range of behaviors such as dropping from plants (Losey and Denno, 1998; Fan et al., 2017). Predation risk can also affect vector feeding behavior and movement (Smyrnioudis et al., 2001; Hodge et al., 2011; Kersch-Becker and Thaler, 2015; Tholt et al., 2018). While such behaviors are effective ways to defend against predation, they may also come at a cost of reduced feeding duration and diet quality, and lower reproductive output for vectors (Preisser et al., 2007; Jones and Dornhaus, 2011; Jeger et al., 2011). However, while reviews show that the non-consumptive effects of predators can be equally or more important in affecting prey demographics as consumptive effects (Preisser et al., 2005), few studies have isolated how consumptive effects and non-consumptive effects of predators might affect vector-borne pathogens.

Direct predator effects on vector-borne pathogens are expected to be straightforward, with reduced vector abundance slowing pathogen

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transmission. However, indirect effects of predators on vectors, cascading from changes to vector abundance and behavior, may affect these same transmission mechanisms in more complex ways. Models suggest that increased vector movement between host plants due to predation risk should accelerate virus transmission, while reduced vector feeding duration should slow transmission (Finke, 2012; Crowder et al., 2019). Empirical support for this has been shown in a system where predators increased transmission of *Barley yellow dwarf virus* in wheat by increasing movement of the aphid vector (Smyrnioudis et al., 2001). In contrast, when predators interrupted feeding by aphid vectors without affecting host-to-host movement, virus transmission was slowed (Long and Finke, 2015). While informative, these studies had variation in vector abundance, and did not measure how vector abundance and behavior interactively affected transmission. Additionally, pathogen transmission mode, or the duration of feeding bouts and retention within vectors necessary for transmission, may determine how certain vector behaviors affect transmission rates (Mauck et al., 2012; Crowder et al., 2019). This highlights the difficulty in isolating tradeoffs between consumptive and non-consumptive predator effects on pathogens, and a need for more targeted assessments of how vector responses to predation risk affect pathogen transmission.

In this study, we addressed these knowledge gaps in a model plant pathosystem comprised of the aphid vector *Acyrthosiphon pisum,* the host *Pisum sativum*, the pathogen *Pea-enation Mosaic Virus* (PEMV), and the ladybeetle predator *Hippodamia convergens*. PEMV is a persistently transmitted virus, vectored primarily by *A. pisum* and economically damaging to legume production in the Northwestern US. We experimentally isolated consumptive and non-consumptive predator effects by manipulating both predator presence and predator lethality. In response to these treatments, we measured how aphid vector populations, and their capacity to transmit the PEMV pathogen, responded to variable lethal and non-lethal predation risk. Data from our experiments were incorporated into structural equation models to isolate the direct and trait-mediated indirect pathways by which predators affected aphid abundance, aphid movement, and pathogen transmission.

2. Material and methods

2.1. Insect and virus maintenance

Pea aphid (*Acyrthosiphon pisum* Harris) colonies were originally collected in commercial pea fields in Washington State, and were maintained on potted pea plants (*Pisum sativum* L. cv. "Banner") in greenhouses in Pullman, WA, USA under controlled conditions ($23 \pm 2\,^{\circ}C$, L16:D8 photoperiod). Our PEMV isolate was obtained from the University of Idaho and was maintained by transferring pea aphids fed on PEMV-infected pea plants into uninfected pea aphid colonies, introducing clean plants as needed. Samples from infectious and uninfectious pea aphid colonies were tested monthly for the presence of PEMV using RT-PCR; these samples confirmed nearly 100% infection levels in the infectious colony and 0% in the uninfectious colony. *H. convergens* predators were collected from pea and alfalfa fields in Washington State 7 days prior to experiment start and stored in a growth chamber at $4\,^{\circ}C$ until needed.

2.2. Effects of predation risk on vectors and PEMV prevalence

To structure our examination of predation effects on pea aphids (both adults and nymphs) and PEMV, we developed an *a priori* interaction network (Fig. 1). We predicted both lethal and risk predator

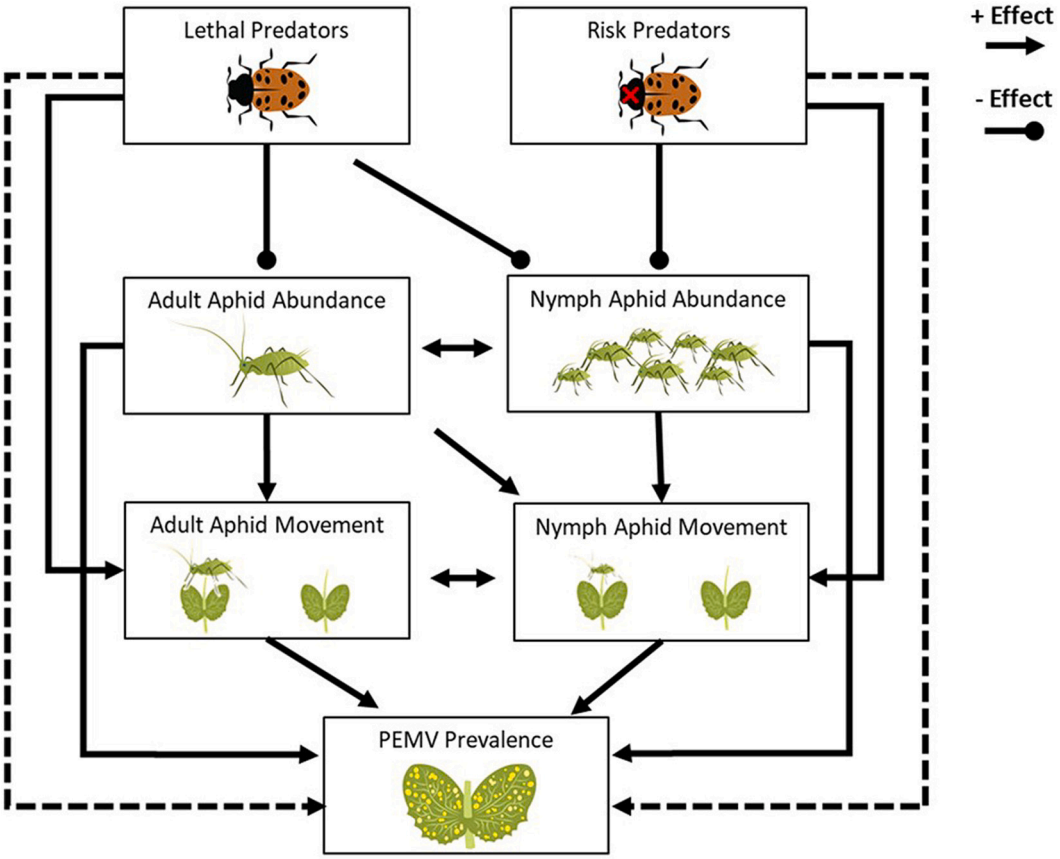

Fig. 1. Interaction network with *a priori* predictions about indirect effects and the direction of effects (positive/negative). Boxes show predator treatments and response variables (adult/nymph aphid abundance/distance from center plant, and PEMV prevalence). We predicted that predation risk would directly reduce aphid abundance and increase aphid movement (full lines), indirectly increasing PEMV prevalence (dotted lines).

treatments would reduce aphid abundance and increase movement. While reduced aphid abundance should decrease PEMV prevalence, increased aphid movement may increase PEMV prevalence by causing vectors to contact more hosts (Finke, 2012; Crowder et al., 2019). We expected vector movement to affect PEMV prevalence more than abundance (Chisholm et al., 2019), resulting in a net indirect positive effect of predators on PEMV prevalence.

To test predictions of our *a priori* model, we conducted a field mesocosm experiment. By manipulating predator mouthparts, we were able to isolate the consumptive and non-consumptive predator effects on the movement and reproduction of vectors, and PEMV transmission. The experiment was conducted on bare-soil plots at the Palouse Conservation Farm in Pullman, WA, USA in two blocks (June, July 2018). Pea plants (*P. sativum* L. cv. "Banner") were planted in 10 cm pots in the greenhouse in potting soil (Sun Gro® Sunshine® LC1 Grower Mix) 2 wk. before the experiment. For each replicate, 25 potted plants were buried in bare soil in a 5×5 square grid within $2 \times 2 \times 2$ m cages with amber mesh Lumite screening (Bioquip, Gardena, California, USA), with 20 cm of space between the centers of each pot. Cages were buried ~10 cm below the soil surface to prevent escape of organisms, and peas were completely buried inside their pots to provide an even, contiguous surface throughout the mesocosm and prevent soil conditions in the field from affecting plant nutritional status.

Each replicate was randomly assigned one of three treatments: Control (no predators); Risk (4 non-consumptive "risk" *H. convergens*); or Lethal (4 unmanipulated "lethal" *H. convergens*). This density reflects ladybeetles observed in commercial pea fields (Lee, 2021). Adult lady beetles were held individually at 25 °C in petri dishes and fed *A. pisum ad-libitum* for 72 h, then starved for 48 h before receiving treatments to standardize hunger levels. "Risk" predators were briefly anesthetized with CO_2 and a small droplet of clear nail polish was applied to seal their mandibles, ensuring palps or antennae were not also restricted. This treatment prevents lady beetles from killing and consuming aphids but allows for movement and prey-seeking behavior (Kersch-Becker and Thaler, 2015). While this treatment can reduce lady beetle movement and foraging rates relative to untreated beetles and thus should be considered conservative of total non-consumptive effects, we observed consistent aphid disturbance and found no effects on mortality over 7-d in greenhouses in treated individuals (Lee, 2021). "Lethal" predators were also anesthetized and received a droplet of polish on the elytra. Predators were then stored at 4 °C overnight before use.

In each cage, 25 7-d old adult PEMV-infectious pea aphids were confined within a fine-mesh frame on the centermost pea plant for 24 h. After 24 h, the mesh was removed, established pea aphids were counted, and predators were released into the mesocosm. Adult and nymph pea aphid populations on each plant were recorded after 5 d. Predators were replaced if found dead or could not be located in mesocosms after 15 min of searching; in total risk and lethal treatments had 29 and 14 predators added back into mesocosms respectively. After 5 d, aphids were removed with an aspirator and pots were treated with a granular formulation of imidacloprid (Bayer Crop Science, NJ, USA) to kill remaining aphids and cease virus transmission. Plants then grew for 5 d to accumulate viral titer before being visually evaluated for PEMV symptoms. For each replicate, aboveground tissue from 3 pea plants from each mesocosm quadrant were destructively sampled (Fig. S1), frozen in liquid nitrogen, and stored at -80 °C. PEMV titer from the 3 plants in each quadrant was determined using rtPCR and quantified with ImageJ (US NIH, Bethesda, Maryland, USA) to measure electrophoresis gel band intensity relative to a positive control (Fig. S2). These pooled titer measurements were used to validate visual evaluations of PEMV presence within each mesocosm, as quantifying titer for all individual plants was unfeasible. Average PEMV titers, however, were highly correlated with visual evaluations of PEMV prevalence (Pearson's Correlation, $r_{46} = 0.80$, $P < 0.001$). Eight replicates for each treatment (Control, Risk, Lethal) were conducted per block.

2.3. Effects of predation risk on vector feeding location

We conducted a second consecutive greenhouse experiment to further assess risk and lethal predator effects on vectors, focusing on individual aphid feeding location. Pea plants were grown for 2 weeks, and pea aphids were raised to adulthood on PEMV-infected peas as previously described. 5 pea plants in 10 cm plastic pots were arranged in a row within $0.3 \times 0.3 \times 0.6$ m black mesh cages (Bioquip, Gardena, California, USA) and potting soil was spread across the tops of pots to provide a contiguous surface. 'Risk' and 'Lethal' *H. convergens* predators were also prepared as described, starved for 48 h, and held at 4 °C for 24 h before use. 15 PEMV-infectious pea adult aphids were confined on the center plant in each row by a mesh bag for 24 h before the bag was carefully removed and 3 'Risk' or 'Lethal' *H. convergens* predators were added. The number, host plant, and feeding locations (top or bottom half of plant) of aphid pea adults and nymphs was recorded daily for three consecutive days. Sixteen replicates were conducted for each treatment.

2.4. Data analyses

We used structural equation modeling (SEM) to test *a priori* predictions for the field mesocosm experiment, using predator treatment ('Risk' or 'Lethal') and block as predictors and pea aphid abundance, pea aphid movement, and PEMV prevalence as responses (Fig. 1). Abundance was the total pea aphid adults or nymphs in the entire mesocosm, and movement was the average distance of aphid adults or nymphs from the center release plant. Parameters were continuous (aphid abundance and movement) or binary counts (number of plants infected out of 25). Block was included in all models as a fixed effect, as hotter temperatures in the second block reduced aphid abundance (GLM, $t_{47} = -5.13$, $P < 0.001$). In our analysis, non-significant paths were dropped if doing so reduced Akaike's Information Criterion (AIC), and paths were added if models without them were rejected by tests of direct separation (Lefcheck, 2016). Predictor coefficients were standardized by their standard deviation to allow for comparisons of effects on different responses (Fig. 3, Table S1). To verify that density-dependent predation was not driving observed patterns in aphid movement by disproportionately removing aphids on the initial release plant, we also ran a linear model to evaluate the proportion of total aphids on the release plant at the end of the experiment using predator treatment and block as fixed effects.

We ran a series of generalized linear mixed models (GLMMs) to evaluate aphid responses to predators in the greenhouse experiment, using predator treatment and day as fixed effects, cage as a random effect, and adult and nymphal aphid abundance, distance from center plant, and proportion feeding on top *vs.* bottom half of host plant as responses. The fit of GLMMs to the observed data distributions were controlled by inspecting residuals and QQ plots. Binomial models for feeding location were weighted by total aphid abundance in each mesocosm to account for differences in abundance between treatments. All data analyses were conducted using R v 3.5.2 (R Core Team, 2022), using the packages 'lme4' for GLMMs (Bates et al., 2015), and 'piecewiseSEM' for structural equation models (Lefcheck, 2016). Posthoc analyses were conducted using the 'emmeans' package (Lenth, 2020) and significance tests were based on analysis of deviance χ^2 tests using the 'car' package (Fox and Weisberg, 2019).

3. Results

3.1. Effects of predation risk on vectors and PEMV prevalence

Plants were more likely to be infected with PEMV in lethal ($\mu = 11.1 \pm 1.4$) and risk ($\mu = 11.6 \pm 1.5$) predator groups than controls ($\mu = 9.7 \pm 1.4$), respectively ($\chi^2 = 4.93$, df = 2, $P = 0.085$) (Fig. 2a), and our best-fit SEM ($P = 0.87$, Fishers's $C = 13.2$, df = 20) showed direct and indirect pathways by which predators affected aphids and PEMV

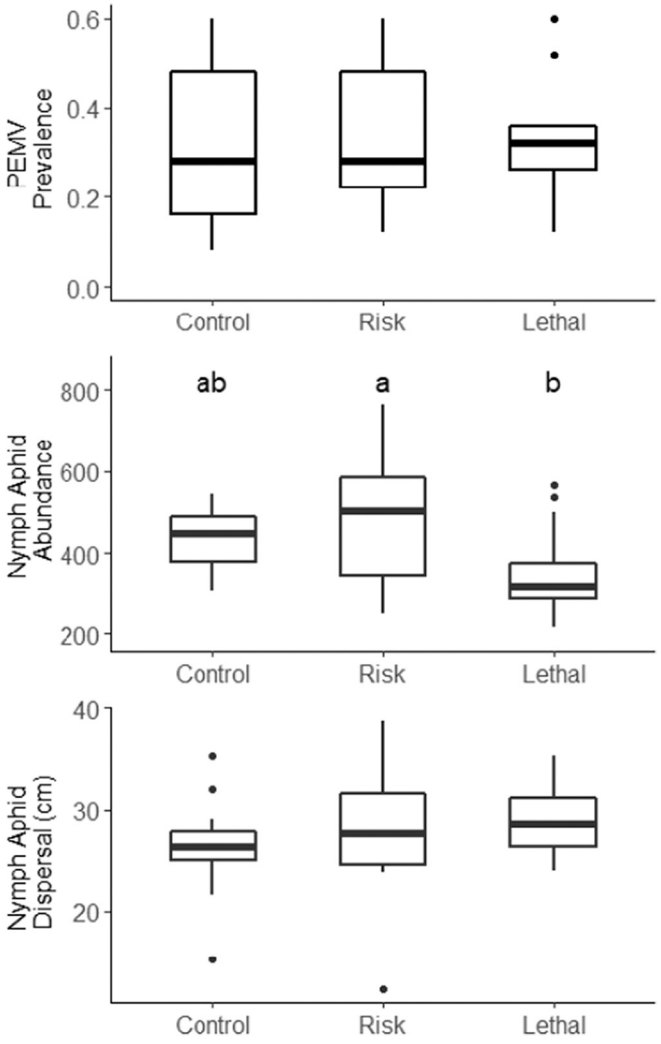

Fig. 2. Effects of predator treatments on a) PEMV prevalence, b) nymph aphid abundance, and c) nymph aphid dispersal in the field. Letters represent significant differences between groups (Tukey's HSD, $\alpha = 0.05$).

(Fig. 3, Table S1). Lethal predators directly reduced adult (βstd = −0.45, df = 46, $P = 0.001$) and nymph abundance (βstd = −0.25, df = 44, $P = 0.037$) but increased adult aphid movement (βstd = 0.34, df = 44, $P = 0.050$) (Fig. 2b). Contrary to predictions, risk predators increased nymph abundance (βstd = 0.25, df = 44, $P = 0.041$) (Fig. 2b). Across aphid responses, adult aphid movement decreased when adult abundance increased (βstd = −0.37, df = 43, $P = 0.027$), and there were positive correlations between adult and nymph abundance (βstd = 0.51, df = 48, $P < 0.001$) and adult and nymph movement (βstd = 0.65, df = 48, $P < 0.001$). PEMV prevalence was directly driven by nymph abundance (βstd = 0.20, df = 43, $P < 0.001$) and nymph movement (βstd = 0.11, df = 48, $P = 0.002$), with a direct effect of lethal predator treatment (βstd = 0.08, df = 48, $P = 0.023$) (Fig. 3). Overall, lethal predators indirectly affected PEMV prevalence positively through increased adult movement (βstd = 0.024) but negatively through reduced nymph abundance (βstd = 0.05), while risk predators indirectly affected PEMV prevalence positively through increased nymph abundance (βstd = 0.05, Fig. 3). Predator treatments did not significantly affect the proportion of total aphids on the release plant (F = 1.14, df = 2, $P = 0.33$).

3.2. *Effects of predation risk on vector feeding location*

Predator treatments increased the proportion of aphid adults feeding on the top half of plants ($\chi^2 = 8.18$, df = 2, $P = 0.017$), though this effect was less pronounced for nymphs ($\chi^2 = 5.09$, df = 2, $P = 0.078$) (Fig. 4, Table S2). Lethal predators reduced adult aphid abundance ($t_{136} = -2.93$, $P = 0.0039$) and nymphs over time ($t_{93} = -7.26$, $P < 0.001$) (Fig. S3a,b), and increased the dispersal of aphid adults ($t_{138} = 3.01$, $P = 0.013$) and nymphs ($t_{45} = 5.10$, $P < 0.001$) (Fig. S3c,d). Risk predators alone did not significantly affect aphid abundance or dispersal (Fig. S3).

4. Discussion

Our study confirms predictions that predation can mediate the transmission dynamics of vector-borne pathogens by affecting aphid abundance and movement. We show the induction of specific aphid behaviors by predators can either promote or interfere with virus transmission. Though lethal predators reduced aphid abundance, the strongest predictor of PEMV prevalence in our model, their effects on aphid movement and feeding behavior appear to have counteracted

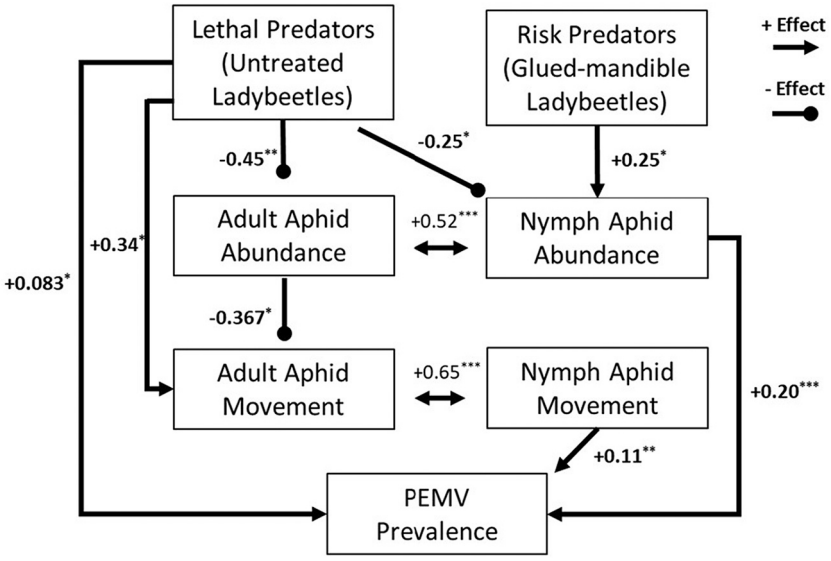

Fig. 3. Accepted model from confirmatory path analysis ($P = 0.87$, Fischer's C = 13.154, df = 20). Bidirectional arrows indicate correlated errors. Lethal predators increase PEMV prevalence directly ($P = 0.02$, Table S1) while decreasing PEMV through density-mediated indirect interaction.

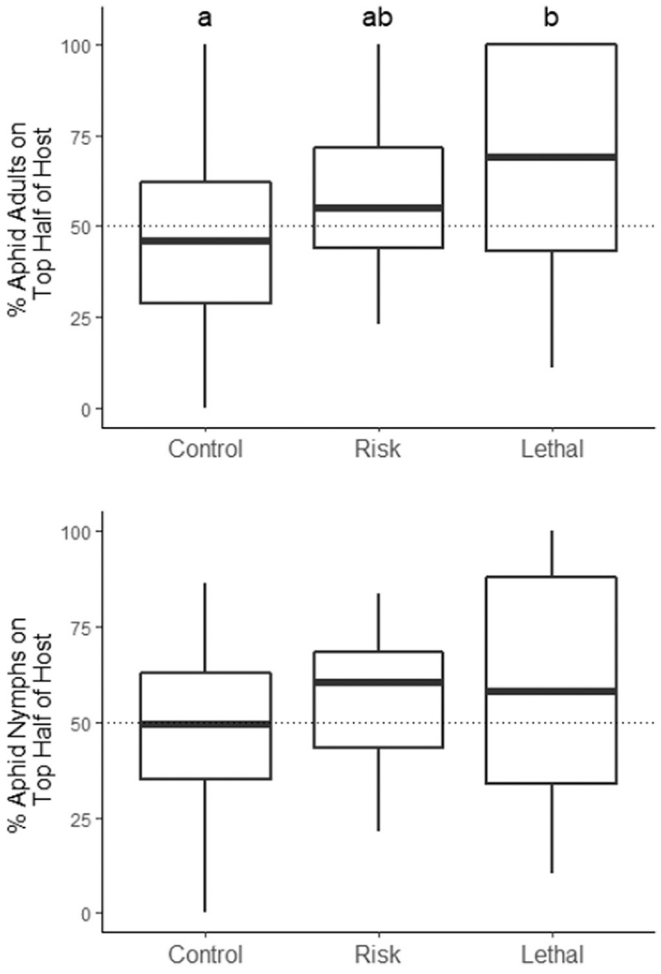

Fig. 4. Effects of predator treatments on the feeding location of aphid adults and nymphs on pea hosts in greenhouse. Dotted lines indicate equal numbers of aphids feeding on top *vs* bottom half of hosts. Letters represent significant differences between groups (Tukey's HSD, $\alpha = 0.05$).

these reductions, resulting in a net increase in PEMV prevalence when predators were present compared to absent. In contrast, risk-only predators had minimal effects on aphid behaviors but affected PEMV prevalence positively through density-dependent mechanisms. These results lend support to model predictions showing that species interactions affecting vector behavior can contribute more to the rates of vector-borne pathogen spread in ecosystems than those affecting vector abundance (Hodge et al., 2011; Crowder et al., 2019). However, our study shows that 'risk' manipulations alone may fail to capture the full range of vector anti-predator responses, which can include responses like compensatory reproduction by herbivores.

Insect herbivores make movement and foraging decisions in response to a 'landscape of fear', where tradeoffs between habitat quality and perceived predation risk are weighed (Fox and Weisberg, 2019). As most plant viruses are dependent on vector dispersal for transmission between hosts (Fereres and Moreno, 2009), responses of herbivores to landscapes of fear are expected to affect pathogen transmission (Finke, 2012; Crowder et al., 2019). In our system, lethal predation increased movement of adult aphids, both directly and *via* reduced aphid abundance, though adult dispersal was not directly linked to increased PEMV. Nymph dispersal, however, may better represent where infectious adult vectors spent time feeding, as nymphs themselves are less likely to leave hosts when threatened by predators (Losey and Denno, 1998). Our results indeed show nymph movement was positively linked with PEMV prevalence, suggesting transmission increased when infectious pea aphids moved to new hosts.

Our model also identified a direct effect of lethal predators on PEMV prevalence, which suggests an important effect on aphids was missing from our *a priori* model, as predators themselves cannot transmit PEMV. Noticing differences in aphid feeding location on host plants between predator treatments in our field study, we recorded feeding location in our greenhouse study. Competitive displacement of aphids to higher locations on individual plants by non-vector herbivores can increase the likelihood of PEMV transmission by causing vectors to feed on more susceptible young tissue (Chisholm et al., 2019). Our results suggest predators may similarly affect virus transmission by displacing aphid vectors upwards on plants (Fig. 4), though such effects could vary in other contexts based on host plant structure and aphid refuge-seeking behavior (Grevstad et al., 2014; Costamagna and Landis, 2011; Northfield et al., 2012).

Predation risk may induce compensatory responses in insects to defer the reproductive and developmental costs of anti-predator behaviors until risk has decreased (Barribeau et al., 2010; Thaler et al., 2012; Hermann et al., 2021). Contrary to our predictions, risk predation increased aphid nymph abundance (Fig. 2), although any long-term negative consequences of compensatory reproduction may not be apparent in our study. It is also possible that disturbance by risk predators induced early dispersal from hosts before aphids reached high densities, at which point reproduction can stall (Agrawal et al., 2004). Moreover, given risk predators' inability to kill prey directly, it is possible they did not induce alarm pheromone release by aphid vectors as substantially as lethal predators (Basu et al., 2021). Alarm pheromone release has been shown to reduce transmission of PEMV independent of effects on aphid reproduction in greenhouses, though whether such effects manifest in the field is unclear (Lee, 2021). Additionally, the conservative nature of our 'risk' manipulation (*i.e.* reduced foraging duration, Kersch-Becker and Thaler, 2015) may have further limited antipredator responses in vectors including alarm pheromone release and movement from hosts. Thus, gross effects of predators reducing vector abundance may be underestimated if non-consumptive effects increase vector abundance.

Risk-manipulated predators performing differently than we predicted highlights the difficulty in establishing how community interactions might affect pathogen transmission *a priori*, as aphid responses to other species can vary based on ecological and environmental context. Aphid's propensity to disperse or drop from hosts when threatened can be affected by colony density, host plant quality, clonal differences, and previous exposure to risk for example (Kersch-Becker and Thaler, 2015; Muratori et al., 2014; Tamai and Choh, 2019). Moreover, aphid perception of the severity of predation risk is dependent on predator foraging strategies, recognition of risk signals, and shared evolutionary history (Preisser et al., 2007; Sih et al., 2010; Basu et al., 2021). Given the potential for interactions and feedback among prey responses to predators (Sheriff et al., 2020), inclusion of multiple vector responses in analyses remains critical to identifying mechanisms behind observed changes in transmission.

Our study provides an experimental and statistical framework for the examination of a broader range of predator effects on vectors and pathogen transmission. Variation in predator communities or density would likely affect the relative magnitude of predators' effects on vector abundance, development, and behaviors (Finke, 2012). Diverse predator communities can act synergistically or antagonistically in suppressing herbivore populations (Losey and Denno, 1998; Snyder and Ives, 2001; Snyder et al., 2008), though the effects of predator diversity on vector behavior and virus transmission have been poorly investigated. Additionally, pathogen characteristics determine the importance of specific vector responses to overall rates of transmission; vector behaviors that accelerate the transmission of certain pathogens (*e.g.* rapid probing and dispersal) may stall the transmission of others (Long and Finke, 2015; Mauck et al., 2018; Crowder et al., 2019). Our results and other accumulating evidence suggest vector abundance alone may not be a suitable

indicator for disease risk in natural systems, and future studies manipulating the characteristics of pathosystems will help to identify the mechanisms through which trophic interactions can affect vector-borne pathogen transmission.

Author contributions

BWL and DWC conceived the experiments. BWL and SB performed the experiments. BWL and REC analyzed the data. All authors wrote the manuscript.

Data and code used for analysis are available from Zenodo (Lee, 2021), doi:https://doi.org/10.5281/zenodo.4546633

Declaration of Competing Interest

None.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.fooweb.2022.e00251.

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