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Reciprocal plant-mediated interactions between a virus and a non-vector herbivore

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Abstract. Vector-borne viruses alter many physical and chemical traits of their plant hosts, indirectly affecting the fitness and behavior of vectors in ways that promote virus transmission. However, it is unclear whether viruses induce plant-mediated shifts in the behavior and fitness of non-vector herbivores, or if non-vectors affect the dynamics of vector-borne viruses. Here we evaluated reciprocal interactions between *Pea enation mosaic virus* (PEMV), a pathogen transmitted by the aphid *Acrythosiphon pisum*, and a non-vector weevil, *Sitona lineatus*. In the field, PEMV-infected plants experienced more defoliation from *S. lineatus* than uninfected plants; behavioral assays similarly showed *S. lineatus* adults preferred to feed on infected plants. In turn, infectious *A. pisum* preferred plants damaged by *S. lineatus*, and *S. lineatus* herbivory led to increased PEMV titer. These interactions may be mediated by plant phytohormone levels, as *S. lineatus* induced jasmonic acid, while PEMV induced salicylic acid. Levels of abscisic acid were not affected by attack from either PEMV or *S. lineatus* alone, but plants challenged by both had elevated levels of this phytohormone. As plant viruses and their vectors often exist in diverse communities, our study highlights the importance of non-vector species in influencing plant pathogens and their vectors through host-mediated effects.

Key words: disease ecology; herbivory; indirect effects; insect behavior; phytohormones; plant-insect-pathogen interactions.

Introduction

Plant viruses drive biodiversity loss in plant communities and can devastate crop productivity (Strange and Scott 2005). Plant viruses rely almost exclusively on insect vectors for transmission to new hosts (Power 2000), and often form mutualistic relationships with vectors (e.g., Belliure et al. 2005, Bosque-Pérez and Eigenbrode 2011, Casteel and Jander 2013). For example, viruses can suppress plant defenses targeting vectors (e.g., Zhang et al. 2012, Luan et al. 2013, Casteel et al. 2015), or increase available nutrients for vectors (Casteel et al. 2014). While such vector-virus mutualisms are well studied, vectors exist in diverse communities, and interact with individuals of many species, including non-vector competitors. Yet, the role of these interactions on disease ecology has received little attention (reviewed by Johnson et al. 2015).

As plant viruses affect plant defenses and nutrient availability, they might also affect non-vector herbivores that co-occur with vectors. Empirical studies have observed positive (Hare and Dodds 1987, Belliure et al. 2010, Thaler et al. 2010, Kersch-Becker and Thaler 2013), negative (Mauck et al. 2010*b*, Van Molken et al. 2012, Pan et al. 2013), and neutral (Sadeghi et al. 2016) effects of a plant virus on the fitness of a non-vector herbivore. However, these studies were primarily conducted in controlled environments and not the field (but see Mauck et al. 2010*b*), and evidence of potential effects of plant viruses on the behavior of non-vector species is scant.

Conversely, non-vector herbivores might affect the susceptibility of plants to pathogens by altering plant physiology (Karban and Baldwin 1997) and phytohormone levels (Abe et al. 2012, Thaler et al. 2012). Herbivores often stimulate the jasmonic acid pathway (Bowles 1991), which can cause a down-regulation of salicylic acid (Thaler et al. 2010, Abe et al. 2012), which often regulates defenses against pathogens (Thaler et al. 2010, Robert-Seilaniantz et al. 2011). Shifts in plant physiology induced by non-vector herbivores might also affect fitness and host selection behavior of vector species (Agrawal 1999). However, the potential direct and indirect (i.e., plant-mediated) effects of non-vectors on viruses and their vectors remain largely unknown.

To address these knowledge gaps, we examined reciprocal interactions between a vector-borne plant virus and a non-vector chewing weevil herbivore in a series of field and greenhouse studies. We hypothesized that herbivory by the non-vector herbivore might affect plant defenses, which could affect host plant susceptibility to the pathogen and behavior of the vector. Conversely, pathogen infection might affect plant physiology and the behavior of the non-vector.

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Methods

Natural history of study system

Pea enation mosaic virus (PEMV) infects multiple crops and native plants (Appendix S1: Fig. S1A) in the Palouse region of eastern WA and northern ID, USA (Clement 2006). PEMV is obligately-transmitted in a persistent manner by pea aphids (*Acrythosiphon pisum;* Appendix S1: Fig. S1B), the primary vector (Clement 2006). *Sitona lineatus* (Appendix S1: Fig. S1C), the pea leaf weevil, is a co-occurring non-vector herbivore that overwinters as an adult and infests fields each spring. *Sitona lineatus* is highly-abundant and occurs in most commercial pea fields, often causing some feeding damage to over 90% of cultivated plants (Vankosky et al. 2011). Although *S. lineatus* cannot transmit PEMV, it co-occurs with *A. pisum*, PEMV, and plant hosts.

Field observations of Sitona lineatus *and PEMV*

We first explored interactions between PEMV and *S. lineatus* by assessing defoliation on 2-month old infected and uninfected pea plants on 12 Palouse farms in June 2014. We identified 3–10 PEMV-infected plants per farm (Appendix S1: Table S1). For each infected plant, an adjacent (<10 cm away) uninfected plant of similar size was identified in a paired design (see Appendix S1). Defoliation on plants was estimated by counting *S. lineatus* feeding notches, a common method for this herbivore (see Appendix S1). The growth stage (number of nodes) and aphid population size on each plant were recorded through visual observation.

Behavioral choice assays

A relationship between *S. lineatus* and PEMV might result from preferences of *S. lineatus* for infected plants, or preferences of infectious *A. pisum* for damaged plants. We tested this with two greenhouse assays (see Appendix S1). The first tested *S. lineatus* preferences by exposing 22 d old plants to one of two treatments: (1) sham-infected or (2) PEMV-infected. The sham treatment involved 8, 6 d old, non-infectious *A. pisum* feeding for 48 h; the PEMV treatment involved 8, 6 d old, infectious *A. pisum* feeding for 48 h. After 48 h, *A. pisum* were removed from plants so interactions with *S. lineatus* were plant-mediated. One sham- and one PEMV-infected plant were potted 10 cm apart in each replicate (10 × 25 × 10 cm cages; nine total). As both treatments included aphid feeding (with or without PEMV), this design allowed us to isolate the effects of PEMV on *S. lineatus*. Fifteen d post-inoculation, when PEMV symptoms manifest, two adult *S. lineatus* were released in each cage, equidistant from the two plants, to feed for 6 d (Appendix S1: Fig. S2A). After 6 d, leaves were excised and analyzed for defoliation (surface area removed in cm^2) with ImageJ (NIH). All plants were tested at the end of the experiment to confirm infectivity using ELISA.

The *A. pisum* preference assay was conducted by exposing 18 d old pea plants to one of two treatments: (1) undamaged – no feeding and (2) damaged – feeding by two adult *S. lineatus* for 48 h. After 48 h, the *S. lineatus* were removed and pairs of plants (one damaged, one undamaged) were added to cages (Appendix S1: Fig. S2B). An undamaged leaf, still connected to each plant, was inserted into opposite ends of a 20 cm vinyl tube, and then 25 adult infectious or non-infectious *A. pisum* were released in the tube center. After 3 h, individual settlement on both plants was recorded. The experiment included eight replicates each of infectious and non-infectious *A. pisum*.

Effects of Sitona lineatus *on susceptibility of host plants to PEMV*

Feeding by *S. lineatus* might also affect host susceptibility to PEMV. We assessed this with assays that explored if feeding by *S. lineatus,* either (1) prior to or (2) after PEMV-inoculation, affected viral titer. We first exposed 40, 14 d old, plants to three treatments: (1) undamaged (20 replicates); (2) low damage: feeding by 1 *S. lineatus* adult for 72 h (14 replicates); and (3) high damage: feeding by two *S. lineatus* adults for 72 h (6 replicates). After removing *S. lineatus*, we added 8, 3 d old, infectious *A. pisum* to each plant for 6 d. For the second assay, individual 14 d old plants were inoculated with PEMV by allowing 8, 3 d old, infectious *A. pisum* to feed for 48 h in a clip cage. After 48 h, *A. pisum* individuals were removed and 0, 1, or 3 (eight replicates each) adult *S. lineatus* were introduced for 6 d, after which defoliation on leaves was analyzed with ImageJ. After each experiment, aboveground biomass of each plant was harvested and analyzed using ELISA and a Pierce BCA protein assay to estimate relative PEMV titer (see Appendix S1).

Effect of PEMV and Sitona lineatus *on the defensive responses of pea*

Herbivore-pathogen interactions can be mediated by plant defenses, and we determined how plant phytohormones (jasmonic, salicylic, and abscisic acid) responded to attack from *S. lineatus* and PEMV. We randomly exposed 24, 9 d old, plants to one of two treatments: (1) sham-infected or (2) PEMV-infected (12 plants each; see *Behavioral choice assays* for methods). Ten d after *A. pisum* removal, either zero or five adult *S. lineatus* were added. After 24 h, undamaged leaves (plants without *S. lineatus*), or damaged leaves with 30–50% leaf area removed (plants with *S. lineatus*), were harvested, weighed, frozen in liquid nitrogen, and homogenized in a paint shaker for 60s. This tissue was tested for phytohormones, and PEMV infection was verified using RT-PCR (Casteel et al. 2015, see Appendix S1). A subsequent experiment exposed 18, 9 d old, plants to one of three treatments: (1) no aphids, (2) sham-infected, or (3) PEMV-infected (six replicates per treatment; see *Behavioral choice assays* for methods). Ten days after *A. pisum* removal, leaves were harvested and tested for phytohormones (see Appendix S1). This allowed us to determine if *A. pisum* feeding alone (compared to healthy plants without *A. pisum*) influenced phytohormones.

Data analysis

From the field study, we used paired *t*-tests to test for effects of PEMV-infection on (1) *S. lineatus* (# of notches),

(2) *A. pisum* (# per plant), and (3) growth stage (# of nodes); the 12 farms served as replicates. Responses reflected differences between uninfected and infected plant pairs. Data from greenhouse behavioral assays was analyzed using paired *t*-tests to determine if *S. lineatus* preferred sham-infected vs. PEMV-infected plants, and if non-infectious and infectious *A. pisum* preferred *S. lineatus*-damaged vs. undamaged plants. Responses reflected differences between plant pairs (either sham-infected vs. PEMV-infected or undamaged vs. damaged). All paired *t*-tests were conducted on raw data without transformation.

We used ANOVA to determine if the density of *S. lineatus* feeding on uninfected pea plants (before inoculation), or infected pea plants (post inoculation), affected PEMV titer; for the first analysis three plants that remained uninfected (two damaged, one undamaged) were excluded. Plant protein content was also compared with ANOVA to determine if it was influenced by *S. lineatus*. We used linear regression to analyze PEMV titer as a function of defoliation (cm^2 removed). We used ANOVA to determine if jasmonic, salicylic, and abscisic acid levels (log transformed due to heteroscedacity, see Appendix S1 for details of two replicates excluded) differed based on PEMV, *S. lineatus*, and their interaction. All analyses were conducted in R v. 3.1.1 (R Core Team 2014).

Results

Relationship between PEMV and Sitona lineatus *in the field*

PEMV-infected plants had more *S. lineatus* feeding notches (i.e., defoliation) than adjacent uninfected plants ($t_{11} = 3.88$, $P = 0.0026$; Fig. 1A). Defoliation levels on infected plants were greater than, or equal to, uninfected plants in each of the 12 fields (Fig. 1A). This occurred even though infected and uninfected plants were similar in terms of growth stage ($t_{11} = -1.36$, $P = 0.20$; Appendix S1: Fig. S3A) and *A. pisum* abundance ($t_{11} = 0.21$, $P = 0.84$, Appendix S1: Fig. S3B).

Behavioral preferences of Sitona lineatus *and* Acrythosiphon pisum

Adult *S. lineatus* preferentially fed on PEMV-infected compared to sham-infected plants ($t_8 = 2.85$, $P = 0.022$, Fig. 1B). As PEMV-infected and sham-infected plants were both attacked by aphids, this indicates an effect of PEMV on *S. lineatus*. Infectious *A. pisum* preferentially settled on *S. lineatus*-damaged compared to undamaged plants (Fig. 1C), although the effect was only marginally significant ($t_7 = -1.90$, $P = 0.099$). Non-infectious *A. pisum,* however, did not prefer *S. lineatus*-damaged compared to undamaged plants ($t_7 = 0.53$, $P = 0.62$, Appendix S1: Fig. S4).

Effects of Sitona lineatus *on PEMV accumulation in plants*

Herbivory by *S. lineatus* prior to PEMV inoculation did not impact relative PEMV titer ($F_{2,34} = 0.32$, $P = 0.73$, Appendix S1: Fig. S5A) or plant protein content ($F_{2,34} = 0.20$, $P = 0.82$, Appendix S1: Fig. S5B). However,

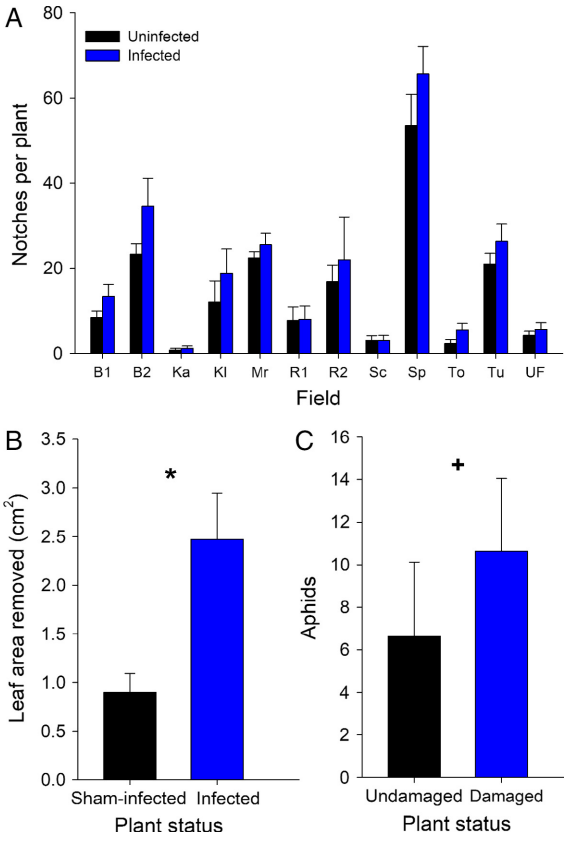

Fig. 1. (A) The amount of defoliation by *Sitona lineatus* (number of feeding notches) on paired uninfected and PEMV-infected plants in commercial pea fields (see Appendix S1 for details on fields and abbreviations used). Mean defoliation levels (± SE) in each field are shown. (B) The amount of defoliation (leaf area removed in cm^2) by *S. lineatus* on paired sham-infected and PEMV-infected plants in greenhouse choice assays. (C) The number of infectious *Acrythosiphon pisum* settling on paired undamaged and *S. lineatus*-damaged plants in greenhouse choice assays. All points represent means ± SE. +: significant at $\alpha = 0.10$; *: significant at $\alpha = 0.05$.

higher *S. lineatus* abundance increased relative PEMV titer for plants damaged after PEMV inoculation ($F_{2,21} = 12.2$, $P = 0.0003$, Fig. 2A). When damage occurred after PEMV inoculation, there was a positive relationship between the amount of leaf defoliation and relative PEMV titer ($t_1 = 4.22$, $P = 0.0004$, Fig. 2B).

Effects of PEMV and Sitona lineatus *on phytohormones*

Salicylic acid levels were higher in PEMV-infected compared to sham-infected plants ($F_{1,18} = 19.2$, $P = 0.0004$), but were not affected by *S. lineatus* ($F_{1,18} = 0.36$, $P = 0.56$) or the PEMV × *S. lineatus* interaction ($F_{1,18} = 0.058$, $P = 0.81$) (Fig. 3A). In contrast, jasmonic acid levels were higher in *S. lineatus*-damaged compared to undamaged plants ($F_{1,18} = 77.8$, $P < 0.0001$), but were not affected by PEMV ($F_{1,18} = 2.35$, $P = 0.14$). The PEMV × *S. lineatus* interaction ($F_{1,18} = 3.30$, $P = 0.086$) was marginally significant (Fig. 3B). Levels of abscisic acid were affected by *S. lineatus* ($F_{1,18} = 6.79$, $P = 0.018$) but not PEMV ($F_{1,18} = 2.33$, $P = 0.14$) or the PEMV × *S. lineatus*

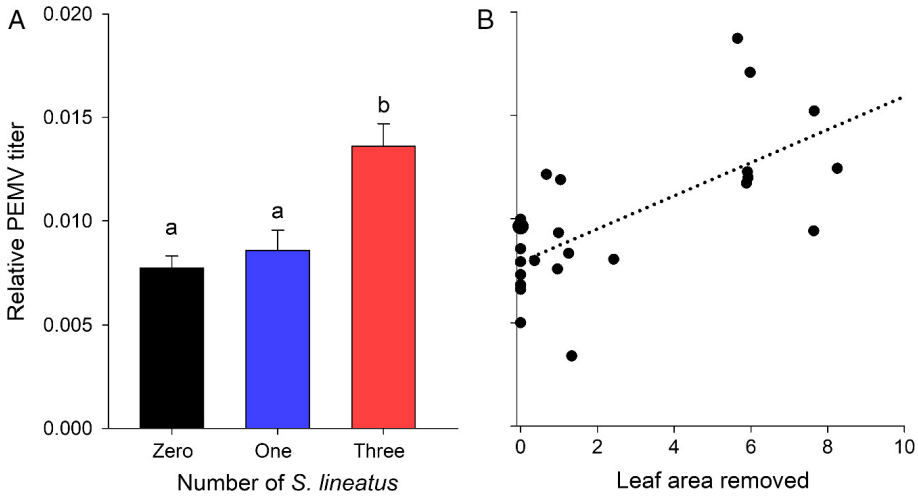

FIG. 2. (A) The relative PEMV titer (sample absorbance/negative control absorbance/total protein, see Appendix S1) after 6 d of *Sitona lineatus* feeding for pea plants that were exposed to either zero, one, or three *S. lineatus* after being inoculated with PEMV. All points represent means ± SE. Different letters represent significant differences at $\alpha = 0.05$. (B) The relative PEMV titer (sample absorbance/negative control absorbance/total protein) for pea plants that experienced varying defoliation from *S. lineatus* (total leaf area removed in cm^2) after being inoculated with PEMV; the dotted line shows the best-fit linear regression line.

interaction ($F_{1,18} = 0.59$, $P = 0.45$). However, compared to undamaged treatments, abscisic acid levels were only higher when both attackers were present (Fig. 3C). For all three phytohormones, there were no differences between healthy control plants (plants not exposed to *A. pisum*) and sham-infected plants exposed to non-infectious *A. pisum* (Appendix S1: Fig. S6).

DISCUSSION

Herbivores that transmit viruses are nested in diverse food webs, where they interact with individuals of many species as they forage. While interactions within food webs can generate complex dynamics for focal populations (Polis and Strong 1996, Berlow et al. 2004), whether interactions between vector and non-vector species affects pathogens remains poorly understood (Johnson et al. 2015). Here we demonstrate strong reciprocal interactions between a plant virus and a non-vector herbivore. While some studies have similarly documented impacts of a plant virus on a non-vector herbivore (Belliure et al. 2010, Mauck et al. 2010*b*, Thaler et al. 2010, Pan et al. 2013), ours is among the first to show a non-vector can affect a pathogen by directly altering plant susceptibility and defense, and by indirectly affecting vector behavior. Our results suggest that PEMV outbreaks are most likely when *S. lineatus* is abundant, as infectious *A. pisum* prefer damaged plants (Fig. 1C) and *S. lineatus* feeding increased PEMV titer (Fig. 2A).

Our data indicates that a non-vector herbivore could alter virus dispersal in at least two ways. First, feeding by a non-vector can directly increase the susceptibility of the host plant to a virus. In our study this was reflected by higher viral titers in host plants fed on by *S. lineatus* (Fig. 2A). Second, feeding by a non-vector can affect the attractiveness of host plants to vectors; infectious aphids preferred plants damaged by *S. lineatus* (Fig. 1C). Since PEMV-infection can promote *A. pisum* fitness (Hodge and Powell 2010), increased titer following *S. lineatus* feeding may explain why damaged plants were preferred by infectious *A. pisum*, while non-infectious individuals had no preferences. Induction of abscisic acid, which was greatest in plants challenged by both PEMV and *S. lineatus*, may also mediate these preferences, as induction of abscisic acid has been shown to benefit the green peach aphid, *Myzus persicae*, in *Arabidopsis thaliana* (Hillwig et al. 2016). By altering host plants in ways that are attractive to vectors, while also increasing pathogen susceptibility, non-vector herbivores can promote pathogen transmission.

Effects of non-vector herbivores on pathogens and vectors may be mediated by changes in host plant physiology after feeding. We observed that feeding by *S. lineatus* did not inhibit the induction of salicylic acid by PEMV (Fig. 3A), but it did induce both the jasmonic and abscisic acid pathways (Fig. 3B, C). This suggests that induction of jasmonic and/or abscisic acid by *S. lineatus* may influence PEMV-plant interactions, although few studies have examined the role of jasmonic or abscisic acid in virus-plant interactions (but see Oka et al. 2013). While plants with elevated jasmonic acid levels often have concurrent reductions in salicylic acid, a key pathway targeting pathogens (Oka et al. 2013), our results indicate that jasmonic acid induction may increase plant susceptibility to a virus even when salicylic acid levels are not suppressed. Similar results have been observed in other systems where treatment of plants with methyl jasmonate increased host plant susceptibility to viruses even when salicylic acid levels were unaffected by the induction of jasmonic acid (Penninckx et al. 1996, Ryu et al. 2004).

Interactions between *S. lineatus* and PEMV may benefit both partners. Although we did not measure *S. lineatus* fitness, their preference for PEMV-infected plants (Fig. 1A, B) suggests they may benefit from feeding on infected plants. While host attractiveness is not always correlated with host quality (Mauck et al. 2010*a*), other chewing herbivores have

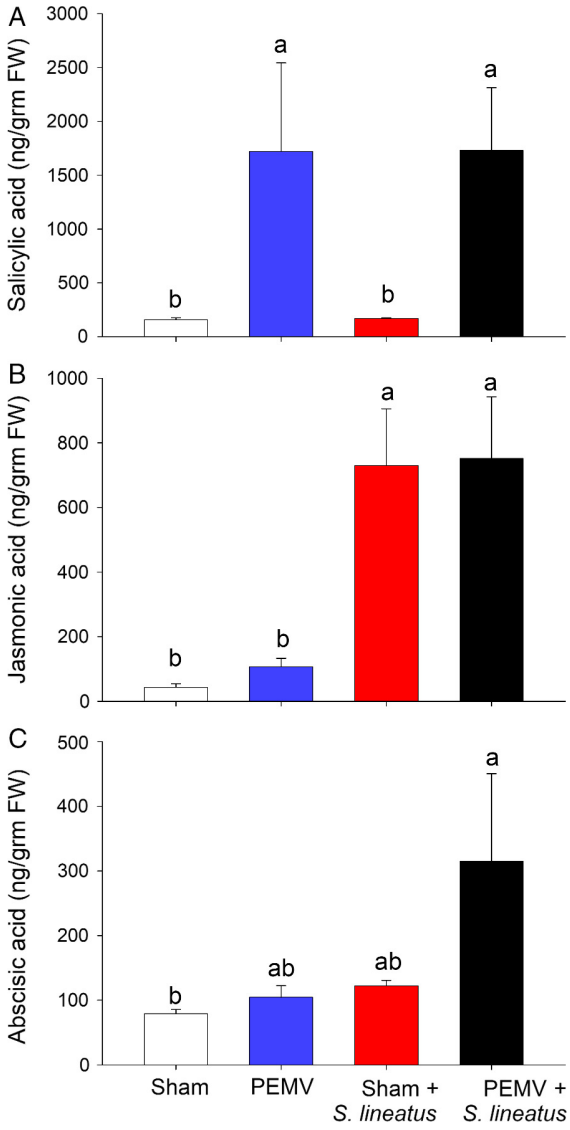

FIG. 3. (A) Salicylic acid, (B) jasmonic acid, and (C) abscisic acid levels (ng/gm fresh weight) for plants that were either sham-infected or infected with PEMV and then exposed to *Sitona lineatus* or not. Means (± SE) for each treatment combination are shown (analyses were conducted on log-transformed data but means and standard errors were back-transformed for presentation). Within each panel, letters above bars indicate significant differences among treatments (Tukey HSD test, $\alpha = 0.05$).

increased performance on infected plants (Hare and Dodds 1987, Musser et al. 2003, Thaler et al. 2010). Such benefits are often attributed to elevated levels of salicylic acid, and suppression of jasmonic acid, a key anti-herbivore defense, in virus-infected plants (Thaler et al. 2010, Ali and Agrawal 2014). Our data similarly show that plants upregulated jasmonic acid in response to attack from *S. lineatus* (Fig. 3A). However, although PEMV-infected plants had increased salicylic acid, PEMV infection did not inhibit induction of jasmonic acid (Fig. 3B). This suggests that mutual antagonism between salicylic and jasmonic acid pathways is not likely the primary mechanism mediating preferences of *S. lineatus* for PEMV-infected plants. Elevated salicylic acid levels induced by PEMV may benefit *S. lineatus* through a mechanism unrelated to chemical defense, however. Studies have shown increased levels of free amino acids following exogenous salicylic acid application (Kováčik et al. 2010). Given positive correlations between free amino acid levels and herbivore performance (Casteel et al. 2014), the attraction of *S. lineatus* to plants exhibiting heightened levels of salicylic acid could be attributable to enhanced nutrition rather than changes in defense.

Our study highlights the importance of studying reciprocal interactions between plant viruses and non-vector species, as these interactions could have widespread implications for virus spread and evolution. Even in highly disturbed agroecosystems, plant pathogens are embedded within complex ecological communities where interactions between vector and non-vector species across multiple trophic levels are common. If chewing herbivores within food webs allow viruses to attain higher titers in their plant hosts, and create feeding damage that benefits vector species, then natural selection may favor viruses that render plants attractive to chewers. Several studies have shown that viruses affect vectors, both through direct and plant-mediated pathways, in ways that enhance viral spread (e.g., Belliure et al. 2005, Mauck et al. 2010*a*, Bosque-Pérez and Eigenbrode 2011). Our results suggest the intriguing possibility that such effects may extend to non-vector species as well. In turn, natural selection might favor non-vector herbivores that provide a more conductive host environment for pathogens whenever plant infection provides reciprocal benefits for the herbivore. More broadly, moving beyond simple studies of vector-virus dynamics will help elucidate how community-wide interactions affect disease ecology.

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Supporting Information

Additional supporting information may be found in the online version of this article at http://onlinelibrary.wiley.com/doi/10.1002/ecy.2449/suppinfo

Data Availability

Data are available from Figshare: https://doi.org/10.6084/m9.figshare.6753317