Antagonistic Effects of Soybean Viruses on Soybean Aphid Performance

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ABSTRACT

Although there is long-standing recognition that pest complexes require different management approaches than individual pests, relatively little research has explored how pests interact. In particular, little is known of how herbivorous insects and plant pathogens interact when sharing the same host plant. The soybean aphid, *Aphis glycines* Mastumura, a recently introduced pest of soybean in the upper midwestern United States, and a complex of plant viruses vectored to soybean by insects have become a major concern for growers in the region. Given the abundance of soybean aphid and the increase in virus incidence in recent years, soybean aphids often use soybean infected by plant viral pathogens. We tested the hypothesis that soybean aphid performance is affected by virus infection of soybean plants. We conducted a series of field and laboratory experiments that examined how infection of soybeans with the common plant viruses, alfalfa mosaic, soybean mosaic, and bean pod mottle viruses, influenced soybean aphid performance. Soybean plants (in the field and laboratory) were hand inoculated with individual viruses, and aphids were allowed to colonize plants naturally in field experiments or added to the plants in clip-cages or within mesh bags in laboratory assays. In the field, aphid density on uninfected control soybean plants was nearly double that on infected plants. In laboratory assays, aphid population growth rates were on average 20% lower for aphids on virus infected compared with uninfected plants. Life table analyses showed that increased mortality on virus-infected plants likely explain differences in aphid population growth. Although there was some heterogeneity in the significance of treatment effects among different experiments, when independent experiments are taken together, there is on average an overall negative effect of these viruses on soybean aphids.

KEY WORDS

*Aphis glycines*, alfalfa mosaic virus, bean pod mottle virus, aphid–plant–virus interactions

Entomologists have long recognized that pest complexes require different management approaches than individual pests (Russin and Boethel 1994). However, relatively little research has focused on pest–pest interactions on a shared host plant, and particularly little is known of how herbivores and plant pathogens interact. Insect–insect, insect–pathogen, and pathogen–pathogen interactions can occur directly through competition or indirectly through the response of the shared host plant to herbivory or pathogen attack. Herbivores or pathogens can alter plant quality in a variety of ways to the detriment or benefit of subsequent consumers of the host. For example, “induced resistance” occurs when plant stresses, such as prior herbivory or mechanical damage, results in chemical or physical changes that deter feeding or inhibit growth or development of a subsequently attacking agent (Karban and Baldwin 1997). Conversely, herbivory and pathogen attack can also lead to “induced susceptibility” in host plants whereby subsequent herbivory is greater on previously stressed plants (Preston et al. 1999). Thus, through effects on the shared host plant, prior herbivory by insects or infection by plant pathogens can influence the performance and preference of later-colonizing species.

Since 2000, the soybean aphid, *Aphis glycines* Mastumura (Aphididae), and the bean leaf beetle, *Cer
toma trifurcata* Forster (Chrysomelidae), have become important pests of soybean (*Glycine max* L.) in the upper Midwest (Geisler et al. 2002, Macedo et al. 2003). In addition to direct damage to plants (Macedo et al. 2003), both species transmit viruses to soybean. *A. glycines* transmits alfalfa mosaic virus (AMV) and soybean mosaic virus (SMV) (Hill et al. 2001), and *C. trifurcata* is a vector of bean pod mottle virus (BPMV) (Krell et al. 2004). Because *A. glycines* populations are active throughout the growing season, the likelihood of aphids encountering previously infected plants is high. However, because of the recent introduction of *A. glycines* to the upper Midwest, virtually nothing is known about how soybean viruses affect the population growth of *A. glycines*.

Plant virus infections have been shown to both increase and decrease plant quality for aphids in a
variety of crops. For example, infected plants can increase aphid population growth rates or increase the attractiveness of host plants compared with uninfected controls in wheat, barley, and oats (Ajayi and Dewar 1982, Fereres et al. 1989, Quiroz et al. 1992, Jimenez-Martinez and Bosque-Perez 2004, Mowry and Ophus 2006), squash (Blua et al. 1994), potatoes (Castle and Berger 1993, Eigenbrode et al. 2002), and beets (Kennedy 1951). Conversely, other studies with Nicotiana sp. (McIntyre et al. 1981), arrowleaf clover (Ellsburpy et al. 1985), and wheat (Mowry 1994, Feibig and Donohue 2004) have found that plant viruses can negatively affect aphid performance and preferences.

Although it is not known how A. glycines will respond to virus-infected soybean, previous research has shown that soybean can gain increased resistance to fungal and bacterial pathogens through an induced systemic acquired resistance (SAR) response (Lin and Kogan 1990, Kogan and Fischer 1991, Underwood et al. 2000, Iverson et al. 2001). Furthermore, there is indirect evidence that soybean-induced responses to pathogens can impact subsequently attacking herbivores. For example, using salicylic acid to chemically stimulate a SAR response in soybean, bean leaf beetle, Ceratoma trifurcata (Forster), preference (Srinivas and Danielson 2001), and Mexican bean beetle, Epilachna varivestis (Mulsant), performance were decreased relative to noninduced plants (Iverson et al. 2001). Although these studies used leaf chewing insects rather than phloem feeding aphids, the studies suggest that pathogen-induced plant responses can impact soybean herbivores. Because A. glycines populations are active throughout the growing season and densities often exceed economic thresholds of 250 aphids per plant and > 80% of plants in a field with aphids, the likelihood of aphids encountering infected soybean plants is high. For example, field plot incidence of SMV can be as high as 80% when aphids are present (Burrows et al. 2005). Similarly, AMV is very common in Wisconsin soybean fields, with as many as 28% of samples from statewide surveys containing the virus (Lee et al. 2001). Early-season activity of C. trifurcata can also cause widespread BPMV infections to soybeans (Krell et al. 2004) and increase the likelihood that soybean aphids will encounter plants previously infected with this virus as well.

In addition to the increased potential of the spread of viruses by aphids, how those viruses affect soybean aphid performance on infected plants is unknown, making it difficult to predict the dynamics of aphid populations and the progression of virus epidemics within soybean fields. In this study, we conducted a diverse series of field and laboratory experiments that examined A. glycines performance (abundance or population growth rate) on control soybean plants (uninfected plants) and plants infected with viruses common in soybean in the upper Midwest (AMV, SMV, and BPMV) to test the hypothesis that viruses have the potential to affect performance of soybean aphid.

Materials and Methods

Experiments were conducted in the field and laboratory to assess the effects of virus infection in soybean on A. glycines population growth. The general approach was to (1) mechanically inoculate soybean plants with different viral pathogens (AMV, SMV, or BPMV), (2) allow symptoms to develop, (3) either (a) allow natural colonization of aphids in the field or (b), manually instert plants with soybean aphids in the laboratory, and (4) monitor aphid population growth on virus-treated plants and compare them to control or mock-inoculated plants. We used a diversity of viruses, soybean varieties, and methodologic approaches to examine the generality of the aphid performance response to virus infection.

Virus Stocks and Mechanical Inoculations

For all experiments, we used Wisconsin field-collected isolates of AMV (Bromoviridae, strain C-Janesville), BPMV (Comoviridae, strain Janesville), and an Iowa strain of SMV (Potyviridae, strain [AI]-5). Virus stocks were maintained in susceptible soybean cultivars in a greenhouse by repeated mechanical inoculations. To establish virus treatments for field and laboratory experiments, soybean plants were mechanically inoculated when seedlings produced the first unifoliate leaves, (V0 stage) (Pedersen 2004). Infectious sap was prepared by grinding the uppermost trifoliate from infected stock plants in 10 ml of phosphate-buffered saline (PBS, pH 7.2). On each plant to be inoculated, V0 leaves were dusted with carborundum powder, and both leaves were rubbed gently with cotton swabs soaked in AMV, BPMV, or SMV sap, respectively (Rabedeaux et al. 2005). Mock-inoculated plants were included in a subset of the experiments to determine whether mechanical damage by the carborundum powder might induce responses that could affect aphid performance independent of the effects of viruses. Plants were treated as described above, but leaves were swabbed with sterile PBS rather than with the virus inoculum. In this study and in other preliminary studies (J.R.D., unpublished data), we consistently found no differences in aphid population growth between mock-inoculated and control soybean plants. Therefore, not all experiments included the mock-inoculation treatment. "Control" treatment plants were untreated.

Before beginning experiments, individual soybean plants (including mock-inoculated and control plants) were visually rated for virus symptoms on a scale from 0 to three (0 = no symptoms, 3 = severe symptoms including stunting, leaf mottling, leaf puckering, and/or lesions). All virus-inoculated plants rated at least 2 (stunted growth, apparent leaf mottling) before establishment of aphids. Mock-inoculated and control plants included in studies did not show symptoms. Presence (or absence) of viruses in the stock plants and plants in experimental treatments were confirmed by a double antibody enzyme-linked immunosorbent assay (ELISA) with commercially avail-
able antibodies (AgDia, Elkhart, IN) for SMV, AMV, and BPMV. Positive and negative controls were used in each assay. Samples with absorbance values (measured at 405 nm) that exceeded the mean plus 2 SD of the negative control were regarded as positive for the target virus. All mechanically inoculated plants were ELISA positive for virus, whereas mock-inoculated plants and controls were always negative for virus.

### Field Experiment

A small-plot field experiment was conducted at the University of Wisconsin West Madison Agricultural Research Station (ARS) in 2003. Four blocks were established, each containing two virus treatments (AMV, SMV), a control, and 18 soybean varieties (see Table 1 footnote) with one plant of each soybean variety. Treatments were arranged in a completely randomized block design with virus treatment randomly assigned to one of three rows in each block and varieties randomly assigned within rows. A buffer strip (one row), containing a single soybean variety, surrounded blocks. Plots were planted on 7 May. When plants reached the V0 growth stage (20 May), they were mechanically inoculated with virus treatments as described above.

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Description</th>
<th>Varieties</th>
<th>Parameter*</th>
<th>Treatments (mean ± SEM)</th>
<th>P</th>
</tr>
</thead>
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<tr>
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<td>18 varieties†</td>
<td>Final density</td>
<td>Control</td>
<td>500 ± 89.27a</td>
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<tr>
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<td>Clip-cage</td>
<td>IA 2021</td>
<td>Pop. growth</td>
<td>Control</td>
<td>0.16 ± 0.02a</td>
</tr>
<tr>
<td>3</td>
<td>Clip-cage</td>
<td>IA 2021</td>
<td>Pop. growth</td>
<td>Control</td>
<td>0.07 ± 0.02</td>
</tr>
<tr>
<td>4</td>
<td>Bagged plant</td>
<td>BSR-101</td>
<td>Pop. growth</td>
<td>Control</td>
<td>0.24 ± 0.02a</td>
</tr>
<tr>
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<td>Life table</td>
<td>BSR-101</td>
<td>r</td>
<td>Control</td>
<td>0.236 ± 0.001a</td>
</tr>
<tr>
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<td>r</td>
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<td>7</td>
<td>Life table</td>
<td>BSR-101</td>
<td>r</td>
<td>Control</td>
<td>0.359 ± 0.002</td>
</tr>
</tbody>
</table>

*P* values are given for overall tests of significance. Within-row means with different letters are significantly different (*α* = 0.05).

† Per capita population growth rates (Pop. growth) was calculated as ln[(final no. aphids + 1)/(initial no. aphids)]/no. days. Intrinsic rate of increase (*r*) was calculated using life table statistics.

‡ Treatments included control and mock (buffer inoculated), SMV-, AMV-, and BPMV-infected plants.

§ Varieties included Bell, Blackhawk, Colfax, Dwight, Merit, Newton, Spansoy 201, IA 1008, IA 2021, A96-494018, M90-184111, M90-217007, M93-326056, LN 92-12033, LN 98-4446, 42386A, 507353P, and 511.356.

### Laboratory Experiments

#### Population growth rates of *A. glycines* were measured under controlled conditions (assayed in clip-cages and fine mesh sleeves) on virus-infected and uninfected plants. For each experiment, soybeans were grown in 0.5-liter pots using a 1:1 mixture ofpeat moss and Metro-Mix 366-P growing medium (Scotts-Sierra Horticultural Products, Marysville, OH). Seeds were inoculated with *Bradyrhizobium japonicum* (Nitragin Inoculants; Liphatech, Milwaukee, WI) by dusting seeds with spores at the time of planting. Osmocote slow release fertilizer was added to each pot at a rate of 3 g/liter (14:14:14, N:P:K + micronutrients; Scotts-Sierra Horticultural Products). Plants were grown in the greenhouse with a 16-h light, 8-h dark photoperiod under 600-W sodium-vapor lights. An *A. glycines* colony was maintained in a separate greenhouse for the duration of the laboratory studies. Aphids were added to the colony regularly from field populations at West Madison and Arlington, WI (University of Wisconsin ARS) to maintain genetic variation.

For all laboratory experiments, initial aphid age was standardized across treatments by starting with same-aged cohorts. Adult aphids from the colony were placed onto freshly excised soybean leaves (10 per leaf) in petri dishes containing moistened filter paper. After 24 h, adults were removed and newly deposited neonates were haphazardly assigned to different treatments. All laboratory assays were conducted with apterous aphids.

#### Clip-Cage Experiments. The first set of laboratory experiments compared *A. glycines* population growth in clip-cages on AMV-infected plants with that of aphids on control plants using intact soybean plants. Clip-cages were constructed of 50 by 9-mm petri dishes (Benton Dickinson and Co., Franklin Lakes, NJ), with the outer surfaces of the tops and bottoms cut out. A fine nylon mesh was affixed to the upper half of the clip-cage, the top and bottom was hinged with 49-mm metal all purpose hair clips (Brentwood Beauty Labs, Hillside, IL), and foam was glued to the inside edges of the plastic to avoid damage to soybean leaves and prevent escape of aphids. Two repetitions...
of this experiment were conducted (Table 1, experiments 2 and 3). In the first, virus treatments were established in a single soybean variety (Table 1) replicated 18 times. The second experiment included three varieties (Table 1) replicated 12 times. In both clip-cage experiments, plants were maintained in a laboratory growth room at 25°C under fluorescent growth lights with a 16:8 light:dark photoperiod. Treatments were randomized within the growth room and haphazardly rearranged at the time of each counting (completely randomized design). Neonate aphids were added to clip-cages (experiments 2 and 3, one and two neonates, respectively) to the underside of the uppermost expanded trifoliate (V4 in these experiments) of each plant. Aphids were counted within each clip-cage on day 18 (experiment 2) and day 20 (experiment 3), respectively. Per capita daily population growth rates were calculated as ln[(final no. aphids + 1)/initial no. aphids]/no. days. The effect of virus infection on aphid population growth rate was analyzed using a t-test for experiment 2, and for experiment 3, we used a mixed-model ANOVA with virus treatment as a fixed effect and soybean variety as a random variable. Post hoc means comparisons were made using Dunnett’s post hoc test to the control (α = 0.05).

Bagged Whole Plant Experiment. This experiment compared A. glycines population growth rate on AMV, BPMV, and mock-inoculated soybeans with that on control plants using whole plants bagged within fine mesh sleeves. Treatments were established on soybean variety BSR-101 as described above (Table 1, experiment 4). Three neonates were added to each of 10 replicate plants per treatment at growth stage V3 (most fully expanded trifoliate). Per capita population growth rates were calculated as above after 17 d, and differences among treatments were assessed using one-way ANOVA. Mean population growth rates of aphids on treatments were compared with that of the control using Dunnett’s post hoc test (α = 0.05).

Life Table Experiments. This final assay examined aphid population growth by following same-aged cohorts of neonates developing in clip-cages on control, AMV, BPMV, and mock-inoculated soybeans (Table 1, experiments 5–7) and constructing life tables. Experimental treatments were established as outlined above and replicated 10 (experiment 5), 20 (experiment 6), and 12 times (experiment 7), respectively. In the first repetition of these experiments (experiment 5), a single neonate was added to clip-cages. Because we were unable to distinguish mortality caused by the physical transfer of neonates to the leaf from potential effects of plant quality, cages in which aphids died on day 2 were excluded from the analysis, which led to loss of an entire replicate. Therefore, in subsequent experiments, three (experiment 6) and five (experiment 7) neonates were placed in clip-cages on the uppermost expanded trifoliate (V3). Therefore, mortality of a single aphid in a replicate in the first day did not result in the loss of an entire replicate. Mortality after the first day after transfer was assumed to be caused by differences in performance. When the first of the neonates reached adulthood, any additional remaining individuals (if still present) were removed from the cage to standardize the number of adults to one per cage. In all experiments, clip-cages were checked daily to record mortality and fecundity. Each day, neonates newly deposited by the mother were counted and removed from each cage. This differed from the “clip-cage” experiment (above) in which all aphids remained in the cages for the duration of the experiment. Life table experiments were run for 35 d. Plants were rearranged within the growth room weekly to minimize the influence of location on aphid development. In addition, clip-cages and aphids were moved to the upper-most (youngest) trifoliate weekly. From these experiments we were able to determine the mean daily survivorship (lx) and fecundity (mX) schedules for each treatment. Kaplan-Meier survivorship analysis (Parmar and Machin 1995) was used to account for censored data for aphids that were lost from the experiment or for those that made it to the end of the experimental period. Time to median mortality of the cohort was calculated, and differences among treatments were analyzed with a Wilcoxon signed-rank test in JMP. From the lx-mX table of each treatment, we calculated the intrinsic rate of increase (r) using PopTools 2.6 (Hood 2003). Under the null hypothesis of no-difference between the treatments, individual replicates from each original treatment were randomly shuffled between two treatments being compared. Life table parameters were recalculated for each iteration (n = 1,000) and a test statistic (difference in r shuffled for treatment A and treatment B, e.g., control versus virus) was computed for each newly shuffled treatment combination. The number of times the test statistic from the shuffled data (r shuffled) exceeded the test statistic from the original data (r), determined the P value for a particular treatment comparison (one-tailed test). This procedure was repeated for each pairwise treatment comparison, and the overall α was adjusted to reflect the multiple comparisons. Randomizations were performed using Poptools 2.6 (Hood 2003).

Meta-Analysis

Because this study included data from seven independent experiments, using four different experimental approaches, a meta-analysis was performed as a way of providing a quantitative overall assessment of the effect of virus infection on soybean aphid population growth. For each experiment, we calculated the log of the P value for the main effects of virus treatments on final density (field study), per capita daily population growth rates, and intrinsic rate of increase. A χ² statistic was calculated as −2 ∑ ln Pi, across the i = 7 experiments, and an overall P value was estimated by comparing this statistic to a χ² distribution with 2k degrees of freedom (k = the total number of separate estimates of P) (Sokal and Rohlf 2001).
**Results**

**Field Experiment.** Approximately 2 mo after inoculation, aphid densities on BPMV- and SMV-infected soybean plants in open field plots were nearly one half (≈265 aphids/plant) that of the controls (500 aphids/plant) in field plots at West Madison (Table 1, \( F_{3,34} = 11.62, P < 0.001 \)). A comparison of statistical models with and without the random term “variety” indicated no significant main effect of variety (\( \chi^2 = 0.57, 1 \text{ df, } P = 0.45 \)).

**Clip-Cage Experiments.** In the first laboratory assay, aphid population growth over an 18-d period within clip-cages was three times higher on control plants compared with that on AMV-infected plants (Table 1, experiment 2; \( F_{1,17} = 12.41, P = 0.003 \)). In the second clip-cage experiment, average population growth rate was lower in virus-infected plants compared with controls, but the effect was not statistically significant (Table 1, experiment 3; \( F_{1,38} = 1.87, P = 0.18 \)). Comparisons of models with and without the random effect of variety indicated no significant main effect of soybean variety (\( \chi^2 = 0.10, 1 \text{ df, } P = 0.75 \)). We note that population growth rate on controls in experiment 2 was on average double that in experiment 3 (0.16 versus 0.07, respectively) which may have contributed to the differences between these experiments.

**Bagged Plant Experiment.** Consistent with the clip-cage experiments, aphid population growth on bagged whole soybean plants was substantially lower on both AMV- and BPMV-infected soybean plants compared with controls (Table 1, experiment 4; \( F_{3,39} = 3.39, P = 0.029 \)). Aphid population growth rate on mock-inoculated plants was not significantly from either treatment.

**Life Table Experiments.** Aphid population growth on virus-infected plants was examined by following fecundity and survivorship of cohorts of individual aphids developing in clip-cages and constructing life tables. In the first clip-cage experiment (Table 1, experiment 5), estimates of aphid intrinsic rate of increase (\( r \)) on AMV-infected plants were almost 20% lower than that of aphids developing on control plants (Table 1; \( P < 0.001 \)). Similarly, \( r \) was ≈15% lower on BPMV-infected plants relative to aphids reared on healthy (control and mock) plants (Table 1, experiment 6; \( P < 0.001 \)). Furthermore, in experiments 5 and 6, survivorship of aphids appeared to be lower on virus-infected plants, because time to median level of mortality for aphids on AMV- and BPMV-infected plants was 11 and 13 d, respectively, compared with an average of 23 d for aphids on control plants. These differences in average survival were, however, only significant for BPMV versus control (\( P = 0.022 \)). A third life table experiment (experiment 7), however, showed no differences in \( r \) or time to median survivorship between virus-infected (AMV and BPMV) and control plants (\( P > 0.50 \) for both viruses).

**Meta-Analysis.** In five of the seven independent experiments, there were significant negative effects of plant virus infections on A. glycines performance as measured by population size, population growth rates, or intrinsic rate of increase, relative to control (uninfected) soybean plants. When probabilities (\( P \)) are summarized across all experiments, an overall effect of plant virus infection on A. glycines development (\( \chi^2 = 92.34, df = 14, P < 0.001 \)) is evident.

**Discussion**

The outcomes of multispecies interactions in crops have the potential to complicate predictions of population dynamics of interacting species based on single species studies. In this study, we examined the potential interactions between plant viruses and an insect pest that share the same host plant species. In a series of diverse field and laboratory experiments, we found that, in general, soybean aphid performance (as measured by overall abundance and population growth rates) on virus-infected soybean plants was significantly lower compared with that of aphids feeding on healthy soybean plants. This finding was observed for plant viruses (AMV, SMV, and BPMV) from three different families (Bromoviridae, Potyviridae, and Comoviridae, respectively), and the effects were manifested across several soybean varieties and using various experimental approaches, suggesting that for this aphid–host plant system, negative effects on aphid performance may be a general response to plant virus infection. On average, virus infection resulted in 50% lower aphid densities compared with controls in the field and ≈25% lower population growth compared with control plants in laboratory assays, findings that could have significant impacts on aphid population dynamics.

Plant pathogen–herbivore interactions are necessarily mediated by the shared host plant and thus are dependent on how the plant responds to and integrates different types of stresses. A simple model of induced responses posits that plant responses to pathogens are typified by signaling cascades characteristic of the shikimic acid pathway and the signaling compound salicylic acid (SA), which can result in systemic acquired resistance (SAR). In contrast, cascades in the octadecanoid pathway with signaling mediated by jasmonic acid (JA) and ethylene production elicit induced resistance (IR) in response to herbivores or wounding. Although initially thought to be separate responses to different plant stressors, recent evidence suggests that multiple pathways can be stimulated by either herbivores or pathogens, and given that “cross-talk” between the pathways is common (Thaler and Bostock 2004), an herbivore/JA–pathogen/SAR model of plant-induced responses may be too simplistic (Maleck and Dietrich 1999, Paul et al. 2000). Preston et al. (1999) gave empirical support for the notion of cross-talk between plant induction pathways by showing that in *Nicotiana tabacum* L., systemic-induced response to tobacco mosaic virus infection leads to inhibition of nicotine production and increased herbivory as SA inhibits the production of JA. Although the mechanism of the interaction is currently not known, our findings suggest that soybean
viruses may induce plant resistance to soybean aphids under laboratory and field conditions. Studies in soybeans using chemical elicitors such as SA, designed to mimic a pathogen response and induced SAR, found negative effects on performance of leaf chewing insects (Srinivas and Danielson 2001). It is possible that such a mechanism is involved in the negative effects of plant viruses on soybean aphids. Further studies are necessary to resolve the underlying mechanisms by which viruses change host quality for aphids.

That plant viruses can affect insect performance has been shown in several other systems. However, the magnitude and even the sign of the interaction can vary depending on the taxa involved. For example, viruses both negatively (Mowry 1994, Fiebig et al. 2004, Eubanks et al. 2005) and positively (Montllor and Gildow 1986, Cardoza et al. 2003) affect host quality, and thus, growth rates of herbivores. Our results stand in contrast to several previous studies that suggest viral infection in plants leads to increased aphid performance and preference (Montllor and Gildow 1986, Costa et al. 1991, Kostiw 1991, Quiroz et al. 1992, Cardoza et al. 2003, Jimenez-Martinez and Bosque-Perez 2004, Mowry and Ophus 2006, Srinivasan et al. 2006), effects that may be attributable to physical symptoms such as yellowing and molting of leaves or increased amino acid and sugar concentrations in the phloem sap (Quiroz et al. 1992, Fiebig et al. 2004, Blua et al. 1994).

Although the overall finding of our experiments shows that, on average, virus infection of soybeans results in significant negative effects on aphid performance, aphid response to viruses was variable in laboratory assays because we failed to find evidence of significant negative effects on aphid population growth rate in two of the seven experiments. Such variation may indicate there are additional factors that interact with plant responses to virus infection to impact aphids. For example, in one clip-cage experiment (Table 1, experiment 3), low overall aphid growth rates across all plants including controls is associated with a lack of difference between aphid performance on infected and uninfected plants. Plant responses to biotic stressors, including viruses, are variable and can depend on interactions with other environmental factors such as light and nutrient availability (reviewed by Balachandran et al. 1997). Although we made every effort to standardize environmental conditions within and among experiments, plant quality factors such as N availability or other nutrient stresses (not measured in this experiment; Myers and Gratton 2006), nodulation rates, time of year, or some other factor affecting aphid performance may have overshadowed or interacted with virus–treatment effects (see also Donaldson and Lindroth 2006). For example, maize dwarf mosaic virus titers increase in response to nutrient and moisture stress (Vangessel and Coble 1993) and severity of tomato mosaic virus symptoms in pepper plants has been shown to respond strongly to moderate differences in temperature (18–24°C; Schuenger and Hammer 1995).

Our findings have implications both for aphid populations in the field and for patterns of virus transmission. Although mechanisms for decreased aphid population growth rate remain unclear, life table analyses suggest that decreased survivorship may lead to decreased aphid performance on infected soybean. Lower population growth rates could prolong the amount of time aphid populations are below economic threshold, potentially decreasing the risk of additional yield losses caused by aphids. Moreover, slower population growth rates may also allow time for natural enemies to aggregate later in the summer, potentially further decreasing aphid populations and minimizing yield losses.

The results of this study provide an important first step toward understanding the complex interactions that occur among soybean, its associated viruses, and soybean aphids. Our data also bring to light the need for in-depth mechanistic studies aimed at understanding plant defensive pathways in soybean, and how induced defenses (against pathogens and herbivores) impact aphid populations and subsequently the epidemiology of vectorborne viruses in this system. In addition to affecting aphid performance, plant viruses may also influence aphid preferences and colonization behavior, which can have significant implications for increasing virus transmission in the field (Eckel and Lampert 1996, Fereres et al. 1999). How soybean aphids orient toward and behave on virus-infected soybean plants is currently unknown. Significantly lower aphid populations in the field experiment (Table 1, experiment 1) could have been caused by both effects on aphid survivorship and increased emigration from virus-infected plants. If infection results in higher emigration rates, we may have underestimated the potential impact of viruses on aphid populations in the caged experiments (emigration was not permitted in laboratory studies). However, increased aphid movement could also result in the greater spread of these nonpersistent viruses within and among fields (Wang and Ghabrial 2002). Given the implications for aphid population distributions and for virus transmission in the field, the effect of viruses on aphid behavior deserves further study in this system. Nevertheless, an understanding of mechanisms underlying the direct negative impacts of plant viruses on aphid populations in soybean may open novel avenues for management of this important agronomic pest. For example synthetic and chemical elicitors or asymptomatic strains of viruses may be used to potentially induce soybean resistance to A. glycines without the negative consequences of viruses on plants.

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