Lack of Effect of Tobacco Mosaic Virus-Induced Systemic Acquired Resistance on Arthropod Herbivores in Tobacco

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ABSTRACT

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The nonspecificity of pathogen-induced systemic acquired resistance (SAR) of tobacco, cucurbits, and other plants against plant pathogens has led to speculation that cross-resistance might also be afforded to arthropod herbivores. However, we found that inoculation of the lower leaves of tobacco (*Nicotiana tabacum*) with tobacco mosaic virus (TMV) had no effect on population growth of tobacco aphids (*Myzus nicotianae*) reared on upper leaves, for which SAR to TMV was active. Newly hatched tobacco hornworms (*Manduca sexta*) were reared to pupation on upper leaves from TMV-inoculated or control plants. Although larval weight gain during the first week was somewhat lower on induced plants in some tests, there were no significant effects on duration of development to pupation or mean pupal weight. In reciprocal tests, previous feeding damage by *M. sexta* did not induce SAR to TMV. However, reproduction of tobacco aphids was reduced on intact leaves of hornworm-damaged plants in two of four trials. These findings suggest the absence of strong, reciprocal interactions in response to induction by pathogens or herbivores and that TMV-activated SAR of tobacco is unlikely to provide significant cross-protection from these arthropod herbivores in the field.

Additional keywords: plant defense.

Many plants respond to localized pathogen attack by activating defenses in noninfected parts of the plant. Restricted (i.e., nonsystemic) infection of tobacco, cucurbits, beans, and other plants with certain viruses, fungi, or bacteria induces persistent, broad-spectrum, systemic acquired resistance (SAR) to subsequent infection by diverse pathogens (14,15,23). Induction by attenuated pathogens, plant extracts, or simple chemical substances underscores the potential practicality of this approach for plant disease management (15).

Restricted infection with tobacco mosaic virus (TMV) of tobacco (*Nicotiana tabacum* L.) cultivars containing the N-gene for resistance to TMV (hypersensitive reaction) induces persistent SAR against a diversity of pathogens, including TMV (19), *Peronospora tabacina* (25), *Phytophthora parasitica* var. *nicotianae*, *Pseudomonas tabaci*, (18) and others. SAR does not confer absolute immunity; rather, it reduces the severity of the disease (e.g., there are smaller and sometimes fewer lesions) in TMV-challenged plants. Induction elicits systemic accumulation of soluble, pathogenesis-related (PR) proteins (25,26), as well as a systemic increase in activities of peroxidase, β-1,3-glucanase, and chitinase. The functions of PR proteins and increased peroxidase activity in SAR of tobacco are equivocal (16,26). Recent work implicates salicylic acid as the endogenous signal in the transmission of SAR in tobacco (16).

The nonspecificity of pathogen-activated SAR suggests that it could provide the added benefit of suppression of arthropod herbivores in the greenhouse or field (8,12,17). McIntyre et al (18) found that restricted infection with TMV on tobacco resulted in a 9–12% reduction in reproduction of the green peach aphid (*Myzus persicae*) on noninfected younger leaves. This was evidently the first demonstration that localized infection by a plant pathogen could induce systemic resistance against a phytophagous insect. Similarly, Hare (8), citing unpublished data, reported a 16% reduction in growth rate of the tobacco hornworm (*Manduca sexta*) on systemically protected leaves of TMV-inoculated plants. In contrast, inoculation of cucumber (*Cucumis sativus*) with the anthracnose fungus (*Colletotrichum lagenarium*) or tobacco necrosis virus (TNV) did not significantly affect performance of arthropod herbivores reared on foliage for which SAR against pathogens was simultaneously confirmed (1,2).

Alternatively, the benefits of SAR could be negated if induced plants are favored by herbivores. For example, infection of tomato with a nearly symptomless strain of TMV resulted in greater survival of the Colorado potato beetle (*Leptinotarsa decemlineata*), an insect for which tomato is a marginal host (9). Striped cucumber beetles (*Acalymma vittata*) consistently fed more on TNV-inoculated cucumber plants than on control plants (2). The consequences of elevated levels of PR proteins for herbivory are unknown; however, increased levels of total soluble proteins have been associated with enhanced performance of some herbivores on stressed plants (22). Thus, the effects of pathogen-activated SAR on host suitability for arthropods presently cannot be predicted (5,17).

Systemic infection by plant pathogens often changes plant quality for arthropod herbivores (7,10,13). However, to our knowledge, the aforementioned two studies with tobacco (8,18) and our more recent work with cucumber (1,2) are the only published tests of the hypothesis that induction of SAR by restricted infection with a plant pathogen can alter host suitability for arthropods. We reexamined this question by inoculating lower leaves of tobacco with TMV and measuring the response of tobacco aphids (*Myzus nicotianae*) and tobacco hornworms to systemically protected upper leaves. We also tested the reciprocal hypothesis that previous herbivory by *M. sexta* would induce systemic resistance to TMV and *M. nicotianae*. Mechanical wounding or herbivore damage to leaves of solanaceous plants induces production of proteinase inhibitors, phytoalexins, and other secondary metabolites, which may be systemically translocated and have been implicated in plant defense (11,20). Recent work has demonstrated short-term, systemic increases in alkaloids of wild tobacco (*N. sylvestris*) induced by real and simulated herbivory (3,4), and it has been suggested that such responses represent a generalized defense against both herbivores and pathogens (13).

MATERIALS AND METHODS

General methods. Burley tobacco cv. KY 14 with the N-gene for resistance to TMV (25) was used in this study. Seeds were sown in Terra-Lite potting soil (W. R. Grace & Co., Fogelsville, PA) in small trays. After 2 wk, groups of 15 seedlings were transplanted to trays in professional growing medium 300 (Pro-Gro Products, Elizabeth City, NC) and fertilized daily with Peters 15-16-17 fertilizer (W. R. Grace & Co.) at 1.5 g/L of water. Two weeks later, individual seedlings were transplanted into plastic pots (15 cm in diameter) in professional growing medium 300. Plants were grown in a greenhouse at 19–33 C with a photoperiod of 14 h supplemented with sodium lamps that provided about 350 μE m^{-2} s^{-1} at canopy level and fertilized three times per week. Some trials were conducted in a growth chamber (Percival WE-95, Percival Manufacturing, Boone, IA) equipped with white fluorescent and incandescent lights and maintained at 24 ± 2 C with a 16 h photoperiod. Plants used in growth chamber trials were moved to the chamber 48 h before inoculation with TMV.

Purified tobacco mosaic virus (Type strain-TMV) in sterile distilled water was kindly provided by Dr. X. S. Ye (Department of Plant Pathology, University of Kentucky). Tobacco aphids used in these experiments were obtained from a colony maintained on KY 14 burley tobacco in the greenhouse. Tobacco hornworms were obtained from a laboratory colony maintained on artificial diet (24).

Effect of TMV-induced SAR on tobacco aphid population growth. Adaxial surfaces of the third, fourth, and fifth fully expanded leaves (leaves 3, 4, and 5) of tobacco plants at the 9- to 10-leaf stage (about 2 mo old) were dusted with Carborundum and gently inoculated by rubbing them with 25 μg TMV per milliliter of sterile distilled water. About 0.1 ml of inoculum was applied per leaf. Control plants were sham-inoculated with water and Carborundum. The inoculated leaves were immediately misted with water. TMV-inoculated and control plants were arranged in a randomized complete block in the growth chamber or on the greenhouse bench.

Eleven days later, a single aphid nymph (<24 h old) was placed on the abaxial surface of leaf 7 of each plant and confined in a ventilated clip-on cage (3.5 cm in diameter). The aphids, all parthenogenetic females, were reared to reproductive age (4–5 days) and allowed to reproduce. Ten days after initial confinement, the number of nymphs that had been produced by each aphid was recorded. This time interval was sufficient for the original females to produce 20–30 offspring during peak SAR (11–12 days after TMV inoculation) (25,26) but short enough to avoid the confounding effects of additional reproduction by their progeny.

All TMV-inoculated and control plants were tested for presence of SAR to TMV on the same day that the aphid nymphs were introduced. Plants were challenged with TMV (25 μg/ml) on leaf 8 as described for the inducing inoculation. Systemic resistance to TMV was assessed by measuring necrotic lesion size 7 days after challenge using a stereomicroscope with a calibrated ocular. Five randomly picked lesions were measured on each side of the leaf, and the individual areas of the 10 lesions were averaged to provide a single value for each plant. On plants challenged with TMV, the number of necrotic lesions typically varies greatly, and lesion size is the most reliable measure of resistance (25,26).

The experiment was performed five times: twice in the growth chamber from April to August and three times in the greenhouse from June to February. There were 10 replicates of TMV-inoculated and control plants per trial. Growth chamber or greenhouse data were subjected to analysis of variance (ANOVA) for main effects of treatment, trial, and replicate and for trial by treatment interaction. Within each trial, numbers of aphid progeny and mean area of TMV necrotic lesions were compared between TMV-inoculated and control plants by paired t tests. When trial by treatment interaction was not significant ($P > 0.05$), data from the separate trials were pooled and results of the combined analysis are also given. Data are presented as means ± standard error (SE).

Effect of TMV-induced SAR on tobacco hornworms. Tobacco plants were inoculated with TMV or water as described for the aphid tests and arranged in the greenhouse or growth chamber as before. Newly hatched (<24 h old), first-instar hornworm larvae were weighed on a microbalance, transferred to individual petri dishes (15 cm in diameter) containing moistened filter paper, and held at 24 ± 2 C under 16 h of light in the growth chamber. Larvae were fed excised foliage from plants that had been inoculated with TMV or water 11 days before the start of the feeding assay. Pathogen-induced SAR has been shown to be effective in excised leaves (19). Furthermore, removal of foliage above the site of inoculation does not reduce SAR of remaining intact leaves (25). After leaf 7 had been fed upon for 2.5 days, it was replaced with freshly excised foliage (leaves 9–15) at 2- to 3-day intervals for 24–29 days, until the larvae had pupated. Larval food was never limited. All plants were tested for presence of SAR to TMV at the beginning of each assay by challenging leaf 8 with virus inoculum as described for the aphid experiments and measuring the size of the resulting lesions as before.

The experiment was performed four times, twice with plants that were held in the growth chamber after the inducing inoculation (May-August) and twice with plants that were left in the greenhouse throughout the test period (July-October). There were 10 replicates per treatment per trial, each consisting of a single larva that was fed foliage from a separate plant. Initial larval weight, net weight gain after 4 and 7 days, developmental period to pupation and pupal weights of hornworms, and mean area of necrotic lesions resulting from the challenge with TMV were compared between TMV-inoculated and control plants as described before. Data were analyzed in the manner described for the aphid experiments. Death of larvae resulted in unbalanced sample sizes for some comparisons. In such cases, treatment means were compared by t tests for independent samples, rather than

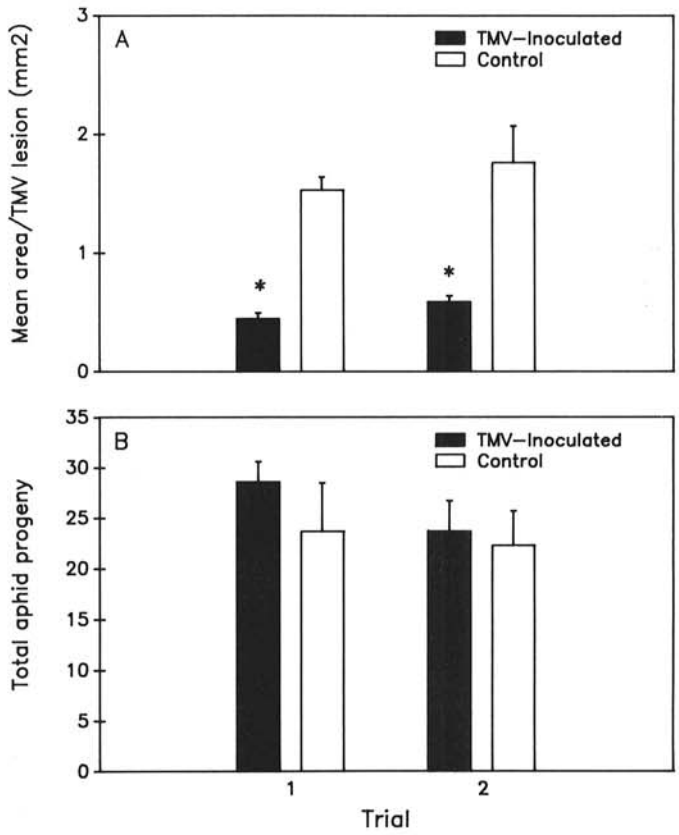

Fig. 1. A, Induction of systemic resistance to tobacco mosaic virus (TMV) by TMV in the growth chamber, and **B,** lack of effect on progeny production by the tobacco aphid, *Myzus nicotianae*, on systemically protected upper leaves. Vertical bars indicate standard error. Means marked with asterisks differ significantly from corresponding control mean (paired t tests, $P < 0.001$).

paired t tests, if the ANOVA indicated the effect of pairing to be nonsignificant (21).

Effect of previous herbivory by tobacco hornworms on plant susceptibility to tobacco aphids and TMV. To test for possible systemic effects of hornworm feeding damage on population growth of tobacco aphids, tobacco plants were grown in the greenhouse as before. Twenty plants (9- to 10-leaf stage, about 2 mo old) were divided into two groups, and half of the plants were damaged by placing three second- and third-instar hornworms on the third, fourth, and fifth leaves (nine total larvae/plant). Larvae were confined to the lower leaves by banding the leaf petioles with cotton. Banding had no apparent effects on plant condition, nor did it affect TMV-induced SAR to the same pathogen in preliminary tests. Treatments were arranged in a randomized complete block in the growth chamber or the greenhouse, with 10 hornworm-damaged and 10 control plants per trial. The hornworms were allowed to feed for 11 days, after which leaf 7 was then challenged with one tobacco aphid nymph (<24 h old) as described before. Aphid progeny were counted 10 days after confinement of the original nymph. The experiment was performed twice in the growth chamber and twice in the greenhouse. Greenhouse and growth chamber experiments were concurrent; trials 1 and 2 ran from August to November and September to December, respectively.

To determine if hornworm feeding damage could induce SAR to TMV, plants were grown and hornworms were confined to leaves 3, 4, and 5 as described above. The larvae were allowed to feed for 11 days, after which intact leaf 8 was challenged with TMV as previously described. Possible induction of SAR was assessed by measuring the areas of 10 necrotic lesions per plant after 7 days as described earlier. The experiment was performed twice in the growth chamber and twice in the greenhouse. Trial dates were 2 wk earlier than those indicated for the tests for hornworm feeding effects on aphids. Data analyses for this experiment and for the preceding one were as described for the tests of TMV-induced SAR on hornworms.

RESULTS

Effect of TMV-induced SAR on tobacco aphid population growth. Tobacco plants inoculated with TMV on the lower leaves were systemically protected against later challenge with TMV on the upper, younger leaves. In the growth chamber, the mean size of necrotic lesions was significantly lower on TMV-inoculated plants than on control plants both in trial 1 ($t = 15.5$, $df = 9$, and $P < 0.0001$) and trial 2 ($t = 3.64$, $df = 9$, and $P < 0.01$), corresponding to average reductions of 68.2 and 70.6%, respectively (Fig. 1). However, progeny production by tobacco aphids did not differ significantly between TMV-inoculated and control plants in either trial 1 ($t = 0.38$, $df = 9$, and $P = 0.71$) or trial 2 ($t = 0.05$, $df = 9$, and $P = 0.96$). ANOVA indicated absence of trial by treatment interaction ($F < 0.1$, $df = 1,26$, and $P = 0.97$) and no significant treatment effect upon aphid reproduction ($F = 0.11$, $df = 1,26$, and $P = 0.74$).

Similarly, aphid reproduction did not differ significantly between TMV-inoculated and control plants in the greenhouse ($t = 0.25$, 0.51, and 0.86 for trials 1–3, respectively; $df = 9$; and $P \geq 0.42$). There was no trial by treatment interaction ($F = 0.28$, $df = 2,38$, and $P = 0.75$), and the overall treatment effect on aphids was not significant ($F = 0.18$, $df = 1,38$, and $P = 0.67$). Reductions in mean area of TMV lesions on TMV-inoculated plants vs. control plants were significant for each trial ($t = 6.22$, 10.0, and 13.3 for trials 1–3, respectively; $df = 9$; and $P \leq 0.002$), indicating that SAR to TMV was present in TMV-inoculated plants (Fig. 2).

Effect of TMV-induced SAR on tobacco hornworms. Tobacco hornworms that were fed leaves from TMV-inoculated plants in the growth chamber trials gained less weight during the first 4 days than did those fed leaves from control plants (Table 1). ANOVA showed a significant overall treatment effect ($F = 10.1$, $df = 1,26$, and $P = 0.02$) but no difference between trials ($F = 0.01$, $df = 1,26$, and $P = 0.91$) and no trial by treatment interaction ($F = 0.16$, $df = 1,26$, and $P = 0.69$). Overall differences between TMV-inoculated and control treatments remained significant after 7 days ($F = 5.53$, $df = 1,25$, and $P < 0.03$) with no trial by treatment interaction ($F = 0.34$, $df = 1,25$, and $P = 0.57$), although paired t tests for data within each trial showed significance only for trial 2 (Table 1). However, mean pupal weight and developmental period (i.e., days to pupation) did not differ significantly between treatments ($F = 0.98$, $df = 1,23$, and $P = 0.33$ and $F = 0.13$, $df = 1,23$, and $P = 0.72$, respectively) (Table 1). Mean area of necrotic lesions resulting from the challenge inoculation was reduced by an average of 59.4 and 73.8% on TMV-inoculated plants in trials 1 and 2, respectively, confirming presence of significant SAR to TMV (Table 1).

In the greenhouse trials, larval weight gain after 4 and 7 days, pupal weight, and days to pupation did not differ significantly between control plants and TMV-inoculated plants for which significant SAR to TMV was simultaneously confirmed (Table 1). There were no significant overall treatment effects upon any of these measures of hornworm performance (ANOVA, $P \geq 0.11$), although trial by treatment interaction was significant for 4-day larval weight gain ($F = 4.29$, $df = 1,25$, and $P = 0.049$). Reduction in mean area of viral lesions on TMV-inoculated plants averaged 45.6 and 61.2% relative to those on control plants in trials 1 and 2, respectively, confirming presence of significant SAR to TMV in TMV-inoculated plants (Table 1).

Effect of previous herbivory by tobacco hornworms on plant susceptibility to tobacco aphids and TMV. ANOVA of the combined growth chamber data indicated a near-significant treatment effect for aphids ($F = 3.98$, $df = 1,26$, and $P = 0.057$), although

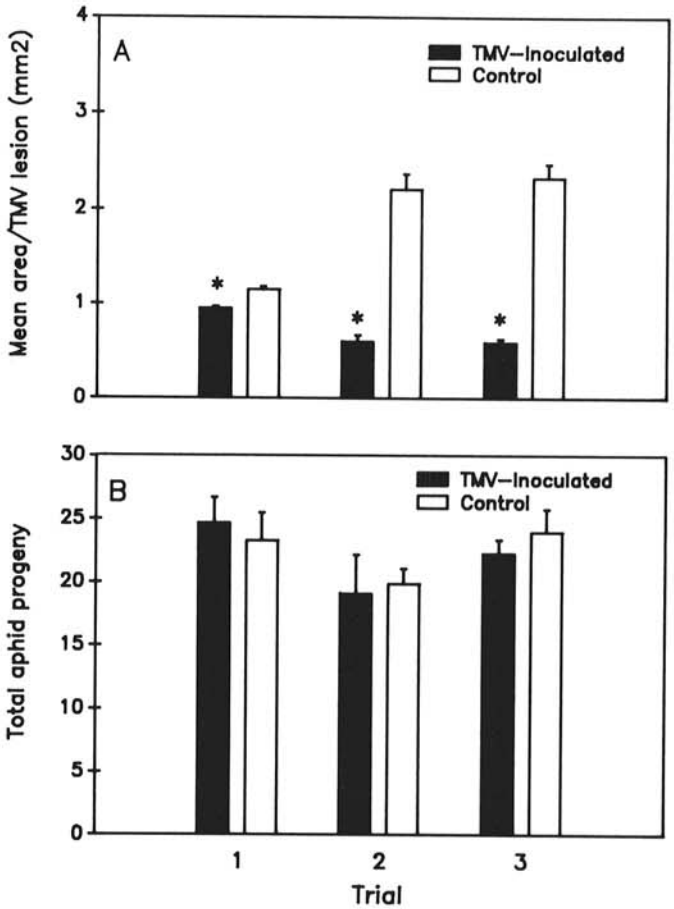

Fig. 2. A, Induction of systemic resistance to tobacco mosaic virus (TMV) by TMV in greenhouse trials, and **B,** lack of effect on progeny production by the tobacco aphid, *Myzus nicotianae*, on systemically protected upper leaves. Vertical bars indicate standard error. Means marked with asterisks differ significantly from corresponding control mean (paired t tests, $P < 0.001$).

the difference in aphid progeny between hornworm-damaged and control plants was relatively small (overall means were 21.8 ± 1.3 vs. 24.9 ± 1.2, respectively). Trial by treatment interaction was also significant ($F = 5.44$, $df = 2,26$, and $P = 0.01$). Analysis of the two trials separately showed that significantly lower numbers of aphid progeny were produced on hornworm-damaged plants in trial 2 but not in trial 1 (Table 2). Similarly, ANOVA for the combined greenhouse trials showed a near-significant treatment effect for aphids ($F = 3.83$, $df = 1,23$, and $P = 0.063$); overall means for hornworm-damaged and control plants were 12.0 ± 1.1 and 15.2 ± 1.9, respectively. Trial by treatment interaction was once again significant ($F = 15.46$, $df = 2,23$, and $P < 0.001$); fewer aphid progeny were produced on hornworm-damaged plants in trial 2 but not in trial 1 (Table 2).

Previous feeding upon lower leaves by *M. sexta* failed to induce systemic protection from TMV in either the growth chamber ($F = 1.17$, $df = 1,27$, and $P = 0.29$) or in the greenhouse ($F = 1.62$, $df = 1,27$, and $P = 0.21$). Paired t tests for treatment differences within individual trials were nonsignificant (Table 2).

DISCUSSION

We found that inoculation of the lower leaves of tobacco with TMV had no measurable effect on reproduction of tobacco aphids reared on upper leaves for which SAR to TMV was active (Figs. 1 and 2). This contrasts with the small but statistically significant differences reported by McIntyre et al (18) for green peach aphids reared on TMV-induced plants. TMV becomes progressively more systemic, and expression of SAR declines at temperatures above 28 C (19,26). Greenhouse temperatures ranged from 23 to 33 C during our first greenhouse trial, conducted from June to August. This may account for the relatively weak SAR to TMV in that trial (Fig. 2). Trials 2 and 3 were conducted during the fall and winter when greenhouse temperatures ranged from 19 to 25 C. Much stronger SAR to TMV was present in these trials (Fig. 2).

Our observation that tobacco hornworms grew somewhat more slowly during the first week on TMV-induced plants in the growth chamber is consistent with Hare's (8) report of a 16% growth reduction of fourth-instar *M. sexta* larvae on induced plants. However, no such differences were evident in our greenhouse tests, despite the fact that the induced plants were protected significantly against TMV (Table 1). Furthermore, the number of larvae surviving after 7 days was similar for TMV-inoculated and control plants (Table 1), and there were no differences between treatments in developmental rate or pupal weight in any trial. Hornworm larvae readily fed upon and consumed large amounts of foliage of both TMV-inoculated and control plants. Hare (8) attributed the effect in his experiment to a reduction in the efficiency of assimilation of ingested food rather than the rate at which food was ingested.

Leaf damage increased the alkaloid content of undamaged leaves of wild tobacco plants resulting in reduced growth of *M. sexta* larvae reared on foliage from previously damaged plants (3,4). Certain alkaloids have been shown to inhibit growth of plant pathogens on nutrient agar (13), and it has been suggested that reciprocal interactions between plant pathogens and herbivores mediated by generalized induced plant defenses may be widespread (12,13,17). However, we found that previous herbivory by hornworm larvae on lower leaves of tobacco

TABLE 1. Induction of systemic resistance to tobacco mosaic virus (TMV) by TMV and effects on tobacco hornworm growth and development

Treatment	Initial weight (mg)	Larval weight gain (mg)[a] 4 days	7 days (n)[b]	Days to pupation[c]	Pupal weight (g) (n)	Area per TMV lesion (mm^2)
Growth chamber trial 1						
TMV	2.4 ± 0.1	15.2 ± 1.1*	84.9 ± 12.0 (10)	26.2 ± 0.4	5.7 ± 0.2 (9)	0.58 ± 0.07*
Control	2.5 ± 0.1	20.7 ± 2.8	103.6 ± 23.3 (8)	25.6 ± 0.5	5.6 ± 0.2 (8)	1.43 ± 0.09
Growth chamber trial 2						
TMV	2.0 ± 0.1	16.2 ± 1.8*	70.2 ± 13.6* (10)	27.2 ± 0.4	5.5 ± 0.2 (9)	0.69 ± 0.04*
Control	2.0 ± 0.1	21.2 ± 2.6	109.7 ± 17.9 (10)	27.2 ± 0.5	5.2 ± 0.1 (10)	2.63 ± 0.20
Greenhouse trial 1						
TMV	2.3 ± 0.2	28.4 ± 6.2	142.5 ± 30.6 (9)	29.5 ± 3.5	5.1 ± 0.5 (2)	1.55 ± 0.07*
Control	2.3 ± 0.2	17.9 ± 2.5	80.9 ± 14.4 (10)	27.2 ± 0.9	5.0 ± 0.3 (6)	2.85 ± 0.24
Greenhouse trial 2						
TMV	1.8 ± 0.1	23.6 ± 2.5	136.2 ± 19.7 (10)	28.0 ± 0.0	5.8 ± 0.2 (4)	0.66 ± 0.02*
Control	1.9 ± 0.1	26.9 ± 4.3	144.8 ± 24.8 (9)	27.4 ± 0.6	5.3 ± 0.2 (5)	1.70 ± 0.18

[a] Within trials, means (±SE) followed by an asterisk differ significantly from corresponding control mean at $P < 0.05$, paired t tests except as indicated below.
[b] n = number of larvae surviving after 7 days.
[c] Within greenhouse trials, treatment comparisons for days to pupation and pupal weight were by two-sample t tests because of differing numbers of survivors and unbalanced data.

TABLE 2. Variable effects of hornworm feeding damage on tobacco aphid reproduction and failure of previous herbivory to induce systemic resistance to tobacco mosaic virus (TMV)

Trial	Total aphid progeny[a] Hornworm damaged[b]	Control	Area (mm) per TMV lesion Hornworm damaged	Control
Growth chamber tests				
Trial 1	20.4 ± 1.5	21.4 ± 1.5	1.31 ± 0.24	1.06 ± 0.13
Trial 2	23.3 ± 2.2*	28.5 ± 1.2	1.02 ± 0.07	0.98 ± 0.07
Greenhouse tests				
Trial 1	9.9 ± 1.0	10.1 ± 0.9	1.13 ± 0.14	0.97 ± 0.07
Trial 2	14.2 ± 1.9*	21.0 ± 2.7	1.04 ± 0.07	0.95 ± 0.07

[a] Within trials, means (±SE) followed by an asterisk differ significantly from corresponding control mean (for growth chamber trial 2, $t = 2.10$; $df = 17$, $P = 0.051$; for greenhouse trial 2, $t = 2.09$; $df = 15$, $P = 0.054$).
[b] Hornworms were confined on leaves 3, 4, and 5 and allowed to feed for 11 days before challenging leaf 8 with TMV and leaf 7 with aphids.

consistently failed to invoke significant SAR against TMV (Table 2). Similarly, previous herbivory by twospotted spider mites or fall armyworm caterpillars on lower leaves of cucumber failed to induce SAR against the anthracnose fungus, *Collectotrichum lagenarium* (1).

We did observe reduced aphid reproduction on hornworm-damaged plants; however, the effect was relatively small and approached statistical significance ($P = 0.05$) in only two of the four trials (Table 2). This is consistent with much of the evidence for effects of rapidly induced changes in damaged plants on herbivorous arthropods, which suggests only relatively small differences in such parameters as larval development time or pupal weights (6). We suggest that the literature probably gives a biased view of the ubiquitousness of such effects because of the hesitancy of investigators to publish negative data.

The present study, together with our earlier work on pathogen-activated SAR of cucumber (1,2) provides a counterpoint to the suggestion by others (12,13,17) that induced plant defenses typically are generalized, diffuse, and have reciprocal effects on both pathogens and insects. Induced plant resistance activated by herbivory is often characterized by low specificity against herbivores (11). Similarly, pathogen-activated SAR typically provides cross-protection from diverse plant pathogens (14). However, our results best support the hypothesis that plants may employ independent mechanisms of induced resistance against pathogens and herbivores. Although our studies suggest that use of pathogen-activated SAR in disease management will probably not result in any additional problems from insects or mites, they also provide little support for the suggestion that manipulation of pathogen-activated SAR will provide significant cross-protection from arthropod pests (12,17,18).

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