Potato Virus Y Transmission Reduced in an Aphid-Resistant Potato Species

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ABSTRACT

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Transmission of potato virus Y (PVY) by the green peach aphid, *Myzus persicae*, was examined on an aphid resistant potato species *Solanum berthaultii* and on susceptible cultivars of *S. tuberosum* in a series of open field, field cage, and greenhouse studies. In open field trials, PVY incidence at the end of the season was not significantly different for *S. berthaultii* compared with *S. tuberosum* 'Chippewa' when both species were exposed to aphid-infested source plants of *S. tuberosum*. However, in field cage trials and in the greenhouse, *S. tuberosum* experienced less PVY infection when exposed to a source plant of *S. berthaultii* compared with *S. tuberosum* exposed to a source plant of *S. tuberosum*. Both source plant and target plant had a significant effect on PVY spread in field cages. The influence of the PVY-infected source plant was consistently greater than the effect of target plant on PVY spread.

Additional key words: glandular trichomes, green peach aphid, PVY spread.

Several wild potato species have been reported to be resistant to the green peach aphid, *Myzus persicae* (Sulzer). In 1946, a Mexican species, *Solanum polyadenium* Greenm., was described as highly resistant to attack by *M. persicae* (1). Resistance was attributed to the action of an "oil" that accumulated on the tarsi of aphids (15). Gibson (4) later determined that glandular hairs on the surface of *S. polyadenium*, *S. berthaultii* Hawkes, and *S. tarijense* Hawkes were the source of an adhesive exudate that restricted aphid mobility.

Two types of glandular trichomes have been associated with aphid resistance in wild *Solanum* L. species. Type A hairs are 120–210 μm long with a tetralobulate, membrane-enclosed gland at their tips. Type B trichomes are considerably longer (600–950 μm), multicellular, and bear a single apical droplet of exudate. Type A exudate undergoes a rapid browning and hardening when the gland is ruptured, resulting in accumulation of exudate on insect tarsi and mouthparts. Aphids placed on *S. berthaultii* become immobilized as a result of exudate accumulation and eventually die of exhaustion and starvation (6,16,17). Type B exudate does not darken or harden, remaining clear and viscous (14). Recent potato breeding efforts have produced aphid-resistant interspecific hybrids of *S. tuberosum* × *S. berthaultii* bearing both types of glandular trichomes (19).

Although *S. berthaultii* has been shown to be highly susceptible to potato virus Y (PVY) infection after mechanical inoculation (9), Gunenc and Gibson (7) reported that glandular hairs on the foliage of *S. berthaultii* hindered departure of alate *M. persicae*, reduced probing, and decreased acquisition of PVY during brief access periods. The presence of type B exudate droplets on leaflet surfaces of *S. berthaultii* contributes to a modification of the probing behavior of *M. persicae* characterized by fewer probes, an increased preprobe time, and decreased total feeding compared with *S. tuberosum* L. cultivars (10).

We report here the results of greenhouse and field studies to examine the possibility that PVY spread is reduced in the aphid resistant species, *S. berthaultii*.

MATERIALS AND METHODS

General. Plants of *S. tuberosum* 'Chippewa' and 'Green Mountain' were propagated from certified seed tubers. Plants of *S. berthaultii* (PI 310927) were sprouted from botanical seed obtained from the Potato Introduction Station, Sturgeon Bay, WI. This accession is highly resistant to *M. persicae* (16) and bears type A and B glandular trichomes on its foliage. True seed was soaked for 24 hr in 1,500 ppm gibberellic acid to increase germination. Plants were grown in 25-cm-diameter plastic pots in a peat-vermiculite soil mix supplemented with controlled-release fertilizer. In the greenhouse, plants were grown under high intensity illumination of 32,000 lx at soil level. Photophase was 16 hr, temperatures fluctuated between 20 and 26 C, and relative humidity ranged between 60 and 90%.

A stock colony of *M. persicae* was initiated from a single alate collected from potato at Freeville, NY, in June 1981. The colony was maintained in a controlled environment chamber at 25 C, under a 16-hr photophase regime on a cultivar of *S. tuberosum*. In preparation for the studies described here, the proportion of alates in the colony was increased by rearing on excised leaves at high population densities.

A local isolate of PVY was maintained in tobacco, *Nicotiana tabacum* L., in the greenhouse.

Greenhouse trials. Replicates of 24 plants each of *S. tuberosum* 'Chippewa' and *S. berthaultii* were arranged in symmetrical arrays on benches in the greenhouse. In the center of each array, approximately 1,000 immature and adult apterous *M. persicae* and 50 adult alates were released on a single PVY-infected source plant of Green Mountain or *S. berthaultii*. Four weeks after infestation with *M. persicae*, pirimicarb was applied (0.02% a.i.) to eliminate the aphid population. Each treatment (source/target plant combination) was run separately during a period from October 1984 to March 1985. Treatments were as follows: (T) *S. tuberosum*; (B) *S. berthaultii*. T/T: Green Mountain source, Chippewa target; B/B: *S. berthaultii* source and target; T/B: Green Mountain source, *S. berthaultii* target; B/T: *S. berthaultii* source, Chippewa target.

Target plants were sampled and assayed for PVY infection at 5, 6, 7, and 8 wk after release of *M. persicae* on source plants by sap inoculation of *S. demissum* Lindl., a local lesion indicator plant. Leaf samples were individually ground with mortar and pestle, and the sap was rubbed onto a Carborundum-dusted leaf. Indicator plants were kept in the greenhouse and examined for lesion development 4 days after inoculation. Three replications of treatments T/T and B/B and two replications of T/B and B/T were completed. Final incidence of PVY was analyzed by two-way analysis of variance (ANOVA). A probability level (α) of 0.05 was used to test main effects (source and target plant).

Open field studies. Field plots of Chippewa and *S. berthaultii* were planted at the H. C. Thompson Vegetable Research Farm at Freeville, NY, during the 1984 growing season. Certified seed tubers of Chippewa were planted on 11 June 1984. Plants of *S. berthaultii* grown from true seed were transplanted at the 10–15-cm stage when the Chippewa tubers had sprouted and first appeared above the soil surface (18 June). Target plants were arranged in symmetrical square arrays of 24 plants spaced 1.8 m apart. Each array consisted of five rows of five plants each, the central position occupied by a PVY source plant. A total of six arrays was planted, consisting of three arrays (replications) of each of the target plant species. A source plant (Green Mountain) that had been mechanically inoculated with PVY in the greenhouse was transplanted to the center of each target plant array on 2 July and infested with 500 immature and adult apterous *M. persicae* from the stock colony. All plants were assayed for PVY infection on 5, 19, and 26 August and 9 September by sap inoculation of *S. demissum*. In addition, visual symptoms were evaluated on each target plant of each array weekly throughout the growing season. PVY infection was assessed by percent infection at the end of the season (9 September) and by comparing the area under disease progress curves (AUDPC) (8).

Field cage studies. A 2 × 2 factorial design was used in 1985 to determine the relative amount of PVY acquisition from and transmission to *S. berthaultii* and Green Mountain. The design consisted of two species of target plant arrays, two species of source plant, and two cages (replications) per target/source combination. Treatments were the same as those described above, i.e., T/T, B/B, T/B, and B/T.

Target plants in each treatment were exposed for 4 wk to a PVY-infected source plant in fiberglass screen cages (2.45 × 2.45 × 1.8 m tall). The experiment was repeated consecutively three times over the growing season. Because all target plants were initiated at the same time, the three experiments were conducted with plants (cohorts) of increasing age. Source plants were infected 3 wk before the start of each experiment and later placed in the center of the target plant array. Five hundred immature and adult apterous *M. persicae* were placed on each source plant immediately after placement in a target array.

Plants of both species were transplanted into 25-cm plastic pots in the greenhouse at Ithaca, NY, on 1 June 1985 when plants were 10 cm tall. Plants were then transferred to a screenhouse. The first and youngest group of plants (cohort) was deployed into field cages on 10 June 1985 and removed 4 wk later on 8 July. The second cohort was deployed on 9 July and removed on 6 August. The third, and oldest, cohort was deployed on 7 August and removed on 4 September. Each age cohort was treated with pirimicarb (0.02% a.i.) before removal from the field cages and again 1 wk later in the screenhouse to eliminate aphids. Mechanical transmission of PVY by leaf contact was prevented by tying the vines to stakes as plants increased in size. The first cohort was deployed with 16 plants per cage. Because of their greater size, only 12 plants of the second and third cohorts were deployed per cage to avoid foliar contact.

Aphids were counted weekly after infestation of the field cages. Six target plants in each cage were examined for 30 sec each, starting with lower leaves and progressing up the stem. A total of 12 counts per sample date were made for each source plant/target plant combination. All aphid species were included in the counts. Aphid populations consisted primarily of *M. persicae* with smaller numbers of the potato aphid, *Macrosiphum euphorbiae* (Thomas), foxglove aphid, *Aulacorthum solani* (Kaltenbach), and buckthorn aphid, *Aphis nasturtii* Kaltenbach. Disposable latex gloves were used and discarded after examination of each plant to prevent PVY transmission. Mean number of aphids per 30-sec count ± standard error of the mean ($N = 12$) was plotted over time for each of the source plant/target plant treatments.

After removal from field cages, all plants were assayed weekly for PVY. The final incidence of PVY infection and aphid counts collected on the last sample date (week 4 after infestation) were analyzed by two-way analysis of variance to determine the influence of source plant and target plant on PVY transmission and total aphid population. A probability level (α) of 0.05 was used to test main effects (source and target plant) and for interaction between main effects.

RESULTS

Greenhouse studies. In the greenhouse, the greatest amount of PVY spread (39%) occurred when Chippewa was both source and target, whereas the least amount of spread (6%) occurred when *S. berthaultii* was both source and target. Greater disease spread (30%) occurred from a Chippewa source to an *S. berthaultii* target than occurred from an *S. berthaultii* source to a Chippewa target (17%) (Table 1). Factorial analysis showed that target species did not influence PVY transmission ($\alpha = 0.05$, two-way ANOVA). Source species, however, had a pronounced effect on amount of PVY spread ($P < 0.001$).

Open field trials. Disease progress curves were constructed for trials conducted during the summer of 1984 (Fig. 1). Final PVY incidence did not differ significantly between *S. berthaultii* and Chippewa target plants ($P = 0.12$ Student's t test). The shape of the disease progress curves is similar. A plateau occurred during weeks 5 through 8 followed by a dramatic increase in infection for both species. During the plateau period, PVY incidence in *S. berthaultii*

TABLE 1. Final potato virus Y (PVY) incidence as percent target plants infected for four source plant/target plant combinations (T = *Solanum tuberosum* 'Green Mountain', B = *S. berthaultii* PI 310927)[a]

Source plant/target plant	PVY incidence (%)	S.E.[b]	N[b]
T/T	38.89	3.67	3
T/B	29.98	9.15	2
B/T	16.67	4.16	2
B/B	5.55	2.78	3

[a] Target plants exposed for 4 wk in the greenhouse at Ithaca, NY, 1985.
[b] S.E. = Standard error of the mean, N = number of replications.

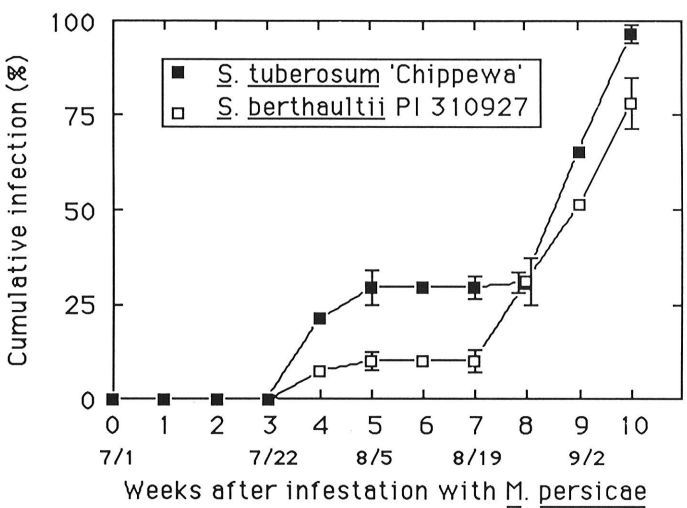

Fig. 1. Cumulative potato virus Y (PVY) infection as a percent of target plants exposed to a PVY-infected *Solanum tuberosum* 'Green Mountain' source plant in the field at Freeville, NY, 1984.

was significantly less than that in Chippewa ($P = 0.016$, Student's t test). The calculated AUDPC value for Chippewa was 1.54-fold greater than that for *S. berthaultii*.

Field cage experiments. Aphid population curves were constructed for each of the three age cohorts (Fig. 2). The aphid final infestation (counted 4 wk after infestation) for each age cohort was analyzed by two-way ANOVA. Influence of target plant species on final aphid populations was large during all three age cohorts ($P < 0.001$). Source plant species affected final aphid populations only for the second age cohort ($P < 0.01$). However, the mean square for target plant effect was approximately four times greater than that for source plant effect.

Aphid populations declined during the third and fourth weeks after initial infestation of the third age cohort (Fig. 2C) resulting from a high incidence of parasitism by hymenopteran parasitoids coupled with an epizootic of entomogenous fungal pathogens that developed in late August. However, the general pattern established during the first two age cohorts was repeated for the third. Source plant had no effect on final aphid populations ($\alpha = 0.05$), and the influence of target plant was large ($P < 0.001$).

Disease progress curves were constructed for the first and second cohorts (Fig. 3). The mean square for source plant effect ($P < 0.005$) was approximately three times larger than the mean square for target plant effect ($0.01 < P < 0.05$) on final disease incidence. Interaction between source plant and target plant effects was not significant ($\alpha = 0.05$, two-way ANOVA). PVY incidence was low during the third cohort. Some infection was detected among third cohort *S. berthaultii* (12.5% for T/B and 8.3% for B/B), but the difference was not significant (Student's t test, $\alpha = 0.05$). Current season infection was not detected in third cohort Green Mountain by the *S. demissum* test, probably because of mature plant resistance and plant senescence. Tubers of the third cohort were harvested, sprouted in the greenhouse, and assayed for PVY infection in January 1986. No infection was detected in third cohort Green Mountain tubers.

DISCUSSION

The wild potato species *S. berthaultii* was highly resistant to infestation by aphids in field cages compared with Green Mountain. Source plant species affected final aphid populations on target plants in field cages in only one case (T/T vs. B/T, second age cohort). The T/T treatment cage had 13 susceptible Green Mountain plants compared with 12 in the B/T treatment cages. This additional target plant may have encouraged overall aphid population development. It is clear, however, that the major determinant of aphid populations in the field cages was target plant species.

Aphid populations were greatest during the second age cohort and least during the third age cohort. The incidence of PVY infection for the first and second cohorts was similar, probably due to the offsetting effects of an increased vector population (inoculum pressure) and a decreased target plant susceptibility to

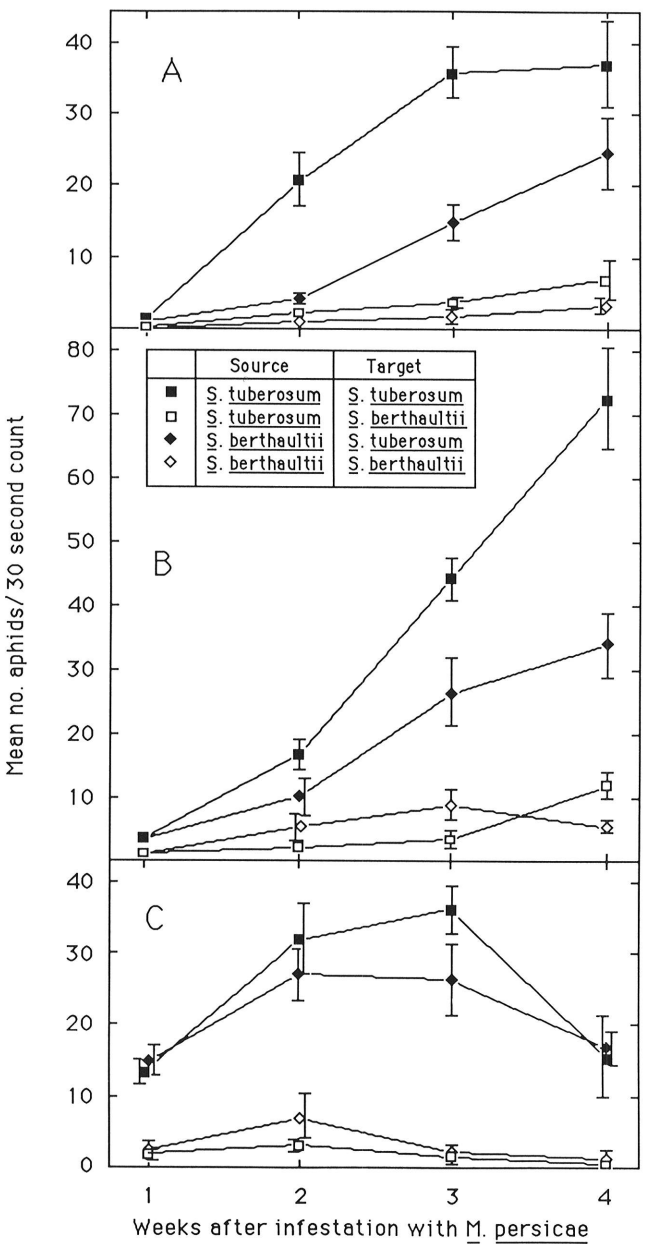

Fig. 2. Mean number of aphids per 30-sec count on target plants for four source plant/target plant species combinations in field cages. Potato virus Y source plants were initially infested with 500 apterous *Myzus persicae* on 10 June, 9 July, and 7 August 1985 for **A, B,** and **C,** respectively. Bars are standard error of the mean ($N = 12$).

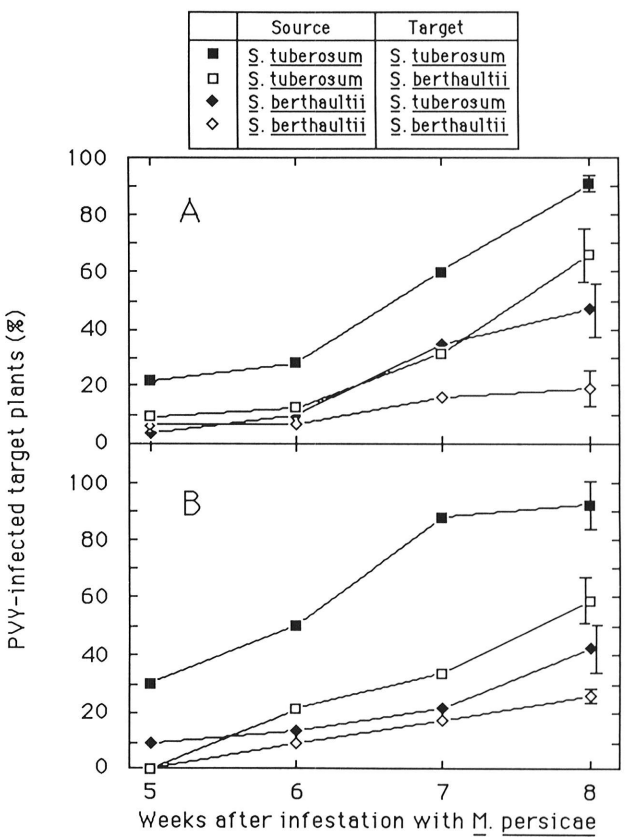

Fig. 3. Cumulative potato virus Y (PVY) infection as a percent of target plants exposed to a PVY-infected source plant in field cages at Freeville, NY. *Myzus persicae* and PVY source plant deployed on 10 June and 9 July 1985, respectively, for **A** and **B.** Bars are standard error of the mean ($N = 2$).

PVY (mature plant resistance) during the second cohort. Declining aphid populations due to natural enemies together with declining target plant susceptibility to PVY may have contributed to low levels of PVY infection for the third cohort.

Incidence of PVY infection at the end of the season was equivalent in open field plots of *S. berthaultii* and Chippewa. However, *S. berthaultii* had less early season infection as reflected in a lower AUDPC value. Results of the greenhouse studies suggested that, to a large extent, any resistance to PVY inoculation (target plant effect) in the open field plots of *S. berthaultii* was overwhelmed by proximity to large numbers of PVY-infected Chippewa inoculum sources later in the season.

In the greenhouse, target plant had a small effect ($P = 0.0628$) on PVY spread. Both source and target plant significantly influenced PVY spread in field cages. In agreement with the results of the greenhouse studies, influence of the source was greater than that of the target. These data support the view that removal or reduction of virus sources (inoculum) is the most effective strategy for limiting virus spread (20).

Gunenc and Gibson (7) reported that glandular hairs on the foliage of *S. berthaultii* hindered departure of alate *M. persicae*. In addition, *S. berthaultii* can be expected to produce fewer alates than *S. tuberosum* because of the absence of a crowding stimulus in the small aphid populations characteristic of the former species. The behavioral modification of aphid probing attributed to type B exudate of *S. berthaultii* (10) is most likely responsible for the target plant effect observed here. With large vector populations, however, the probability increases that at least one viruliferous aphid will conduct a successful inoculation probe within the retention period of PVY. Apparently, the reduced probability that an alate aphid is able to alight on an infected *S. berthaultii* plant, acquire PVY, and then return to flight plays a larger role in determining spread of PVY than does the effect of glandular trichomes on probing behavior of a viruliferous aphid once it has landed on a healthy target plant. Breeding for resistance to aphids as vectors of nonpersistent viruses should emphasize the mechanical properties of glandular trichomes that entrap aphids and hinder departure of alates. Primary spread of PVY from a remote source will be reduced by factors that interfere with probing after the vector has alighted on a potato plant. Secondary spread within a field will be affected by availability of virus to potential vectors and by size of the vector population. We have demonstrated that PVY acquisition from and transmission to *S. berthaultii* by *M. persicae* is reduced compared with *S. tuberosum*.

Allomonal compounds may play a role in both entrapment of aphids and modification of probing behavior. However, little evidence is available to indicate that volatile compounds are effective in deterring probing by *M. persicae*. Gibson and Pickett (5) suggested that volatile trans-β-farnesene (EβF) in glandular trichome exudate of *S. berthaultii* repelled *M. persicae* and might reduce virus spread. Other investigations into the effect of EβF on host selection by *M. persicae* and the inhibition of EβF activity by other common plant sesquiterpenes cast doubt on the utility of EβF as a means to decrease nonpersistent virus spread. Phelan and Miller (13) reported that *M. persicae* were not deterred from making test probes in the presence of EβF, although settling (uninterrupted probing of more than 10 min duration) was reduced compared with an untreated control. Increased restlessness coupled with an increased frequency of probes of short duration should enhance rather than retard nonpersistent virus transmission. Furthermore, Dawson et al (2) demonstrated that β-caryophyllene, a ubiquitous plant sesquiterpene, inhibited the behavior-altering properties of EβF. β-Caryophyllene occurs in much greater concentrations in glandular exudate of *S. berthaultii* than EβF (5; D. A. Avé, *personal communication*). To date, the simplest explanation for the reduced vector potential of *M. persicae* on *S. berthaultii* and the adverse influence of glandular trichomes on probing behavior is the mechanical interference with probing by trichome exudate and reduced ability of alates to disperse from the adhesive foliage of these plants.

Transfer of glandular trichomes from *S. berthaultii* into commercial cultivars has great potential for incorporating resistance to several of the major insect pests of potato, i.e., aphids (17), Colorado potato beetle (3,19), fleabeetles (18), and leafhoppers (16,18). The purpose of the epidemiological studies presented here was to assess the effect that eventual release of a cultivar with glandular trichome-based resistance might have on epidemiology of PVY, the major nonpersistent aphid-borne virus of potatoes. Existence of immunity (12) to PVY might seem to make the issue irrelevant. However, glandular trichomes will provide a first line of defense, reducing inoculum pressure and thereby the possibility of the occurrence of a new, resistance-breaking virus strain.

M. persicae is more economically important as a virus vector than as a foliar parasite of potatoes (11). Breeding for resistance to aphids in potatoes should ideally focus on resistance mechanisms that interfere with the insects' ability to transmit virus. We believe that current efforts to produce new potato cultivars incorporating glandular trichomes as a defense against aphids and other insect pests will also be effective in reducing PVY transmission.

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