Quantification of Disease Resistance that Reduces the Rate of Tobacco Etch Virus Epidemics in Bell Pepper

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ABSTRACT


Tobacco etch virus, caused by tobacco etch virus (TEV), was monitored in three pepper genotypes over a 3-yr period at two locations in northeast Georgia. The three genotypes were Yolo Wonder B (susceptible), Tambel 2 (moderately resistant), and Asgrow-XPH-5021 (moderately resistant). The effect of host resistance on the development of TEV epidemics was indicated by the following results: 1) final TEV incidence was 45% less resistant genotypes, 2) relative area-under-the-disease-progress curve was 42-68% less in resistant genotypes, 3) apparent infection rate was about 50% less in resistant genotypes, and 4) time for TEV disease incidence to reach 50% was delayed 23-37 days in resistant genotypes. All four methods of quantifying the effect of host resistance on TEV disease progression were highly correlated. The consequence of the rate-reducing resistance in Tambel 2 and Asgrow-XPH-5021 was to decrease fruit yield (average of 24%), fruit weight (14%), and number of fruit when compared to susceptible Yolo Wonder B.

Tobacco etch virus (TEV), a member of the potyvirus group that is transmitted nonpersistently by aphids, causes severe epidemics in bell pepper (Capsicum annuum L.) throughout the southeastern United States and also in Arizona, California, Texas, and Mexico (2,12,17,20,31,35). In North Carolina, disease incidence approaches 75% each season (17). Since 1984, detailed surveys have shown that TEV incidence exceeded 90% at harvest in virtually all pepper fields in northeast Georgia (2,20). A yield loss model based on time of infection in relation to crop development has been developed, and actual yield losses in experimental plots ranged from 23 to 50% (18). Early infection reduces fruit set as well as fruit size and weight, whereas late-season infection has little effect on fruit set or fruit weight. Thus, control strategies that delay TEV infection could greatly limit yield losses experienced by growers.

Resistance that reduces the epidemiologic rate of diseases caused by fungi has been shown to play an important role in Integrated Pest Management programs (3,27,28,34). Slow wilting (24,26) and slow rusting (11) are terms that were coined to describe the resistance of host genotypes that possess this form of resistance to fungal pathogens. An analogous type of resistance to plant viruses may be found in hosts that restrict viral infection at different stages of development: virus inoculation, virus multiplication, virus translocation within plants, and virus acquisition by vectors (5,6,8-10,13-15,23,32,36). The terms “partial resistance” (15,23,27), quantitative resistance (8,9), and field resistance (9) have been employed to describe resistances of these types, but their epidemiological (rate-reducing) effects on the rate of epidemic development in the field have not often been quantified in plant virus pathosystems. It is suspected that virus restriction is controlled polygenically, which can have several advantages over the monogenically controlled resistance that is currently used in breeding programs to control virus diseases (7,22). Monogenic resistance can “break down” when new viral (2,13) or fungal strains (29,34) increase in frequency, whereas rate-reducing resistance in fungal pathosystems has been shown to remain stable and effective against all pathogen races or strains (9,29,30).

Kuhn et al (10) identified multiple levels of resistance to TEV in bell pepper genotypes. Two genotypes, Tambel 2 and Asgrow-XPH-5021, have a moderate level of resistance, which appeared to be effective under field conditions. However, the effect of these two genotypes on TEV epidemics has not been determined. The specific objectives of this study were to quantify the rate-reducing effect of moderately resistant pepper genotypes on TEV disease progress and yield compared with a susceptible pepper genotype.

MATERIALS AND METHODS

Experimental genotypes. Three bell-type pepper genotypes were used: Yolo Wonder B, a cultivar grown commercially in northeast Georgia, is considered highly susceptible to TEV; initial symptoms appear 3-5 days after inoculation, viral antigen concentration is high within 7 days, and symptoms are severe (mosaic, leaf curling, stunting) (10). Tambel 2, is a cultivar developed at Texas A&M University (32), and Asgrow is an experimental hybrid (XPH-5021) produced by Asgrow Seed Company (Kalamaoo, MI); both have moderate resistance to TEV; initial symptoms occur 2-3 wk after inoculation, viral antigen concentrations are low to medium for 2-3 wk, and after inoculation, only moderate symptoms develop (mosaic, little or no stunting) (10).

Field experiments. Experiments were conducted at two locations: Northeast Georgia Mountain Station, near Blairsville, where TEV epidemics occur annually, and University of Georgia Plant Sciences Farm, near Athens, where TEV epidemics have not been observed. At both sites, infection of test plants depended on natural sources of inoculum and vectors.

Pepper seedlings grown in 7-cm paper cups in the greenhouse were fertilized weekly with Peter's 20-20-20 (N-P-K) soluble fertilizer (6 g/L of H₂O). About 6 wk after seeding, soluble fertilizer and the fungicide Terrachlor 75% wettability powder (pentachloronitrobenzene, 2.7 g a.i./L of H₂O) were added to the soil at the time of hand transplanting. Before transplanting,
experimental plots were fertilized (broadcast) with 120 kg/ha of total nitrogen. Thereafter, cultivation and irrigation were applied as needed.

Each experimental unit consisted of a single pepper genotype planted in a plot 6 rows wide with 24 plants (30 cm apart)/row. The overall plot size was 6.0 × 8.0 m. Plots were replicated four times and arranged in a randomized complete block design. Plots were spaced 3.0 m apart both within and between blocks. In Blairsville, pepper genotypes were transplanted on 27 May, 3 June, and 28 May in 1985, 1986, and 1987, respectively. Peppers were transplanted in Athens on 23 May, 6 June, and 1 June in 1985, 1986, and 1987, respectively.

Disease progress curves. TEV incidence, expressed as the percent of plants exhibiting TEV symptoms, was recorded weekly for each plot. The visual ratings began the week of 23 June, 22 June, and 21 June and continued until the time of the first harvest which was on 11 August, 1 September, and 9 August, in 1985, 1986, and 1987, respectively. Disease progress curves (TEV incidence versus time) were plotted for each replicate of each genotype for each year. These curves provided the basis for the four other epidemiology measurements: final TEV incidence, relative area under the disease progress curve (RAUDPC), apparent infection rate (r), and time to reach 50% TEV incidence (T50). A computer software program (3) was written to calculate an RAUDPC for each pepper genotype for each year. The RAUDPC for each resistant genotype was then expressed as a percentage of the RAUDPC calculated for Yolo Wonder B. The apparent infection rate, expressed in units per day, was calculated by first converting disease incidence values to a proportion on a scale of 0 to 1 and then transforming the data to logits, $\ln \left[ Y / (1 - Y) \right]$, where $Y$ represents the proportion of infected plants and $1 - Y$ represents the proportion of healthy plants remaining in the plot. Logit values ($y$) were regressed against time ($x$) to obtain a regression line, the slope of which is the apparent infection rate $r$ (29). Time for TEV disease incidence to reach the 50% level was calculated from the regression equation used to obtain the apparent infection rate. The equation used to derive the $T_{50}$ was: $T_{50} = \log(Y_0) / r$ in which $Y_0$ is the estimated disease proportion at the first date of assessment, and $r$ is the apparent infection rate.

Fruit yield. The center four rows of each plot were harvested beginning when fruit turned brown to red in color and continued until the majority of the pepper plants stopped bearing fruit. The harvests were conducted every 10–14 days in 1985 and weekly in 1986 and 1987. In addition to total yield (kg/ha), two yield components were measured: number of fruit per plot and average fruit weight.

Data analysis. Analysis of variance was used to determine if final disease incidence, RAUDPC, apparent infection rate, $T_{50}$, fruit yield, and yield components, were affected by genotype. The Waller-Duncan K-ratio t-test was then used to determine if significant differences existed among genotypes ($k = 100$; $P = 0.05$). Pearson’s correlation coefficients were calculated to test the strength of relationships among the methods used to quantify TEV epidemics (25).

ELISA. Enzyme-linked immunosorbent assays (ELISA) were conducted, as described previously (4), to confirm serologically that field plants with viruslike symptoms were infected with TEV. On each sampling date, a newly expanded leaf was collected from a stem terminal of each plant in the center two rows of each plot (about 50 plants). Each leaf (0.3–0.5 g) was extracted in 3.0–5.0 ml of ELISA extraction buffer using a motor-driven leaf press. Plants were considered TEV positive if the 410-nm absorbance reading (Dynatech Microelisa MR590, Dynatech Instruments, Inc., Santa Monica, CA) was at least 0.1 and also at least two times greater than negative controls (leaves from healthy pepper plants).

RESULTS

Disease progress curves. Symptoms of TEV were first observed on day of year 179, 178, and 170 in 1985, 1986, and 1987, respectively (Fig. 1). Only a very few diseased plants of both susceptible and resistant genotypes were found during these initial observations. In 1985 and 1987, disease incidence was significantly greater in susceptible Yolo Wonder B plots by 1 wk after the first observation compared with TEV incidence levels in resistant genotypes, while significant differences between susceptible and resistant genotypes did not occur until the week 4 in 1986 (Fig. 1). In all 3 yr, disease incidence increased more rapidly in Yolo Wonder B than in the resistant genotypes. Disease progress was more rapid in Tambel 2 than in Asgrow in 1986, but it was similar in both resistant genotypes in 1987 (Fig. 1). TEV disease incidence in pepper genotypes planted near Athens never reached incidence levels greater than 1% in any of the three growing seasons; therefore, disease progress curves were not obtained.

Fig. 1. Tobacco etch virus disease progress curves for Yolo Wonder B (YWB), Tambel 2, and Asgrow XPH-5021 (Asgrow) during three growing seasons (1985–1987) in field plots near Blairsville, GA. Transplants were planted in the field on 27 May, 3 June, and 28 May in 1985, 1986, and 1987, respectively. Day 170 = June 19. Each point is the mean of four replications.
Each year, final TEV incidence was near 100% in Yolo Wonder B and always significantly greater than in Asgrow and Tambel 2 (Table 1). The final disease incidence in Asgrow was almost the same in each year (range of 48–52%). Final incidence in Tambel 2 was greater in 1986 than in 1987. The RAUDPc values for resistant Asgrow and Tambel 2 were lower than susceptible Yolo Wonder in each of the three test years, ranging from 32 to 56% (Table 1).

Disease progress data were fitted to linear, monomolecular, logistic, and Gompertz growth models. Based on coefficients of determination ($R^2$), standard errors of the estimate, and examination of residuals, the logistic model was found to provide the best fit and, therefore, this model was used to quantify disease progression to make comparisons among the three genotypes (Fig. 2). Apparent infection rates were relatively similar within pepper genotypes for each of the test years ranging from 0.15 to 0.18 per day for susceptible Yolo Wonder B and from 0.06 to 0.09 per day for the two moderately resistant genotypes (Table 1). Thus, Tambel 2 and Asgrow reduced the apparent infection rate by 40–67% relative to susceptible Yolo Wonder B. When $T_{90}$ values for both genotypes and years were considered, $T_{90}$ for the two resistant genotypes was delayed 23–37 days compared with Yolo Wonder B (Table 1).

Fruit yield. At Blairsville, Tambel 2 and Asgrow produced greater fruit yields (average of 23.6% higher) and average fruit weight (average of 13.9% higher) in each of the three test years than Yolo Wonder B (Table 2). More fruit/plot were produced by the two resistant genotypes in 1986 and by Asgrow only in 1987 than by Yolo Wonder B. No differences involving fruit measurements were observed between Tambel 2 and Asgrow in any of the 3 yr.

At Athens, all yield and yield component measurements were similar among the three test genotypes, with one exception (Table 2). The average fruit weight of Yolo Wonder B was greater than Tambel 2 and Asgrow in 1987.

Correlation analyses. There were significant ($P<0.05$) to highly significant ($P<0.01$) correlations among the variables used to quantify TEV epidemics (Table 3). In 1985, 1986, and 1987 correlation coefficients between final TEV incidence and RAUDPc were 0.90 or greater. High correlation coefficients ($P<0.01$) were observed also between $T_{90}$ and final TEV incidence and $T_{90}$ with RAUDPc (Table 3).

ELISA. For all three test genotypes, serological tests in 1985 and 1986 demonstrated a high correlation (0.94, $P<0.01$) between visual observations and ELISA evaluations of the proportion of plants infected with TEV. Early in the growing season when the first plants were observed with symptoms, some plants with symptoms gave negative ELISA readings. However, the readings were positive 1 wk later.

ELISA tests for viruses other than TEV were conducted periodically during each of the three test years. Similar to other studies (2,10) in the same geographical area, only a few plants (<0.2%) were found with other viruses, particularly cucumber mosaic virus (CMV) and tomato spotted wilt virus. Thus, there was essentially no interference with other viruses in our TEV epidemiological studies.

![Logit TEV incidence vs. Day of Year](image.png)

**Fig. 2.** Logit lines obtained from tobacco etch virus disease progress curves for Yolo Wonder B (YWB), Tambel 2, and Asgrow XPH-5021 (Asgrow) during three growing seasons (1985–1987) in field plots near Blairsville, GA. Transplants were planted in the field on 27 May, 3 June, and 28 May in 1985, 1986, and 1987, respectively. Day 170 = June 19. Disease incidence values >5% were transformed to logits and regressed against day of year.

**TABLE 1.** Effect of bell pepper genotype on final incidence of tobacco etch virus (TEV) incidence at time of harvest, relative area under the disease progress curve (RAUDPc), time for TEV incidence to reach 50% ($T_{90}$), and apparent infection rate for TEV epidemics in Blairsville, 1985–1987

<table>
<thead>
<tr>
<th>Year</th>
<th>Genotype</th>
<th>Final TEV incidence</th>
<th>RAUDPc</th>
<th>Apparent infection rate</th>
<th>Incidence $T_{90}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1985</td>
<td>Yolo Wonder B</td>
<td>97.2 a</td>
<td>100.0 a</td>
<td>0.15 a</td>
<td>205 b</td>
</tr>
<tr>
<td></td>
<td>Asgrow XPH-5021</td>
<td>52.4 b</td>
<td>55.7 b</td>
<td>0.09 b</td>
<td>228 a</td>
</tr>
<tr>
<td></td>
<td>SED</td>
<td>5.8</td>
<td>5.6</td>
<td>0.01</td>
<td>1.8</td>
</tr>
<tr>
<td>1986</td>
<td>Yolo Wonder B</td>
<td>98.6 a</td>
<td>100.0 a</td>
<td>0.16 a</td>
<td>221 c</td>
</tr>
<tr>
<td></td>
<td>Tambel 2</td>
<td>73.4 b</td>
<td>46.4 b</td>
<td>0.08 b</td>
<td>246 b</td>
</tr>
<tr>
<td></td>
<td>Asgrow XPH-5021</td>
<td>51.4 c</td>
<td>32.3 c</td>
<td>0.07 b</td>
<td>255 a</td>
</tr>
<tr>
<td></td>
<td>SED</td>
<td>3.7</td>
<td>4.8</td>
<td>0.02</td>
<td>3.2</td>
</tr>
<tr>
<td>1987</td>
<td>Yolo Wonder B</td>
<td>99.8 a</td>
<td>100.0 a</td>
<td>0.18 a</td>
<td>195 c</td>
</tr>
<tr>
<td></td>
<td>Tambel 2</td>
<td>48.5 b</td>
<td>39.2 b</td>
<td>0.06 b</td>
<td>230 b</td>
</tr>
<tr>
<td></td>
<td>Asgrow XPH-5021</td>
<td>47.7 b</td>
<td>39.5 b</td>
<td>0.06 b</td>
<td>232 b</td>
</tr>
<tr>
<td></td>
<td>SED</td>
<td>4.4</td>
<td>4.3</td>
<td>0.02</td>
<td>3.8</td>
</tr>
</tbody>
</table>

Within each column and each year, means followed by a different letter are significantly different using the Waller-Duncan $K$-ratio test ($P<0.05$).

Standard error of difference between means.
TABLE 2. Yield, number of fruit, and average fruit weight of pepper genotypes evaluated in field plots near Athens and Blairsville, GA, 1985–1987

<table>
<thead>
<tr>
<th>Genotype</th>
<th>Blairsville*</th>
<th>Athens*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yield (kg/ha × 10^3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yolo Wonder B</td>
<td>16.9 b</td>
<td>10.9 b</td>
</tr>
<tr>
<td>Tambell 2</td>
<td>...</td>
<td>13.2 a</td>
</tr>
<tr>
<td>Asgrow XPH-5021</td>
<td>19.7 a</td>
<td>14.3 a</td>
</tr>
<tr>
<td>Number of fruit/plot</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yolo Wonder B</td>
<td>467 a</td>
<td>317 b</td>
</tr>
<tr>
<td>Tambell 2</td>
<td>...</td>
<td>351 a</td>
</tr>
<tr>
<td>Asgrow XPH-5021</td>
<td>426 a</td>
<td>364 a</td>
</tr>
<tr>
<td>Average fruit weight (g)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yolo Wonder B</td>
<td>123.0 b</td>
<td>116.8 b</td>
</tr>
<tr>
<td>Tambell 2</td>
<td>...</td>
<td>128.0 a</td>
</tr>
<tr>
<td>Asgrow XPH-5021</td>
<td>150.0 a</td>
<td>123.4 a</td>
</tr>
</tbody>
</table>

* Tobacco etch virus epidemics were observed each year; TEV incidence always exceeded 99% in susceptible Yolo Wonder B.
* Tobacco etch virus incidence never exceeded 1%.
* Within each column of each of the three measurements, means followed by a different letter are significantly different using the Waller-Duncan k-ratio test (P = 0.05).

TABLE 3. Correlations among epidemiological measurements used to quantify tobacco etch virus (TEV) epidemics in Blairsville, GA, 1985–1987

<table>
<thead>
<tr>
<th>Year</th>
<th>Method of measurement</th>
<th>r*</th>
<th>RAUDPC*</th>
<th>T90</th>
</tr>
</thead>
<tbody>
<tr>
<td>1985</td>
<td>Final TEV incidence</td>
<td>0.88*</td>
<td>0.81*</td>
<td>-0.98*</td>
</tr>
<tr>
<td></td>
<td>r</td>
<td>0.75*</td>
<td>-0.91*</td>
<td>-0.84*</td>
</tr>
<tr>
<td></td>
<td>RAUDPC</td>
<td>0.91*</td>
<td>-0.87*</td>
<td>-0.92*</td>
</tr>
<tr>
<td>1986</td>
<td>Final TEV incidence</td>
<td>0.87*</td>
<td>0.93*</td>
<td>-0.89*</td>
</tr>
<tr>
<td></td>
<td>r</td>
<td>0.91*</td>
<td>-0.87*</td>
<td>-0.92*</td>
</tr>
<tr>
<td></td>
<td>RAUDPC</td>
<td>0.87*</td>
<td>0.99*</td>
<td>-0.88*</td>
</tr>
<tr>
<td>1987</td>
<td>Final incidence</td>
<td>0.87*</td>
<td>0.99*</td>
<td>-0.88*</td>
</tr>
<tr>
<td></td>
<td>r</td>
<td>0.87*</td>
<td>-0.88*</td>
<td>-0.98*</td>
</tr>
</tbody>
</table>

* Apparent infection rate.
* Relative area under the disease progress curve.
* Time for TEV incidence to reach 50%.
* Significantly different from 0 (P ≤ 0.01).

DISCUSSION

Disease progress curves indicated that epidemics at Blairsville in susceptible Yolo Wonder B progressed slowly at first, then increased rapidly, after which epidemics slowed as fewer healthy plants remained to be infected. Epidemics that progress in this fashion are best described by a logistic (or similar) growth function and have been termed compound interest or polycyclic diseases (28,34). We found the logistic model to provide the best fit to quantify TEV disease progression in bell pepper and, therefore, we used this model to compare genotypes. Madden et al (16) also found that the logistic model provided the best fit to describe the temporal increase of combined incidence data for TEV and tobacco vein mottle virus (TVMV) in tobacco.

The logistic growth pattern of TEV disease progression indicates that infected pepper plants within each plot were contributing to new infected plants (28,34). To lessen the effects of epidemics that increase logistically, it is important to reduce the rate of plant-to-plant spread of the virus within the field, thereby allowing more time for plants to set and produce marketable fruit. A study with watermelon showed that a 5- to 20-day delay in the onset of watermelon mosaic epidemics resulted in a substantial increase in yield (1), while Nutter et al (19) showed that a reduction in the apparent infection rate of barley stripe mosaic virus in Dickson barley also significantly increased yield. The moderately resistant pepper genotypes that reduced the apparent infection rate of TEV in this study also provided a substantial benefit in terms of pepper yield and quality. A 40–67% reduction in the apparent infection rate resulted in a 23- to 37-day delay in the time to reach 50% TEV disease incidence.

Correlations were found between all epidemiological measurements used to quantify the effect of host resistance on TEV epidemics in the field. For the purpose of screening a large number of pepper genotypes for rate-reducing resistance to TEV in the field, determining final TEV incidence would be the simplest method since a single visual rating of a large number of genotypes could be made late in the growing season. Final disease incidence values are often considered to represent the summation of virus-host-vector-environment interactions over the course of a season (29,34). However, our study shows that TEV incidence data to compare genotypes could also be obtained over a period of several weeks before harvest since the greatest differences in TEV incidence among genotypes occurred when TEV incidence was between 50 and 95% in Yolo Wonder B. Since r, RAUDPC, and T90 were also positively correlated with one another, genotypes found to reduce r relative to susceptible genotypes should also reduce RAUDPC and the time to reach 50% TEV incidence. Therefore, each of these measurements could be used to provide quantitative information concerning the effect of disease control tactics on TEV epidemics. Each of these types of measurements may better lend themselves to specific applications. For example, Lecoq and Pitrat (14) developed a protective effect index, which represented the delay in days for 50% of field-grown muskmelon plants to become infected with CMV in treated plots compared with nontreated controls while Nutter et al (18) have used RAUDPC as a means to quantify losses due to TEV.

TEV affected pepper yield by reducing both the average weight of the fruit and the number of fruit. The effect of TEV on fruit weight is especially important because fruit are graded according to size and shape before being sold and a lower grade will result in lower profits. When compared to Yolo Wonder B, TEV epidemics were sufficiently delayed in Tambell 2 and Asgrow to allow the plants to produce more marketable fruit. At the Athens location where TEV epidemics have not been observed, fruit yield of Yolo Wonder B, Tambell 2, and Asgrow did not differ. In fact, in 1987 fruit weight was less in the resistant genotypes than in Yolo Wonder B, a reversal of the ranking of genotypes for fruit weights in all three test years at the Blairsville location where TEV was prevalent. These Athens data support the view that TEV is mainly responsible for the fruit yield loss at Blairsville.

TEV epidemics progressed similarly in 1985 and 1987. The delay in the initial occurrence of TEV and the time to reach 50% incidence in 1986 may have been the result of a reduced aphid population or reduced aphid activity caused by severe dry weather during May and June (20). Pepper growth was slow during this period and possible reduced virus replication and transmission may also have contributed to a delay in TEV epidemics.

Under laboratory/greenhouse conditions, Kuhn et al (10) found that Asgrow was somewhat more resistant than Tambell 2. The current field studies do not support a difference in the rate-
reducing effects between the two genotypes. Fruit yield and most disease progress measurements were similar for the two genotypes in each of the three test years. In 1986, however, differences observed in final disease incidence, RAUDPC, and 29 incidence indicated Asgrow might be more resistant than Tamlor 2, perhaps because of the delay in 29.

Host resistance to aphid vectors is another important factor that could slow virus epidemics. Although, similar numbers of Myzus persicae and total aphids were counted on yellow sticky traps placed in field plots of Yolo Wonder B, Tamlor 2, and Asgrow throughout the three growing seasons, this suggests only that there were no differences for aphid preference among pepper genotypes (20). Although resistance to aphid vectors in Asgrow XPH-5021 and Tamlor 2 remains a possibility, resistance to aphid vectors, by itself, has not been found to effectively reduce the spread of nonpersistently transmitted viruses (23).

In breeding programs, the effect of host genotype on resistance to plant viruses is frequently studied at the individual plant level and such studies usually consist of a limited number of test plants of a single genotype. Such programs tend to select for an extreme level of resistance (10), which is often monogenic and strain-specific (7, 22). Host genotypes that can be infected but exhibit reduced symptom severity may be discarded without full consideration for their epidemiological effects at the population level in the field.

In this study, quantification of TEV epidemics showed that genotypes Tamlor 2 and Asgrow, which possess moderate levels of resistance, were effective in reducing the apparent infection rate of TEV epidemics; therefore, this resistance can be termed rate-reducing resistance (11). Resistance that reduces the rate of epidemics has been found to be effective against all pathotypes in other disease pathosystems and should result in reduced selection pressure for resistance-breaking strains (21, 29, 30, 34). Because horticulturally acceptable pepper cultivars with extreme resistance to TEV are not readily available, it would be desirable to screen current cultivars for rate-reducing resistance that might be effective alone or in conjunction with other control strategies such as reflective plastic mulch (1).

LITERATURE CITED


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