Multiple Levels of Resistance to Tobacco Etch Virus in Pepper

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ABSTRACT

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Resistance to tobacco etch virus (TEV) was identified in pepper genotypes by comparing symptom severity; incubation period; viral antigen concentration, quantified by enzyme-linked immunosorbent assay (ELISA); and ability to become infected relative to the reaction of the susceptible Yolo Wonder B. Two genotypes, FL-XVR-3-25 and GA-C44-V22, exhibited extreme resistance under greenhouse conditions: no symptoms developed, and the virus could not be detected by ELISA or infectivity tests in either mechanically inoculated or uninoculated leaves. Under field conditions, however, 50–85% of the FL-XVR-3-25 plants developed a mild disease (mild chlorosis and leaf roll symptoms), and the TEV antigen could be detected in less than 15% of the plants with symptoms. A mild mottle developed on 15–25% of the GA-C44-V22 plants, but the viral antigen could not be detected in them. Moderate resistance, as in the genotypes Tambel-2 and Asgrow-XPH-5021, was characterized by mosaic and little or no stunting under both greenhouse and field conditions, a 2- to 3-wk incubation period, and low to medium concentrations of the viral antigen for 2–3 wk after inoculation. These studies demonstrate that there are multiple levels of resistance to TEV in pepper.

In 1985 tobacco etch virus (TEV) was identified as the most important virus infecting bell pepper (*Capsicum annuum* L.) in northeastern Georgia (1). In this 5-yr study, TEV disease incidence in susceptible genotypes was 90–100% by the end of each growing season, with yield losses in the range of 15–50% (F. W. Nutter, Jr., *unpublished data*). The virus also occurs in other pepper-growing areas of the United States (9,15,17).

The main sources of primary inoculum of TEV in Georgia are apparently perennial species of *Solanum* and *Physalis* (1). Since these species are common and widespread in forests and other uncultivated areas near pepper fields, it seems unlikely that their eradication can be achieved. Therefore, studies were initiated to define the nature of resistance to TEV in pepper and to determine its usefulness in controlling the TEV disease. A variety of sources of resistance to TEV in pepper have been reported (5,8,11).

Specific objectives of this study were to determine potential levels of resistance to TEV in pepper genotypes, to determine virus-host factors related to resistance, and to compare the effectiveness of resistance under both greenhouse and field conditions.

MATERIALS AND METHODS

Pepper genotypes. Seeds of 53 pepper genotypes were obtained from several sources. Twenty-three field selections came from the University of Georgia breeding line C44, which originated from crosses including Truhart Perfection, Yolo Wonder, PI 163192, and PI 264281 (7). Five genotypes were obtained from Texas A&M University: Tambel-1, Tambel-2 (16), Grand Rio 66, TAM 80011-6B-1, and TAM 8105A-2-1. Four were obtained from the University of Florida: Florida VR-2, Florida BG-1, FL-XVR-2-34, and FL-XVR-3-25. George Park Seed Co. and Brawley Seed Co. provided 14 commercial cultivars. Two commercial hybrids (Melody and Skipper) and five experimental hybrids

(XPH-5017, XPH-5018, XPH-5019, XPH-5020, and XPH-5021) came from Asgrow Seed Co.

Virus isolates and plant maintenance. During each year from 1983 to 1986, several isolates of TEV were obtained from infected pepper plants in fields in northeastern Georgia. On the basis of serology and limited host range tests, all the isolates were similar; therefore, one isolate, designated TEV-GA-85, was used in most greenhouse tests. TEV-GA-85 and six other isolates—one from pepper fields in 1986, one from horsenettle (*Solanum carolinense* L.), one from pepper in Florida (provided by John Simons), and three from plants of the resistant pepper genotypes Tambel-2, FL-XVR-3-25, and GA-C44-V22 growing in Georgia—were tested for their reaction on resistant pepper genotypes.

In greenhouse tests, pepper plants were grown singly in plastic pots (10 cm in diameter) containing a mixture of soil, sand, and vermiculite (2:1:1, v/v) (pH 6.8) previously treated with methyl bromide. The plants were fertilized weekly with a complete (N-P-K) fertilizer solution. Greenhouse temperatures ranged from 21 to 35 C in the daytime and from 18 to 24 C at night.

Inoculation and infectivity test. For mechanical inoculation, sap from infected Yolo Wonder B plants in 0.01 M potassium phosphate buffer (pH 7.0) containing 1% Celite was applied to test plants with a cheesecloth pad. Furthermore, mechanical inoculation of Yolo Wonder B plants was used to test for TEV in inoculated symptomless plants or any plants that required virus identification.

Enzyme-linked immunosorbent assay. The double-antibody sandwich enzyme-linked immunosorbent assay (ELISA) (2) was used for both quantitative and qualitative tests. For quantitative tests, leaf samples were weighed to the nearest 0.01 g and then placed in a leaf press to extract sap in ELISA extraction buffer (9 ml/g); the buffer was 0.02 M potassium phosphate (pH 7.3) containing 0.15 M NaCl, 0.03 M KCl, 0.05% Tween 20, and 2.0% polyvinylpyrrolidone (mol wt 40,000). When two or more treatments were compared, four or more replications per treatment were assayed. The samples were placed in precoated ELISA microtiter plates in a randomized complete block design. The antibody protein concentration for coating the ELISA plates was 1.25 μg/ml, and the enzyme conjugate dilution was 1/500. After 1–3 hr, the reaction between alkaline phosphatase and *p*-nitrophenyl phosphate was stopped with 3 M NaOH, and absorbance readings were taken at 405 or 410 nm (two ELISA readers).

The ELISA procedure for qualitative tests was similar, except sample weights were estimated, extracted sap was diluted 1/5 to 1/20, and the enzyme-substrate reaction was not stopped. For a sample to be considered positive, the absorbance was required to be at least 0.1 and at least twice as great as that of sap samples from healthy pepper plants. Furthermore, infectivity tests were conducted with many samples to confirm that plants were infected with TEV.

Incubation period and viral antigen concentration. Plants of five pepper genotypes (Yolo Wonder B, Tambel-2, Asgrow-XPH-5021, FL-XVR-3-25, and GA-C44-V22) were grown singly in pots 10 cm in diameter (one plant per pot) and inoculated with TEV-GA-85 at the three- to four-leaf stage. The pots were placed in a randomized complete block design on a greenhouse bench. Inoculated leaves were assayed quantitatively by ELISA at 4 and 11 days after inoculation. New, expanded uninoculated leaves were tested at 25 days. Plants without symptoms were assayed for infectivity. Two similar tests were conducted in January and August 1986.

Plant growth experiment. Plants of three susceptible genotypes (Yolo Wonder B, California Wonder, and Keystone) and four resistant genotypes (Tambel-2, Asgrow-XPH-5021, FL-XVR-3-25, and GA-C44-V22) were grown in pots 15 cm in diameter (one plant per pot) and inoculated with TEV-GA-85 at the three- to four-leaf stage. Plant height was measured weekly, and the dry weights of shoots and roots and quantitative ELISA values were determined 7 wk after inoculation.

Test for extreme resistance. When no plants within a single genotype developed symptoms after sap inoculation with TEV and the virus could not be detected by ELISA, various procedures were used in an attempt to cause infection. These procedures included modifications of the inoculation buffer (increases in molarity or the addition of reducing agents), the use of test plants of different ages, multiple inoculations, alterations of the environment both before and after inoculation, grafting, and the promotion of new leaf growth on mature inoculated plants.

Field experiments. A field experiment, centrally located in a commercial pepper-growing area in northeastern Georgia, was established on 4 June 1986, with 17 pepper genotypes (results for only five genotypes are reported here). The experiment was designed to expose the test genotypes to intense inoculum pressure. In each of four blocks (replications), there were 35 rows, with 12 plants per row. Alternate rows (18 total) were planted with the TEV-susceptible Yolo Wonder B. Each of the other 17 rows, planted with the experimental genotypes, contained two Yolo Wonder B plants in the center. The two plants in the center of each of the 35 rows were mechanically inoculated with TEV-GA-85 on 12 June. The rows with the 17 genotypes were randomized in each block. Symptoms were recorded weekly, and disease indices for the degree of symptom expression were determined on 31 July and 30 August. Both qualitative and quantitative ELISA tests were conducted on a young expanded leaf selected from each of the approximately 40 plants of each genotype. Leaves were selected on 9 July and 12 September and tested within 48 hr.

A second field test was conducted in 1987 at a site about 300 m from the 1986 test. The 1987 test included four replications each (150 plants per replication) of genotypes GA-C44-V22 and FL-XVR-3-25. The plants were observed for TEV symptoms and evaluated for infection by infectivity tests and ELISA.

RESULTS

Comparison of ELISA and infectivity tests. In many cases ELISA was used to determine the presence of the TEV antigen and to estimate its concentration. Therefore, results of the serological procedure were compared with results of infectivity tests (sap inoculation of Yolo Wonder B) as a means to determine the dilution end point. Infectivity could be detected at a sap dilution of 10^{-6}, and the end point of antigen detection was 10^{-5}. It appears, therefore, that ELISA measures the relative level of infectious TEV.

Screening for resistance. Forty-five of the 53 pepper genotypes tested were susceptible to TEV. All plants of the susceptible genotypes developed mosaic symptoms 3–8 days after inoculation, and sap from uninoculated leaves with symptoms had ELISA absorbance values (405 nm) of 1.00 or higher. Eight genotypes showed resistance to TEV. No symptoms developed on four of them (GA-C44-V22, GA-C44-GC, FL-XVR-2-34, and FL-XVR-3-25), and their ELISA absorbance values (0.01–0.04) were similar to those of the uninoculated controls. At 10–21 days after inoculation, mild symptoms were observed on four other genotypes (Asgrow-XPH-5019, Asgrow-XPH-5020, Asgrow-XPH-5021, and Tambel-2), and their ELISA absorbance values were relatively low (0.12–0.73).

Incubation period and virus concentration. The incubation period for TEV symptom appearance was 4 days in the susceptible Yolo Wonder B (Table 1). In Tambel-2 and Asgrow-XPH-5021, symptoms did not appear until 11–15 days after inoculation, and no symptoms developed on FL-XVR-3-25 or GA-C44-V22 (Table 1).

In Yolo Wonder B, the TEV antigen was readily detected in inoculated leaves 4 days after inoculation and reached a maximum concentration at 11 days (Table 1) and at 10–15 days in other experiments. At 4 days, the virus was barely detectable in Tambel-2 and not detectable in Asgrow-XPH-5021. Maximum TEV antigen concentrations similar to those in Yolo Wonder B were attained in both genotypes, but only after 3–4 wk (Table 1). Neither ELISA nor infectivity tests could detect TEV in FL-XVR-3-25 or GA-C44-V22 in these greenhouse tests.

Plant growth experiment. At 6 wk after inoculation with TEV, plants of the susceptible genotypes Yolo Wonder B, California

TABLE 1. Time-course study of incubation period and viral antigen concentration in uninoculated new leaves of pepper genotypes mechanically inoculated with tobacco etch virus

Genotype[v]	Symptom appearance (days after inoculation)			Absorbance (410 nm) in enzyme-linked immunosorbent assay[x]		
	Initial	50%[w]	100%	4 days[y]	11 days	25 days
Yolo Wonder B	4	4	5	0.81 a	1.65 a	1.47 a
Tambel-2	11	15	20	0.15 b	0.35 b	0.91 b
Asgrow-XPH-5021	15	16	17	0.04 c	0.23 c	1.01 b
FL-XVR-3-25	—[z]	—	—	0.01 c	0.03 c	0.02 c
GA-C44-V22	—	—	—	0.02 c	0.02 c	0.02 c

[v] Eighteen plants per genotype.
[w] Percentage of plants showing symptoms.
[x] Absorbance values are the average of data from 18 plants. Values for uninoculated pepper controls ranged from 0.00 to 0.04. Values in the same column followed by the same letter are not significantly different according to the Waller-Duncan k-ratio test ($P = 0.05$).
[y] Days after inoculation. Sampled inoculated leaves at 4 and 11 days and uninoculated new leaves at 25 days.
[z] No symptoms developed throughout the experiment.

TABLE 2. Plant height and dry weights of shoots and roots of seven pepper genotypes and their viral antigen concentrations 42 days after inoculation with tobacco etch virus

Genotype	Inoculation[x]	Plant height[y] (cm)	Dry weight[y] (g)		Mean absorbance (405 nm)[z]
			Shoots	Roots	
Yolo Wonder B	No	26.5 a	7.6 a	2.8 a	0.08 d
	Yes	21.0 b	3.6 b	0.9 b	0.83 bc
California Wonder	No	29.5 a	8.7 a	2.4 a	0.10 d
	Yes	21.0 b	3.9 b	0.9 b	1.02 ab
Keystone	No	28.3 a	6.9 a	2.6 a	0.09 d
	Yes	23.6 b	4.9 b	1.5 b	0.99 bc
Tambel-2	No	28.5 a	5.9 a	1.7 a	0.07 d
	Yes	24.0 a	4.4 b	1.8 a	0.70 c
Asgrow-XPH-5021	No	30.7 a	6.7 a	3.0 a	0.08 d
	Yes	29.5 a	7.2 a	2.8 a	1.31 a
FL-XVR-3-25	No	29.5 a	8.1 a	4.1 a	0.13 d
	Yes	30.7 a	7.0 a	3.5 a	0.15 d
GA-C44-V22	No	34.2 a	8.1 a	2.9 a	0.09 d
	Yes	31.3 a	7.0 a	2.6 a	0.08 d

[x] Pepper plants, grown in the greenhouse in pots 15 cm in diameter, were inoculated with the virus or (in the case of control plants) rubbed with inoculation buffer 5 wk after seeding. Six replications (plants) per treatment were tested.
[y] Paired comparisons using Student's t-test ($P = 0.05$); for each genotype, values in the same column followed by the same letter are not significantly different.
[z] Values followed by the same letter are not significantly different according to the Waller-Duncan k-ratio test ($P = 0.05$).

TABLE 3. Infection of plants of susceptible and resistant pepper genotypes after mechanical inoculation with tobacco etch virus at five inoculum concentrations

Inoculum concentration[a]	Plants infected[b] (%)					
	Test 1			Test 2		
	Yolo	Tambel	Asgrow	Yolo	Tambel	Asgrow
10^{-1}	100	53	93	100	93	100
10^{-2}	100	67	60	100	87	87
10^{-3}	67	27	0	73	20	0
10^{-4}	7	0	0	0	0	7
10^{-5}	0	13	0	13	7	13

[a] Leaf tissue from Yolo Wonder B plants infected for 15 days was ground in 0.01 M potassium phosphate buffer (pH 7.0) containing 1% Celite. Dilutions were made in the same buffer and rubbed onto 15 plants of each cultivar.
[b] Yolo = Yolo Wonder B; Tambel = Tambel-2; Asgrow = Asgrow-XPH-5021.

Wonder, and Keystone were 22% shorter than the uninoculated controls (Table 2). Differences in height between inoculated and uninoculated plants were first observed 3–4 wk after inoculation (data not shown). The shoot and root dry weights of the susceptible genotypes were an average of 46 and 57%, respectively, less than those of the uninoculated controls (Table 2). Although the shoot dry weight of infected Tambel-2 was 25% less than that of its uninoculated controls, its root weight was unaffected by the TEV infection. Virus inoculation of Asgrow-XPH-5021, FL-XVR-3-25, and GA-C44-V22 did not cause plant height or the dry weights of shoots and roots to be less than those of the uninoculated controls (Table 2).

At 6 wk after inoculation, Asgrow-XPH-5021 had a higher level of TEV antigen concentration than Yolo Wonder B, California Wonder, Keystone, and Tambel-2 (Table 2). No antigen was detected in inoculated plants of FL-XVR-3-25 and GA-C44-V22.

Inoculum concentration. At sap dilutions of 10^{-1} and 10^{-2}, all plants of Yolo Wonder B and most plants of Tambel-2 and Asgrow-XPH-5021 became infected with TEV (Table 3). At a dilution of 10^{-3}, 70% of the Yolo Wonder B plants became infected, whereas only 24% of the Tambel-2 plants and none of the Asgrow-XPH-5021 plants became infected. A few plants of all genotypes became infected at sap dilutions of 10^{-4} and 10^{-5}.

TEV isolates. Seven isolates of TEV infected 93% of the plants of Yolo Wonder B, Tambel-2, and Asgrow-XPH-5021 (Table 4), and the incubation period and degree of symptom expression were similar for all isolates. The incubation period was longer and symptoms milder in Tambel-2 and Asgrow-XPH-5021 than in Yolo Wonder B.

Only three of 108 plants of the resistant genotypes FL-XVR-3-25 and GA-C44-V22 became infected with the isolates of TEV (Table 4). The three infected plants had symptoms similar to those of Yolo Wonder B. About 64% of the progeny of the three infected plants were susceptible, whereas none of the progeny of symptomless plants became infected with TEV. Since seeds of the two genotypes were produced in the field, we suspect that outcrossing was responsible for the few plants that developed symptoms and that pure lines of FL-XVR-3-25 and GA-C44-V22 react with extreme resistance to all seven isolates.

Extreme resistance. Seeds collected from progeny of FL-XVR-3-25 and GA-C44-V22 plants that developed no symptoms and tested negative by ELISA consistently produced plants that did not become infected when inoculated under greenhouse conditions. A variety of greenhouse tests (six plants or more per genotype in each test) were conducted to attempt to infect these genotypes: six rubs per leaf on three to five leaves of plants of different ages; multiple inoculations per plant at 1, 2, and 4 days after the first inoculation; maintenance of inoculated plants at a constant 21 C, at a constant 33 C, and at 35 C during the day and 25 C at night; preinoculation dark and 35 C for 24–48 hr; and grafts of scions to infected stocks of Yolo Wonder B. Furthermore, the top one-third was removed from plants that had been inoculated 55 days previously, and new axillary leaf growth was observed for 46 days. None of the plants developed

TABLE 4. Infection of susceptible and resistant pepper genotypes by seven isolates of tobacco etch virus

Genotype	Isolates[a,b]						
	GA-85	GA-86	SIM	HN	TAM	FL-R	GA-R
Yolo Wonder B	11/12[c]	6/6	12/12	6/6	6/6	—[d]	6/6
Tambel-2	9/12	6/6	11/12	4/6	6/6	6/6	3/6
Asgrow-XPH-5021	5/6	6/6	6/6	—	5/6	6/6	6/6
FL-XVR-3-25	1/12	0/6	0/12	0/6	0/6	0/6	0/6
GA-C44-V22	0/12	0/6	1/12	0/6	0/6	1/6	0/6

[a]GA-85 and GA-86 = isolates collected from Georgia pepper fields in 1985 and 1986; SIM = isolate from John Simons; HN = isolate from horsenettle; TAM, FL-R, and GA-R = isolates from Tambel-2, FL-XVR-3-25, and GA-C44-V22 plants growing in Georgia, respectively.
[b]Isolates were cultured in Yolo Wonder B for 15 days prior to inoculation. Sap inoculum from young expanded leaves was diluted to a concentration of 10^{-1} in 0.01 M potassium phosphate buffer (pH 7.0) containing 1% Celite.
[c]Plants infected/plants inoculated.
[d]Combination not tested.

TABLE 5. Disease development in plants of five pepper genotypes inoculated with tobacco etch virus in the field

Genotype[a]	Plants with symptoms[b] (%)	Plants positive by enzyme-linked immunosorbent assay[b] (%)	Disease index[b,c]	
			31 July	30 Sept.
Yolo Wonder B	100	98	4.0	4.0
Tambel-2	98	90	2.5	4.0
Asgrow-XPH-5021	100	95	1.0	2.0
FL-XVR-3-25	85	8	2.5	1.8
GA-C44-V22	18	0	0.0	1.0

[a]Plants were transplanted on 4 June 1986. Inoculum source plants within the plots were inoculated with the virus on 12 June.
[b]Values are based on four replications, each consisting of 10 plants.
[c]0 = No symptoms; 1 = 1–25% of plants with mottle but no stunting; 2 = 26–75% of plants with mottle and apparent stunting; 3 = mottle and moderate stunting; 4 = mosaic and severe stunting.

symptoms, ELISA tests were negative, and sap from both inoculated and uninoculated leaf tissue, extracted in buffer of different molarities and containing additives, did not cause Yolo Wonder B plants to become infected.

Field experiment. When grown in the field, over 94% of the plants of Yolo Wonder B, Tambel-2, and Asgrow-XPH-5021 developed TEV symptoms (mosaic, upward leaf rolling, or stunt) and had positive ELISA reactions (Table 5). Late in the growing season, 18% of the GA-C44-V22 plants developed a mild mottle with no stunting. Beginning about mid-season, the new growth of 85% of the FL-XVR-3-25 plants was mildly chlorotic, and upwardly rolled leaves were smaller than normal. The TEV antigen could not be detected in any of the GA-C44-V22 plants and was detected in only 8% of the FL-XVR-3-25 plants exhibiting symptoms (Table 5).

Two disease index evaluations indicated that Yolo Wonder B is affected more severely and earlier by TEV than Tambel-2, Asgrow-XPH-5021, FL-XVR-3-25, and GA-C44-V22 (Table 5). The disease was very mild on GA-C44-V22 and progressively more severe on FL-XVR-3-25, Asgrow-XPH-5021, and Tambel-2.

Late in the growing season (15 August–1 September), Yolo Wonder B, Tambel-2, and Asgrow-XPH-5021 had mosaic and leaf rolling on the newest growth, but only Yolo Wonder B plants had symptoms on the oldest growth and were stunted 20% or more. The TEV antigen concentration was similar in all leaf tissues of all three genotypes. The leaf tissue tested was from the youngest leaves (test 1); from the youngest, oldest, and intermediate-aged leaves tested individually (test 2); and from leaves of different ages selected at random and combined (test 3).

Symptoms of resistant genotypes in the field. Since the cause of symptoms on GA-C44-V22 and FL-XVR-3-25 was not clearly evident, about 600 plants of each genotype were planted in the field in 1987. Mild mottle developed on 25 and 50% of the GA-C44-V22 and FL-XVR-3-25 plants, respectively. The ELISA results for TEV were negative or questionable for 96 GA-C44-V22 plants with symptoms and positive for 14 of 96 FL-XVR-3-25 plants with symptoms. Sap from mottled plants of the two resistant genotypes caused TEV symptoms (and was also TEV-positive by ELISA) in only two of about 100 Yolo Wonder B plants tested.

DISCUSSION

Our studies demonstrate multiple levels of resistance to TEV in pepper. Genotype GA-C44-V22 exhibited extreme resistance under greenhouse conditions: no symptoms developed, and the virus could not be detected by either ELISA or infectivity tests in either mechanically inoculated or uninoculated leaves. However, a few plants developed a mild mottle in the field, perhaps because of intense inoculation pressure by multiple aphids per plant. It seems clear that the plants can become infected, but very little viral antigen accumulates in them. The disease reaction and relative viral antigen content in GA-C44-V22 were consistent under field conditions with intense inoculum pressure for five consecutive years, 1984 through 1988 (12; F. W. Nutter, Jr., *unpublished data*).

A moderate level of resistance to TEV was found in the genotypes Asgrow-XPH-5021 and Tambel-2. When they were compared with the susceptible genotype Yolo Wonder B, the resistance was characterized by a 1- to 2-wk delay in symptom appearance, a slow rate of accumulation of the viral antigen, a less severe effect on plant growth, and a lower susceptibility to mechanical inoculation. All plants of Asgrow-XPH-5021 and Tambel-2 eventually developed symptoms, and the viral antigen concentration continued to increase until it was similar to that in Yolo Wonder B, after a delay of approximately 2 wk. Field studies by Padgett (12) in 1985, 1986, and 1987 demonstrated that the moderate resistance in Asgrow-XPH-5021 and Tambel-2 delayed the development of TEV epidemics, and fruit yield was significantly higher than in Yolo Wonder B. Under both greenhouse and field conditions, Asgrow-XPH-5021 is more resistant than Tambel-2 but much less resistant than GA-C44-V22. We believe the moderate resistance in both Asgrow-XPH-5021 and Tambel-2 should be termed rate-reducing resistance (14) and warrants consideration in pepper-breeding programs. Rate-reducing resistance in fungal pathosystems is associated with a durable type of resistance, which may be effective against variants within pathogen populations (14).

Under greenhouse conditions, genotype FL-XVR-3-25 appeared to have extreme resistance similar to that of GA-C44-V22. However, under field conditions, FL-XVR-3-25 was more susceptible to TEV than GA-C44-V22 and more resistant than Asgrow-XPH-5021 and Tambel-2.

The mild mottle symptoms on a low percentage of field-grown plants of the resistant genotype GA-C44-V22 caused us to consider other viruses as the cause of the symptoms. Mechanically transmitted viruses other than TEV were ruled out, because the inoculation of Yolo Wonder B, susceptible to several pepper viruses (1), with sap from mottled plants caused no symptoms. Furthermore, when sap from the resistant mottled plants was

tested serologically (by ELISA or gel diffusion), negative results were obtained with antisera to alfalfa mosaic virus, beet western yellows virus, cucumber mosaic virus, pepper mottle virus, potato leaf roll virus, potato virus Y, and tobacco mosaic virus. Tests for a virus persistently transmitted by an aphid were negative: adults of *Myzus persicae* Sulz. were first fed for 24 hr on mottled tissue from resistant genotypes and then transferred to Yolo Wonder B plants (five aphids per plant). We cannot entirely disregard the possibility of a causal agent other than TEV, but at this time we surmise that the symptoms on GA-C44-V22 were caused by TEV and that the plants had a very low concentration of the virus.

A few sources of resistance to TEV in *Capsicum* spp. have been reported previously. Two genotypes of *C. annuum*, South Carolina (SC) line 46252 and PI 264281 (the same as genotype P11), react with extreme resistance to TEV (3,5,8,10,11); their reaction is probably similar to that of GA-C44-V22. Symptomless systemic infections can be detected in the genotypes, and a small proportion of plants develop a mild mottle. Agronomico 8, a derivative of PI 264281, and accession 2120 reacted with no symptoms to TEV in a California study (10). Nagai and Smith (10) reported a similar reaction in the genotypes PI 152225 and PI 159236 of *C. frutescens* L. Greenleaf (8), however, believes PI 152225 has a lower level of resistance than SC 46252.

Inheritance studies (3,8) have shown that SC 46252, PI 152225, and PI 264281 each have a single recessive gene controlling resistance to TEV. Furthermore, Greenleaf (8) suggested that modifying genes in SC 46252 and PI 152225 may be responsible for altering the disease reaction caused by TEV. Apparently, no genetic crosses between resistant genotypes have been attempted; therefore, it is not known if resistance expressed in these genotypes is the result of the same gene or different genes. All of the resistance studies reported above were conducted in the greenhouse, and resistance was determined on the basis of symptomatology and, in some cases, tests for infectivity by mechanical or graft inoculation. This extreme type of resistance has been incorporated into a few cultivars (4,6,18), but they apparently have not been widely accepted by commercial growers because of poor fruit quality.

It is not clear at this time if the multiple levels of resistance to TEV in pepper are due to different major genes or if modifying genes, as suggested by Greenleaf (8), might be responsible. The pedigree of the moderately resistant Tambel-2 includes the extremely resistant Agronomico 8 (10,16). Smith (13) differentiated isolates of TEV on the basis of the disease reaction they caused in five pepper genotypes, thus suggesting more than one gene for resistance. We do not know if multiple genes are responsible for the moderate resistance expressed by Tambel-2; however, the hypothesis of polygenic control is consistent with what is known about other pathosystems characterized as having rate-reducing resistance (14). Future studies should attempt to clarify the host gene or genes that contribute to the multiple levels of resistance reported herein.

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