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Inheritance of a gene for resistance to tomato spotted wilt virus (TSWV) from *Lycopersicon peruvianum* Mill

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Summary

Inheritance of resistance to tomato spotted wilt virus (TSWV) derived from the cultivar 'Stevens' was studied. Five TSWV isolates, which differ in geographic origin and elicit different symptoms on tomato, were used to screen the resistant parent plants. Enzyme-linked immunosorbent assay (ELISA) was used to distinguish healthy and infected plants. Two susceptible advanced breeding lines were crossed with four F_4 plants of a 'Stevens' × 'Rodade' obtained from South Africa (SA). There were no differences in the progeny responses of the four SA parents to TSWV. The inheritance of TSWV resistance was found to be a single dominant gene. The SA, F_1, and the backcrosses to the resistant parent were found to have eight out of 612 plants infected four months after the inoculations, which indicates a 98.7% penetrance of the resistance gene.

Abbreviations: BCP_1-1 – Backcrossed to resistant parent; BCP_1-1 – Backcrossed to resistant parent from the first selected F_1; BCP_1-2 – Backcrossed to resistant parent from the second selected F_1; BCP_2 – Backcrossed to susceptible parent; BCP_2-1 – Backcrossed to susceptible parent from the first selected F_1; BCP_2-2 – Backcrossed to susceptible parent from the second selected F_1; ELISA – enzyme-linked immunosorbent assay; F_1-1 – first selected F_1 plant; F_1-2 – second selected F_1 plant; F_2-1 – F_2 progeny from the first F_1 selection; F_2-2 – F_2 progeny from the second F_1 selection; P_1 – resistant parent; P_2 – susceptible parent; SA – F_4 plants from the South African cross 'Stevens' × 'Rodade'; SA#2, SA#5, SA#7, and SA#8 – selections of the F_4 plants; SDS – sodium dodecyl sulfate; TSWV – tomato spotted wilt virus; 87 – Arkansas advanced breeding line 87-68-2; 89 – Arkansas advanced breeding line 89-31-M.

Introduction

Tomato spotted wilt virus (TSWV) was first described in Australia by Brittlebank (1919). It is now recognized as a cause of disease worldwide and has been reported to affect over 192 dicotyledonous species in 33 families and eight monocotyledonous species in five families (Best, 1968; Iwaki et al., 1984; Cho et al., 1986; Cho et al., 1987; Cho et al., 1989).

The cultivated tomato (*Lycopersicon esculentum* Mill.) is seriously affected by this virus. In addition to plant stunting and yield reduction, the fruit is blemished with necrotic or chlorotic ringspots, usu-

ally appearing only after the fruit begins to show color leaving it unmarketable. Although this disease is somewhat sporadic in Arkansas, losses have been reported as high as 38% in commercial tomato fields (Paterson et al., 1989).

Cho et al., (1989) reported that thrips, the natural vectors of TSWV, migrate into a field from numerous weed hosts and transmit the virus before insecticides have time to incapacitate the insect. They also reported that cultural practices such as fallowing, rotation, and control of alternate weed hosts were marginally effective in TSWV management with cross protection proving to be totally ineffective. Consequently, resistance to the virus appears to be the most promising means of controlling the disease.

Isolate-specific resistance to TSWV has been found in *L. pimpinellifolium* Mill. (Samuel et al., 1930; Smith, 1944; Finlay, 1953). The resistance to TSWV found in 'Pearl Harbor' was developed from this species (Kikuta et al., 1945). Holmes (1948) found that the selection *L. esculentum* cultivars 'Rey de los Tempranos' and 'Manzana' also had TSWV isolate-specific resistance. Finlay (1953) tested all of the above-mentioned sources of resistance against ten different TSWV isolates from Australia and identified five isolate-specific resistance genes. He concluded, however, that it was impossible to breed a tomato cultivar containing resistance to all TSWV isolates tested due to the presence of gene linkage and recessive alleles.

The wild species *L. peruvianum* Mill. has been reported by Wenholz (1939), Smith (1944), Norris (1946), and Finlay (1952) to have broad resistance to isolates of TSWV. Norris (1946) reported that "*L. peruvianum* Mill. possesses true resistance amounting almost to immunity" to TSWV. Gilbert & Tanaka (1971) released 'Anahu' as a TSWV resistant tomato cultivar that had *L. peruvianum* in its background.

In our laboratory, 'Rey de los Tempranos', 'Manzana', 'Pearl Harbor', and 'Anahu' were found to be susceptible to local isolates of TSWV when evaluated for infection using visual symptoms and an enzyme-linked immunosorbent assay (ELISA) (Paterson, 1987; Paterson et al., 1989). However, when testing the accessions of *L. peruvianum* in which Smith (1944) found TSWV resistance, Paterson (1987) found only 24 plants out of 573 that tested positive for TSWV infection using ELISA.

Watterson et al., (1989) described an unreleased tomato cultivar that contains resistance to TSWV derived from *L. peruvianum.* The inheritance was reported to be "a major dominant gene ... with one or more modifying genes". However, this germplasm was not available for testing.

The fresh market cultivar 'Stevens' was reported to be resistant to TSWV (van Zijl et al., 1986; J.J.B. van Zijl, personal communication). This cultivar was developed from a cross between *L. esculentum* and *L. peruvianum* (Stevens, 1964).

F_4 seed of a 'Stevens' × 'Rodade' cross was obtained from J.J.B. van Zijl (Vegetable & Ornamental Plant Research Institute, Pretoria, South Africa). This population, which was designated as "SA", was resistant to a total of five TSWV isolates from Arkansas, Texas, and Hawaii as indicated by ELISA and visual symptoms. Because the TSWV resistance found in this source was demonstrated to be effective against all isolates tested, a thorough understanding of its inheritance and resistance properties would be useful in creating isogenic lines, new cultivars, and hybrids.

The objective of this study was to determine the inheritance of the TSWV resistance found in the SA population.

Materials and methods

Origin and maintenance of TSWV isolates

TSWV isolates were maintained in young *Nicotiana rustica* L. by rub-inoculating Carborundum-dusted leaves with sterile cheesecloth pads. The inoculum was symptomatic tissue that was homogenized in a sterile ice-cold mortar and pestle with cold inoculation buffer (0.1 M phosphate buffer, pH 7.4, containing 0.01 M sodium sulfite).

Isolates 85-9 and 87-34 originated from infected tomatoes in southeastern Arkansas in 1985 and 1987, respectively. The symptoms of 85-9 in tomato are similar to the "tip blight" symptoms described

by Norris (1946) with 87-34 being closer to the "mild" strain he described. Isolate Glox originated from Texas in Gloxinia (*Sinningia speciosa* Lodd.) shipped commercially to Arkansas. Symptoms of this isolate in tomato are the mildest of the isolates used. Isolate T-1 and T-2 originated from Hawaii with T-2 showing symptoms similar to isolate 85-9. T-1 produces tip blight symptoms, and it appears less severe in tomato than both 85-9 and T-2.

Testing resistance of the SA population

Rooted cuttings of 11 of the reported TSWV-resistant SA F_4 tomato plants were tested on two occasions using the five TSWV isolates. A total of 12 rooted cuttings from each of 11 SA F_4 tomato plants were inoculated. A single cutting from each of the 11 SA plants along with six TSWV susceptible 'VF Pink' plants were treated as a set and inoculated with one of the five TSWV isolates along with an isolate mix. The isolate mix was prepared by taking approximately equal weights of infected *N. rustica* tissue from each of the five isolates and homogenizing them in inoculation buffer. This mixture was rub-inoculated on young *N. rustica,* and the resulting infected plants were used as inoculum. This was done to allow for possible pseudo-recombination of the TSWV genome.

Inoculation of parents and progeny for inheritance tests

To help identify the possibility of a heterozygous condition for TSWV resistance, four SA plants were selected as resistant parents: SA#2, SA#5, SA#7, and SA#8. Also, two separate F_1 plants per cross were selected as parents for 416 BCP_2 plants (backcrossed to susceptible parent), 353 BCP_1 plants (backcrossed to resistant parent), and 744 F_2 plants tested. The susceptible parents selected were two Arkansas advanced breeding lines with different parentages: these were 87-68-2 (87) and 89-31-M (89). Reciprocal crosses were not made because *L. peruvianum* can only act as the staminate parent when crossed with *L. esculentum* (Rick, 1979) thus precluding any cytoplasmic inheritance of the TSWV resistance.

Parents and progeny from one of the eight original Arkansas × SA crosses were planted in six, 48-cell flats. This was repeated for each of the eight original crosses. The layout of the six flats was as follows: eight of the exterior cells of each flat were planted to TSWV susceptible 'VF Pink' tomatoes for visual monitoring of the virus infection. Each flat contained two rooted cuttings of each of the two F_1 parents (F_1-1, F_1-2), two rooted cuttings of the resistant parent (P_1), four plants from the susceptible parent (P_2), four backcross plants from each of the F_1 parents to the resistant parent (BCP_1-1, BCP_1-2), four backcross plants from each of the F_1 parents to susceptible parent (BCP_2-1, BCP_2-2), and seven F_2 plants from each of the F_1 parents (F_2-1, F_2-2). Each generation was kept as a distinct group within the flat; however, groups were randomized within the center 40 cells of a flat.

Because of the severe symptoms induced by isolate 85-9, it was used as the screening isolate for all the generations. Inoculations were accomplished using a touch-up paint sprayer at 3.56×10^5 N/m^2. Infected *N. rustica* leaves were homogenized in a blender in cold inoculation buffer (10% w/v) followed by filtration through sterile cheesecloth. Carborundum (600-mesh) was then added at 1% w/v. Volumes of 90–115 ml of cold agitated filtrate and Carborundum were used immediately to inoculate each flat. The sprayer nozzle was held 3–5 cm from the apical growing point, and inoculation was done with 0.4–0.8 second bursts of the sprayer. The possibility of escapes or skips was reduced by repeating the inoculation after 6–8 days. If the daytime temperatures in the greenhouse exceeded 30 C, the inoculations were performed in the evening when the greenhouses were cooler. Samples for ELISA were taken 1–2 weeks after the final inoculation.

During the initial data collection, the results of the F_1-2 progeny for the 87 × SA#5 cross indicated a deviation from the ratio observed for other parental combinations, so 100 additional F_2-2 plants and 60 additional BCP_2-2 plants were screened as above. The aberrant ratio was not observed in the larger sample, so the data were

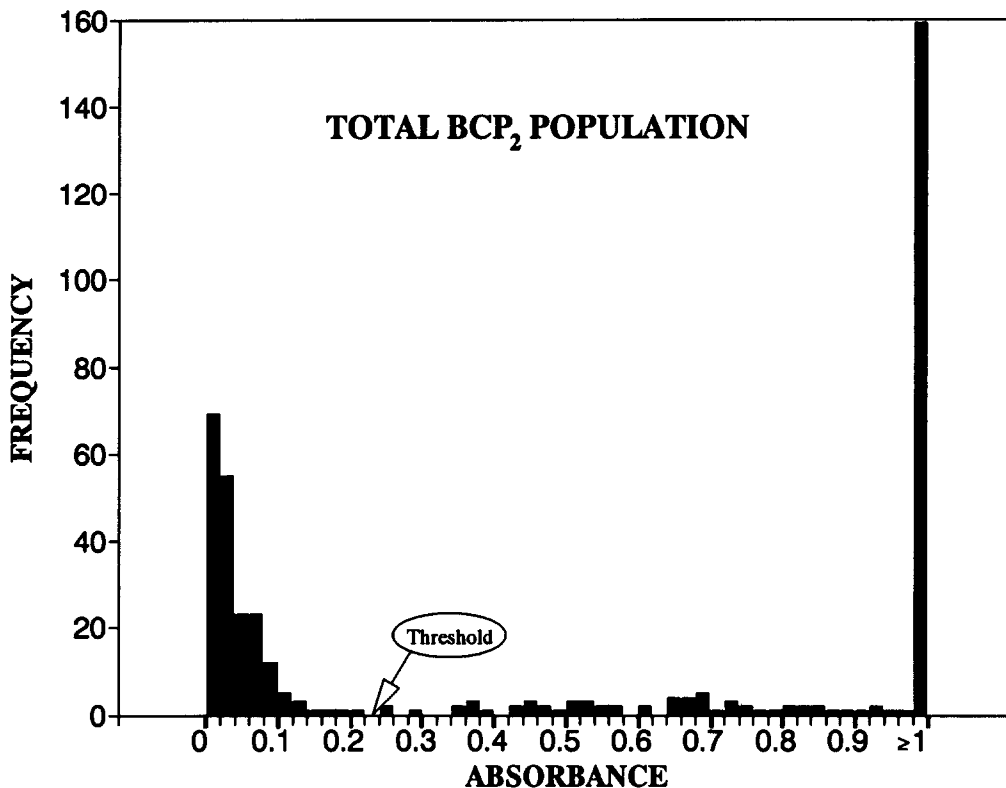

Fig. 1. BCP_2 population frequencies of ELISA absorbance values at A_{490} nm. All plants with values ≤ 0.23 were considered uninfected with TSWV.

pooled in the results. Also, there was no evidence of heterogeneity for TSWV resistance in the four SA parents, so the results of the F_1, BCP_1, and BCP_2, within each of the eight Arkansas × SA crosses were pooled in the results.

Sample preparation for ELISA testing

Rapid efficient extraction of plant sap for virus evaluation by ELISA was accomplished with a sap extractor from Erich Pollähne (Hannover, West Germany). Preliminary results showed that some samples had TSWV contamination after the recommended water washes; this was especially apparent when healthy plants were extracted following infected *N. rustica*. This problem was eliminated by first washing the rollers with a water wash, followed by approximately a 5-ml wash of 1% w/v sodium dodecyl sulfate (SDS) and another water wash. The sensitivity of the TSWV ELISA test was not reduced even with deliberate SDS contamination of extracted sap samples.

All plants were evaluated for TSWV by ELISA using TSWV-L antibody from Agdia Inc. (Mishawaka, Indiana). The protocol was altered to use 150 μl per well instead of the recommended 200 μl, and albumin was omitted from the extraction buffer. Tissue for ELISA testing was selected from the leaves nearest the growing point. However, in some instances, plants were collapsing, which required sampling from other living leaf or stem tissue. Dead tissue was not tested. The absorbance value at A_{490} nm was read on a MR 600 Microplate Reader from Dynatech Laboratories, Inc. (Alexandria, Virginia).

Each 96-well ELISA plate contained four wells of each of the following controls placed at intervals across the plate: TSWV-infected *N. rustica;* healthy *N. rustica;* and healthy *L. esculentum.*

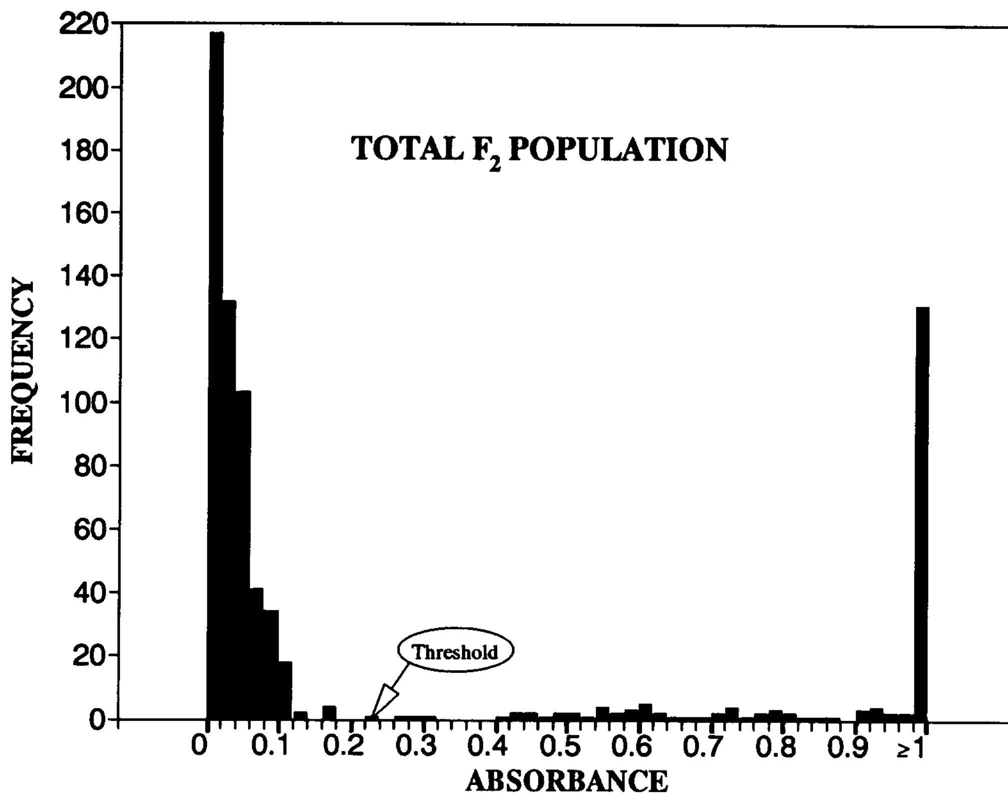

Fig. 2. F_2 population frequencies of ELISA absorbance values at A_{490} nm. All plants with values ≤ 0.23 were considered uninfected with TSWV.

Each plate was duplicated and the mean absorbance value for the two duplicate wells was used for analysis.

Interpretation of ELISA and statistical analysis

The mean of the absorbance values for all the healthy *L. esculentum* samples used in ELISA tests was 0.057. The absorbance values from all ELISA plates were plotted on a histogram (frequency distribution, Figs 1 & 2) as suggested by Sutula et al., (1986). The threshold for distinguishing infected and healthy plants was selected at four times the mean absorbance values for the healthy controls. Any sample with an absorbance value less than or equal to the threshold value of 0.23 was interpreted as healthy.

To help clarify the status (healthy or infected) of those plants that had absorbance values found between the two modes in the histogram (those between >0.20 and ≤ 0.80) a retesting method was designed. The questionable plants were selected, allowed to grow for at least two weeks, then retested with ELISA. The results of the second test were subjected to the absorbance threshold criteria. In the few cases in which plants again had intermediate absorbance values, the plants were tested a third time. Not all the plants having intermediate absorbance values could be retested because the plants died after the first sampling.

The genetic model for inheritance of TSWV resistance was tested by Chi-square.

Results and discussion

TSWV resistance in the original 11 SA plants

None of the cuttings from the 11 SA parents in-

dicated infection in the initial testing with any of the five TSWV isolates or the isolate mix. However, all 73 of the TSWV susceptible 'VF Pink' check plants inoculated at the same time as the SA parent plants became severely infected as judged by symptoms and ELISA. This initial test indicated that there was TSWV resistance in the SA plants and that it was not isolate specific.

Effectiveness of the inoculations

All 384 susceptible 'VF Pink' plants used for visual monitoring for the effectiveness of the inoculation procedure in the inheritance tests developed severe systemic infection. All 168 TSWV susceptible P_2 plants were found to be severely infected as judged by symptoms and ELISA (Table 1). This indicates that the TSWV inoculation procedure was able to infect 100% of the known susceptible plants.

Five of 83 SA parents, 14 of 179 F_1 cuttings, 41 of 358 BCP_1 plants, 25 of 416 BCP_2 plants, and 60 of 744 F_2 plants had ELISA absorbance values > 0.23 in the initial screening. Subsequent ELISA testing indicated that all but three F_1, five BCP_1, four BCP_2, and four F_2, still had absorbance values > 0.23 three months after their initial inoculations (Table 1). The absorbance values of the susceptible check plants either remained > 1.00 for the three months after inoculation or the plants collapsed and died. These data suggest that a plant that carries this resistance can sometimes be infected, but the resistance mechanism suppresses the virus after initial infection of the plant.

Inheritance of the TSWV resistance

The pooled BCP_2 population had 222 infected plants and 194 healthy plants (Table 1). These data indicate a ratio of 1 resistant: 1 susceptible with a probability of 0.17 (pooled $\chi^2 = 1.8846$). This ratio suggests the resistance to the tested TSWV isolates is a single dominant gene.

There were 744 F_2 plants tested for TSWV susceptibility with 193 becoming infected. The data in Table 1 reveal that the pooled F_2 population has a ratio of 3 resistant: 1 susceptible with a probability of 0.56 (pooled $\chi^2 = 0.3513$). Like the BCP_2 population, thses data also indicate the TSWV resistance is controlled by a single dominant gene.

Finlay (1953) described five genes for TSWV resistance. These genes were identified as Sw_1^a, Sw_1^b, sw_2, sw_3, and sw_4. Unlike those genes, the TSWV resistance gene identified in this study came from a different species and has been found to have broad resistance to isolates from many geographical areas. We have tested the cultivars and species identified as resistant by Finlay and found them susceptible to our isolates (Paterson, 1987; Paterson et al., 1989). These studies imply that the TSWV resistance gene identified in this study is different. It is therefore proposed to tentatively identify this TSWV resistance gene as *Sw*-5 until linkage tests can be made.

Penetrance of the TSWV resistance gene

When results indicate a single dominant gene, the F_1, P_1, and BCP_1 populations are expected to be resistant and the susceptible parent population fully infectible. However, eight out of 612 plants tested from these generations were found to be infected three months after the initial inoculation. This represents 1.3% of the tested population known to carry at least a single resistant allele. Thus, the penetrance of this resistance gene was estimated to be 98.7%. These results concur with van Zijl et al., (1986) who conducted a field study on the resistant parent cultivar of 'Stevens'; 2.6% were found to be infected with TSWV. They found no TSWV symptoms in two other resistant populations under field conditions in which 80–90% of the susceptible plants became infected.

All of the plants from the resistant generations (F_1, and BCP_1) that became infected had the potential to be heterozygous for the resistance gene. None of the resistant homozygous SA parent plants had absorbance values > 0.23 in ELISA tests beyond the second screening. It is not unusual to have reduced levels of resistance when the plant is heterozygous for a dominant resistance gene. When the *Tm-1* gene for resistance to tobacco mosaic virus

Table 1. Resistance reactions to TSWV isolate 85-9 of the P_1 (resistant parent), P_2 (susceptible parent), F_1, BCP_1 (backcrossed to the resistant parent), BCP_2 (backcrossed to the susceptible parent) and the F_2 populations, based on ELISA results

	Number of Plants (%)			χ^2	df	P
	Resistant	Infected	Total			
P_1 Cuttings						
SA#2	20 (100%)	0 (0%)	20			
SA#5	23 (100%)	0 (0%)	23			
SA#7	24 (100%)	0 (0%)	24			
SA#8	16 (100%)	0 (0%)	16			
P_2 Plants						
87-68-2 (87)	0 (0%)	86 (100%)	86			
89-31-M (89)	0 (0%)	82 (100%)	82			
F_1 Cuttings						
87 × SA#2	22 (92%)	2 (8%)	24			
87 × SA#5	24 (100%)	0 (0%)	24			
87 × SA#7	23 (100%)	0 (0%)	23			
87 × SA#8	24 (100%)	0 (0%)	24			
89 × SA#2	23 (100%)	0 (0%)	23			
89 × SA#5	20 (100%)	0 (0%)	20			
89 × SA#7	23 (96%)	1 (4%)	24			
89 × SA#8	17 (100%)	0 (0%)	17			
BCP_1 Plants						
SA#2 × (87 × SA#2)	40 (91%)	4 (9%)	44			
SA#5 × (87 × SA#5)	44 (100%)	0 (0%)	44			
SA#7 × (87 × SA#7)	45 (100%)	0 (0%)	45			
SA#8 × (87 × SA#8)	42 (100%)	0 (0%)	42			
SA#2 × (89 × SA#2)	48 (100%)	0 (0%)	48			
SA#5 × (89 × SA#5)	39 (98%)	1 (2%)	40			
SA#7 × (89 × SA#7)	48 (100%)	0 (0%)	48			
SA#8 × (89 × SA#8)	47 (100%)	0 (0%)	47			
BCP_2 Plants						
87 × (87 × SA#2)	19 (40%)	29 (60%)	48	2.0833	1	0.15
87 × (87 × SA#5)	46 (44%)	59 (56%)	105	1.6095	1	0.21
87 × (87 × SA#7)	14 (39%)	22 (61%)	36	1.7778	1	0.18
87 × (87 × SA#8)	30 (64%)	17 (36%)	47	3.5957	1	0.06
89 × (89 × SA#2)	25 (56%)	20 (44%)	45	0.5556	1	0.46
89 × (89 × SA#5)	15 (33%)	30 (67%)	45	5.0000	1	0.03
89 × (89 × SA#7)	24 (53%)	21 (47%)	45	0.2000	1	0.66
89 × (89 × SA#8)	21 (47%)	24 (53%)	45	0.2000	1	0.66
BCP_2 Ratio	1	1				
BCP_2 Total				15.0219	8	
BCP_2 Pooled	194 (47%)	222 (53%)	416	1.8846	1	0.17
BCP_2 Heterogeneity				13.1373	7	0.07
F_2 Plants						
87 × SA#2	58 (69%)	26 (31%)	84	1.5873	1	0.21
87 × SA#5	130 (71%)	52 (29%)	182	1.2381	1	0.27
87 × SA#7	61 (75%)	20 (25%)	81	0.0041	1	0.95
87 × SA#8	57 (79%)	15 (21%)	72	0.6667	1	0.41
89 × SA#2	58 (71%)	24 (29%)	82	0.7967	1	0.37
89 × SA#5	58 (69%)	26 (31%)	84	1.5873	1	0.21
89 × SA#7	66 (80%)	17 (20%)	83	0.9036	1	0.34
89 × SA#8	63 (83%)	13 (17%)	76	2.5263	1	0.11
F_2 Ratio	3	1				
F_2 Total				9.3101	8	
F_2 Pooled	551 (74%)	193 (26%)	744	0.3513	1	0.56
F_2 Heterogeneity				8.9588	7	0.26

was studied in depth, it was found that the resistance to the virus was slightly less when it was in a heterozygous condition (Fraser & Loughlin, 1980). It is unknown if this TSWV resistance has a similar reaction. The *Sw-5* gene should be considered incompletely dominant, if it is true that the resistance to TSWV is less in the heterozygous state.

Besides the observation of the possible heterozygous resistance weakness, two other characteristics of the resistance were noted. First, after TSWV inoculations, many plants demonstrated a necrosis of the inoculated tissue followed by growth of apparently uninfected tissue. These plants, when tested with ELISA, were found to have absorbance values <0.23, and systemic symptoms never appeared. Second, plants selected because they were found to have intermediate absorbance values did not always show visual symptoms.

Derivation and broad usage of the TSWV resistance gene

The PI number of the *L. peruvianum* used in the original cross was lost after the death of J.M. Stevens in 1966. The *L. peruvianum* accessions mentioned in the remaining records were PI 126928, PI 126929, PI 126944, PI 128645, PI 128654, and PI 129109. There were also two *L. peruvianum* var. 'dentatum' mentioned, but the PI numbers were not included. The PI number most frequently mentioned in all remaining records was PI 128654 (J.J.B. van Zijl, personal communication).

After the completion of this study we learned that 'Stevens' showed field resistance to TSWV in trials conducted in Australia, Brazil, and New York (R.F. Heisey, Asgrow Seed Co., personal communication). This information coupled with the findings of this study and the results of van Zijl et al. (1986) from South Africa indicate that the TSWV resistance found in 'Stevens' is not isolate specific.

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