Solanum habrochaites Accession LA1777 Recombinant Inbred Lines Are Not Resistant to Tomato Yellow Leaf Curl Virus or Tomato Mottle Virus

Aliya Momotaz¹, John W. Scott^{2,4}, and David J. Schuster³

Gulf Coast Research and Education Center, IFAS, University of Florida, 14625 CR 672, Wimauma, FL 33598

Additional index words. CAPS (cleaved amplified polymorphic sequence) marker, *Lycopersicon esculentum*, *Lycopersicon hirsutum*, tomato, *Solanum lycopersicum*

Abstract. Cultivated tomato (*Solanum lycopersicum* L.) accessions have been susceptible to the whitefly-transmitted begomoviruses *Tomato yellow leaf curl virus* (TYLCV) and *Tomato mottle virus* (ToMoV) that can cause serious crop damage. *S. habrochaites* accession LA1777 has been reported to be resistant to TYLCV. To locate putative virus resistance genes, 89 recombinant inbred lines (RILs) previously developed from LA1777 in a tomato background, LA1777 and the susceptible RIL parent E6203, were screened against the begomoviruses TYLCV and ToMoV. An initial study showed 18 RILs had less disease severity to TYLCV or ToMoV. Eight RILs had *S. habrochaites* alleles at TG27 (restriction fragment length polymorphism marker) on chromosome 1, three RILs had *S. habrochaites* alleles at TG202 on chromosome 7, and one RIL had *S. habrochaites* alleles at both marker loci. The RILs with these regions were intercrossed in 10 different cross combinations and F₂ seeds were then obtained. The F₂ progenies were inoculated separately with both viruses and then evaluated in the field. The F₂ plants with less disease severity were selected, but most did not have the markers from the hypothetical resistance regions. The F₃ progenies were then inoculated and rated for disease severity to both viruses. None of the F₃s demonstrated any increased level of resistance, even if derived from F₂s homozygous for the target regions from both chromosomes. All plants from every cross combination were susceptible for both TYLCV and ToMoV, suggesting that there is no begomovirus resistance in the LA1777 RIL population. Some limitations of capturing all genes in an RIL population derived from an outcrossing accession are discussed.

The begomoviruses, *Tomato yellow leaf curl virus* (TYLCV) and *Tomato mottle virus* (ToMoV), vectored by the silverleaf whitefly (*Bemisia argentifolii* Bellows & Perring) can cause severe losses in tomato production. Most cultivated tomatoes are extremely susceptible to these diseases, and yield losses can reach 100% if plants are infected in early growth stages, especially by TYLCV (Green and Kallo, 1994; Picó et al., 1996). Accessions of several wild *Solanum* species, including *S. habrochaites* S. Knapp & D.M. Spooner., *S. chilense* (Dunal) Reiche., *S. pimpinellifolium* (Jusl.) Mill., *S. cheesmanii* (L. Riley), and *S. peruvianum* (L.) Mill., have been reported by many authors to have

TYLCV resistance, and intensive breeding efforts have been undertaken to develop cultivars with TYLCV resistance (Scott, 2006). Several accessions of *S. habrochaites* have been reported to be resistant to TYLCV (Ji et al., 2007), and some of these accessions have been used in breeding programs for developing TYLCV-resistant lines. The accession of *S. habrochaites* B6013 has demonstrated excellent resistance to TYLCV from Taiwan to south India (Kallo and Banerjee, 1990), and resistance in this accession was reported to be controlled by two epistatic genes (Banerjee and Kallo, 1987). One resistant line, H24, has been developed from this accession and a gene controlling TYLCV (Ty-2) in it has been mapped to chromosome 11 (Hanson et al., 2000, 2006). The accession LA1777 was reported to be highly resistant to TYLCV (Fargette, 1991; Hanson, 1999; Ioannou, 1985; Moustafa, 1991) and also to ToMoV (Hanson, 1999). Another accession of *S. habrochaites*, LA0386, has also been reported as resistant to TYLCV with dominant control by more than one gene (Hassan et al., 1984). Picó et al. (2000) reported that LA0386 had a higher level of resistance than LA1777 when using agroinoculation. Vidavsky and Czosnek (1998) developed a TYLCV-resistant line named Ih902 from a cross between *S. habrochaites* accessions

LA1777 and LA0386 followed by crossing to *S. lycopersicum* and selfing resistant, symptomless individuals. The authors suggested that the resistance in this line is controlled by one major dominant gene with some minor genes. The line Ih902 has been evaluated and used as a broad-based source of resistance for begomovirus resistance breeding programs in Guatemala, where seven bipartite begomoviruses have been reported (Mejía et al., 2005; Nakhla et al., 2005), and for TYLCV resistance breeding programs in Middle Eastern countries (Maruthi et al., 2003). However, there is no report specifically describing the origin and chromosomal location of resistance gene(s) in line Ih902.

Recombinant inbred lines (RILs) derived from LA1777 in *S. lycopersicum* (Monforte and Tanksley, 2000), that cover ≈83% of the *S. habrochaites* genome, are available and provide a means to locate genes with resistance to TYLCV and other begomoviruses. Therefore, we screened 89 of these RILs for resistance to TYLCV and ToMoV. In an initial study, none of the RILs demonstrated a high level of resistance to either disease (Momotaz et al., 2005). However, 18 lines displayed potentially low levels of resistance to ToMoV or TYLCV. Nine of these lines had *S. habrochaites* alleles at TG27 [restriction fragment length polymorphism marker (RFLP)] on chromosome 1 and four of the lines had *S. habrochaites* alleles at TG202 on chromosome 7 (Momotaz et al., 2005). We hypothesized that minor genes in these regions on chromosomes 1 and 7 might provide better resistance when combined. Thus, the objective of this work was to combine RILs expressing low levels of resistance to determine if higher levels of resistance could be obtained and, thus, identify the location of genes from LA1777 resistant to TYLCV and ToMoV.

Materials and Methods

Spring and Fall 2004

In the Spring of 2004, crosses were made between RILs with markers that were correlated with the low levels of resistance from 2003 testing (Table 1). The developed hybrids were designated 401 to 410. Seeds were bulked from 10 plants of each F₁ hybrid grown in the field to obtain F₂ seeds in the Fall of 2004.

Spring 2005

In the Spring of 2005, seeds of the 10 F₂ lines, the susceptible control 'Horizon', and the resistant control line 927 (advanced breeding line derived from *S. chilense* accession LA1932) were sown in flats containing Black Beauty spent coal (Reed Minerals Div., Highland, IN) on 25 Jan. Seedlings of these lines were transplanted at the cotyledon stage into two sets of Speedling trays (3.8-cm² cell size; Speedling, Sun City, FL) in the greenhouse on 7 Feb. On 25 Feb., trays were moved into whitefly-proof greenhouses for virus inoculation.

Received for publication 18 Jan. 2007. Accepted for publication 14 Mar. 2007.

This research was funded by T-Star grant PL 89-106 from the USDA.

We are grateful to the Tomato Genetic Resource Center for providing the RIL seeds, to Cathy Provenzano for making crosses to Rosa Ayala, and other crew members for taking care of the plants.

¹Postdoctoral Research Associate

²Professor of Horticultural Science

³Professor of Entomology and Nematology

⁴To whom reprint requests should be addressed; e-mail jwsc@ufl.edu

Table 1. Crosses of recombinant inbred lines (RILs) that had partial begomovirus resistance from previous testing and restriction fragment length polymorphism markers (RFLPs) associated with the RILs.

Designation	RIL crosses	RFLP markers
401	LA3963 × LA3993	UI ² × TG27 (1) ³
402	LA3986 × LA3993	TG202 × TG27 (1)
403	LA3968 × LA3993	TG180 (12) × TG27 (1)
404	LA4001 × LA3993	TG27, TG202 × TG27 (1)
405	LA3913 × LA3950	TG27 × TG202 (7)
406	LA3915 × LA3950	TG27 × TG202 (7)
407	LA3970 × LA3950	TG27 × TG202 (7)
408	LA3993 × LA3950	TG27 × TG202 (7)
409	LA3963 × LA3950	UI × TG202 (7)
410	LA4001 × LA3950	TG27, TG202 × TG202 (7)

²Unidentified introgressed region in RIL LA3963.

³The numbers in parentheses includes the chromosome number.

Inoculation. Inoculation was done according to the method described by Griffiths and Scott (2001). Two *B. argentifolii* colonies viruliferous for TYLCV or ToMoV were maintained separately on plants of the dwarf tomato 'Florida Lanai'. Plants at the three leaf stage were inoculated separately for 16 d by placing them in growth chambers with TYLCV- or ToMoV-infested 'Florida Lanai' plants. There were two virus-infected source plants per ≈550 test plants. Each inoculation plant had eight to 12 leaves and each was heavily infested with whitefly adults and immature life stages. The source plants were shaken daily to disperse whitefly adults onto the test plants. On 14 Mar., whiteflies were killed with a foliar application of a 1% v/v solution of M-Pede® (potassium salts of fatty acids; Dow AgroSciences LLC, Indianapolis, IN). The trays were then drenched with Admire 2F® (imidacloprid; Bayer Crop-Science LP, Research Triangle Park, NC) at the rate of ≈3 mL/1000 plants. On 21 and 22 Mar., the inoculated seedlings of TYLCV and ToMoV, respectively, were transplanted into separate field plots for each disease.

Twenty plants per F₂ were planted and spaced 46 cm apart within rows with 152 cm between rows. The beds had been fumigated with 67% methyl bromide:33% chloropicrin at 197 kg·ha⁻¹ and covered with white plastic mulch. The plants were fertilized with a total of 293 kg·ha⁻¹ of nitrogen and 486 kg·ha⁻¹ of potassium placed in two bands 15 cm from the plants on top of the beds using granular fertilizer. The plants were staked and tied and irrigated by seepage ditches adjacent to three experimental beds. Plants were sprayed with pesticides as needed throughout the season.

Resistance assessment was began 45, 60, and 70 d after inoculation (DAI) in the field. Disease severity was rated using a 0 to 4 scale of Scott et al. (1996) in which 0 = no symptoms, 1 = slight symptoms visible only on close inspection, 2 = mild symptoms on part of the plant, 3 = severe symptoms over the entire plant, and 4 = severe symptoms and stunting. The plants were also subjectively rated for general vigor. A plant was considered tolerant if it had a lower virus disease severity rating with greater vigor. Data from the rating 70 DAI were used for analysis.

Fall 2005

Twenty F₂ plants with less disease were selected during the Spring of 2005 and their F₃ progenies were inoculated and evaluated in the Fall of 2005. Seeds of the 20 F₃, susceptible control 'Horizon', and resistant control 927 were sown in Black Beauty spent coal on 27 July. Seedlings were transplanted on 8 Aug. (17 d) into two sets of Speedling trays, and inoculation began on 26 Aug. On 12 Sept., whiteflies were killed and on 3 and 4 Oct., the seedlings inoculated with TYLCV and ToMoV, respectively, were then transplanted in separate field plots. Each of the genotypes had 10 plants. Fertilization, irrigation, and all other cultural practices were the same as in Spring of 2005.

DNA extraction and polymerase chain reaction amplification. Genomic DNA was isolated from fully expanded leaf tissues of the F₂ plants (selected for lower disease severity and more vigor) using a simple DNA isolation procedure (Fulton et al., 1995).

Cleaved amplified polymorphic sequence (CAPS) markers of RFLP clones TG27 (forward sequence 5'-TTGGGCGTTTAAAAA GTTGG; reverse sequence 5'- GCGGTTTT CAACAACCAAAA) and TG202 (forward sequence 5'-AGGATGCAATGGTGAGGA AG-3'; reverse sequence 5'-CACTGCATT GTGTCGAAC CT-3') were designed from the DNA sequences available at Sol Genomics Network (<http://sgn.cornell.com>) and the other CAPS TG180 markers used in this experiment were selected from Sol Genomics Network. The polymerase chain reaction (PCR) amplification reactions for each of the markers were performed according to the procedures of Momotaz et al. (2004) in a 10-μL reaction volume. PCR amplification was carried out with a DNA automated thermocycler (Gene Amp; Applied Biosystems, CA). Approximately 4 μL of the amplified DNA fragments obtained with oligonucleotides designed from the probes TG27 and TG202 were digested in a total volume of 10 μL for 2 h with 1.5 units of the *Tsp5091* and *DpnII* restriction endonuclease to yield codominant markers. Restriction fragments were separated on 2% (w/v) agarose gels (Genetic analysis grade; Fisher Scientific, Fair Lawn, NJ) containing ethidium bromide (1.0 μg/mL⁻¹) in 1 × TBE buffer visualized under ultraviolet transillumination and photographed using the AlphaImager Imaging System (San Leandro, CA).

Results

The resistant control 927 was highly resistant to both TYLCV and ToMoV (Table 2). A very high percentage of the F₂ plants were

Table 2. TYLCV and ToMoV disease severity frequency distributions and means at 70 d after inoculation began for F₂ genotypes in the Spring of 2005.

Genotypes	Disease	Disease severity						Mean ^a
		0	1	2	2.5	3	4	
401	TYLCV	0	0	0	0	1	18	3.8
	ToMoV	0	0	0	0	0	20	3.9
402	TYLCV	0	1	0	0	7	12	3.5
	ToMoV	0	0	0	0	1	19	3.8
403	TYLCV	0	0	0	0	5	15	3.6
	ToMoV	0	1	0	1	1	17	3.6
404	TYLCV	0	0	0	2	4	14	3.5
	ToMoV	0	0	1	0	2	17	3.6
405	TYLCV	0	0	0	1	5	14	3.7
	ToMoV	0	0	0	0	1	19	4.0
406	TYLCV	0	0	0	0	0	20	3.7
	ToMoV	0	1	0	0	0	19	3.8
407	TYLCV	0	0	0	0	5	15	3.5
	ToMoV	0	0	0	0	0	18	4.0
408	TYLCV	0	0	0	0	2	18	3.9
	ToMoV	0	0	0	0	0	20	4.0
409	TYLCV	0	0	0	0	2	17	3.8
	ToMoV	0	0	0	0	3	17	3.7
410	TYLCV	0	0	2	2	2	14	3.5
	ToMoV	0	0	0	0	1	19	3.9
Horizon	TYLCV	2	0	4	1	3	25	3.3
	ToMoV	6	7	2	0	0	17	2.3
927 ^b	TYLCV	18	2	0	0	0	0	0.1
	ToMoV	14	4	0	1	0	0	0.4

^aBased on 20 plants per line per disease.

^bHorizon is the susceptible control and 927 is the resistant control.

TYLCV = Tomato yellow leaf curl virus; ToMoV = Tomato mottle virus.

Table 3. Marker analysis of some selected F₂ plants with their individual disease severity ratings for TYLCV and ToMoV.

Line	Possible marker (s) ^z	TG27	TG202	TG180	Disease severity ^y		
					45 DAI	60 DAI	70 DAI
401-Y10 ^x	TG27 only	—	—	—	1.5	2.0	3.0
409-6	TG202 only	—	e ^w	—	0.5	2.5	3.5
409-19	—	—	e	—	2.0	3.0	3.5
403-Y3	TG180 × TG27	h	—	—	2.0	2.0	3.0
403-Y18	—	he	—	—	2.0	2.0	3.0
403-Y19	—	he	—	—	1.5	2.0	3.0
403-7	—	he	—	e	0.5	1.5	2.5
403-8	—	he	—	e	0.5	2.0	3.0
403-14	—	he	—	e	0.5	0.5	1.0
402-Y4	TG202 × TG27	h	e	—	1.0	1.0	1.0
402-Y10	—	h	e	—	2.0	2.0	3.0
402-Y11	—	he	e	—	2.5	2.5	3.0
402-3	—	h	e	—	0.0	2.0	3.0
405-Y4	—	—	—	—	1.5	1.5	2.5
405-7	—	e	e	—	0.0	2.0	3.5
406-14	—	he	e	—	1.0	1.0	1.0
404-Y3	TG27, TG202 × TG27	h	he	—	2.0	2.0	2.5
404-Y20	—	h	he	—	1.0	1.5	2.5
404-3	—	h	e	—	0.5	2.0	3.0
404-13	—	h	he	—	1.0	1.5	2.0
410-Y4	TG27, TG202 × TG202	—	—	—	1.5	1.5	2.0
410-Y18	—	h	h	—	1.5	1.5	2.0
410-2	—	h	h	—	0.0	2.0	3.0

^zSee Table 1.

^yDisease severity for TYLCV or ToMoV depending on selection; see footnote to z. Nonselected plants were 3.0 at 45 DAI.

^xGenotypes with a “Y” in the suffix were selected from TYLCV-inoculated plants; if no y, then selected plants from ToMoV-inoculated plots.

^wh = homozygous for *S. habrochaites* allele; e = homozygous for *S. lycopersicum* allele; he = heterozygote.

TYLCV = *Tomato yellow leaf curl virus*; ToMoV = *Tomato mottle virus*; DAI = days after inoculation.

severely infected with both diseases and only 4% of TYLCV-inoculated and 2% of ToMoV-inoculated plants had early ratings less than 3 (Table 2). The lack of plants with intermediate ratings is a pattern favoring a few escapes as opposed to the few plants having resistance. Accordingly, most of the plants with earlier lower ratings eventually became more heavily infected and were considered susceptible in later ratings. Nevertheless, 23 plants that had lower ratings in the early season were selected and analyzed for their potential markers (Table 3). Very few were homozygous for marker(s) from the hypothetical resistance regions. Thus, it does not appear that these regions are linked to resistance genes. Seeds were sown from 20 of the 23 F₂ plants and F₃ progenies were evaluated for both diseases in the following season (Table 4). The F₃s had very high percentages (97% TYLCV inoculated and 92% ToMoV inoculated) of severely infected plants and some had a few plants less severely infected. There was no increase in resistant progeny as would be expected if parents had resistance. The genotype 927 was highly resistant like in the previous season. ‘Horizon’ had some escapes, especially for ToMoV, and actually had more plants with intermediate disease severity for TYLCV than did the F₃s for reasons unknown to us.

Discussion

We found no evidence for resistance to TYLCV or ToMoV in lines derived from RILs that were combined based on their more vigorous growth after inoculation in an earlier study (Momotaz et al., 2005). Further evidence that regions on chromosomes 1 and 7 were not associated with TYLCV or ToMoV resistance was found when CAPS markers linked to *S. habrochaites* introgressions were absent in line lh902 (data not shown). Line lh902 was derived from *S. habrochaites* (Vidavsky and Czosnek, 1998) and has been highly resistant to begomoviruses in Guatemala, where it has been used in a begomovirus resistance breeding program (Mejía et al., 2005). The present study along with our previous one (Momotaz et al., 2005) strongly indicate that resistance genes for TYLCV or ToMoV are not present in the 83% of the *S. habrochaites* genome covered by the RIL population used in our experiments. Resistance gene(s) derived by Vidavsky and Czosnek (1998) may be introgressed from LA386 or from part of the LA1777 genome not present in the RILs. This could include the ≈17% not present in RILs or other genes not represented by the RILs. The latter is possible because *S. habrochaites* accession LA1777 is an outcrossing species,

Table 4. TYLCV and ToMoV disease severity frequency distributions and means 70 d after inoculation began for 20 F₃ genotypes in the Fall of 2005.

Genotypes ^z		Disease severity						
		0	1	2	2.5	3	4	Mean ^y
401-Y10	TYLCV	0	0	0	0	3	7	3.6
	ToMoV	0	1	1	0	0	7	2.7
402-3	TYLCV	0	0	0	0	6	4	3.3
	ToMoV	0	0	0	0	2	8	3.6
402-Y4	TYLCV	0	0	0	1	2	7	3.3
	ToMoV	0	0	0	0	2	7	3.5
402-Y10	TYLCV	0	0	1	0	6	2	2.7
	ToMoV	0	1	1	0	0	8	3.2
402-Y11	TYLCV	0	0	0	1	0	7	3.4
	ToMoV	0	2	1	0	0	7	2.9
403-7	TYLCV	0	0	1	0	5	4	3.1
	ToMoV	0	0	0	0	2	8	3.0
403-8	TYLCV	0	0	0	0	3	7	3.7
	ToMoV	0	0	0	1	0	9	3.4
403-14	TYLCV	0	0	0	0	3	6	3.3
	ToMoV	0	0	0	0	0	10	3.6
403-Y3	TYLCV	0	0	0	0	0	9	3.5
	ToMoV	0	0	0	0	0	9	3.5
403-Y18	TYLCV	0	0	0	0	0	1	3.5
	ToMoV	0	0	0	0	0	7	3.5
403-Y19	TYLCV	0	0	0	0	2	7	3.4
	ToMoV	0	0	0	0	0	10	3.5
404-3	TYLCV	0	0	0	0	8	1	3.1
	ToMoV	0	0	0	1	4	5	3.2
404-13	TYLCV	0	0	0	0	9	1	3.1
	ToMoV	0	0	0	1	2	7	3.3
404-Y3	TYLCV	0	0	0	0	1	6	3.8
	ToMoV	0	0	0	0	1	9	3.5
404-Y20	TYLCV	0	0	0	0	0	10	3.5
	ToMoV	0	0	0	0	1	9	3.5
405-7	TYLCV	0	0	0	1	2	4	3.3
	ToMoV	0	0	0	0	2	8	3.4
405-Y4	TYLCV	0	0	0	0	0	2	4.0
	ToMoV	0	0	0	0	0	1	3.5
406-14	TYLCV	0	0	0	0	0	7	3.9
	ToMoV	0	0	1	2	0	7	3.2
409-6	TYLCV	0	0	0	0	5	3	3.2
	ToMoV	0	0	0	0	1	9	3.6
410-2	TYLCV	0	0	0	0	1	7	3.5
	ToMoV	0	0	0	0	1	9	3.6
Horizon	TYLCV	1	0	5	9	5	10	2.9
	ToMoV	11	1	1	1	16	8	2.2
927	TYLCV	24	0	0	0	0	0	0.1
	ToMoV	17	4	2	0	0	0	0.4

^zGenotypes were F₃ lines from F₂ plants selected for less disease severity; those with a “Y” in the suffix were from plants inoculated with TYLCV; if no y, then selected from ToMoV-inoculated plots. Horizon = susceptible control; 927 = resistant control.

^yBased on 10 plants per line per disease except for controls; horizon = 30 and 38 plants for TYLCV and ToMoV, respectively; 927 = 24 and 23 plants for TYLCV and ToMoV, respectively.

TYLCV = *Tomato yellow leaf curl virus*; ToMoV = *Tomato mottle virus*.

in which resistance alleles may not be fixed. The RILs were derived from a single plant (Bernacchi and Tanksley, 1997) that likely did not have all of the genetic variation of LA1777. Further evidence for this possibility emerged from our LA1777 whitefly resistance work. We detected regions that may be associated with silverleaf whitefly resistance based on CAPS markers and an interspecific F₂ population. However, three of the RILs that should have had a CAPS marker did not have the polymorphism (data not shown). At present, the source(s) of resistance reported

by Vidavsky and Czosnek (1998) remain unknown. Picó et al. (2000) provided evidence that LA0386 had better TYLCV resistance than LA1777 and it may be that the resistance was derived from LA0386. In this work, agroinoculation was used, which would circumvent infection by the whitefly. This is important because LA1777 has a high density of type IV glandular trichomes and is not attractive to the whitefly (see Muigai et al., 2003). Some previous studies reporting resistance to TYLCV from LA1777 have not addressed this issue. However, resistance from LA1777 cannot be ruled out unequivocally, although we have shown that the resistance is not present in the LA1777 RILs. Other researchers should be aware of this before they attempt to locate resistance in the LA1777 RIL population.

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