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Resistance to Race T2 of the Bacterial Spot Pathogen in Tomato

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Abstract. **Thirty-two tomato (*Lycopersicon esculentum* Mill.) or *L. pimpinellifolium* (L.) Mill. accessions were inoculated with race T2 of *Xanthomonas campestris* pv. *vesicatoria* (Xcv) in a field experiment at Wooster, Ohio, in 1995. Plants from accessions which segregated for race T2 resistance in greenhouse tests were selected and these are designated by hyphenated extensions below. The eight most resistant accessions from 1995 and PI 262173 were retested in 1996. *Lycopersicon esculentum* accession PI 114490-1-1 had virtually no Xcv symptoms either year. *Lycopersicon pimpinellifolium* accessions LA 442-1-Bk and PI 128216-T2 expressed a high level of resistance in 1995, but only partial resistance in 1996. Accessions with partial resistance for both seasons were PI 79532-S1, PI 155372-S1, PI 126428, PI 271385, PI 195002, PI 262173, Hawaii 7998, and Hawaii 7983. PI 79532-S1 is a *L. pimpinellifolium* accession and the remaining seven are *L. esculentum*. Twenty accessions tested in 1995 for T2 plus 10 other accessions were also tested for race T1 resistance in Presidente Prudente, Sao Paulo, Brazil, in 1993. Hawaii 7983, PI 155372-S1, PI 114490, PI 114490-S1, and PI 262173 had greater resistance to T1 than the susceptible control, 'Solar Set'. Comparisons with earlier experiments, in which accessions were inoculated with race T1 or T3, indicated that the most consistent source of resistance to all three races was PI 114490 or selections derived from it.**

Bacterial spot incited by *Xanthomonas campestris* pv. *vesicatoria* (Xcv) causes yield losses (Pohronezny and Volin, 1983) in tomato under periods of high temperature and rainfall (Cox, 1966; Nayudu and Walker, 1960). Despite extensive breeding efforts (Scott et al., 1991, 1995), breeding for resistance to bacterial spot in tomato is difficult, as evidenced by the lack of resistant commercial cultivars. Three known races of the pathogen that attack tomato contribute to the problem (Jones et al., 1995). In Florida, only one race (T1) was known prior to 1989. Strains of this race induce a hypersensitive response in Hawaii 7998 (Bouzar et al., 1994; Jones and Scott, 1986). Breeding for resistance to bacterial spot in Florida was based on incorporation of resistance from Hawaii 7998 into improved inbreds. However, a strain from Brazil was discovered that did not induce a hypersensitive response on Hawaii 7998 (Wang et al., 1990), and this strain was designated T2 (Bouzar et al., 1994). More recently, another strain was isolated in Florida that did not induce hypersensitivity on Hawaii 7998, was distinct from race T2, and was designated race T3 (Jones et al., 1995). In vitro studies indicated that race T3 is antagonistic to T1 (El Morsy et al., 1994) and T3 is becoming the predominant race of Xcv in Florida tomato fields (J.B. Jones, unpublished data). Resistance to T3 strains was discovered in several accessions, with the highest level in Hawaii 7981 (Scott et al., 1995). Hawaii 7981 was susceptible to T1 in a 1984 field trial (J.W. Scott and J.B. Jones, unpublished). No resistance has been reported to race T2.

Since no known Xcv resistant cultivars have been released, it appears that the three races have evolved independently of host resistance. If a cultivar resistant to races T1 and T3 were to be developed and widely grown, race T2 or another virulent race might emerge. Therefore, resistance to race T2 must be identified and incorporated into tomato along with resistance to T1 and T3. The primary objective of this paper is to report the field reactions of *Lycopersicon* germplasm screened for resistance to Xcv race T2. The secondary objective is to report their field reactions to race T1 and compare these data to published results with races T1 and T3.

Materials and Methods

Germplasm. We tested 32 accessions of *Lycopersicon* for bacterial spot race T2 resistance at Wooster, Ohio, in 1995 (Table 1). 'Solar Set' was the susceptible control. Hawaii 7998 and Hawaii 7981 were the race T1 and race T3 resistant controls, respectively. 'Ontario 7710' was the bacterial speck [*Pseudomonas syringae* pv. *tomato* (Okabe) Young, Dye, and Wilkie] resistant control. 'Campbell-28' was tested since it has partial resistance to T1 and, possibly, T3. Other entries included six additional Hawaiian lines originally bred for bacterial wilt [*Pseudomonas solanacearum* (Smith) Smith] resistance, as were Hawaii 7998 and Hawaii 7981. Plant introduction (PI) 127805 was tested since it may be the progenitor of the above Hawaiian lines (Rouamba et al., 1988). Two entries with observed Xcv resistance in Brazil, PI 195002 and M22, were supplied by K. Kurosawa at Botucatu Univ., Botucatu, Sao Paulo, Brazil, and V. Barbosa, respectively. Five accessions with observed Xcv resistance in Bulgaria (LCH 233, FG 198, LCH 162, LCH 165, and LCH 167) were provided by V. Sotirova of the Institute of Genetics and Plant Breeding, Sofia, Bulgaria.

The remainder of the accessions were tested in Wooster as a result of screening for race T2 resistance in greenhouses at Gainesville, Fla.

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Table 1. Bacterial spot tomato race 2 (T2) disease severity for genotypes at Wooster, Ohio, Summer 1995.

Accession[z]	Species	Disease severity[y]
LCH 233	*Lycopersicon esculentum*	6.5 a[x]
Ontario 7710	*L. esculentum*	6.5 a
PI 273445	*L. esculentum*	6.3 ab
FG 198	*L. esculentum*	6.3 ab
LCH 162	*L. esculentum*	6.3 ab
Solar Set	*L. esculentum*	6.0 a–c
LCH 165	*L. esculentum*	5.8 a–d
Hawaii 7976	*L. esculentum*	5.8 a–d
Hawaii 7997	*L. esculentum*	5.5 b–e
Hawaii 7975	*L. esculentum*	5.3 c–e
PI 126932-1-2	*L. pimpinellifolium*	5.3 c–e
Hawaii 7981	*L. esculentum*	5.3 c–e
PI 127805	*L. pimpinellifolium*	5.0 d–f
Hawaii 7982	*L esculentum*	5.0 d–f
M22	*L esculentum*	5.0 d–f
PI 127807	*L. pimpinellifolium*	5.0 d–f
Hawaii 7983	*L. esculentum*	5.0 d–f
LCH 167	*L. esculentum*	5.0 d–f
PI 262173	*L. esculentum*	5.0 d–f
Hawaii 7996	*L. esculentum*	4.8 e–g
Campbell 28	*L. esculentum*	4.8 e–g
Hawaii 7998	*L. esculentum*	4.8 e–g
PI 126428	*L. esculentum*	4.3 f–h
PI 195002	*L. esculentum*	4.0 gh
PI 155372-S1	*L. esculentum*	4.0 gh
1218 F_3	*L. esculentum*	3.8 h
PI 79532-S1	*L. pimpinellifolium*	3.5 h
PI 271385	*L. esculentum*	3.5 h
216155 F_3	*L. esculentum* x *L. pimpinellifolium*	2.3 i
PI 114490-1-1	*L. esculentum*	2.0 i
PI 128216-T2	*L. pimpinellifolium*	2.0 i
LA 442-1-BK	*L. pimpinellifolium*	2.0 i

[z]Accessions with hyphenated extensions were derived from the accession by previous selection for race T2 resistance in the greenhouse.
[y]Horsfall–Barratt scale (1–12), lower value indicates less disease.
[x]Mean separation in column by Duncan's multiple range test at $P \leq 0.05$.

More than 200 accessions with some reported bacterial disease resistance were originally field tested for resistance to race T1 (Scott and Jones, 1985). Wang (1992) tested these in Gainesville for race T2 hypersensitivity by injecting 10^8 colony-forming-units (cfu)/mL of inoculum into a leaflet and observing for necrosis during 48 h of incubation. Wang also tested the accessions for quantitative resistance by a dip test where plant foliage was immersed for 15 s into inoculum of 10^6 cfu/mL containing 0.025% Silwet L-77 (Loveland Industries, Greeley, Colo.). No hypersensitivity occurred among the accessions, but selections among and within accessions were made for quantitative resistance based on expression of small and few lesions. Eight accessions, or selections derived from them (those with hyphenated extensions below), were ultimately selected for field testing (PI 128216-T2, PI 114490-1-1, PI 271385, PI 79532-S1, PI 126428, PI 262173, PI 127807, and PI 126932-1-2). Additionally, 134 accessions consisting of wild species obtained from C.M. Rick, Tomato Genetics Resource Center, Davis, Calif., representing *L. pimpinellifolium*, *L. cheesmanii* Riley, *L. chmielewskii* Rick et al., *L. parviflorum* Rick et al., and *L. esculentum* var. *cerasiforme*, were tested by R.E. Stall for resistance to T1, T2, and T3. From this group, *L. pimpinellifolium* accession LA 442 was selected for T2 resistance based on a slow hypersensitive response (confluent necrosis) and small and few lesions from the dip test. Two F_3 lines were derived by intercrossing accessions followed by the above selection procedure for T2 resistance. Line 1218 was derived from a cross of PI 114490 and Hawaii 7998, while line 216155 was derived from a cross of PI 128216 and PI 155372.

Race T2 —Wooster, 1995. Seed from the above cultigens were sown on 5 Apr. 1995 in a greenhouse at Wooster, Ohio. On 23 May they were transplanted to the field in a randomized complete-block design with four blocks and five plants per plot and grown on bare ground, unstaked. There was 31 cm between plants, 1.5 m between plots, and 1.5 m between rows. The highly susceptible variety 'Easy Harvest' was planted between treatments as border rows. Chlorothalonil (tetrachloroisophthalonitrile) was used to control foliar fungal pathogens and insecticides were used to control insect pests.

Plants were spray-inoculated with a mixture of the following T2 strains; 500, 519b, 561, and 572a. The bacteria were suspended in 0.01 M $MgSO_4 \cdot 7\ H_20$ adjusted to $\approx 10^8$ cfu/mL and sprayed with a backpack sprayer between 20:30 and 21:30 HR on 10 July. Plants were reinoculated as above, on 17 July, with the same strains and inoculum concentration.

Plants were rated for disease severity on 18 Aug. by inspecting each plant and then giving an overall rating for the plot using the scale of Horsfall and Barratt (1945). The Horsfall–Barratt scale translates percentage of diseased tissue to numbers, where 1 = 0%, 2 = 0% to 3%, 3 = 3% to 6%, 4 = 6% to 12%, 5 = 12% to 25%, 6 = 25% to 50%, 7 = 50% to 75%, 8 = 75% to 87%, 9 = 87% to 94%, 10 = 94% to 97%, 11 = 97% to 100%, and 12 = 100% diseased tissue. Data were tested by analysis of variance, and significant differences among treatment means were determined using Duncan's multiple range test at $P \leq 0.05$.

Race T2 —Wooster, 1996. Controls were the same as for the 1995 experiment, except Ohio 88119 was added as another susceptible control. The eight accessions with the lowest 1995 ratings, except for the two F_3 lines (Table 1), were tested in 1996, as were PI 262173 and Hawaii 7983. Seed was sown on 3 Apr. 1996 in a greenhouse at Wooster, Ohio. On 30 May, seedlings were transplanted to the field without border rows between the treatment rows. Plants were inoculated on 2 and 11 July and rated for disease severity on 22 Aug. All other procedures were the same as described for Wooster–1995. The data for this experiment were included after submission of this report, but before final review and acceptance.

Race T1—Brazil, 1993. This experiment was conducted at the CICA Research Station, Presidente Prudente, Sao Paulo, Brazil. There

Table 2. Bacterial spot tomato race 2 (T2) disease severity for genotypes at Wooster, Ohio, Summer 1996.

Accession[z]	Species	Disease severity[y]
Ontario 7710	*Lycopersicon esculentum*	8.3 a[x]
Ohio 88119	*L. esculentum*	7.8 a
Solar Set	*L. esculentum*	5.8 bc
Hawaii 7981	*L. esculentum*	5.0 c–e
PI 128216-T2	*L. pimpinellifolium*	4.8 de
PI 262173	*L. esculentum*	4.5 d–g
LA 442-1-BK	*L. pimpinellifolium*	4.3 d–g
Hawaii 7998	*L. esculentum*	4.3 d–g
Hawaii 7983	*L. esculentum*	4.3 d–g
PI 271385	*L. esculentum*	4.3 d–g
PI 195002	*L. esculentum*	4.0 e–g
PI 126428	*L. esculentum*	3.8 f–h
PI 79532-S1	*L. pimpinellifolium*	3.5 gh
PI 155372-S1	*L. esculentum*	3.0 h
PI 114490-1-1	*L. esculentum*	2.0 i

[z]Accessions with hyphenated extensions were derived from the accession by previous selection for race T2 resistance in the greenhouse.
[y]Horsfall–Barratt scale (1–12), lower value indicates less disease.
[x]Mean separation in column by Duncan's multiple range test at $P \leq 0.05$.

Table 3. Bacterial spot tomato race 1 (T1) disease severity for genotypes at Presidente Prudente, Sao Paulo, Brazil, Spring 1993.

Accession[z]	Species	Disease severity[y]
PI 126932	*Lycopersicon esculentum*	6.0 a[x]
PI 128643	*L. peruvianum*	5.7 ab
PI 79532	*L. pimpinellifolium*	5.7 ab
PI 128216-S1	*L. pimpinellifolium*	5.7 ab
PI 340905	*L. pimpinellifolium*	5.7 ab
PI 79532-S1	*L. pimpinellifolium*	5.3 ab
PI 127807	*L. pimpinellifolium*	5.3 ab
Hawaii 7981	*L. esculentum*	5.3 ab
Hawaii 7997	*L. esculentum*	5.3 ab
PI 306216	*L. esculentum*	5.3 ab
PI 324707	*L. esculentum*	5.3 ab
PI 244672	*L. esculentum*	5.3 ab
PI 128216	*L. pimpinellifolium*	5.0 a–c
PI 273445	*L. esculentum*	5.0 a–c
Solar Set	*L. esculentum*	5.0 a–c
Hawaii 7982	*L. esculentum*	4.7 a–c
PI 99782	*L. esculentum*	4.7 a–c
Campbell 28	*L. esculentum*	4.7 a–c
Hawaii 7975	*L. esculentum*	4.7 a–c
PI 126428	*L. esculentum*	4.3 b–d
Hawaii 7996	*L. esculentum*	4.3 b–d
PI 271385	*L. esculentum*	3.7 c–e
Hawaii 7998	*L. esculentum*	3.7 c–e
PI 155372	*L. esculentum*	3.7 c–e
Hawaii 7976	*L. esculentum*	3.7 c–e
PI 114490	*L. esculentum*	3.0 d–e
PI 262173	*L. esculentum*	3.0 d–e
PI 114490-S1	*L. esculentum*	3.0 d–e
PI 155372-S1	*L. esculentum*	2.7 e
Hawaii 7983	*L. esculentum*	2.7 e

[z]Accessions with hyphenated extensions were derived from the accession by previous selection for race T2 resistance in the greenhouse.
[y]Horsfall–Barratt scale (1–12), lower value indicates less disease.
[x]Mean separation in column by Duncan's multiple range test at $P \leq 0.05$.

were 30 accessions tested; these included Hawaii 7998 and 21 others that were tested for race T3 resistance in the 1992 trial at Bradenton (Scott et al., 1995), PI 128216, and the seven other Hawaiian lines tested in Wooster–1995. The seed were sown on 22 Dec. They were transplanted to the field in a randomized complete-block design with three blocks and five plants per plot on 19 Jan. and grown on bare ground, unstaked. Spacings were 40 cm between plants, 80 cm between plots, and 1.3 m between rows. Disease was from natural infection since no artificial inoculation was carried out. Disease severity was rated on 13 Apr. using the Horsfall–Barratt scale noted earlier. The data were also analyzed as previously described. Analysis of 11 leaf samples taken from five locations in the field indicated that only race T1 was present.

Results and Discussion

Race T2 —Wooster 1995 and 1996. The disease pressure in Wooster was high in both years as indicated by the reaction of the susceptible control 'Solar Set' both years (Tables 1 and 2). The T2 strains also caused susceptible reactions in the genotypes with resistance to bacterial speck (Ontario 7710) and to bacterial spot race T3 (Hawaii 7981) both years. Plants of the race T1 resistant Hawaii 7998 and Hawaii 7983 had significantly less disease than 'Solar Set' both years, but more disease than would be expected if the pathogen had been race T1. T1 resistant genotypes possibly have a low level of resistance to T2. Sampling of infected leaves in 1996 indicated only T2 was present.

PI 114490-1-1 had no visible lesions in either season and was designated as resistant to T2 (Tables 1 and 2). Early blight (*Alternaria solani,* Ell. and Mart.) often occurred on the plants, sometimes causing defoliation of lower leaves. Thus, no scores of 1 were assigned since there might have been some Xcv lesions on the shed leaves. LA 442-1-Bk and PI 128216-T2 expressed no Xcv lesions in 1995, but were only slightly more resistant than 'Solar Set' in 1996. Accessions with partial resistance for both years were PI 79532-S1, PI 155372-S1, PI 126428, PI 271385, PI 195002, PI 262173, Hawaii 7998, and Hawaii 7983. Several accessions had less disease than 'Solar Set' in 1995 but were not tested in 1996. Most of these were rated 4.8 or 5.0 (Table 1). Whereas these accessions may indeed have some resistance to T2, this level of resistance probably would not be useful to tomato breeders. The two F_3 selections, 216155 and 1218, had good resistance in 1995, indicating the

Table 4. Summary of the reactions of *Lycopersicon* accessions to the three known tomato races of the bacterial spot pathogen.

		Disease severity[z]					
		Race T1		Race T2		Race T3	
Accession[y]	Species	Fla. 1982[x]	Brazil 1993	Ohio 1995	Ohio 1996	Fla. 1992[w]	Fla. 1993[w]
Hawaii 7998[v]	*L. esculentum*	---	3.7	4.8	4.3	4.0	3.5
Hawaii 7981[u]	*L. esculentum*	---	5.3	5.3	5.0	---	1.3
PI 114490-1-1	*L. esculentum*	4.3	3.0	2.0	2.0	2.3	3.5
PI 128216-T2	*L. pimpinellifolium*	5.2	5.7	2.0	4.8	3.7	2.2
LA 442-1-Bk	*L. pimpinellifolium*	---	---	2.0	4.3	---	---
PI 79532-S1	*L. pimpinellifolium*	5.2	5.3	3.5	3.5	4.0	4.3
PI 155372-S1	*L. esculentum*	5.2	2.7	4.0	3.0	3.7	3.0
PI 126428	*L. esculentum*	4.8	4.3	4.3	3.8	3.0	3.3
PI 271385	*L. esculentum*	4.1	3.7	3.5	4.3	2.8	4.3
PI 195002	*L. esculentum*	---	---	4.0	4.0	---	---
PI 262173	*L. esculentum*	4.4	3.0	5.0	4.5	4.2	3.8

Solar Set		---	5.0 (3.7)[t]	6.0 (5.3)	5.8 (5.0)	4.7 (4.0)	4.7 (4.0)

[z]Horsfall–Barratt scale (1–12), lower value indicates less disease.
[y]Accessions with hyphenated extensions were derived from the accession by previous selection for resistance to T2 in the greenhouse. (See footnote x).
[x]Data are the ratings designated RE in Scott and Jones (1985). Values <5 probably indicate some resistance. All data are for original accessions, not selections of them.
[w]Data from Scott et al. (1995).
[v]Resistance to T1 documented elsewhere (Jones and Scott, 1986).
[u]Susceptible to race T1 in 1984 testing where Horsfall–Barratt score was 7 (J.W. Scott and J.B. Jones, unpublished).
[t]Number in parentheses indicates lowest value that was not significantly different from the susceptible control 'Solar Set' by Duncan's multiple range test at $P \leq 0.05$.

greenhouse dip test for T2 was effective in these two cases.

Race T1—Brazil, 1993. The only accessions with significantly less Xcv T1 disease symptoms than 'Solar Set' were Hawaii 7983, PI 155372-S1, PI 114490, PI 114490-S1 (PI 114490-1-1 is a selection out of PI 114490-S1), and PI 262173 (Table 3). All three replications of 'Solar Set' were rated 5, whereas all accessions with ratings <4 did not have any replications with ratings of 5, except for Hawaii 7976 (data not shown). This group included Hawaii 7998, a known source of resistance to T1. Thus, accessions with ratings of <4 may have at least partial resistance to T1. This group consisted of PI 155372 and PI 271385.

Comparison with earlier T1 and T3 experiments. Many of the accessions that expressed resistance to T2 and/or T1 in the experiments described above had been rated at Bradenton, Fla., for T1 disease severity in 1982 (Scott and Jones, 1985) and for T3 in 1992 and 1993 (Scott et al., 1995) and are presented for comparison with the recent data (Table 4). Accessions that had relatively good T1 resistance in Brazil and Florida were PI 114490, PI 271385, and PI 262173 (Table 4). No extension designations have been used since the original PIs were tested in 1982. Both PI 155372 and PI 155372-S1 were partially resistant (based on the reaction of Hawaii 7998) in Brazil, but PI 155372 was not resistant in Florida. Further testing of PI 155372 and PI 155372-S1 with T1 would be needed to better characterize their responses.

Scott et al. (1995) reported that accessions PI 128216, PI 114490, PI 126428, and PI 155372 were resistant to T3 in 2 years of testing (Table 4). The first accession was hypersensitive to T3 while the latter three accessions were not. The most consistent source of resistance to all three known tomato races of Xcv is from PI 114490 or selections derived from it (Table 4). A second possible source of resistance to all three races is PI 155372-S1. However, PI 155372-S1 was not as resistant as PI 114490-1-1 to T2, and its reaction to T1 should be tested further. PI 262173 had partial resistance to all three races, but the levels were not impressive, especially for T2 or T3. LA 442, which was inconsistent in response to T2 inoculation, has not been tested in the field for T1 resistance. Plants of LA 442 were hypersensitive to T3 (R.E. Stall, unpublished data), but it did not appear to be resistant in the field in the summers of 1995 and 1996 at Bradenton (J.W. Scott et al., unpublished). PI 128216-T2 and PI 126428 were partially resistant to T2 and T3, but not to race T1 (Table 4). PI 79532 expressed partial resistance only to T2 (Table 4). PI 195002 needs to be tested for resistance to T1 and T3.

Three races of Xcv that are virulent on tomatoes have been identified without any selection pressure from monocultures of resistant cultivars, since none exist. The use of race specific resistance could accelerate development of new virulent races. Thus, a logical breeding approach would be to use a broad spectrum resistance, like that of PI 114490-1-1, in future breeding efforts, either alone or combined with T1 and/or T3 resistance.

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