Tomato Bacterial Spot Resistance Derived from PI 114490; Inheritance of Resistance to Race T2 and Relationship across Three Pathogen Races

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ABSTRACT. Crosses were made between tomato (*Lycopersicon esculentum* Mill.) inbreds susceptible to races T2 and T3 of bacterial spot (*Xanthomonas vesicatoria* and *Xanthomonas campestris* pv. *vesicatoria*, respectively) and accession PI 114490 with resistance to races T1, T2, and T3. Resistance to race T2 was analyzed using the parents, F₁, and F₂ generations from one of the crosses. The F₁ was intermediate between the parents for disease severity suggesting additive gene action. The segregation of F₂ progeny fit a two-locus model ($\chi^2 = 0.96$, $P = 0.9-0.5$) where four resistance alleles are required for a high resistance level, two or three resistance alleles provide intermediate resistance, and zero or one resistance allele results in susceptibility. The narrow sense heritability of resistance to T2 strains was estimated to be 0.37 ± 0.1 based on F₂ to F₃ parent-offspring regression. A second cross was developed into an inbred backcross (IBC) population to facilitate multilocation replicated testing with multiple races. Segregation for T2 resistance in the inbred backcross population also suggested control was by two loci, lending support to the two-locus model hypothesized based on the F₂ segregation. To determine if the same loci conferred resistance to the other races, selections for race T2 resistance were made in the F₂ and F₃ generations and for race T3 resistance in the F₂ through F₄ generations. Six T3 selections (F₃), 13 T2 selections (F₄'s that diverged from seven F₂ selections), and control lines were then evaluated for disease severity to races T1, T2, and T3 over two seasons. Linear correlations were used to estimate the efficiency of selecting for resistance to multiple races based on a disease nursery inoculated with a single race. Race T1 and race T2 disease severities were correlated ($r \geq 0.80$, $P < 0.001$) within and between years while neither was correlated to race T3 either year. These results suggest that selecting for race T2 resistance in progeny derived from crosses to PI 114490 would be an effective strategy to obtain resistance to both race T1 and T2 in the populations tested. In contrast, selection for race T3 or T2 will be less likely to result in lines with resistance to the other race. PI 114490 had less resistance to T3 than to T2 or T1. Independent segregation of T2 and T3 resistance from the IBC population derived from PI 114490 suggests that T3 resistance is not controlled by the same genes as T2 resistance, supporting the linear correlation data.

Bacterial spot is a serious disease of tomato (*Lycopersicon esculentum* Mill.) and pepper (*Capsicum annuum* L. var. *annuum*) worldwide. The disease is difficult to control under hot and rainy conditions typical of many tomato and pepper growing regions (Cox, 1966; Nayudu and Walker, 1960). At least two species of *Xanthomonas* cause bacterial spot on these hosts (Jones et al., 2000). Bacterial spot of pepper is caused primarily by *Xanthomonas campestris* pv. *vesicatoria*. At present there are 11 races ([http://](http://henderson.ces.state.nc.us/newsletters/veg/00-05/)

henderson.ces.state.nc.us/newsletters/veg/00-05/) that infect pepper (Astua-Monge et al., 2000; Bouzar et al., 1994; Kousik and Ritchie, 1996; Sahin and Miller, 1995). Pepper cultivars have been developed with three dominant resistance genes that provide hypersensitive resistance to nine (0, 1, 2, 3, 4, 5, 7, 8, 9) of the eleven *X. campestris* pv. *vesicatoria* races. Another gene not yet present in pepper cultivars provides resistance to five pepper races, including race 6 (Sahin and Miller, 1998). The resistant pepper cultivars have been effective for control on many farms, but the emergence of virulent races has caused problems on some farms and is reason for concern for future control (Gassmann et al., 2000).

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In contrast, both *X. campestris* pv. *vesicatoria* and *X. vesicatoria* are known to cause bacterial spot in tomato. These two species contain three reported races (T1 and T3 in *X. campestris* pv. *vesicatoria* and T2 in *X. vesicatoria*). For simplicity, these species will be referred to by their race designation. The tomato races have developed in the absence of resistant cultivars. Resistance to race T1 from Hawaii 7998 is complex being controlled by multiple hypersensitive genes (Wang et al., 1994; Whalen et al., 1993; Yu et al., 1995) and other genes (Scott and Jones, 1989). Before cultivars resistant to race T1 could be developed in Florida, T3 emerged and has now largely replaced T1 (Jones et al., 1998). Hypersensitive resistance to race T3 was discovered in several accessions (Jones et al., 1995) and the inheritance from one of the sources, Hawaii 7981, is conferred by an incompletely dominant hypersensitivity gene (Scott et al., 1996) and other genes (Scott et al., 2001). Recently a fourth race has been identified that overcomes T3 resistance (Minsavage et al., unpublished). Resistance to this race was found in *L. pennellii* accession LA716 (Astua-Monge et al., 2000) but little breeding work with this resistance source has been done to date. A durable resistance in tomato that would be effective across races would be desirable. A non-hypersensitive resistance to race T2 from PI 114490 has been reported and this accession was also resistant to race T1 and T3 (Scott et al., 1997). PI 114490 is an indeterminate cherry (var. *cerisforme*) tomato with yellow fruit sent to the USDA in 1936 from the Royal Botanic Garden, U.K. In Summer 2002 there was a severe outbreak of the fourth race at Bradenton, Fla. and it was determined that PI 114490 had resistance (Scott, unpublished data). As PI 114490 provides the only known source of resistance to race T2 and may provide a broad-spectrum resistance, it would be useful to determine how many genes are involved. It would also be useful to know if the genes controlling resistance are the same or different for each race. If they are the same, one could select for one race and thereby obtain resistance to other races. The objectives of this report are 1) to report on the inheritance of resistance to tomato race T2, and 2) to present information on the relationship of resistance derived from PI 114490 to three pathogen races in the populations studied.

Materials and Methods

INHERITANCE STUDY. Tomato inbred Fla. 7600 (7600), resistant to race T1 from Hawaii 7998 and susceptible to races T2 and T3, was crossed with bacterial spot resistant accession PI 114490 in Fall 1993 and the F_1 was self-pollinated in Spring 1994. In 1996 the parents and F_1 were grown in the field at Wooster, Ohio in a randomized complete-block design with four blocks and five plants per plot along with other genotypes described previously (Scott et al., 1997). The F_2 was grown in an adjacent area along with some other F_2 lines in a similar design except there were six blocks of 20 plants per plot. A few plants died, so there were 94 F_2 plants. Details of the experiment are described by Scott et al. (1997), but the plants were inoculated with the T2 pathogen and rated for disease severity using the scale of Horsfall-Barratt (1945). This 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. The F_2 population was partitioned into resistant (R), intermediate (I), and susceptible (S) groups based on the segregation of the parents and the F_1 . A chi-square analysis was then conducted testing a two-gene model

described in the Results and Discussion section. Eighteen selections were made in the 7600 x PI 114490 F_2 population. In 1997 two blocks (plots) of five plants of F_3 lines derived from the 18 F_2 selections were inoculated with T2 and rated for disease severity at Wooster as described above.

Analysis of regression between disease response data from progeny in the F_2 and F_3 generations was used to estimate the heritability of T2 resistance in the 7600 x PI 114490 cross. The covariance of F_2 and F_3 generations was used since F_2 plants and F_3 families were evaluated in separate years. The regression coefficient was adjusted for inbred parents as suggested by Smith and Kinman (1965), and a standard error was calculated as described by Foolad and Jones (1992).

INBRED BACKCROSS (IBC) STUDY. The F_1 from 7600 x PI 114490 was backcrossed twice to bacterial spot susceptible Ohio 9242 and 166 plants were advanced by single seed descent for four generations. In Summer 2001, 85 BC_2S_4 lines and control lines were grown in 10-plant plots at Bradenton where they were inoculated with race T3 strains. The same 85 lines were grown in Wooster in replicated trials during 1999 and 2000 and as single plots with the entire 166 lines in 2001. Plots in Wooster were inoculated with race T2 strains by methods described previously. At Bradenton, each plant was rated for disease severity on the Horsfall-Barratt scale and means were calculated for each line. At Wooster each line was given an overall disease severity rating using the Horsfall-Barratt scale, and line means were based on the average rating of the plot.

The number of loci affecting resistance was estimated in the IBC population using the approach of Eskridge and Coyne (1996). Estimates of effective loci were obtained based on 85 lines common to T2 evaluations in nurseries during 1999, 2000, and 2001, the complete population of 166 lines evaluated in the T2 nursery in 2001, and the 85 lines evaluated in the T3 nursery in 2001. Broad sense heritability for T2 resistance was estimated according to Cotterill (1987), and standard errors for heritability were calculated according to Hallauer and Miranda (1988). Heritability estimates for T2 resistance in the IBC population were obtained by partitioning variation and obtaining mean square estimates for genotype, year, replicate, genotype by year interaction, and error. Heritability estimates assumed the selection unit was an individual inbred backcross line (IBL).

SPECIFIC-RACE SELECTION EXPERIMENTS. To select for resistance to race T2, the 7600 x PI 114490 population was used and selections were made in the F_2 and F_3 generations in the summers of 1996 and 1997, respectively, at Wooster, Ohio. To select for race T3 resistance, crosses were made between PI 114490 and four inbreds (7481, 7598, 7599 and 7655), in 1995. The former three inbreds were susceptible to bacterial spot and 7655 was susceptible to races T2 and T3 but had race T1 resistance derived from Hawaii 7998. The F_1 's were self-pollinated in Spring 1996. Selections for race T3 resistance were made in the F_2 , F_3 , and F_4 generations in Summer 1996, Spring 1997, and Summer 1997, respectively, at Bradenton, Fla.

In Summer 1998, experiments were conducted that consisted of six inbreds (F_3 generation) selected for resistance to race T3 at Bradenton, 13 inbreds (F_4 generation) selected for T2 resistance at Wooster, Ohio, and five controls; 'Solar Set' (susceptible), Hawaii 7998 (T1 resistant), Hawaii 7981 (T3 resistant), PI 114490 (T1, T2, and T3 resistant), and 'Campbell 28' (race non-specific, partial resistance). These same genotypes were used in Summer 1999 experiments along with Fla. 7835 and Fla. 7839, two inbreds with resistance to T1 from Hawaii 7998 and T3 from PI 126932

and Hawaii 7981, respectively. Both years the genotypes were grown in three replicated trials that were separately inoculated with the three races of the pathogen. The T1 experiments were conducted in Wooster, Ohio in 1998 and in Fremont, Ohio in 1999. The T2 experiments were conducted in Wooster, Ohio, while the T3 experiments were conducted in Bradenton, Fla.

WOOSTER 1998. Seed were sown in a greenhouse at Wooster, Ohio. On 27 May they were transplanted to the two field locations in randomized complete-block designs with four blocks. The T1 and T2 field plots were established on separate fields more than 3 km apart. There were five plants per plot for the T1 experiment and 10 plants per plot for the T2 experiment. They were grown on bare ground unstaked with 0.31 m between plants, and 1.22 m (T1) or 1.52 m (T2) between rows. Chlorothalonil (2,4,5,6-tetrachloroisophthalonitrile) was used to control foliar fungal pathogens and insecticides were used to control insect pests.

Plants in the T2 experiment were spray-inoculated with a mixture of two strains of T2, Xcv544b and Xcv572a. Plants in the T1 experiment were inoculated with strain Xcv110c. Inoculum was produced by growing the bacteria on nutrient agar for 24 h at 28 °C. Bacterial cells were removed from the agar plates and suspended in 10 mM MgSO₄·7H₂O and the suspensions were standardized to A₆₀₀ = 0.15 (a concentration of $\approx 3 \times 10^8$ CFU/mL). Inoculum was applied with a backpack sprayer in the late evening on 1 Jul.

The T1 and T2 inoculated plants were rated for disease severity on 27 and 28 Aug., respectively, by inspecting each plant and then giving an overall rating for the plot using the scale of Horsfall and Barratt (1945) described previously.

WOOSTER 1999. Methods were the same as for the 1998 experiment with the exception of the following; on 8 June plants were transplanted to the field, and plants were spray-inoculated with a mixture of T2 strains (Xcv544b, Xcv572a, Xcv788, Xcv791) on 9 July. Plants were rated for disease severity on 3 Sept.

FREMONT 1999. Methods were the same as for the 1998 experiment except on 7 June plants were transplanted to the field. There were 10 plants per plot in rows 1.52 m apart. Plants were spray-inoculated with T1 strain Xcv767 on 9 June and rated for disease severity on 2 Sept.

BRADENTON 1998. Seed were sown in a greenhouse and plants were transplanted to the field on 27 July on 20-cm-high, 81-cm-wide beds of EauGallie fine sand that had been fumigated with 67% methyl bromide: 33% chloropicrin at 392 kg·ha⁻¹ and covered with white polyethylene mulch two weeks before transplanting. A randomized complete-block design was used with four blocks and 10 plant plots. Plants were spaced 0.46 m apart within plots that were 0.91 m apart in rows, with 1.52 m between rows. Recommended fertilizer and insecticide programs were followed (Hochmuth et al., 1988), and the label rate of chlorothalonil was applied to control fungal pathogens. Plants were grown with stake culture and irrigated by seepage from ditches adjacent to the six experimental beds. Race T3 inoculum (a mixture of strains XV938, T54, and T56) was prepared as described previously and applied with a backpack sprayer early in the morning on 17 Aug. On 21 Sept. each plant was evaluated for bacterial spot disease severity by rating the two aisle sides of the staked plants and averaging the two ratings per plant.

BRADENTON 1999. Methods were the same as for the Bradenton 1998 experiment with the exception of the following; plants were transplanted to the field on Aug. 3, then were spray-inoculated with T3 strains on 19 Aug. and were rated for disease severity on 22 Sept.

RACE VERIFICATION IN T1 AND T2 PLOTS. At the end of the growing season, 10 fruit and 10 leaf samples with bacterial spot lesions were arbitrarily selected from T1 and T2 plots. Bacteria were isolated from the lesions and two *Xanthomonas* colonies per sample were purified and subjected to polymerase chain reaction (PCR) analysis using primers specific for *Xanthomonas* spp. (Rst 65/69) and race T1 (Rst 27/28) (Jones and Stall, unpublished). Race T2 strains were positive in PCR with primers Rst 65/69 and negative with Rst 27/28. All strains from T1 and T2 plots were confirmed as race T1 and T2, respectively. No cross-contamination of strains was observed.

SPECIFIC RACE SELECTION STATISTICAL ANALYSIS. Disease severity for genotypes in all experiments were tested by analysis of variance, and significant differences among treatment means were determined using Duncan's multiple range test at $P < 0.05$. A correlation analysis of disease severities between the three races was performed within and between years for PI 114490 and the T3 and T2 selected lines using the Corr procedure of the Statistical Analysis System (SAS Institute, Cary, N.C.).

Results and Discussion

INHERITANCE OF RACE T2 RESISTANCE. Race T1 resistant 7600 was susceptible to race T2 while PI 114490 had a high level of resistance to T2 (Table 1). The latter genotype had no typical bacterial spot lesions but was rated 2 rather than 1 since there were some (<3%) early blight (*Alternaria solani*) lesions that may have masked possible bacterial spot lesions. The F₁ was intermediate to the parents with a mean rating (3.5) very close to the mid-parent value (3.63). The F₂ segregation is shown in Fig. 1. There were 6.38% of the plants rated 2, similar to PI 114490. This is very close to the 6.25% expected with a two-gene model requiring all four alleles for maximum resistance (R). If two or three resistance alleles resulted in intermediate resistance (I) like the F₁ (3 to 4 Horsfall-Barratt ratings) and zero or one resistance alleles resulted in susceptibility (S) (≥ 5 Horsfall-Barratt ratings) a 1R:10I:5S ratio would be expected. This model was tested and the data had a good fit ($\chi^2 = 0.96$, $P = 0.9$ to 0.5) so the model was accepted.

Further support for a two-locus model came from analysis of segregation in the inbred backcross lines. Of the 85 lines evaluated in both T2 and T3 disease nurseries, eleven lines had intermediate resistance (3 to 4 Horsfall-Barratt ratings) for race T2 (Table 2). No inbred backcross lines (IBL) demonstrated resistance to bacterial spot equivalent to that found in PI 114490, though most lines had superior plant type and yield index (data not shown). For the three years of T2 trials with the IBC population, genetic models ranging from 1–4 loci were found to be consistent with the data. For combined data on the 85 lines, an estimate of 2 genes

Table 1. Bacterial spot race T2 disease for tomato breeding lines 7600, PI 114490, their F₁ and susceptible 'Solar Set' at Wooster, Ohio, in Summer 1995.

Genotype	Generation	Disease severity ^z
7600	P ₁	5.25 a ^y
PI 114490	P ₂	2.00 c
7600 x PI 114490	F ₁	3.50 b
Solar Set		5.75 a

^zRated on Horsfall-Barratt scale where lower numbers indicate less disease (see text).

^yMean separation in columns by Duncan's multiple range test at $P \leq 0.05$.

Fig. 1. Race T2 disease severity frequency distribution for the F₂ generation derived from a cross of tomato breeding line 7600 (P₁) and PI 114490 (P₂) inoculated with race T2 at Wooster, Ohio in Summer 1996. A two-gene resistance model was proposed where the allele number from PI 114490 of; four equals resistance (R) (2 Horsfall-Barratt rating), two or three equals intermediate resistance (I) (3 to 4 Horsfall-Barratt ratings), and one or zero equals susceptibility (S) (≥ 5 Horsfall-Barratt ratings). The expected 1R:10I:5S ratio had an acceptable fit ($\chi^2 = 0.96$, $P = 0.9$ to 0.5) and the model was accepted.

(± 0.33) provided the best fit. Both two and three gene models are consistent with the observed segregation for the complete population of 166 lines ($P < 0.05$). Based on the selection unit of an individual IBL, the heritability is 0.58 (± 0.25). This estimate of heritability should not be considered an accurate representation of breeding value for predicting genetic gain under selection. Rather, it represents an upper estimate of the total variation attributable to genotype in these trials assuming that the selection unit is an individual IBL.

A genetic model that postulates loci unique to T2 and T3 resistance and resistance loci in common to both races is suggested by the data for the IBC population. Two to three loci explain resistance to race T3. Lines with low disease ratings in both T2 and T3 trials may contain distinct loci from PI 114490. However, we would expect only a small proportion of lines, 1.56%, to share any two unlinked loci in the IBC population. Thus, it is likely that the five to six lines that share tolerance to both T2 and T3 (Table 2) share a single locus that confers some tolerance to both races. Linear correlations between T2 and T3 data are rather low and marginally nonsignificant ($r = 0.19$, $P = 0.084$) for the IBC population, suggesting that a different locus or loci play an important role despite the potential for shared alleles.

The response to selection of progeny derived from PI 114490 is predicted to be high to moderate in T2 nurseries. The linear correlation between F₂ plants and their corresponding F₃ families

was highly significant ($P = 0.001$). Narrow sense heritability was estimated at 0.37 ± 0.1 using the regression coefficient adjusted by a factor of two-thirds (Smith and Kinman, 1965) to account for inbred parents. Parent-offspring regression provides a conservative estimate of heritability and is a better predictor of how populations will perform under selection than estimates based on variance components (Foolad and Jones, 1992). However, the method provides only an estimate of narrow sense heritability when the means of F₂ individuals and F₃ families are used as variance since dominance still contributes to the term for genetic variation (Foolad and Jones, 1992). The estimated heritability suggests that field selection will be an effective approach for improving the resistance against race T2 strains. Plant habit (*sp* locus) did not appear to play a role in bacterial spot resistance in the IBC population.

SPECIFIC RACE SELECTION. There was low disease pressure for race T1 in 1998 as evidenced by lower disease ratings for 'Solar Set' and Hawaii 7981 that are both susceptible to T1 (Table 3). The T3 rating in 1999 was higher than normal for Hawaii 7981 that usually has virtually no symptoms to T3 (Scott et al., 1995; Scott, unpublished data). Samples of lesions on Hawaii 7981 were analyzed and determined to be a mutant form of T3 (Astua-Monge et al., 2000). Thus, this mutant may have played a role in the higher ratings of the T3 data in 1999.

Despite variation in disease pressure, there was a high level of consistency between individual lines throughout the experiment. PI 114490 (resistant control) had excellent resistance to races T1 and T2 both years (Table 3). PI 114490 was significantly more resistant to race T1 than T1 resistant Hawaii 7998 in 1998, but the two genotypes were similar in 1999. For race T3, PI 114490 was less resistant than Hawaii 7981 in 1998, but they were similar with more disease in 1999. Both Hawaiian lines were intermediate to PI 114490, the resistant control, and 'Solar Set', the susceptible

Table 2. Severity of bacterial spot disease for inbred backcross tomato lines with partial resistance to race(s) T2 at Wooster, Ohio, and/or T3 at Bradenton, Florida in 2001.

Inbred	Disease severity ²	
	Race T3	Race T2
Partial resistance to T2 and T3		
6137	3.9	4
6040	4.1	4
6033	4.6	4
6084	4.7	4
6082	5.0 ³	3
6080	5.4 ³	4
Partial resistance to T3 only		
6050	4.1	7
6146	4.2	6
6022	4.9	6
Partial resistance to T2 only		
6046	5.9	4
6099	6.0	4
6072	6.9	4
6088	7.0	4
6096	7.0	4
Controls		
Ohio 9242	7.0	6
PI 114490	4.5	1.3

²Plants rated using Horsfall Barratt scale (1 to 12), lower values indicate less disease.

³Lines appeared to segregate for partial resistance.

Table 3. Tomato race T1, T2, and T3 bacterial spot disease severity ratings for tomato genotypes selected for T3 or T2 resistance and controls in Summer 1998 and 1999.

Genotype	Race selected for	Bacterial spot rating 1998 ^z			Bacterial spot rating 1999 ^z		
		T1	T2	T3	T1	T2	T3
1. (7481 x PI 114490)-BK-9-SBK-3	T3	2.00 e ^y	3.44 e	2.85 i-k	5.25 bc	3.00 cd	3.36 lm
2. (7598 x PI 114490)-BK-1-2-SBK-SBK	T3	4.10 a	5.73 a	3.13 g-i	5.50 a-c	4.50 b	5.34 cd
3. (7599 x PI 114490)-BK-12-1-4	T3	3.30 b	4.84 bc	3.23 f-h	5.50 a-c	4.75 b	3.99 hi
4. (7655 x PI 114490)-BK-2-1-SBK	T3	2.12 de	2.21 ij	2.93 h-j	2.00 i	2.00 f	3.44 kl
5. (7655 x PI 114490)-BK-7-2-10	T3	2.05 e	2.71 f-h	2.55 kl	3.50 e-g	2.00 f	3.54 j-l
6. (7655 x PI 114490)-BK-16-1-2	T3	2.10 de	2.69 f-h	3.53 c-e	3.25 f-h	3.00 cd	4.31 fg
7. (7600 x PI 114490)-BK-36-1	T2	2.00 e	2.25 ij	2.69 j-l	2.50 hi	2.00 f	3.11 m
8. (7600 x PI 114490)-BK-36-2	T2	3.12 bc	3.57 e	3.67 cd	4.25 de	3.00 cd	5.63 bc
9. (7600 x PI 114490)-BK-39-1	T2	2.00 e	2.36 h-j	3.16 f-i	2.25 i	2.25 ef	4.47 f
10. (7600 x PI 114490)-BK-39-2	T2	2.00 e	2.24 ij	2.45 l	2.50 hi	2.00 f	3.83 h-j
11. (7600 x PI 114490)-BK-41-1	T2	2.13 de	2.48 g-j	3.47 c-g	2.75 g-i	2.75 de	4.82 e
12. (7600 x PI 114490)-BK-41-2	T2	2.00 e	2.41 g-j	3.61 cd	2.00 i	2.00 f	4.85 e
13. (7600 x PI 114490)-BK-41-3	T2	---	2.82 fg	3.13 g-i	2.00 i	2.25 ef	5.01 de
14. (7600 x PI 114490)-BK-41-4	T2	2.00 e	2.95 f	3.31 d-g	2.00 i	2.25 ef	5.31 cd
15. (7600 x PI 114490)-BK-49-BK	T2	2.35 de	2.70 f-h	3.50 c-f	4.75 cd	2.50 d-f	4.92 e
16. (7600 x PI 114490)-BK-51-1	T2	2.65 cd	2.58 f-i	4.11 b	2.50 hi	2.75 de	4.32 fg
17. (7600 x PI 114490)-BK-55-BK	T2	2.06 e	2.41 g-j	3.70 c	2.00 i	2.00 f	5.24 d
18. (7600 x PI 114490)-BK-26-1	T2	2.00 e	2.56 f-i	2.75 j-l	2.00 i	2.00 f	4.36 fg
19. (7600 x PI 114490)-BK-26-2	T2	2.00 e	2.53 g-j	3.46 c-g	2.38 hi	2.25 ef	3.95 hi
PI 114490	-	2.06 e	2.12 j	2.48 l	2.00 i	2.00 f	3.88 hi
Solar Set	-	4.00 a	5.94 a	5.51 a	6.25 a	5.50 a	6.31 a
Hawaii 7998	T1	3.37 b	4.68 c	5.19 a	3.00 f-i	4.75 b	5.86 b
Hawaii 7981	T3	3.10 bc	5.15 b	1.18 m	6.25 a	4.75 b	4.13 gh
Campbell 28	---	4.25 a	4.09 d	5.31 a	6.00 ab	4.25 b	5.31 cd
7835	T1, T3	---	---	---	3.75 ef	3.50 c	3.71 i-k
7839	T1, T3	---	---	---	3.25 f-h	3.50 c	3.74 i-k

^zPlants rated using Horsfall-Barratt scale (1 to 12), lower value indicates less disease. Data for each race taken in different nurseries specifically inoculated with the indicated race.

^yMean separation in columns by Duncan's multiple range test at $P \leq 0.05$.

Table 4. Correlation coefficients for bacterial spot disease severity ratings between races T1, T2, and T3 in Summer 1998 and 1999 for PI 114490 and T2 or T3 selected tomato lines derived from PI 114490.

Race/year	Race/year				
	T1/1998	T2/1998	T3/1998	T1/1999	T2/1999
T2/1998	0.90***	---	---	---	---
T3/1998	0.24 ^{NS}	0.10 ^{NS}	---	---	---
T1/1999	0.70**	0.80***	0.04 ^{NS}	---	---
T2/1999	0.86***	0.92***	0.23 ^{NS}	0.82***	---
T3/1999	0.36 ^{NS}	0.27 ^{NS}	0.63*	-0.01 ^{NS}	0.16 ^{NS}

^{NS}, *, **, *** Nonsignificant or significant at $P = 0.05$, 0.01, or 0.001, respectively.

control, for race T2 both years. In previous experiments Hawaii 7998 was intermediate, but Hawaii 7981 was similar to 'Solar Set' (Scott et al., 1997). The two lines bred for a combination of T1 and T3 resistance, Fla. 7835 and Fla. 7838, were more resistant than 'Solar Set' to those two races as expected. They both had significantly less disease than 'Solar Set' and the Hawaiian lines for race T2, which was unexpected. In Summer 2000 Fla. 7835 was tested again for T2 and it had the same result, whereas both Hawaiian lines were similar to 'Solar Set' (Scott and Miller, data not shown). Numerous lines with T1 or T3 resistance have been tested and they were susceptible to T2 (Table 1; Scott and Miller, unpublished data). However, the combined resistance to T1 and T3 provided some resistance to T2. The apparent epistatic reaction is intriguing with regard to possible future gene pyramiding via linked molecular markers.

In both years, all six lines selected for resistance to race T3 had more T3 resistance than the susceptible control and were equally or more resistant to T3 than the resistant control PI 114490 (Table 3). The lines selected for T3 resistance generally had T2 and T1 resistance intermediate to the parents or similar to PI 114490, but occasionally some, especially line 2, were similar to the susceptible control (Table 3). The 13 lines selected for T2 resistance were more resistant than the susceptible control and were intermediate to the parents or similar to PI 114490 for all three races both years (Table 3).

For PI 114490, and the lines selected for T2 or T3 resistance, there were very highly significant correlations between T1 and T2 disease severity both between and within years ($r > 0.80$, $P < 0.001$) (Table 4). However, there were no significant correlations between T3 disease severity and either T1 or T2 disease severity

between or within years. Within races, over different seasons, there were very highly significant, highly significant, and significant correlations for disease severity for T2, T1 and T3, respectively. The data suggest a strong relationship between resistance genes for races T1 and T2 that should allow for breeders to select for resistance to either race and obtain resistance to the other one. Line 8 had less resistance than the resistant control for all three races both years (Table 3). For the remainder, only line 16 in 1998 and line 15 in 1999 had less T1 resistance than the resistant control suggesting either an environmental effect or a difference in resistance genes for T2 and T1. Even here line 15 was not as resistant to T2 as the resistant control in 1998 and it may not have the highest level of T2 resistance. In no case where T2 selections were similar to the resistant control both years was there any problem with the race T1 resistance being less than the resistant control. It appears from the data that if high levels of T2 resistance were selected, one would also obtain a high level of T1 resistance. Still it would be advisable to assay lines occasionally for the other race if selecting for only one to ensure retention of high resistance levels.

However, the data suggest it will be difficult to screen for T2 or T3 and obtain resistance to other race. Lines 9, 10, 12, 17, and 19 were all similar to the resistant control for T2 both years, but only line 10 was similar to resistant control for T3 both years. Of the six lines selected for T3 resistance, only line 4 was similar in disease severity to the resistant control for T1 and T2 both years. These results do indicate that selection for T2 or T3 resistance can result in resistance to the other race, but selections would have to be tested for the other race in a breeding program to determine which lines carried the other resistance. The mean ratings for the T2 selected lines for T2 were 2.6 in 1998 and 2.3 in 1999 while the T3 selected lines had mean T3 ratings of 3.0 and 4.0 in 1998 and 1999, respectively. PI 114490 also had higher ratings for T3 than for T2 or T1 (Table 3). Part of this could have been due to the mutant race in 1999, but even so, Table 2 and other data support observations over the last few years that resistance from PI 114490 is not as effective for T3 as it is for the other two races. A concerted breeding effort with PI 114490 has resulted in only a few lines with good resistance to T3 (Scott, unpublished results). Another approach would be to combine resistance from PI 114490 with the Hawaii 7981 or other sources of T3 resistance. Although this will be difficult without molecular markers to identify the genes selected, such combinations should give good levels of T3 resistance and the gene combinations may be effective even in the face of virulent pathogen mutations. In fact, in the summer of 2002 at Bradenton where there was a heavy infestation of race T3 and the fourth race in the field, a line with both Hawaii 7981 and PI 114490 in its pedigree had a good level of resistance, whereas hundreds of lines with only T3 resistance were susceptible (data not shown).

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