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Inheritance of Resistance in Tomato to Race T3 of the Bacterial Spot Pathogen

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Abstract. Hawaii 7981 tomato (*Lycopersicon esculentum* Mill.), resistant to race T3 of the bacterial spot pathogen [*Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye], was crossed to the susceptible tomato inbred, Fla. 7060, and subsequently F_2 and backcross seed were obtained. These generations were planted in the field, inoculated with the race T3 pathogen and evaluated for disease severity over two summer seasons. Data were tested for goodness-of-fit to a model based on control by the incompletely dominant gene *Xv3* that confers hypersensitivity. The F_1 was intermediate in disease severity to the parents for both seasons. When data were combined over both seasons, the backcrosses fit the expected 1:1 ratios although each deviated from the expected ratio in one of the 2 years tested. The F_2 did not fit the expected 1:2:1 ratio in either year or when data from the two years were combined due to a deficiency of resistant plants. Thirty-three F_2 plants representing an array of disease severities and hypersensitivity reactions were selected in the second season and their F_3 progeny were inoculated and evaluated for disease severity. Hawaii 7981 was significantly more resistant than the 12 most resistant F_3 selections even though all expressed hypersensitivity. A hypersensitive F_3 with intermediate field resistance was crossed to Hawaii 7981 and subsequently, F_2 and backcross generations were obtained. These generations were field inoculated with the race T3 pathogen and evaluated for disease severity. Hawaii 7981 was significantly more resistant than the F_3 parent as in the previous year. The data did not fit an additive-dominance model and epistatic interactions were significant. Thus, it appears that field resistance to race T3 of bacterial spot found in Hawaii 7981 is conferred quantitatively by *Xv3* and other resistance genes. Breeding implications are discussed.

Three races of bacterial spot, incited by *Xanthomonas campestris* pv. *vesicatoria*, infect tomato (*Lycopersicon esculentum*). These have been designated T1, T2, and T3 as described by Jones et al. (1995). In Florida, T1 was the endemic race, but it has been largely replaced by T3 probably due to antagonism of T3 over T1 (Jones et al., 1998). This has occurred in the absence of T1 resistant cultivars. Because bacterial spot is difficult to control by chemical means, especially during hot, rainy weather common in Florida early in the fall production season, host resistance seems an attractive control strategy. Field resistance to race T1 was found in Hawaii 7998 (Scott and Jones, 1986). This resistance was reported to be complex and unusual. Scott and Jones, (1989) reported field resistance to be largely additive and controlled by three to five effective factors. Whereas hypersensitivity is generally controlled by single dominant genes, the race T1 hypersensitive response of Hawaii 7998 was controlled by either two (Whalen et al., 1993) or three genes (Wang et al., 1994). Later, Yu et al. (1995) discovered three regions of the genome were in fact associated with the hypersensitive response. However, field resistance was not explained by the hypersensitive response alone. Scott and Jones (1989) reported only 5% of F_2 plants were resistant in the field, but ≈60% were hypersensitive. Wang (1992) reported correlation coefficients of only 0.39 to 0.41 between hypersensitivity and field resistance in two field F_2 populations. Later, Somodi et al. (1996) found correlation coefficients of 0.31 to 0.52 between hypersensitivity and field resistance in two F_2 populations.

Hypersensitive resistance to race T3 was discovered in Hawaii 7981 and *L. pimpinellifolium* L. accessions PI 126932 and PI 128216 (Jones et al., 1995). The highest level of resistance in the field was in Hawaii 7981 (Scott et al., 1995). In a growth chamber experiment, the hypersensitive response in Hawaii 7981 was determined to be controlled by an incompletely dominant gene designated *Xcv-3* (Scott et al., 1996) although *Xv*3 is now the preferred gene designation and will be used herein. The objective of this study was to characterize inheritance of race T3 bacterial spot resistance in the field. This information will be useful in developing breeding strategies to incorporate T3 resistance into commercial cultivars.

Materials and Methods

1994 Experiment. The race T3 susceptible inbred Fla. 7060 was crossed to Hawaii 7981, and subsequently the F_1 was self-pollinated to produce F_2 seed and crossed to each parent to produce backcrosses. These generations were used for inheritance studies in 1994 and 1995. Seed were sown in a greenhouse in Black Beauty spent coal (Reed Minerals Div., Highland, Ind.)

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Table 1. Summer 1994, 1995, and combined disease ratings for tomato genotypes Hawaii 7981 (resistant), Fla 7060 (susceptible) and derived generations after inoculation with *Xanthomonas campestris* pv. *vesicatoria* race T3 and chi-square tests for control of resistance by a single incompletely dominant gene.

Genotype	Generation	Years(s)	Total plants	Disease rating[z] <3	3–4	>4	Expected ratio	χ^2	*P*
Hawaii 7981 (7981)	P_1	1994	28	23	5	0	1:0:0	---	---
		1995	29	29	0	0		---	---
		Σ	57	52	5	0		---	---
Fla. 7060 (7060)	P_2	1994	30	1	0	29	0:0:1	---	---
		1995	30	0	8	22		---	---
		Σ	60	1	8	51		---	---
7060 x 7981	F_1	1994	30	5	25	0	0:1:0	---	---
		1995	30	5	25	0		---	---
		Σ	60	10	50	0		---	---
(7060 x 7981) 7981	BCP_1	1994	88	40	43	5[y]	1:1:0	0.73	0.5–0.1
		1995	119	72	47	0		4.03	0.05–0.025
		Σ	207	112	90	5[y]		1.39	0.5–0.1
(7060 x 7981) 7060	BCP_2	1994	90	0	37	53	0:1:1	2.84	0.1–0.05
		1995	120	5[y]	66	49		5.25	0.025–0.01
		Σ	210	5[y]	103	102		0.17	0.9–0.5
(7060 x 7981)-Bk	F_2	1994	169	28	83	58	1:2:1	10.69	<0.005
		1995	208	37	118	53		6.23	0.025–0.01
		Σ	377	65	201	111		12.88	<0.005

[z]Rated on scale of Horsfall-Barratt (1945) where <3 = 0% to 3%, 3 to 4 = 3% to 12%, >4 = 12% to 50% infected tissue.
[y]Combined with 3 to 4 disease rating for chi-square analysis.

medium on 16 June and seedlings were transplanted into Todd planter flats (3.8 cm^3 cell size) (Speedling, Sun City, Fla.) on 27 June. On 25 July plants were transferred to 385 cm^3 pots so that hypersensitivity could be measured by confluent necrosis as described elsewhere (Jones et al., 1995; Klement, 1982). Results of the hypersensitivity tests were ambiguous and will not be presented. Plants were transplanted to the field on 22 Aug. on 20-cm-high, 81-cm-wide beds of EauGallie fine sand (sandy, siliceous, hyperthermic Alfic Alaquod) that had been fumigated with 67% methyl bromide : 33% chloropicrin at 392 $kg \cdot ha^{-1}$ and covered with white polyethylene mulch 2 weeks before transplanting.

A randomized complete-block design was used with three blocks and 10 plant plots for the P_1, P_2, and F_1 generation, 30 plant plots for the backcrosses, and 60 plant plots for the F_2 generation. Plants were spaced 46 cm apart within plots that were 91 cm apart in rows, with 152 cm between rows. Recommended fertilizer and insecticide programs were followed (Hochmuth et al., 1989), and the label rate of 2,4,5,6-tetrachloroisophthalonitrile (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 was produced by growing the bacteria on Difco nutrient agar (Becton Dickinson and Company, Sparks, Md.) for 24 h at 28 °C. Bacterial cells were removed from the agar plates and suspended in 10 mM $MgSO_4 \bullet 7\ H_2O$ and the suspensions were standardized to A_{600} = 0.15 (a concentration of $\approx 10^8$ colony forming units (cfu)/mL). Inoculum was applied with a backpack sprayer early in the morning on 31 Aug. On 19 Sept. each plant was evaluated for bacterial spot disease severity by rating the two aisle sides of the staked plants using the scale of Horsfall and Barratt (1945) and then averaging two ratings per plant. 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 partitioned into resistant, intermediate, and susceptible categories based on distribution of the P_1, P_2, and F_1 generations so that chi-square analysis could be used to test for genetic control by *Xv3,* the single incompletely dominant gene that confers the hypersensitive response (Scott et al., 1996).

1995 Experiment. Seed were sown on 12 June**,** seedlings were transplanted to planter flats in the greenhouse on 21 June, and plants were transplanted to 385 cm^3 pots on 27 July. Leaflets on each plant were injected in the greenhouse on 10 Aug. with a suspension containing 10^8 cfu/mL so that hypersensitivity could be measured as in the 1994 experiment. Plants were transplanted to the field on 17 Aug., inoculated on 25 Aug., and rated for disease severity on 26 Sept. A randomized complete-block design with three blocks and 10-plant plots was used. Each block had one plot for the P_1, P_2, and F_1 generations, four plots for the backcrosses, and seven plots for the F_2. Hypersensitivity reactions were evaluated in the field by the same method as used in the greenhouse in 1994. A new fully formed leaf was tagged and injected on 18 Sept. and confluent necrosis was evaluated at 24 and 48 h. Seed was saved from 33 F_2 plants that were selected on 16 Oct. They were selected for various combinations of disease severity and hypersensitivity and included nine highly resistant (Horsfall-Barratt <3) plants. All other procedures were as described for the 1994 experiment.

1996 Experiment. Seed of Hawaii 7981, Fla. 7060, their F_1 and 33 F_3 lines from the F_2 selections made in 1995 were sown on 10 June, seedlings were transplanted to planter flats on 20 June, and plants were transplanted to the field on 22 July. They were planted in a randomized complete-block design with three blocks and eight plants per plot. The plants were inoculated in the field by spraying a suspension (10^8 cfu/mL) of a mixture of T3 strains on the foliage in the morning of 12 Aug. Disease severity was rated on 24 Sept. Data were subjected to analysis of variance procedures and significant differences between F_3 genotypes were

determined by Duncan's multiple range test using "SAS For Windows" (SAS Inst., Inc., 1997). All other procedures were as described for the 1994 experiment.

1997 Experiment. To test for existence of resistance genes other than *Xv3*, Hawaii 7981 was crossed with E228 in Fall 1996. E228 was an F_3 line selected for a strong hypersensitivity response comparable to that of Hawaii 7981 with less bacterial spot race T3 field resistance in the 1996 experiment. In Spring 1997 the F_1 was self-pollinated to produce F_2 seed and backcrosses were made with the parents. These generations and Fla. 7060, a susceptible control, were sown on 19 June. Seeds were transplanted to planter flats on 27 June, plants were transplanted to the field on 21 July and inoculated on 1 Aug. They were arranged in a randomized complete-block design with four blocks and 10 plants per plot for the control, P_1, P_2, and F_1, and 25 plants per plot for backcrosses and F_2 with two plots per block for the F_2. Disease severity was rated 29 Aug. Hypersensitivity was evaluated in the field as described previously. A new fully formed leaf was tagged and injected on 19 Sept. and confluent necrosis was evaluated at 24 and 48 h. Data were subjected to generation means analysis (Mather and Jinks, 1982) using a Lotus spreadsheet program (Ng, 1990). All other procedures were as described for the 1994 experiment.

Results

Disease severity of the F_1 was intermediate between the resistant and susceptible parents in 1994 and 1995, supporting

Table 2. Bacterial spot race T3 disease severity and hypersensitivity reactions for Hawaii 7981, Fla. 7060, their F_1, 33 F_2 plants selected for various levels of disease severity and hypersensitivity reactions in 1995, and their F_3 progeny in 1996.

	Summer 1995 F_2 selection		Summer 1996 F_3 progeny		
F_3 plot	Disease			Disease severity	
designation	severity	Hypersensitivity[z]	Hypersensitivity[y]	Mean	Range
E212	5	–	+/–	5.4 a[x]	4–7
E218	5	–	–	5.2 a	3–6
E215	4	–	–	5.0 a–c	4–6
E207	5	–	–	4.8 a–d	4–6
E225	3	+	+/–	4.7 a–d	2–6
Fla. 7060	(4.7)	(–)	(–)	4.7 b–e	4–5
E234	4	–	–	4.5 b–f	2–6
E208	4	++	+	4.3 c–g	1–6
E216	3	+++	+	4.3 c–g	3–5
E232	3	+	+	4.3 c–g	2–5
E220	3	++	+/–	4.1 d–h	2–5
E204	3	+	+/–	4.0 e–h	3–5
E209	3	++	+	4.0 e–h	1–6
E236	3	+	+/–	3.9 f–h	1–6
E229	3	+	+/–	3.8 g–i	1–5
E223	4	–	–	3.8 g–i	2–6
E228	3	+++	++	3.7 g–j	3–5
E230	3	+++	+	3.6 g–k	1–5
E210	5	+	+/–	3.5 h–k	1–5
E235	3	+	+/–	3.5 h–l	1–5
E214	1	++	+/–	3.5 h–l	1–6
E221	5	+++	+/–	3.2 i–m	1–5
E211	1	+++	++	3.0 j–m	1–5
E231	1	++	+	3.0 j–m	1–4
7060 x H7981 F_1	(2.9)	(+)	(+)	3.0 j–m	2–4
E226	3	+++	+	2.9 k–m	1–6
E206	1	++	+	2.8 l–n	1–4
E213	3	+++	+	2.6 m–o	1–5
E222	3	++	+/–	2.5 m–o	1–4
E205	1	++	+	2.5 m–o	1–5
E219	1	+++	++	2.2 n–p	1–4
E217	1	+++	++	2.1 op	1–4
E224	1	++	++	2.0 op	1–4
E227	3	+++	++	1.9 op	1–6
E233	2	++	+	1.7 p	1–4
Hawaii 7981	(1.1)	(+++)	(++)	1.0 q	1–1

[z]Based on speed and intensity of confluent necrosis from two tests (greenhouse and field) where – indicates no necrosis and +, ++, and +++ indicate increasingly rapid and intense necrotic responses, respectively.

[y]Based on the speed and intensity of the response of 6 to 12 plants per genotype in the greenhouse where – indicates no necrosis, +/– indicates segregation for hypersensitivity, + indicates necrosis 48 h after injection, and ++ indicates necrosis 24 h after injection.

[x]Mean separation within column by Duncan's multiple range test at $P < 0.05$.

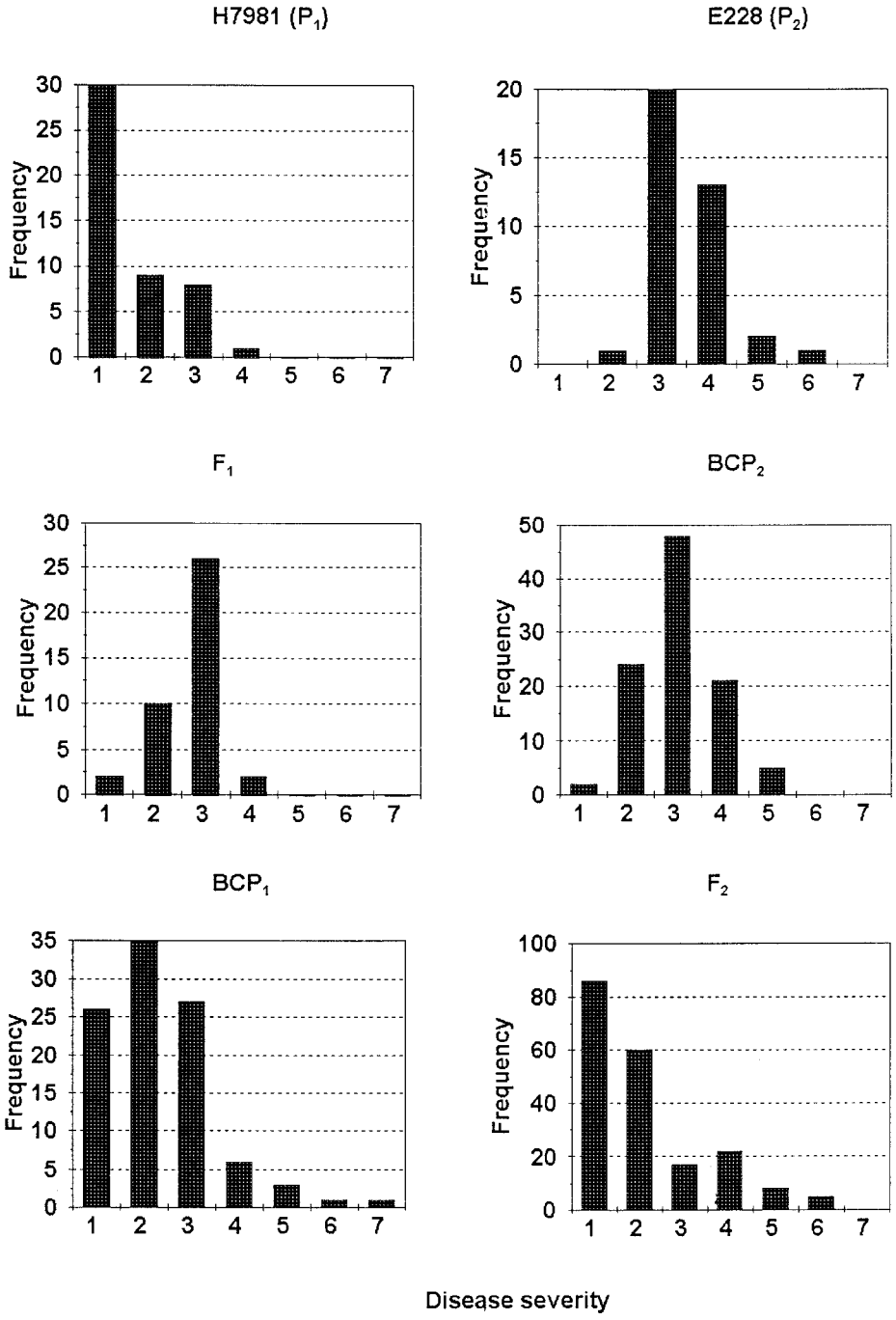

Fig. 1. Bacterial spot race T3 disease severity frequency distributions for tomato parents Hawaii 7981 (H7981), E228 and generations derived from them at Bradenton, Fla., during Summer 1997. BC = backcross.

additive inheritance as would be expected with incomplete dominance (Table 1). The backcross to the resistant parent had an acceptable fit to the expected 1:1 ratio of resistant to intermediate plants in 1994 and when data were combined over both seasons. In the 1995 experiment, the observed frequency deviated significantly from the expected 1:1 ratio because of an excess of resistant plants. For the backcross to the susceptible parent the chi-square test was barely acceptable in 1994 because of a higher number of susceptible plants. In 1995 there were more intermediate plants than susceptible plants and the observed frequency deviated significantly from the expected ratio. However, when data were combined for the 2 years, there was an acceptable fit to the expected 1:1 ratio of intermediate to susceptible plants (Table 1). The combined F_2 data deviated significantly from the expected 1:2:1 ratio because of a deficiency of resistant plants and an excess of susceptible plants (Table 1). There was a deficiency of resistant plants both years. In 1995 there was an excess of intermediate plants, whereas in 1994 there was an excess of susceptible plants. From the backcross and F_2 generations it was apparent that there was more disease pressure in 1994 than 1995. The deficiency of resistant plants in the F_2 generation and inconsistent ratios for the backcrosses suggested that there might be more than one gene controlling resistance to race T3 from Hawaii 7981.

Data for the F_3 progeny of the selected F_2 plants lend support to the existence of genes other than a single hypersensitivity gene controlling resistance. Although nine F_2 plants were selected for hypersensitivity and disease severity of <3, none of their F_3 progeny nor any other F_3 were as resistant as Hawaii 7981 (Table 2). This is reflected in their significantly higher means and their ranges that might indicate lack of homozygosity. Also, E223 did not express hypersensitivity, but was more resistant than susceptible Fla. 7060.

To explore the existence of resistance genes other than *Xv3*, a family derived from E228 crossed with Hawaii 7981, was evaluated in summer 1997. In 1997, E228 was not as resistant as Hawaii 7981 as was the case in 1996 (Tables 2 and 3; Fig. 1). As in 1996, E228 had significantly less disease than Fla. 7060 that had a rating of 5.68 in 1997. The F_1 was intermediate between the parents and was skewed slightly towards susceptibility (Table 3; Fig. 1). The backcross to Hawaii 7981 (BCP_1) was distributed primarily between the F_1 and Hawaii 7981, but there were some plants more susceptible than the F_1 which resulted in higher than expected disease severity. The backcross to E228 was distributed between the F_1 and E228 with less variation than BCP_1. The F_2 had a continuous distribution with a relatively high percentage of plants in the most resistant categories. This resulted in a F_2 mean that was lower than expected and it was on the opposite side of the midparent value than the F_1 mean. The data did not fit an additive-dominance model, primarily due to deviations in BCP_1 and F_2 generations (Table 3).

Thus, an interaction analysis was performed that revealed significant homozygous × homozygous ([i]), homozygous × heterozygous ([j]), and heterozygous × heterozygous ([l]) interactions (Table 4). Because the [i] and [l] interactions had opposite signs, a duplicate dominant or recessive suppressor type of epistasis was indicated (Mather and Jinks, 1982). Both additive and dominant effects were significant. The epistasis prevented the estimate of effective factors, narrow sense heritability, and broad sense heritability.

Table 3. Bacterial spot race T3 disease severity for Hawaii 7981 (P_1), E228 (P_2), F_1, F_2, and backcross generations and joint scaling test for goodness of fit to an additive-dominance model.

Generation	Plant no.	Mean disease severity[z] Observed	Mean disease severity[z] Expected	Variance	Goodness of fit
P_1	40	1.28	1.29	0.25577	0.022
P_2	35	3.51	3.37	0.59009	1.275
F_1	40	2.70	2.63	0.42051	0.493
F_2	198	2.09	2.48	1.73186	16.636
BCP_1	99	2.31	1.96	1.38054	9.071
BCP_2	100	3.03	3.00	0.73646	0.144
Midparent	---	2.39			$\chi^2 = 27.64$ $P<0.001$

[z]Rated on the Horsfall-Barratt (1945) scale where higher number indicates more disease.

Table 4. Estimates of additive, dominance, and interaction parameters for the Hawaii 7981 x E228 family.

Parameter[z]	Estimate (±SE)	*t* test
m	0.092 ± 0.481	0.19^{NS}
[d]	–1.119 ± 0.076	-14.68^{***}
[h]	5.408 ± 1.179	4.59^{***}
[i]	2.302 ± 0.475	4.85^{***}
[j]	0.805 ± 0.329	2.44^{*}
[l]	–2.800 ± 0.739	3.79^{***}

[z]Definitions: m = midpoint (between AA and aa), [d] = difference of AA and aa from midparent, [h] = difference of Aa from midparent value, [i] = homozygote × homozygote interaction, [j] = homozygote × heterozygote interaction, and [l] = heterozygote × heterozygote interaction.
^{NS,*,***}Nonsignificant or significant at $P \leq 0.05$ or 0.001, respectively.

Discussion

It is generally believed that hypersensitive responses are controlled by single dominant genes. However, this was not the case with bacterial spot race T3 resistance derived from Hawaii 7981. Earlier, Scott et al. (1996) reported that race T3 hypersensitive response was controlled by a single incompletely dominant gene, *Xv3*. However, evidence is presented herein that genes in addition to *Xv3* control bacterial spot race T3 resistance in the field. There was a deficiency of resistant segregates over two seasons in the F_2 generation that caused rejection of the expected 1:2:1 incompletely dominant inheritance ratio (Table 1). Furthermore, F_3 progeny from the most resistant F_2 selections were not as resistant as was Hawaii 7981 (Table 2) despite the fact that these selections generally expressed homozygous hypersensitivity. Moreover, one F_3 without hypersensitivity (E223), had more resistance than the susceptible control. Generation means analysis from a family derived from E228 crossed with Hawaii 7981 indicated significant additive, dominant, and epistatic effects that would by necessity require two genes. An actual estimate of the effective factor number was precluded by the epistasis. Due to overlap of the parents with the F_1 generation, it is not possible to provide evidence for a particular epistatic ratio. A more complex model involving three gene interactions or linkage would require generations not available in these studies (Mather and Jinks, 1982).

Control of resistance to race T3 by *Xv3* plus other genes presents some difficulties for development of resistant cultivars. It would be straightforward to backcross *Xv3* into recurrent parents by conducting hypersensitivity tests on plants from each backcross generation. However, if this was done the other resistance genes would likely be lost, and the final resistance obtained might resemble resistance of E228 more than resistance of Hawaii 7981. If such a parent were then crossed to a susceptible parent to make a hybrid cultivar, the intermediate resistance of the hybrid would probably have little value. If both parents had resistance equal to E228, the hybrid would be an improvement over susceptible cultivars; and such hybrids would probably have resistance similar to a hybrid between a susceptible parent and a parent with Hawaii 7981 type resistance. To obtain inbreds with the resistance level of Hawaii 7981 will require modified backcrossing and field selection under high disease pressure. Also, to insure that resistance genes are not lost, F_3 or possibly F_4 generations would have to be screened between backcrosses so considerable time would be involved. Ultimately resistance in both parents would provide the best resistance. Yet, the first commercially acceptable hybrids would likely have one resistant and one susceptible parent. Although the hybrid would be intermediate (Table 1), this is a good level of resistance which by observation is superior to hybrids heterozygous for race T1 resistance (Scott and Jones, 1989; Scott et al., 1989, 1991).

Control of hypersensitivity to race T3 by a single incompletely dominant gene (Scott et al., 1996) is simpler than has been reported for race T1, where it appears that three genes are involved (Wang et al., 1994; Yu et al., 1995) although two genes have also been reported (Whalen et al., 1993). Field studies with race T1 also indicated that genes for resistance components other than hypersensitivity are required for high levels of resistance (Scott and Jones, 1989; Somodi et al., 1996; Wang, 1992). Thus, breeding for race T1 resistance is also difficult as has been discussed (Scott et al., 1989, 1991). Without horticulturally acceptable race T1 recurrent parents, combining race T1 and race T3 resistance into an acceptable cultivar becomes a formidable task unless tightly linked molecular markers could be found for all the resistance genes. It has not even been possible to evaluate breeding lines for race T1 resistance in Florida because T3 has essentially replaced T1 (Jones et al., 1998; Jones and Scott, personal observations). Perhaps developing cultivars with only T3 resistance would provide acceptable bacterial spot control in production areas such as Florida. The first cultivars would likely have only one resistant parent, thus allowing some T3 to develop that would probably inhibit T1 proliferation. However, if cultivars with two resistant parents were developed, T3 could be suppressed to the point that T1 might emerge.

Given the rather complicated inheritance from the Hawaiian

resistant sources to races T1 and T3, it might be easier to utilize the nonrace specific resistance of PI 114490 (Scott et al., 1997). Such resistance may not be overcome by mutation of new races of *X. campestris* pv. *vesicatoria* as has been the case with single gene resistance in pepper (*Capsicum annuum* L. var. *annuum*) cultivars (Kousik and Ritchie, 1996). In tomato, emergence of T3 overcame the race specific, yet multigenic T1 resistance from Hawaii 7998 and became the predominant race in Florida in the absence of a T1 resistant cultivar (Jones et al., 1998). Inheritance of resistance and breeding strategies for using PI 114490 resistances has not been reported as yet. Resistance from PI 114490 is not as effective to race T3 as races T1 and T2, thus combining resistance from various sources may be another approach for developing broad based and stable resistance to this destructive bacterial disease.

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