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BACTERIAL SPOT RESISTANCE IS NOT ASSOCIATED WITH BACTERIAL WILT RESISTANCE IN TOMATO

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Abstract. **Hawaii 7998, resistant to bacterial spot (caused by *Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye) [XCV] and bacterial wilt (caused by *Pseudomonas solanacearum* E. F. Smith) [PS] was crossed with 'Walter,' which is susceptible to both diseases. Thereafter, backcross and F_2 seed were obtained. Plants of the parents and derived generations were inoculated with XCV in 2 field experiments. Cuttings were taken from each plant, rooted, inoculated with PS, and transplanted to the field. Thus, each plant was rated for both diseases. Both diseases were more severe in the higher temperature experiment. In the segregating F_2 generation, 65.7% of the plants were resistant to bacterial wilt and 16.9% of the plants were resistant to bacterial spot over both experiments. The percentage of bacterial spot resistant plants was similar for both bacterial wilt resistant and susceptible groups. Thus, the resistances were inherited independently and incorporation of both resistances into susceptible backgrounds requires screening for both diseases. Bacterial wilt resistance from Hawaii 7998 was largely controlled by a single dominant gene. However, verification of resistance beyond the F_2 generation was required for both diseases due to their rather complex inheritances and because of the environmental influences on disease expression.**

Bacterial wilt, caused by *Pseudomonas solanacearum* E. F. Smith, has wide distribution, limiting tomato (*Lycopersicon esculentum* Mill.) production in the humid tropics (9). Bacterial spot, caused by *Xanthomonas campestris* pv. *vesicatoria* (Doidge) Dye (XCV), is not as common as bacterial wilt in the tropics, but it is a major problem in some areas and is the most serious disease of tomato in Florida (5). Since neither disease is easily controlled by cultural or chemical means, genetic resistance may hold the best potential for prevention of these diseases.

In 1983, Scott and Jones (8) discovered field resistance to bacterial spot in a bacterial wilt resistant accession, Hawaii 7998. Plants of Hawaii 7998 were found to react in a hypersensitive manner to the bacterial spot pathogen (1). The resistance was found to be inherited multigenically (6), and it was presumably controlled by a gene that promotes hypersensitivity plus other resistance genes. The latter genes conceivably might also confer bacterial wilt resistance. Laterott (3) reported that accessions of tomato that were resistant to bacterial wilt were also resistant to bacterial canker caused by *Corynebacterium michiganense* (Smith) Jensen. There are other examples of accessions with some resistance to more than one bacterial pathogen, but linkages between the resistances have not been explored (7). The objective of this report was to present the results of experiments which tested if resistance to bacterial spot was related to resistance to bacterial wilt.

Materials and Methods

Hawaii 7998 (H), resistant to both bacterial spot and bacterial wilt, was crossed with 'Walter' (W), susceptible to both diseases, to obtain F_1 (WxH) seed. Subsequently, the F_1 was backcrossed to both parents [W(WxH) and H(WxH)] and F_2 L(WxH)] ⊗ seed was obtained. The 6 genotypes were used in 2 experiments. Seeds for these experiments were sown in an inert medium and transplanted to planter flats (Todd) with 1.5 inch^2 (3.8 cm^2) cells 10 to 14 days later. After one month, transplants were set in the field on beds of EauGallie fine sand that were 6 inches (15 cm) high and 30 inches (75 cm) wide. The soil had been fumigated with methyl bromide:chloropicrin (67%:33%) at the rate of 350 lb/acre (392 kg/ha) 2 weeks earlier and covered with white polyethylene mulch. The beds were 55 inches (137 cm) from center to center and plants were spaced 25 inches (61 cm) apart within the rows. In the first experiment, plants were transplanted in a completely randomized design with 3 replicates and 10 plants per plot of the parents and F_1, 20 plants per plot of the backcrosses, and 90 plants per plot of the F_2. In the second experiment, a completely randomized block design was used with 3 blocks consisting of 10 plants per plot of the parents and F_1, and 40 plants per plot of the backcrosses and F_2. Recommended fertilizer and insecticide practices were used and chlorothalonil was used to control fungal pathogens. Plants were staked and tied. Water was provided by seepage irrigation.

Plants were inoculated with XCV by spraying a bacterial suspension on the plants in the early morning a few weeks after field transplanting. Inoculum of XCV was prepared from a culture that had been grown on nutrient-yeast-dextrose agar for 48 hr at 83F (28C). The bacteria were washed from the plates, suspended in 0.01 M $MgSO_4$ and adjusted to 10^8 cfu/ml (2). Approximately one month after inoculation, individual plants were rated for percentage defoliation by bacterial spot. Prior to the rating, 2 cuttings were taken from each plant and labeled as to genotype and plant number. Cuttings were rooted in flats of sand in a chamber with intermittent mist for approximately 2 weeks. Plants were transplanted to 4 inch (10.2 cm) pots after rooting and placed on a greenhouse bench. About 2 weeks after transplanting, when the roots had spread through the soil, a knife was inserted straight down into the soil an inch (2.5 cm) away from the stem. Ten and 30 ml of inoculum for the first and second experiment, respectively, were added to the soil at the cut area. Preparation of *P. solanacearum* inoculum was similar to that described for XCV after virulent colonies were selected on tetrazolium chloride medium. A week later, the best cutting from each mother plant was transplanted to the field in the same design as was used for the respective bacterial spot portion.

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Once symptoms of bacterial wilt first appeared, weekly ratings over a 2 month period were made. At the conclusion of each experiment an estimate of plant vigor was made, using a 0 (dead) to 5 (no symptoms) scale.

In the first experiment, henceforth referred to as Summer-Fall 1986, the bacterial spot rating was made in Sept. 1986 and the bacterial wilt vigor ratings were made in Oct. 1986. In the second experiment, henceforth referred to as Fall '87-Spring '88, the bacterial spot rating was done in Oct. 1987, cuttings from the mother plants were held over the winter, and bacterial wilt vigor rating was done in May 1988.

During the Summer-Fall 1986 experiment, temperatures ranged from 83 to 96F (28 to 36C) during the day, with night temperatures ranging from 69 to 78F (21 to 25C) during the bacterial spot portion. For the bacterial wilt portion, temperatures ranged from 84 to 94F (29 to 34C) during the day, and from 68 to 75F (20 to 24C) during the night. During the Fall '87-Spring '88 experiment, temperatures ranged from 80 to 96F (26 to 36C) during the day, with night temperatures ranging from 58 to 75F (14 to 24C) during the bacterial spot portion and from 58 to 89F (14 to 31C) during the day, with night temperatures ranging from 38 to 74F (3 to 23C) during the Spring 1988 bacterial wilt portion.

Plants with bacterial wilt vigor ratings of 4 or 5 were considered healthy (resistant), whereas those with ratings of less than 4 were considered diseased. Chi-square tests were performed on W (WxH) and (WxH) ⊗ generations to determine the goodness-of-fit to a single dominant gene model. Bacterial wilt vigor ratings were used in correlation analysis with percentage defoliation data from XCV inoculations. Within each experiment F_2 plants were considered resistant to XCV if they had defoliation in the range of Hawaii 7998 for that experiment. Chi-square contingency tests were used to determine if the occurrence of bacterial spot resistance in the F_2 corresponded with resistance to bacterial wilt.

Results and Discussion

Bacterial wilt. The percentage of healthy plants for each generation at the conclusion of the bacterial wilt tests is shown in Table 1. There was greater disease development during the hotter Summer-Fall 1986 experiment than in the Fall '87-Spring '88 experiment. This is in agreement with the report of Mew and Ho that the disease is more severe under higher temperatures (4). We postulate that resistance to bacterial wilt from Hawaii 7998 is primarily controlled by a single dominant gene. The percentage of healthy plants for the F_1 was more similar to the resistant parent than the susceptible parent. The theoretical ratio 1:1 of healthy to diseased plants in the backcross to 'Walter' (BCP_1) had an acceptable fit in both experiments (Table 1). The backcross to Hawaii 7998 was at least as resistant as Hawaii 7998. The F_2 segregated 3:1 (healthy to diseased plants) in the Fall '87-Spring '88 experiment (Table 1). However, in the Summer-Fall 1986 experiment the F_2 had an unacceptable fit to a 3:1 (healthy to diseased plants) due to a deficiency of healthy plants. From our test results, although not unequivocal, resistance to bacterial wilt from Hawaii 7998 is largely controlled by a single dominant gene. However, a quantitative genetic analysis would probably support a more complex genetic model. Some disease developed even in Hawaii 7998, especially in the warm Summer-Fall 1986 experiment (Table 1). Furthermore, it has been difficult to get resistant selections to breed true in breeding work. Incomplete penetrance and/or action of modifier genes may be responsible. Thus, although a single dominant gene was a major factor in conferring resistance, breeders should not handle this disease resistance as a single gene system.

Bacterial spot. Results of bacterial spot tests are in Table 2. Disease was more severe in the Summer-Fall 1986 experiment, when the weather was hotter and wetter than in the other experiment. For both experiments, the F_1 was intermediate between the parents, although not skewed toward the resistant parent as in previous experiments (6). In fact, the disease severity for the F_1 in 1986 was unexpectedly high, being skewed towards the susceptible parent and similar to the backcross to 'Walter.' In the F_2, the percentage of resistant plants for bacterial spot was much lower than the percentage of resistant bacterial wilt plants (Tables 1 and 2).

Segregation of resistances. The 2 resistances segregated independently in the F_2 generation, and thus were not as-

Table 1. Total plants and percentage of healthy plants for 'Walter', Hawaii 7998 and derived generations after inoculation with the bacterial wilt pathogon in two experiments at Bradenton, Florida.

Genotype	Generation	Total plants	Healthy plants (%)	Expected ratio	X^2	P
Experiment 1 (Summer-Fall 1986)						
Walter (W)	P_1	18	0	—	—	—
Hawaii 7998 (H)	P_2	25	69.0	—	—	—
W x H	F_1	21	75.7	—	—	—
W (W x H)	BCP_1	53	52.3	1:1	0.090	0.90-0.95
H (W x H)	BCP_2	44	81.0	—	—	—
(W x H) ⊗	F_2	249	58.7	3:1	35.568	>0.001
Experiment 2 (Fall 1987-Spring 1988)						
Walter (W)	P_1	5	16.5	—	—	—
Hawaii 7998 (H)	P_2	22	95.3	—	—	—
W x H	F_1	24	72.0	—	—	—
W (W x H)	BCP_1	51	55.0	1:1	0.490	0.20-0.50
H (W x H)	BCP_2	105	95.3	—	—	—
(W x H) ⊗	F_2	97	83.3	3:1	3.742	0.05-0.10

Table 2. Bacterial spot disease severity and percentage of resistant plants for 'Walter', Hawaii 7998 and derived generations from two field experiments at Bradenton, Florida.

Genotype	Generation	Disease severity			
		Summer-Fall 1986		Fall 87-Spring 88	
		Defoliation (%)	Resistant[z] (%)	Defoliation (%)	Resistant[z] (%)
Water (W)	P_1	41.3	0	14.2	0
Hawaii 7998 (H)	P_2	4.8	100	1.1	100
(W x H)	F_1	32.0	7	8.2	18
W (W x H)	BCP_1	31.0	6	13.3	0
H (W x H)	BCP_2	11.2	59	3.6	60
(W x H) ⊗	F_2	25.6	18	9.5	16

[z]Plants considered resistant if disease severity was in the range for Hawaii 7998.

Table 3. Segregation of F_2 Plants derived from Hawaii 7998 x 'Walter' for bacterial spot and bacterial wilt resistance for two field experiments at Bradenton, Florida.

Experiment	Bacterial wilt	Bacterial spot		Contingency X^2	p
		Resistant	Susceptible		
Summer-Fall 1988	Resistant	19 (16.8%)	94	0.026	.75-.90
	Susceptible	15 (18.8%)	65		
Fall '87-Spring '88	Resistant	13 (16.0%)	68		
	Susceptible	3 (17.6%)	14	0.121	.50-.75

sociated (Table 3). The percentage of bacterial spot resistant plants was similar for both bacterial wilt resistant and susceptible categories ($p > 0.5$). Furthermore, there was no correlation between disease ratings for the 2 diseases in the F_2 (Table 4). Thus, genes that conferred resistance to bacterial wilt were not the same as, or linked to, those conferring resistance to bacterial spot.

It should be noted that F_2 plants rated resistant to either disease in these experiments should have their F_3 progeny tested to verify resistance since genetic control for both is rather complex and environmental factors alter disease expression. However, the lack of association between the resistances was apparent from both the data presented and from other work where several bacterial spot resistant breeding lines were not resistant to bacterial wilt (data not shown). We had hoped that screening only for bacterial spot resistance would insure bacterial wilt resistance, but this is not the case. The results indicate that screening for resistance to both diseases is necessary to insure incorporation of both resistances.

Table 4. Correlation coefficients between bacterial spot and bacterial wilt infection for F_2 plants derived from 'Walter' x Hawaii 7998 in two experiments at Bradenton, Florida.

Experiment	Correlation coefficient	Probability
Summer-Fall 1986	0.03	0.65
Fall '87-Spring '88	–0.03	0.81

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