Inheritance of Collar Rot Resistance in the Tomato Breeding Lines C1943 and NC EBR-2

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ABSTRACT

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Collar rot is a tomato seedling disease caused by the fungus *Alternaria solani*. Resistant and susceptible parents, F_1, F_2, and backcross generations were evaluated for collar rot resistance in a greenhouse. Genetic analyses included midparent-hybrid comparisons, diallel analysis, and generation mean analysis. The genotypes C1943 and NC EBR-2 were most resistant to collar rot. Additive and dominant effects were important in controlling the trait, and collar rot resistance was incompletely recessive to susceptibility.

Alternaria solani Sorauer causes two distinct phases of disease on tomato plants: early blight and collar rot. Early blight, which defoliates mature plants and contributes to major economic losses by growers, is considered a more serious problem than collar rot. Collar rot is mainly a seedbed disease carried to the field on tomato transplants and has been associated with the southern production of tomato seedlings in open fields (16). Seedlings shipped north for transplanting develop characteristic dark, sunken stem lesions close to the soil line (13). As the disease progresses, the lesions girdle the stems, forming "collars." Many diseased transplants break at the point of infection, resulting in poor stands, and those that survive generally have impaired growth and fruit production (9). Therefore, collar rot has serious implications to tomato growers as a form of disease or as a source of inoculum for an early blight epidemic.

The disease is currently controlled through seedbed sterilization and fungicide application. Genetic resistance was identified in several varieties and accessions in 1942 (1,2), but all commercial cultivars presently grown are susceptible to collar rot. The genetics of collar rot resistance has been studied as a means to understand early blight resistance; however, the collar rot and leaf blight phases may be controlled through independent genetic factors (2–5). Some genotypes exhibit resistance to one or both phases of *A. solani* attack (1,4,7).

A 1945 study by Reynard and Andrus (17) suggested that collar rot susceptibility is incompletely dominant to resistance and that a single gene controls the trait. The symbol a_d was designated for the collar rot gene (17). This resistance was identified in the old cultivar, Devon Surprise, but early blight resistance was not evaluated. Since 1945, several advanced breeding lines with early blight resistance have been released (4–6). Some have been evaluated for collar rot resistance or susceptibility, whereas others have not. In the current study, the most advanced early blight resistant breeding lines were evaluated for collar rot resistance and the inheritance of collar rot resistance was determined for these newer sources of germ plasm. Several tomato breeding lines and F_1 progeny screened for collar rot resistance in this study were previously evaluated for field resistance to early blight (10,11). In those studies, early blight resistance was quantitatively inherited. Specific objectives for the current study were to estimate the genetic factors controlling collar rot resistance and to compare the results with those of the early blight inheritance studies.

MATERIALS AND METHODS

Plant material. The tomato genotypes 71B2, C1943, MD 165, Castlejay, and Rutgers were crossed in a half diallel mating design in experiments 1 and 2. Parents and the resulting 10 hybrids, as well as three reciprocal genotypes, were evaluated for resistance. Experiments 3 and 4 used C1943, NC EBR-1, NC EBR-2, 87B187, Castlejay, Rutgers, and Jackpot as parents; nine hybrid, six F_2, and two backcross generations [C1943 × (C1943 × Castlejay) and Castlejay × (C1943 × Castlejay)] were screened. In all experiments, inbred and hybrid genotypes had 10 plants per replicate and segregating genotypes had 25 plants per replicate. Plots were arranged in a randomized complete block design with three replications.

Castlejay, Rutgers, MD 165, and Jackpot are susceptible to both early blight and collar rot. The genotypes 71B2, C1943, NC EBR-1, NC EBR-2, and 87B187 have some degree of early blight resistance (5,6,10,11,15), but only C1943 and NC EBR-2 have good collar rot resistance (5,6,7). C1943 was developed by the Campbell Institute for Agricultural Research and was used as a parent for NC EBR-2, released by the North Carolina Agricultural Research Service in 1988 (6).

Inoculation and rating. Four-week-old tomato seedlings were transplanted into a sterilized seedbed in a greenhouse at the USDA Beltsville Agricultural Research Center, Beltsville, MD. Row spacing was approximately 15 cm, and plant spacing was approxi-

TABLE 1. Analysis of variance for collar rot resistance of inbred and hybrid tomato genotypes

Source	df	Mean Square	F value
Experiments 1 & 2:			
Experiment	1	2.68	0.24
Rep (Experiment)	4	11.15	1.83
Genotype	17	615.84	101.13***[z]
Experiment × Genotype	17	7.38	1.21
Error	68	6.09	
Experiments 3 & 4:			
Experiment	1	14.18	0.77
Rep (Experiment)	4	18.41	0.59
Genotype	15	783.35	24.96***
Experiment × Genotype	15	22.00	0.70
Error	60	31.39	

[z] *** = Significantly different at the 0.1% level.

mately 15 cm. One week after transplanting, plots were inoculated with spore suspensions of *A. solani*. Ten isolates endemic to the eastern United States were grown on lima bean agar for 6 days at 22 C under normal diurnal light conditions. Aerial mycelium was scraped and the cultures were uncovered, inverted, and placed in diurnal light at ambient room temperature for 24 hr to induce sporulation. Spores from all 10 isolates were mixed with distilled water to produce a spore suspension of approximately 20,000 spores per ml. The suspension was applied with a hand-held sprayer directed at the base of the stems. Following inoculation, the soil was mounded around the plant stems and the seedbed was covered with clear plastic for 18 hr the first night to insure adequate humidity for infection.

Plants were removed from the seedbed and soil was washed from the stems 2 wk after inoculation. Ratings for collar rot severity were according to Andrus, et al (1), in which 0 = dead plant, 25 = broken stem, 50 = well-developed lesions, 75 = slight flecking, and 100 = no symptoms. Plants with a rating of 75 or 100 were considered resistant, whereas those with 0, 25, or 50 were susceptible.

Statistical analyses. Parent and hybrid means were separated using the Waller-Duncan k-ratio *t* test, and midparent and hybrid means for each genotype were compared using the Student's *t* test. Genotypes included in the diallel mating system in experiments 1 and 2 were analyzed for general combining ability (GCA) and specific combining ability (SCA) according to Griffing's model 1 method 4 procedure (8). In model 1 method 4, one set of F_1 progeny are included in the analysis, and valid inferences can be made only for the experimental material used. Joint three-

TABLE 2. Collar rot mean values for tomatoes inoculated with *Alternaria solani* in experiments 1 and 2

Genotype	Mean[y]
C1943	92.5 a[z]
71B2 × C1943	59.3 b
C1943 × Castlejay	57.1 bc
Rutgers × C1943	56.7 c
MD 165 × C1943	54.6 c
Castlejay × C19543	54.4 c
C1943 × Rutgers	54.2 c
71B2	50.5 d
MD 165 × 71B2	50.4 d
Castlejay	50.0 d
Rutgers	50.0 d
Castlejay × Rutgers	50.0 d
71B2 × Castlejay	50.0 d
71B2 × MD 165	50.0 d
71B2 × Rutgers	50.0 d
MD 165 × Castlejay	49.2 d
MD 165 × Rutgers	48.4 d
MD 165	47.5 d

[y] A rating of 0 = dead, 25 = broken stem, 50 = well-developed lesions, 75 = slight flecking, 100 = healthy.
[z] Mean separation in columns by Waller-Duncan k-ratio *t* test, 5% level.

TABLE 3. Collar rot mean values for tomatoes inoculated with *Alternaria solani* in experiments 3 and 4

Genotype	Mean[y]
NC EBR-2	83.3 a[z]
C1943	77.1 b
Rutgers × C1943	52.5 c
Castlejay × NC EBR-2	51.8 c
NC EBR-1	51.7 c
87B187 × NC EBR-2	51.7 c
NC EBR-2 × 87B187	51.7 c
C1943 × Castlejay	50.9 c
C1943 × NC EBR-1	49.2 cd
Castlejay	48.7 cd
NC EBR-2 × Castlejay	48.8 cde
Castlejay × 87B187	47.9 cde
Rutgers	47.1 cde
87B187 × Castlejay	44.9 def
87B187	42.8 ef
Jackpot	40.0 f

[y] A rating of 0 = dead, 25 = broken stem, 50 = well-developed lesions, 75 = slight flecking, 100 = healthy.
[z] Mean separation in columns by Waller-Duncan k-ratio *t* test, 5% level.

TABLE 4. Student's *t* test of hybrid and midparent values for collar rot resistance in tomatoes for experiments 1 and 2

Genotype	Midparent	Hybrid
71B2 × C1943	71.5[y]	59.3*[z]
C1943 × Castlejay	71.3	57.1*
Castlejay × C1943	71.3	54.4*
Rutgers × C1943	71.3	56.7*
C1943 × Rutgers	71.3	54.2*
MD165 × C1943	70.0	54.6*
71B2 × Castlejay	50.3	50.0
71B2 × Rutgers	50.0	50.0
Castlejay × Rutgers	50.1	50.0
71B2 × MD165	49.0	50.0
MD165 × 71B2	49.0	50.4
MD165 × Castlejay	48.8	49.2
MD165 × Rutgers	48.8	48.4

[y] A rating of 0 = dead, 25 = broken stem, 50 = well-developed lesions, 75 = slight flecking, 100 = healthy.
[z] Significantly different from the midparent at the 5% level.

TABLE 5. Student's *t* test of hybrid and midparent values for collar rot resistance in tomatoes for experiments 3 and 4

Genotype	Midparent	Hybrid
Castlejay × NC EBR-2	66.1[y]	51.8*[z]
NC EBR-2 × Castlejay	66.1	48.8*
C1943 × NC EBR-1	64.4	49.2*
87B187 × NC EBR-2	63.1	51.7*
NC EBR-2 × 87B187	63.1	51.7*
C1943 × Castlejay	63.0	50.9*
Rutgers × C1943	62.1	52.8*
Castlejay × 87B187	45.9	47.9
87B187 × Castlejay	45.9	44.9

[y] A rating of 0 = dead, 25 = broken stem, 50 = well-developed lesions, 75 = slight flecking, 100 = healthy.
[z] Significantly different from the midparent at the 5% level.

TABLE 6. General combining ability and specific combining ability variance components for resistance to collar rot

Source	df	Mean Square	F value
Experiment	1	0.82	0.06
Rep (Experiment)	4	14.62	2.33
Genotype	9	84.04	13.37***[z]
GCA	4	174.88	27.83***
SCA	5	11.37	1.81
Experiment × Genotype	9	8.26	1.31
Error	36	6.28	

[z] *** = Significant at the 0.1% level.

TABLE 7. Generation means and number of plants in a class for collar rot resistance in the cross C1943 × Castlejay

Generation	n	Mean[y]	Standard error	Number of plants[x] 0	25	50	75	100
P_1 (Castlejay)	59	48.7 a[z]	1.65	3	0	53	3	0
BCP_1	133	52.7 a	1.10	2	3	112	13	3
F_1	58	50.9 a	0.60	0	0	56	2	0
F_2	150	57.5 b	1.39	2	2	106	29	11
BCP_2	149	65.6 c	1.62	0	0	85	35	29
P_2 (C1943)	60	77.1 d	1.91	0	0	8	39	13

[x] Number of plants in a rating class.
[y] A rating of 0 = dead, 25 = broken stem, 50 = well-developed lesions, 75 = slight flecking, 100 = healthy.
[z] Mean separation in columns by Waller-Duncan k-ratio *t* test, 5% level.

factor scaling tests were performed for five tomato families evaluated in experiments 3 and 4 to predict fitness of the data to a simple additive-dominance model (12).

RESULTS

As assumptions for homogeneity of variance were met, combined analyses of variance were performed for data from experiments 1 and 2 and from experiments 3 and 4 (Table 1). Genotypic effects were highly significant for all experiments. Experiment effects and experiment × genotype interactions were nonsignificant.

Genotypic mean separation for experiment 1 and 2 (Table 2) showed that C1943 was resistant to collar rot, whereas 71B2, MD 165, Castlejay, and Rutgers were susceptible. Hybrids of C1943 and susceptible parents were also susceptible. The overall infection severity was greater in experiments 3 and 4 (Table 3), but the results were consistent with those of experiments 1 and 2. C1943 and NC EBR-2 were the only resistant genotypes. NC EBR-1, 87B187, Castlejay, Rutgers, and Jackpot were susceptible, as were crosses between resistant and susceptible parents. Hybrid means for resistant × susceptible crosses were significantly lower than their respective midparent values (Tables 4 and 5), whereas means of susceptible × susceptible crosses were not.

Diallel analysis of hybrids included in experiments 1 and 2 provided estimates of GCA and SCA (Table 6). GCA is the average performance of a line in a hybrid combination, whereas SCA denotes the presence of hybrids that perform better or worse than the average performance of the lines involved (8). The nonsignificant SCA suggests that no exceptional hybrids could be identified among the genotypes evaluated. The GCA mean square was highly significant and accounted for 92.5% of the total genotypic variation. Individual GCA effects for each parent were calculated as 5.32, 0.21, −1.45, −1.90, and −2.18 for C1943, 71B2, Rutgers, Castlejay, and MD 165, respectively, with a higher GCA value indicating greater resistance. C1943 had the best general combining ability, which was also reflected in the mean separation data.

Parent, hybrid, F_2, and backcross generation means and variances were determined for the resistant × susceptible cross C1943 × Castlejay (Table 7). The F_1 mean was not significantly different from the susceptible parent Castlejay or from the backcross to Castlejay. The F_2 mean was significantly different from the other five generation means and had a value toward the susceptible genotypes. The mean value of the backcross to the resistant C1943 was midway between the F_1 and C1943 values.

Generation means for parent, F_1, and F_2 generations of the resistant × susceptible crosses C1943 × NC EBR-1, Rutgers × C1943, NC EBR-2 × Castlejay, and NC EBR-2 × 87B187 are shown in Table 8. All hybrids in the four families were susceptible. F_1 means of the NC EBR-2 × Castlejay and C1943 × NC EBR-1 families were not significantly different from their respective susceptible parents, whereas NC EBR-2 × 87B187 and Rutgers × C1943 hybrid means were significantly different from the susceptible parents. F_2 generation means in all families were significantly different from parent and hybrid means. F_2 values were higher than F_1 and susceptible parent values but were generally in the susceptible range.

Estimates of midparent values (m), additive effects ([d]), and dominance effects ([h]) for the five families were determined using joint three-factor scaling tests (Table 9). A simple additive-dominance model was fitted to the data with the scaling test (12). If the data fit the model, the relationships among generation means depend on additive and dominant genetic effects and epistasis is absent (12). All families had significant m, [d], and [h] parameters and fit the simple model as verified with a goodness-of-fit (X^2) test.

TABLE 8. Generation means and number of plants in a class for collar rot resistance in four tomato families

Generation	n	Mean[y]	Standard error	Number of plants[x] 0	25	50	75	100
Family: NC EBR-2 × Castlejay								
P_1 (Castlejay)	59	48.7 a[z]	1.65	3	0	53	3	0
F_1	60	48.8 a	1.92	3	1	54	0	2
F_2	146	57.4 b	1.63	2	7	100	20	17
P_2 (NC EBR-2)	60	83.3 c	1.85	0	0	3	34	23
Family: NC EBR-2 × 87B187								
P_1 (87B187)	59	42.8 a	2.37	8	3	46	2	0
F_1	58	51.7 b	0.84	0	0	54	4	0
F_2	150	58.3 c	1.68	4	7	89	35	15
P_2 (BC EBR-2)	60	83.3 d	1.85	0	0	3	34	23
Family: C1943 × NC EBR-1								
P_1 (NC EBR-1)	60	51.7 a	1.31	1	0	53	6	0
F_1	58	49.2 a	0.60	0	2	56	0	0
F_2	150	56.3 b	1.44	4	2	105	30	9
P_2 (C1943)	60	77.1 c	1.91	0	0	8	39	13
Family: Rutgers × C1943								
P_1 (Rutgers)	60	47.1 a	1.34	2	3	55	0	0
F_1	60	52.5 b	1.14	0	0	55	4	1
F_2	150	58.2 c	1.47	1	3	108	22	16
P_2 (C1943)	60	77.1 d	1.91	0	0	8	39	13

[x] Number of plants in a rating class.
[y] A rating of 0 = dead, 25 = broken stem, 50 = well-developed lesions, 75 = slight flecking, 100 = healthy.
[z] Mean separation in columns by Waller-Duncan k-ratio t test, 5% level.

DISCUSSION

The resistant breeding lines C1943 and NC EBR-2 were evaluated in hybrid combinations with several susceptible genotypes. Performance of the resistant × susceptible crosses approached that of the susceptible parents, and hybrid means were significantly lower than respective midparent means. Also, resistant × susceptible hybrid means were higher than the susceptible × susceptible means. These results indicate that resistance to collar rot is incompletely recessive to susceptibility and support the conclusions of Reynard and Andrus (17), although different sources of resistance were evaluated.

A simple hybrid breeding program with this genetic material would not produce good resistant genotypes, because resistance was recessive and no hybrids performed as well as C1943 or NC EBR-2. Nevertheless, hybridization between C1943 and NC EBR-2 followed by further breeding for horticultural characteristics may be promising.

Diallel analysis for hybrids of C1943, 71B2, MD 165, Rutgers, and Castlejay showed that SCA was nonsignificant, providing further evidence that no superior hybrids were present among the genotypes evaluated. The highly significant GCA indicated the importance of selecting genotypes with good general combining ability for parents in a breeding program.

Analyses of generation means showed that F_2 population means were higher than susceptible parent and F_1 means in each of five families, and resistant plants were present among each F_2 population. Thus, breeding progress by selection of resistant F_2 individuals followed by backcrosses to resistant parents with high GCA values and desirable horticultural traits would appear feasible.

Joint scaling tests for five tomato families revealed that both additive and dominant genetic components controlled the collar rot resistance trait, although dominance effects appeared more

TABLE 9. Scaling tests for a simple additive-dominance model for collar rot resistance in five tomato families

Family	Parameter m	[d]	[h]	chi-square
C1943 × Castlejay	64.29*[z]	−13.51*	−13.16*	4.758
C1943 × NC EBR-1	64.27*	−12.65*	−15.10*	0.100
Rutgers × C1943	62.32*	−15.07*	− 9.61*	0.287
NC EBR-2 × Castlejay	66.09*	−17.20*	−17.31*	0.001
NC EBR-2 × 87B187	63.90*	−20.00*	−12.85*	1.820

[z] Scaling factors significantly different from zero at the 5% level.

important as evidenced by the hybrid means data. This differs from results of the early blight inheritance studies where additive factors were most important, epistasis was present, and dominance effects were minimal (9,10,14,15). Previous reports that early blight resistance may be inherited independently from collar rot resistance (2–5) are supported here, although only for the NC EBR-1, 71B2, and 87B187 genotypes. NC EBR-1, 71B2, and 87B187 were highly susceptible to collar rot, but showed good resistance to early blight (9,10). However, the C1943 source of resistance (also present in NC EBR-2) has foliar resistance and stem lesion resistance closely associated and possibly linked, as reported recently by Gardner (6). When using C1943 or NC EBR-2 as sources of early blight resistance, selection in the greenhouse for collar rot resistance identifies foliar resistance as well. Early blight resistance appears to be controlled by many genes, one of which may be a collar rot gene in C1943 and NC EBR-2.

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