Inheritance of Resistance to Anthracnose Caused by *Colletotrichum coccodes* in Tomato

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ABSTRACT. Inheritance of resistance to tomato anthracnose caused by *Colletotrichum coccodes* (Wallr.) S.J. Hughes was evaluated in parental, F₁, F₂, and backcross populations developed from crosses between adapted resistant (88B147) and susceptible (90L24) tomato (*Lycopersicon esculentum* Mill.) breeding lines. Resistance was evaluated via measurement of lesion diameters in fruit collected from field-grown plants and puncture inoculated in a shaded greenhouse. Backcross and F₂ populations exhibited continuous distributions suggesting multigenic control of anthracnose resistance. Anthracnose resistance was partially dominant to susceptibility. Using generation means analysis, gene action in these populations was best explained by an additive-dominance model with additive × additive epistatic effects. A broad-sense heritability (H) of 0.42 and narrow-sense heritability (h²) of 0.004 was estimated for resistance to *C. coccodes*. One gene or linkage group was estimated to control segregation for anthracnose resistance in the cross of 90L24 × 88B147.

Tomato anthracnose, caused by *Colletotrichum* spp., is a disease which primarily affects ripe fruit. The causative organisms include *C. coccodes* (Wallr.) S.J. Hughes, *C. gloeosporioides* (Penz.) Penz. and Sacc. in Penz., and *C. dematium* (Pers.) Grove. *C. coccodes* is the organism most commonly isolated from infected fruit. Anthracnose occurs in North America, Asia, Europe, Africa, and the East Indies and causes crop losses where moisture conditions favor disease development Stevenson and Pohronezny, 1991). In North America, anthracnose is the most serious ripe fruit disease of tomatoes grown for processing in the northeastern and northcentral regions.

Fruit lesions appear as circular sunken areas with concentric ring markings dotted with small black microsclerotia. Infected fruit have a very short vine or shelf life and are susceptible to secondary infections. Although less common, leaves, stems, and roots may also be infected. If left uncontrolled, serious crop losses and reduced crop quality occur. In commercial production fields subjected to standard fungicide spray schedules, incidence of fruit infection may approach 15% (Sherf and McNab, 1986). Barksdale and Stoner demonstrated that fungicide applications could be reduced without reducing yield in breeding lines with genetic resistance to anthracnose.

A number of anthracnose resistant breeding lines have been developed by the USDA with resistance derived from tomato (*Lycopersicon esculentum*) plant introduction (PI) 272636 collected in Costa Rica. Transfer of appreciable levels of genetic resistance from PIs to breeding lines and elite commercial materials has proven difficult. This problem may be attributed to the occurrence of multiple genetic factors important in conferring anthracnose resistance and their presumed partial loss during cultivar development. Resistance to *C. dematium* induced anthracnose in breeding lines developed from PI 272636 was reported to be heritable (Miller et al., 1983) and attributable primarily to additive genetic effects (Ng et al., 1990). Previous studies (Barksdale, 1971, 1972) have demonstrated that resistance to *C. coccodes* derived from PI 272636 was partially dominant to susceptibility but have not examined heritability and additive and

nonadditive genetic variance for resistance. Utilizing an advanced anthracnose resistant tomato breeding line with resistance derived from PI 272636, the objectives of this study were to 1) ascertain the genetic effects which contribute to *C. coccodes* resistance, 2) estimate the number of resistance genes involved in the expression of *C. coccodes* resistance, and 3) determine the heritability of resistance in this breeding material.

Materials and Methods

PLANT MATERIAL. The tomato breeding lines 88B147 and 90L24 were chosen as anthracnose resistant and susceptible parents, respectively, to generate F₁ and segregating F₂ and backcross populations. Line 88B147 is a determinate-vined processing type tomato released by the USDA with anthracnose resistance derived from *L. esculentum* PI 272636 (T.H. Barksdale, personal communication; Ng et al. 1990). 90L24 is an adapted early maturing line chosen for its susceptibility to anthracnose. 90L24 was used as the female parent in crosses with 88B147 to produce F₁ seed. F₁ plants were self-pollinated to produce the F₂ population. 90L24 and 88B147 were used as female parents in crosses with F₁ plants to construct the respective backcross populations. All crosses were conducted using greenhouse-grown plants and standard emasculation practices. Plants were grown using standard greenhouse cultural conditions.

Plants raised for anthracnose resistance evaluations were grown from seedlings in the greenhouse and transplanted to the field at Beltsville, Md., into Comus silt loam in 1994 and into Keyport fine loam soil in 1995. Field grown plants were spaced at 0.6-m intervals in single rows on black polyethylene covered raised beds, with beds positioned on 1.5-m centers. The number of plants evaluated for each generation are given in Table 1. Supplemental water and fertilizer were supplied using trickle irrigation. Standard cultural practices for tomato production and pest control in Maryland were followed except application of fungicide (tetrachloroisophthalonitrile; i.e., chlorothalonil) was discontinued at first fruit set. Five fruit from each plant of the respective lines were harvested at the red ripe stage of development and transferred to the greenhouse for evaluation of anthracnose resistance using a hypodermic puncture inoculation technique (Barksdale and Koch 1969; Robbins and Angell 1970a).

INOCULATION AND DISEASE EVALUATION. Cultures of *C. coccodes*

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Table 1. Anthracnose lesion diameters in parental, F_1 , F_2 , and backcross populations from the tomato cross 90L24 x 88B147.

Generation ^z	1994		1995	
	n	Lesion diam (mm)	n	Lesion diam (mm) ^y
P_1	20	9.6 ± 0.8	15	19.9 ± 0.9
P_2	20	4.9 ± 0.6	15	12.6 ± 1.0
F_1	20	7.6 ± 0.8	15	14.2 ± 0.7
F_2	100	6.7 ± 0.2	100	13.4 ± 0.4
$BC_1 P_1$	---	---	50	15.2 ± 0.6
$BC_1 P_2$	---	---	50	13.5 ± 0.6

^z P_1 = 90L24, susceptible parent; P_2 = 88B147, resistant parent.

^yPopulation lesion diameters are the average for n number of plants. Lesion size for each plant is the mean for five inoculated fruit.

Table 2. Parameterization coefficients for gene effects in an analysis of generation means.

Generation ^z	Mean phenotype ^y					
	m	[d]	[h]	[i]	[j]	[l]
P_1	1	1	-1/2	1	-1	1/4
P_2	1	-1	-1/2	1	-1	1/4
F_1	1	0	1/2	0	0	1/4
F_2	1	0	0	0	0	0
$BC_1 P_1$	1	1/2	0	1/4	0	0
$BC_1 P_2$	1	-1/2	0	1/4	0	0

^z P_1 = 90L24, susceptible parent; P_2 = 88B147, resistant parent.

^ym = midparent; [d] = additive; [h] = dominance; [i] = additive × additive; [j] = additive × dominance; [l] = dominance × dominance

were isolated from infected tomato fruit, maintained by routine transfer on potato dextrose agar at ≈6-month intervals, and stored at 2 to 4 °C. Cultures were increased for inoculation studies in 100 × 15-mm petri plates on medium containing clarified 30% V-8 tomato juice (Campbell Soup Co., Camden, N.J.) and 2% Difco Bacto-agar. V-8 tomato juice was clarified by mixing 1 Celite diatomaceous earth filter aid : 5 juice (v/v) and filtering the resulting suspension through Whatman no. 2 filter paper. Culture medium was sterilized by autoclaving. Sporulation was promoted by growing cultures under continuous cool white light at room temperature (Barksdale, 1967). Spores were washed from the plates with sterile distilled water and adjusted to a final concentration of 5×10^6 conidia/mL with distilled water. Vine-ripened fruit were transported directly from the field in brown paper bags to a shaded greenhouse. Fruit were placed on brown paper on greenhouse benches and inoculated by placing a droplet of spore

suspension on the fruit and puncturing through the droplet with a 25-gauge needle to a depth of ≈2 mm (Barksdale and Koch, 1969; Robbins and Angell, 1970a). The greenhouse was vented to maintain average minimum daytime temperatures of 24 to 30 °C day and a minimum of 18 °C night. Six days after inoculation, fruit lesion diameters (mm) were measured to the nearest half-millimeter. Check fruit inoculated with distilled water did not develop lesions.

GENETIC ANALYSIS. Statistical analyses were performed using the SAS (SAS Institute, Cary, N.C.) Mixed and General Linear Models procedure. Assessment of genotype × year effects was limited to analysis of parental and F_1 generations since these populations were genetically uniform. Backcross populations of the F_1 to each parental line were not available for evaluation in 1994, so estimation of best fit models were restricted to the 1995 season and plot location. Relevant to the best fit model, related estimates of heritability and gene number were similarly restricted to the 1995 data set. Using generation means and their expectations (Table 2), generation means analysis (Mathers and Jinks, 1982) was performed to produce a best fit model describing the genetic components influencing anthracnose resistance. The analysis was performed using lesion size and three separate transformations of lesion size (natural logarithm, logarithm, square root). The variable lesion size is reported since it exhibited the highest R^2 value (0.93) in the best-fit model and had the lowest sum of squared residuals vs. the transformed data.

Broad-sense heritability (H) estimates were derived using population variance components in the formula described by Wright (1968): $H = \{V_{F2} - [(V_{P1} + V_{P2} + 2V_{F1})/4]\}/V_{F2}$.

Narrow-sense heritability (h^2) was estimated using F_2 and backcross generation variance components as described by Mather (1949) and Warner (1952): $h^2 = [2V_{F2} - (V_{B1} - V_{B2})]/V_{F2}$, where V_{P1} = variance of P_1 (90L24, susceptible parent), V_{P2} = variance of P_2 (88B147, resistant parent), V_{F1} = variance of the F_1 generation, V_{F2} = variance of the F_2 generation, and V_{B1} and V_{B2} = variance of the F_1 backcrossed to P_1 and P_2 , respectively.

An estimate of the number of genes (n) segregating for anthracnose resistance was estimated as described by Wright using the formula $n = \{(P_1 - P_2)^2[1.5 - 2h(1 - h)]\}/\{8[V_{F2} - (V_{P1} + V_{P2} + 2V_{F1})/4]\}$, where P_1 = the mean of P_1 , P_2 = the mean of P_2 , $h = (F_1 - P_2)/(P_1 - P_2)$, and F_1 = the mean of F_1 . V_{P1} , V_{P2} , V_{F1} , and V_{F2} are defined as described above.

Results and Discussion

Anthracnose lesion diameters in fruit of the resistant tomato parent 88B147 (P_2) were 96% less than those observed in the susceptible 90L24 (P_1) line in 1994 and 58% less than P_1 in 1995

Table 3. Population distributions of anthracnose lesion diameters for inoculated fruit of parental, F_1 , F_2 , and backcross plants from the tomato cross 90L24 x 88B147 grown in 1995 field plots.

Population ^z	Upper class limit [lesion diam (mm)] ^y										
	6	8	10	12	14	16	18	20	22	24	26
	Plants (no.)										
P_1					1	2	1	4	2	3	2
P_2		1	4	3	4		2		1		
F_1	1	3	1	2	2		2	1	3		
F_2	3	7	20	18	15	11	10	14	2		
$BC_1 P_1$		1	4	10	10	3	7	6	6	3	
$BC_1 P_2$	1	5	3	11	10	10	5	3	1		1

^z P_1 = 90L24, susceptible parent; P_2 = 88B147, resistant parent.

^yLesion size for each plant is the mean for five inoculated fruit.

Table 4. Analysis of variance estimates of anthracnose lesion diameter estimated from the tomato cross 90L24 x 88B147. Generations were considered fixed effects and fruit within generations were considered random effects.^z

Source	1994 ^y		1995 ^x	
	Type III F	P > F	Type III F	P > F
Generation	9.04	0.0002	42.78	0.0001

Source	1994 ^y			1995 ^x		
	Variance	Z	P > Z	Variance	Z	P > Z
Fruit (generation)	39.51	10.02	0.0001	24.67	10.00	0.0001

^zAnalysis of variance conducted using Proc Mixed in SAS.

^y1994 generations comprised of P₁, P₂, F₁, and F₂.

^x1995 generations comprised of P₁, P₂, F₁, BC₁P₁, BC₁P₂, and F₂.

in fruit collected from field plots and puncture inoculated with a spore suspension of *C. coccodes* (Table 1). Observed lesion diameters were ≈ 2 to 2.5 times greater in 1995 tests in comparison to 1994 evaluations. Relative rankings of respective generations, however, remained the same from year to year. Evaluation of lesion diameter for year and genotype $\times$ year effects did not indicate significant year ($Z = 0.69$; $P > 0.487$) or genotype $\times$ year effects ($Z = 0.73$; $P > 0.465$). Although nonsignificant, observed differences in relative lesion size could be attributed to a number of environmental effects. Since greenhouse cooling capacity was limited, warmer ambient temperatures experienced during 1995 vs. 1994 tests likely contributed in large part to the larger lesions observed in 1995. Similar variation in lesion size of susceptible and resistant breeding lines between respective tests was apparent in the results of Robbins and Angell (1970a). Although not true for all varieties, Barksdale and Koch (1969) noted that significantly more disease often occurred on fruit produced on sandy soil than on clay soil when evaluating natural infection.

Use of the puncture inoculation technique to screen for anthracnose resistance is a more severe test to identify lines with field resistance, but generally preferred over techniques which rely on natural infection (Barksdale and Koch 1969; Robbins and Angell 1970a). Although tomato lines such as 88B147 with useful levels of field resistance typically exhibit slower growing lesions upon puncture inoculation in comparison to fruit of susceptible lines, tomato lines reported to be field resistant based on natural or nonwounding techniques have been reported to be susceptible in the field (Barksdale 1970, Barksdale 1971, Robbins and Angell 1970a). Using the puncture technique, fruit are uniformly inoculated and sample sizes can be small because the frequency of escapes is much lower. Although many isolates of the fungus can penetrate the fruit epidermis without prior wounding, lack of infection in anthracnose susceptible fruit has been commonly reported when using nonwounding inoculation techniques (Barksdale 1967, Fulton 1948, Hoadley 1960, Kendrick and Walker 1948).

Frequency distributions for 1995 parental, F₁, F₂, and backcross populations developed from these parental lines are shown in Table 3. As expected, analysis of variance demonstrated significant differences between generations in both 1994 and 1995 tests (Table 4). Mean lesion size of the F₁ and F₂ populations is intermediate to the two parents and skewed toward the resistant parent (Table 1). Backcrosses of the F₁ to the respective susceptible and resistant parents produced mean lesion sizes intermediate between the F₁ and P₁ or P₂, respectively. Similar to the F₁ and F₂ populations, lesion sizes of the backcross populations evaluated in

1995 were skewed toward the resistant parent, suggesting that resistance is partially dominant to susceptibility. Barksdale (1971) noted partial dominance for resistance to anthracnose caused by *C. coccodes* in populations developed between crosses of the anthracnose resistant PI 272636 and a susceptible tomato cultivar. Data describing resistance in tomato to anthracnose caused by *C. coccodes* and *C. dematium* suggests that the direction of gene action is influenced by the choice of parents used to develop the segregating populations.

A series of genetic models were tested for goodness of fit to anthracnose lesion diameter in the populations available from the cross of 90L24 x 88B147. The data obtained from ripe fruit infected with *C. coccodes* did not fit a simple additive-dominance model (m[d][h]), suggesting that nonallelic interactions or epistatic effects accounted for a portion of the variability (Table 5). The addition of additive $\times$ additive ([i]), additive $\times$ dominance ([j]) or dominance $\times$ dominance ([l]) to the additive-dominance model resulted in acceptable four-parameter models based on their chi-square values (m[d][h][i], m[d][h][j] and m[d][h][l], respectively). The additive-dominance model with additive $\times$ additive epistatic effects (m[d][h][i]) was chosen as the best fit model based on its lower chi-square value, high R² value, and lowest error variance (s²). Estimates of genetic effects in this model indicated small but significant additive effects [d] (Table 6). Although additive $\times$ additive effects [i] were nonsignificant, their addition to the additive-dominance model explained a larger portion of the variability (i.e., larger R² values and smaller s²) and improved the precision of the parameter estimates as evidenced by their lower standard errors. Dominance effects [h] were not significant in the best fit model for anthracnose lesion diameter. Parameter esti-

Table 5. Genetic models tested for goodness of fit to anthracnose lesion diameter in tomato populations developed from the cross 90L24 x 88B147.

Model tested ^a	χ^2	χ^2 critical	R ²	s ²
m[d][h]	12.35 [*]	7.81	0.74	4.12
m[d][h][i]	3.24	5.99	0.93	1.62
m[d][h][j]	5.87	5.99	0.88	2.94
m[d][h][l]	5.87	5.99	0.88	2.94
m[d][h][i][j]	3.24	3.84	0.93	3.24
m[d][h][i][l]	3.24	3.84	0.93	3.24

^am = midparent; [d] = additive; [h] = dominance; [i] = additive $\times$ additive; [j] = additive $\times$ dominance; [l] = dominance $\times$ dominance

^{*}Significant at $P \leq 0.05$. Models significant at $P \leq 0.05$ deviate from expectations and are rejected.

Table 6. Estimates of genetic effects in the best fit model $m[d][h][i]$ for anthracnose lesion diameter in the tomato cross 90L24 x 88B147.

Source ^a	Parameter estimate	Standard error
m	13.36	0.48
[d]	2.91*	0.66
[h]	1.74	1.96
[i]	3.77	1.59

^am = midparent; [d] = additive; [h] = dominance; [i] = additive x additive

*Significant *t* test at $P \leq 0.05$

mates for additive, dominance, and epistatic effects were nonsignificant in the $m[d][h][j]$ and $m[d][h][l]$ models. The more complex five-parameter models, $m[d][h][i][j]$ and $m[d][h][i][l]$, were judged to be inappropriate models based on their relatively higher, although nonsignificant, chi-square values, nonsignificant additive, dominance and interaction effects, and their higher error variance.

Robbins and Angell (1970b) determined that genetic variance in tomato for *C. coccodes* resistance derived from two *L. pimpinellifolium* (Jusl.) Mill. accessions was primarily nonadditive with a large dominance component. Resistance to anthracnose caused by *C. dematium* in PI 272636 derived lines was reported to be primarily additive but dominance and epistatic effects were also present (Miller et al., 1983; Ng et al., 1990). General combining ability in the latter study accounted for >90% of the genetic variation, supporting a predominant role for additive effects in resistance. In *L. esculentum*, resistance to *C. coccodes* is similar but not identical to resistance to *C. dematium* (Miller et al., 1984). Although it would be advantageous to examine genetic effects from a larger number of accessions of the respective species, anthracnose resistance has been identified in only a few accessions of each species. Available data indicates that genetic effects derived from anthracnose resistant *L. pimpinellifolium* accessions are primarily nonadditive, or dominant, whereas genetic effects contributed by resistant *L. esculentum* accessions are primarily additive.

A broad-sense heritability (*H*) of 0.42 was estimated for *C. coccodes* resistance in 88B147 which is derived from PI 272636. Narrow-sense heritability was estimated as 0.004. An average broad-sense heritability of 0.57 and narrow-sense heritability of 0.42 for resistance to tomato anthracnose caused by *C. dematium* was estimated in *L. esculentum* populations with resistance derived from PI 272636 (Ng et al., 1990). Robbins and Angell (1970b) reported a broad-sense heritability of 0.81 for *C. coccodes* resistance in *L. esculentum* derived from *L. pimpinellifolium*. The moderate to high heritabilities reported suggest that reasonable progress could be made in a breeding program to introgress useful levels of anthracnose resistance into *L. esculentum*. Our low estimate of narrow-sense heritability is consistent with the relatively low but significant additive effects estimated from our best fit model. Low or negative heritability estimates may also be exacerbated by the variance component method used to estimate heritability (Cassol and St. Clair 1994). Departures from normality and large environmental effects which result in data inaccuracies cause small errors to become extremely inaccurate when partitioning variance, thus resulting in poor heritability estimates which may be zero or negative (Simmonds 1979). Environmental bias in heritability estimates would be limited by replication over different environments. Using Warner's estimate, however, this bias cannot be eliminated by replication over environments since a different sample of annual plants is used in each environment

(Nyquist 1991). These heritability estimates do not take into account potential linkage among resistance loci, which can further bias heritability estimates and the difficulty of introgressing quantitatively inherited traits into cultivated forms of the *L. esculentum* with an intensity equal to that observed in the wild donor parent. Linkage between resistance loci and undesirable characters may also limit the rate of progress realized in a breeding program. Intermating of promising early generation selections may permit recombination of desirable resistance genes and maximize subsequent progress in the breeding program. In light of strong additive effects in their materials, Ng et al. (1990) suggested that maximum progress in improving anthracnose resistance in tomato could be attained by minimizing dominance and epistatic relationships.

The number of genes or effective factors segregating for anthracnose resistance in the cross of 90L24 x 88B147 was estimated as 1.09. The apparent complex inheritance observed for anthracnose resistance and the difficulty encountered in transferring high levels of resistance from unadapted PI lines to elite cultivated materials, suggests that the number of genes or factors governing resistance is underestimated and that our estimate may reflect a single linkage group of a number of genes. As discussed by Zeng et al. (1990), underestimation of the true number of loci occurs when Wright's assumptions are violated. These assumptions include additive gene action with equivalent allelic effects at all loci, absence of linkage, and parental lines which are at opposite phenotypic extremes. Alternatively, since the resistant parent 88B147 is an adapted processing type tomato, far removed from its resistant wild donor parent, which exhibits slow growing, small one to two millimeter diameter lesions (Barksdale 1970; Stommel, unpublished results), resistance genes may have been lost in the breeding process and only a single major resistance gene or a small linkage group of genes remains.

Based on observations of frequency distributions for segregating populations, Barksdale (1971) suggested that *C. coccodes* resistance in *L. esculentum* PI 272636 was quantitatively inherited. In populations developed between crosses of susceptible *L. esculentum* parents and two resistant *L. pimpinellifolium* accessions, Robbins and Angell (1970b) estimated via components of variance analysis that at least six genes influenced anthracnose resistance. However, by arbitrarily assigning plants to a discrete resistant or susceptible class with 11.5 mm assigned as the point of separation, they were able to fit their data to a single gene model using a chi square test. The disparity in gene number of the latter estimates likely reflects the difficulty of assigning plants from a continuously distributed population to discrete classes when such classes cannot be clearly distinguished.

Our results support a quantitative mode of inheritance for anthracnose resistance. The variability in gene number estimates and heritability reported for anthracnose resistance likely reflect genetic differences in resistant parental sources and their relative stage of development for commercial use, which ranged from unadapted or wild to advanced breeding lines. The significant contribution of additive effects to resistance in *L. esculentum* suggests that high levels of resistance may be attainable in adapted lines if individual genes which influence resistance can be combined.

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