Comparison of Inheritance of Resistance to Tomato Anthracnose Caused by Two *Colletotrichum* spp.

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ABSTRACT

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Inheritance in tomato of resistance to anthracnose caused by *Colletotrichum coccodes* and *C. dematium* was compared using a six-parent diallel in 1982. The parents, one set of F_1 hybrids, and five reciprocal crosses were grown at the USDA Beltsville Agricultural Research Center. Reciprocal effects were not present in either set of inoculations of the two pathogens for the five crosses studied. Mean squares for general combining ability accounted for most of the genetic variability in both sets of inoculations. Mean squares for specific combining ability were only significant for the inoculations involving *C. dematium*. According to the analysis of variance and covariance of the parental arrays, partial dominance appeared to be in the direction of susceptibility. Narrow-sense heritabilities were 78% for *C. coccodes* and 64% for *C. dematium*. The correlation coefficient between inoculations with *C. coccodes* and *C. dematium* was 0.87.

Additional key words: *Lycopersicon esculentum*

The fungus *Colletotrichum coccodes* (Wallr.) Hughes has traditionally been cited as the causal organism of tomato anthracnose, a rot of ripe fruit of tomato (*Lycopersicon esculentum* L.). However, a number of other *Colletotrichum* and *Glomerella* spp. have also been shown to cause typical anthracnose lesions on tomato (4,6,7). *C. dematium* (Pers. ex Fr.) has been isolated from field-infected fruit in the midwestern and eastern processing tomato production regions (14).

Resistance to *C. coccodes* has been studied by Robbins and Angell (11) and Barksdale (3,5). These researchers determined that this trait was quantitatively inherited with partial dominance in the direction of resistance. Currently, no tomato cultivar is resistant to *C. coccodes* or *C. dematium*, although PI 272636 is resistant to both (2,4).

The purpose of this study was to compare the inheritance of resistance to tomato anthracnose caused by *C. coccodes* and *C. dematium*.

MATERIALS AND METHODS

Six inbred tomato lines were crossed in every possible combination in a diallel mating design. Three inbreds were USDA breeding lies (81B416-1, 81B1105-2, and 625-3-1) resistant to *C. coccodes* and *C. dematium*; resistance in these three lines was derived from PI 272636. The fourth inbred was a tolerant selection from a line (Ark79-90) developed by Joe McFerran, University of Arkansas. The susceptible parents used were the commercially grown cultivar US141 and a USDA breeding line 81B9, both developed by Allan K. Stoner, USDA, Beltsville Agricultural Research Center.

The six parents, one set of F_1 hybrids ($\frac{1}{2}p\ (p-1)$, where p = number of parents), and five selected reciprocal crosses (Ark79-90 × US141, 81B1105-2 ×US141, 625-3-1 × 81B9, 625-3-1 × Ark79-90, and 81B1105-2×625-3-1) were transplanted in the spring of 1982 into a Hatboro silt loam at the USDA Beltsville Agricultural Research Center (BARC) at Beltsville, MD. Plots were arranged in a randomized complete-block design with four replicates and 10 plants per plot. Recommended cultural practices and pesticide applications were performed. On 24 August, two sets of 20 mature, blemish-free, red fruit were harvested from each plot and transported in paper bags to a shaded greenhouse in Beltsville. The fruits were placed on brown paper on benches in preparation for inoculation.

Two isolates of *C. coccodes* and two of *C. dematium* were grown on 30% filtered V-8 juice agar under continuous light at room temperature (1). Isolates of each species were combined in distilled water to provide an inoculum of 5×10^6 and 9.6×10^6 spores per milliliter of *C. coccodes* and *C. dematium*, respectively. The hypodermic inoculation technique developed by Robbins and Angell (10) was used. Six days after inoculation, lesion diameters were measured.

Mean lesion diameter was calculated for each 20-fruit sample. Orthogonal contrasts were performed on the plot means for each inoculation between selected crosses and their reciprocals. A $\log_{10}$ transformation was performed on plot means because variance heterogeneity was found in an F_{max} test. Transformed data gave homogeneity of variance and were used in further analyses.

An analysis of variance was performed on the parent and F_1 hybrid data for each species. Means of the parents and hybrids within each inoculation were compared using a Waller-Duncan Bayesian k-ratio t test (13). General combining ability (GCA) and specific combining ability (SCA) were calculated according to Griffing's Model 1 Method 4 (8) by the Schaffer and Usanis computer program (12), where only the data from the F_1 hybrids were used. GCA effects were also calculated for each parent within each inoculation.

The type of gene action was estimated by a diallel analysis described by Hayman (9). In this analysis, the variance of the parental means (V_p), mean variance of the offspring of each parental array (V_r), mean variance of the arrays (V_a), and the covariance of the arrays with the nonrecurring parent (W_r) were calculated. A t test was performed on the W_r-V_r values to determine whether certain assumptions (ie, diploid segregation, no reciprocal differences, independent action of nonallelic genes, no multiple allelism, homozygous parents, and independent distribution of genes) were met. Also, the regression coefficients of the lines produced in the V_r-W_r graphs were tested for significant differences from unity. A difference would indicate a nonallelic interaction for some arrays. Last, theoretical limiting parabolas, within which all array points must lie, were calculated.

The parent-offspring regression technique (15) was used to calculate narrow-sense heritability. This value provided an estimate of the proportion of the phenotypic variance that resulted from the additive effects of the genes. Finally, a correlation coefficient was calculated between the mean lesion diameters resulting from inoculations with two *Colletotrichum* spp.

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RESULTS AND DISCUSSION

The analysis of variance containing the planned comparisons of five selected crosses (US141 × Ark79-90, US141 × 81B1105-2, 81B9 × 625-3-1, Ark79-90 × 625-3-1, and 625-3-1 × 81B1105-2) and their reciprocal crosses determined that there were no significant differences within each comparison. Therefore, the assumption of no reciprocal effects was made, thereby permitting the use of Griffing's Model 1 Method 4 for the diallel analysis.

Mean lesion diameters of the parents and F_1 hybrids obtained 6 days after inoculation with both *Colletotrichum* spp. are presented in Table 1; however, the Waller-Duncan *k*-ratio *t* test was performed on the transformed data. Four parents and most of the F_1 hybrids responded similarly to inoculation by the different species. Of the remaining two parents, 81B416-1 had a smaller mean lesion diameter and 81B1105-2 had a larger mean lesion diameter when inoculated with *C. dematium* than with *C. coccodes*. Thus, the resistance to *C. coccodes* in tomato may not be identical to the resistance to *C. dematium*.

The analyses of variance with GCA and SCA mean squares are presented in Table 2. Most of the genetic variability in both sets of inoculations was accounted for by the significant GCA mean squares (Table 2). Fruit inoculated with *C. dematium* also had a small but significant SCA mean square. GCA can be interpreted in terms of additive genetic variance and SCA in terms of nonadditive genetic variance. Therefore, the significant variation noted among the F_1 hybrids within each inoculation was primarily a result of additive genetic effects. *C. dematium*-inoculated fruits may have a small nonadditive component contributing to the variation.

GCA effects were also calculated for each parent within each inoculation (Table 3). A negative GCA effect was desired because resistance was expressed as smaller lesion diameter. The most negative GCA effect was obtained with 625-3-1, followed by Ark79-80, 81B416-1, and 81B1105-2. The two susceptible lines, 81B9 and US141, had positive GCA effects. There were no differences in ranking of the GCA effects of the parents between inoculations of the *Colletotrichum* spp.

The degree of dominance controlling the trait was estimated by the genetic analysis described by Hayman (9). The *t* tests performed to test the appropriateness of Hayman's analysis were not significant for either set of inoculations of the two pathogens; therefore, the assumptions of the analysis were probably met. For *C. coccodes* (Fig. 1) and *C. dematium* (Fig. 2), the *y*-intercepts of the V_r-W_r regression lines were greater than zero; partial dominance was therefore assumed for each case. For the set of fruit inoculated with *C. coccodes*, 625-3-1 (the most resistant parent) was furthest from the origin, having the largest V_r and W_r values; a relatively high proportion of recessive alleles was therefore assumed. The remaining parents had lower V_r and W_r values characteristic of a relatively high proportion of dominant alleles. However, in inoculations with *C. dematium*, US141, 81B9, Ark79-80, 625-3-1, and 81B1105-2 had the lowest V_r-W_r values, whereas 81B416-1 had the highest. Again, the resistance to both species may not be exactly the same. In both cases, however, there was a tendency for partial dominance to be in the direction of susceptibility to anthracnose.

The parent-offspring regression technique was used to calculate narrow-sense heritability (h^2) (15). Narrow-sense h^2 was 78 and 64% for *C. coccodes*- and *C. dematium*-inoculated fruit, respectively. Again, in both cases, the additive variance was relatively high, which was in agreement with the combining ability analyses.

Last, the responses of the tomato fruit to inoculation by *C. coccodes* and *C. dematium* were correlated (r = 0.87). Because the correlation coefficient was highly significant (P = 0.01) and the reactions of the fruit to the two organisms were directly related, either *C. coccodes* or *C. dematium* could be used for screening for resistance in a breeding program. As a precaution, however, if one species is more prevalent in a particular growing region, breeding lines developed for that region should be screened with that species.

In earlier studies (3,5,11), resistance to anthracnose as caused by *C. coccodes* was inherited quantitatively with resistance

Table 1. Fruit mean lesion diameters (mm) for parents and crosses from a six-parent diallel of tomato inoculated with *Colletotrichum coccodes* or *C. dematium*

Genotype	*C. coccodes*	*C. dematium*
US141	18.9 a[z]	20.7 ab
81B9	17.6 a	21.1 ab
US141 × 81B9	17.5 ab	18.4 abcd
81B9 × 81B1105-2	10.9 bc	10.5 ef
US141 × 81B416-1	10.5 cd	13.9 bcde
US141 × Ark79-90	8.2 cde	8.5 fgh
US141 × 81B1105-2	7.8 cde	6.2 hijk
81B9 × Ark79-90	7.5 cdef	9.9 efg
81B9 × 81B416-1	7.9 cdef	13.7 bcde
81B9 × 625-3-1	7.0 cdef	5.7 hijk
US141 × 625-3-1	6.9 defg	3.9 lmno
81B416-1	5.2 efgh	1.5 qr
Ark79-90 × 81B416-1	4.9 fghi	2.9 nop
Ark79-90 × 81B1105-2	4.2 ghi	5.9 hijk
81B416-1 × 81B1105-2	4.4 hi	5.1 ijkl
Ark79-90	4.2 hi	3.6 no
81B1105-2	3.7 hi	6.6 ghij
625-3-1 × 81B1105-2	3.5 ij	3.3 mno
81B416-1 × 625-3-1	2.4 jk	1.4 qr
Ark79-90 × 625-3-1	2.1 jk	2.5 op
625-3-1	1.6 k	1.0 r

[z]Mean separation was performed on $\log_{10}$ transformed data by Waller-Duncan *k*-ratio *t* test.

Table 2. Combining ability analysis of a six-parent diallel of tomato for resistance to *Colletotrichum coccodes* and *C. dematium*

Source of variation	df	Mean square[a] *C. coccodes*	Mean square[a] *C. dematium*
Replicates	3	0.03 ns	0.01 ns
GCA[b]	5	0.07 **	0.97 **
SCA	9	0.03 ns	0.09 **
Error	42	0.03	0.02

[a]Mean lesion diameter subjected to a $\log_{10}$ transformation before analysis of variance; ns = not significant and ** = significant at P = 0.01.
[b]GCA = general combining ability and SCA = specific combining ability.

Table 3. Estimates of general combining ability effects for resistance to *Colletotrichum coccodes* and *C. dematium* for each parent in the tomato diallel

Parent	*C. coccodes*	*C. dematium*
81B9	0.20[a]	0.27[a]
US141	0.20	0.16
81B1105-2	−0.04	−0.01
81B416-1	−0.06	−0.05
Ark79-90	−0.09	−0.07
625-3-1	−0.21	−0.30
$\bar{x}$[b]	0.76	0.76

[a]Mean lesion diameter subjected to a $\log_{10}$ transformation.
[b]Mean lesion diameter of F_1 hybrids from transformed data.

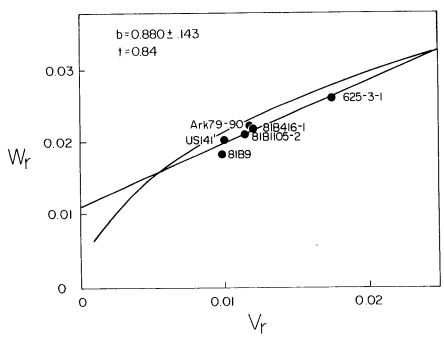

Fig. 1. Variance-covariance (V_r-W_r) graph with theoretical, limiting parabolas for susceptibility to *Colletotrichum coccodes* in a six-parent diallel of tomato.

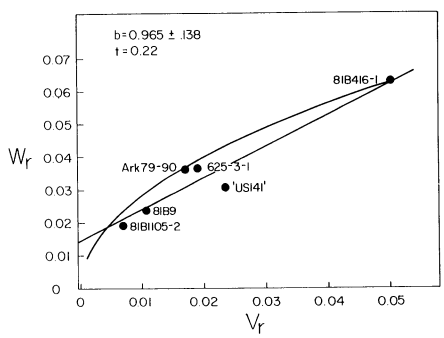

Fig. 2. Variance-covariance (V_r-W_r) graph with theoretical, limiting parabolas for susceptibility to *Colletotrichum dematium* in a six-parent diallel of tomato.

being partially dominant. In this study, however, the variation in the F_1 hybrids was primarily due to additive gene effects and partial dominance was in the direction of susceptibility. This probably was because different plant material was used. The assumptions of this study allowed inferences only to be made on the experimental material.

The most significant component in this population was the additive variance; therefore, relatively rapid genetic advance should be possible in breeding and selection of resistant phenotypes. We did not test the possibility of combining both species in inoculum used for screening.

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