

Single Gene Control of Anthracnose Resistance in *Citrullus*?

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Previous reports (1, 3, 6, 7) indicate that resistance to Colletotrichum lagenarium (Pass.) Ell. and Halst. in watermelon is governed by a single dominant gene. These studies differed in classification of resistance, race identification, inoculum levels and susceptibility of greenhouse-grown seedlings.

If a single gene controls resistance to race 2, it should be simple to incorporate the high level of resistance found in resistant into commercially accepted cultivars. Actually, no cultivar has been developed with as much resistance to race 2 anthracnose as the resistant source. A greenhouse study of inheritance of race 2 resistance in documented resistant and susceptible Citrullus genotypes was conducted in 1982. A field study was conducted in 1983. Methods were modified in the second study as indicated.

Methods. An isolate of the fungus was obtained from the EREC and determined to be race 2 by the method of Jenkins et al. (2). Culture and spore production methods were similar to those of Littrell and Epps (4). Randomized blocks of parents and progeny of the host were mist inoculated with spore suspensions: 50,000 spores/ml in the greenhouse and 20,000 spores/ml in the field study. Greenhouse seedlings were inoculated at the 2-4 leaf stage. Field plants were inoculated at fruit set. Greenhouse seedlings were rated 8 days after inoculation on a 0-10 scale (0 = no lesions, 10 = dead) by comparing with a set of 11 plants representing 1 point on the scale. Plants rated 0-6 were considered resistant, 7-10 susceptible. Field plants were rated in two ways five weeks after inoculation: 1 - the oldest branch on each plant was rated for percent defoliation and 2 - percent of remaining leaves showing lesions. A disease index was a composite of both ratings. Plants indexed 0-85 were considered resistant, above 85 susceptible. The average between resistant and susceptible parent means was used as the division between resistant and susceptible plants.

Treatments assigned to each block were F_1 , F_2 and BC_1 generations from a diallel cross among the resistant watermelon plant introduction PI 189225, PI 299379, the susceptible watermelon cv. New Hampshire Midget and a resistant line of Citrullus colocynthis (1) Schrad., designated R309.

Results. The greenhouse seedling study could not distinguish resistant plants from susceptible plants. Anthracnose symptoms developed on even the most resistant lines. This result is consistent with the report of Winstead et al. (7), who reported that PI 189225 was susceptible to anthracnose race 2 and Sowell et al. (5), who reported the PI resistant. Unlike Sowell et al., Winstead et al. depended entirely on greenhouse seedling inoculations to screen for resistance and used an inoculum level of 50,000 spores/ml instead of 20,000 spores/ml.

Resistant and susceptible plants were more easily identified in the field study. Table 1 shows the ratios of resistant and susceptible plants for each of the 3 susceptible x resistant set of parent-progeny populations. Chi-square analysis of F_2 and backcross progenies of the NHM x PI 299379 and NHM x PI 189225 crosses cannot reject the hypothesis that resistance in PI 299379 and 189225 are controlled by a single dominant gene. However, the NHM x R309 progeny does not fit any expected ratio for a 1 or 2 gene dominant, recessive or additive trait.

Frequency distributions of resistance levels in PI 299379 and PI 189225 and their progeny suggest that the inheritance of resistance is more complex than that suggested by Chi-square analysis. The shift of the F_1 populations toward the susceptible parent does indicate complete dominance. Also, the F_2 populations are not divided into discrete classes.

Comparison of means using orthogonal contrasts for each of the 6 sets of parent-progeny populations is found in Table 2. A comparison of the F_1 and F_2 populations with the midparent (average of the 2 parental means) is a test for dominance. Comparison of the F_1 and F_2 with the midparent from PI 299379 x NHM supports the conclusion that resistance is largely due to dominance. However, the failure of the F_1 and backcross generations to show as much resistance as PI 299379, the lack of a bimodal distribution in the F_2 and the failure of the F_2 to produce plants as susceptible as the susceptible parent indicates the presence of other modifying genes. The same conclusion can be drawn from the progeny populations from PI 189225 x NHM.

Comparison of the F_1 and backcross populations from R309 x NHM to the resistant parent indicates resistance is due to complete dominance. However, the F_2 is not significantly different from the midparent and fails to support the conclusion of either 1 or 2 dominant genes. Instead, it is more representative of the distribution expected from several dominant genes acting in concert.

The F_1 population from PI 299379 x PI 189225 (Table 2) is significantly more susceptible than either of the resistant parents, indicating the presence of additive or recessive resistance factors in each line that are not found in the other. The failure of the F_2 population to show distinct segregation for susceptible plants suggests that both lines share a common major dominant gene factor for resistance and that the unshared factors were minor modifiers.

Comparison of progeny means from R309 x PI 189225 indicates that the higher level of resistance displayed by PI 189225 is due to dominance. Thus, it follows that R309 owes its resistance to a set of factors separate from PI 189225. The same conclusion can be reached from the comparison of progeny means from R309 x PI 299379. Small populations, due to poor germination, made statistical analysis of this family difficult. Nevertheless, the involvement of separate genetic factors was apparent from the segregation of the progeny.

In conclusion, two types of inheritance for resistance was identified in the 3 resistant lines. R309 (*C. colocynthis*), demonstrated an intermediate level of resistance attributed to several dominant genes acting in concert. In PI 299379 and PI 189225 resistance is controlled largely by a single dominant gene. In the latter 2 lines, resistance is modified by minor genes.

Literature Cited

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Table 1. Reaction of the parents and progeny from each of the 3 susceptible x resistant crosses to inoculation with *C. lagenarium* race 2 in the 1983 field study. The Chi-square analysis tests the hypothesis that resistance is due to a single dominant gene².

Population	Total Plants	Resistant	Susceptible	Expected Ratio	χ^2	P
NHM x PI 299379						
NHM	64	1	63	0:64		
PI 299379	5	5	0	5:0		
F ₁	58	50	8	58:0		
F ₂	71	55	16	3:1	.230	.50-.70
BC to NHM	68	28	40	1:1	2.118	.10-.20
BC to PI 299379	70	61	9	70:0		
NHM x PI 189225						
NHM	72	5	67	0:72		
PI 189225	58	58	0	58:0		
F ₁	40	39	1	40:0		
F ₂	74	60	14	3:1	1.459	.20-.30
BC to NHM	70	33	37	1:1	.229	.50-.70
BC to PI 189225	74	74	0	74:0		
NHM x R309						
NHM	49	13	36	0:49		
R309	9	9	0	9:0		
F ₁	45	44	1	45:0		
F ₂	69	36	33	3:1	19.174	.01
BC to NHM	34	22	12	1:1	2.941	.05-.10
BC to R309	10	9	1	10:0		

²All plants were indexed on a 0-200 scale with 0 being immune and 200 being dead. Plants with an index from 0-85 were considered resistant, those above 85 susceptible.

Table 2. Comparison of the mean disease index of parents and progeny from each of 6 susceptible x resistant or resistant x resistant crosses in the 1983 field study².

Population	Mean	Comparison ³	
NHM x PI 299379			
NHM	154.35	NHM vs PI 299379	**
PI 299379	26.57	PI 299379 vs F ₁	**
F ₁	72.79	PI 299379 vs BC to PI 299379	**
F ₂	70.21	Midparent vs F ₁	**
BC to PI 299379	58.85	Midparent vs F ₂	**
Midparent	90.46		
NHM x PI 189225			
NHM	142.93	NHM vs PI 189225	**
PI 189225	25.46	PI 189225 vs F ₁	**
F ₁	49.11	PI 189225 vs BC to PI 189225	**
F ₂	57.92	Midparent vs F ₁	**
BC to PI 189225	27.86	Midparent vs F ₂	**
Midparent	84.20		
NHM x R309			
NHM	115.24	NHM vs R309	**
R309	48.70	R309 vs F ₁	**
F ₁	45.86	R309 vs BC to R309	**
F ₂	87.57	Midparent vs F ₁	**
BC to R309	74.82	Midparent vs F ₂	**
Midparent	81.97		
PI 299379 x PI 189225			
PI 299379	26.56	PI 299379 vs PI 189225	**
PI 189225	32.10	PI 299379 vs F ₁	**
F ₁	42.36	PI 189225 vs F ₁	**
R309 x PI 189225			
R309	55.32	R309 vs PI 189225	**
PI 189225	32.99	PI 189225 vs F ₁	**
F ₁	34.03	PI 189225 vs BC to PI 189225	**
F ₂	47.78	R309 vs F ₁	**
BC to PI 189225	32.82		
R309 x PI 299379			
R309	55.80	R309 vs PI 299379	**
PI 299379	33.25	PI 299379 vs F ₁	**
F ₁	44.41	PI 299379 vs BC to PI 299379	**
F ₂	43.02	R309 vs F ₁	**
BC to PI 299379	39.24		

²Plants were indexed on a 0 to 200 scale with 0 being immune and 200 being dead.
³Non-significant (ns) or significant of 5% (*) or 1% (**) levels. Comparisons were made using orthogonal contrasts.

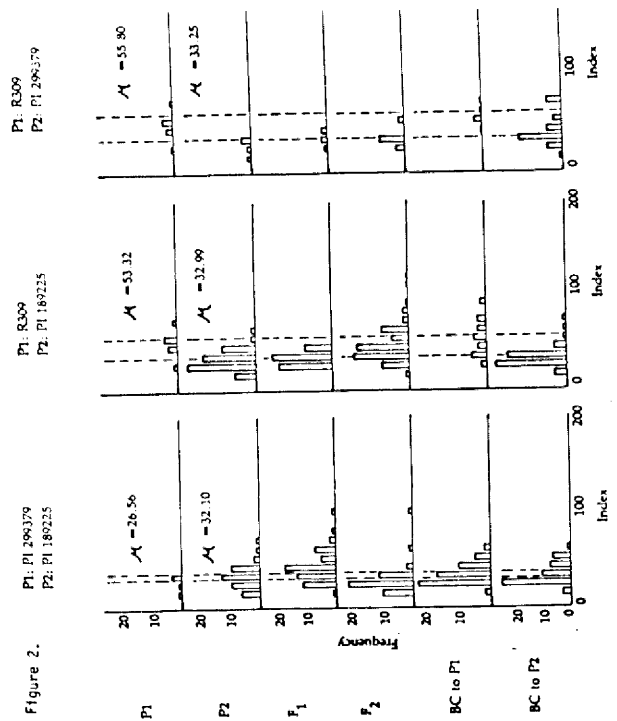


Figure 2.

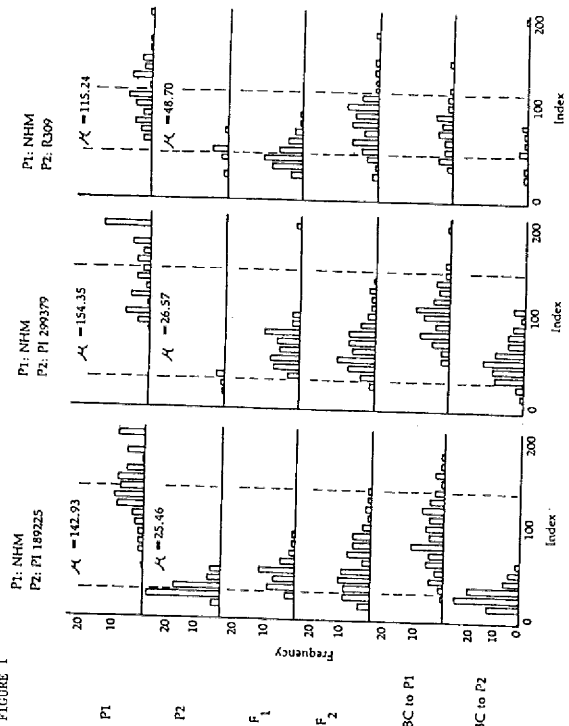


FIGURE 1