Inheritance of Resistance to *Alternaria cucumerina* in *Cucumis melo* Line MR-1

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ABSTRACT

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The resistant reaction of muskmelon line MR-1 to Alternaria leaf blight is characterized by the production of small necrotic lesions in response to infection by the pathogen. These lesions remain restricted and do not expand to support abundant sporulation, as is the case with susceptible cultivars. The F_1, F_2, and BC_1 from crosses of the Alternaria leaf blight-resistant inbred line MR-1 and the susceptible cultivars Perlita and PMR 6 were used to determine inheritance of resistance to *Alternaria cucumerina*. All plants in the F_1 populations were resistant. F_2 phenotypic ratios were 3 resistant:1 susceptible. The BC_1 to the resistant parent populations were all resistant and the BC_1 to the susceptible parent segregated 1 resistant:1 susceptible. The reactions of parental lines and progenies to conidial inoculation with *A. cucumerina* support the hypothesis that the resistance of line MR-1 is conferred by a single dominant gene designated *Ac*.

Alternaria leaf blight, incited by *Alternaria cucumerina* (Ellis & Everh.) J. A. Elliott, is an important foliar disease of muskmelon (*Cucumis melo* L.) in the southeastern and midwestern production areas of the United States (13). Because of the presence of primary inoculum from previous muskmelon crops, Alternaria leaf blight is a perennial problem that must be controlled through the application of protective fungicides (3,11). A more economical and environmentally acceptable means of control of the disease would be the use of resistant cultivars. Several cultivars have been released (1,5–7) that have various levels of resistance to the disease, but these have not been successful in commercial production because of poor shipping quality or the lack of sufficient levels of resistance to downy mildew incited by *Pseudoperonospora cubensis* (Berk. & M.A. Curtis) Rostovzev and powdery mildew incited by *Sphaerotheca fuliginea* (Schlechtend.:Fr.) Pollacci (4).

Muskmelon line MR-1 was released as a source of resistance to both downy and powdery mildews (14). This line has high levels of resistance to five pathotypes of *P. cubensis*, three races of *S. fuliginea* (15), and races 0, 1, and 2 of *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *melonis* W.C. Snyder & H.N. Hans. (18). Because MR-1 is a source of high levels of resistance to several important diseases, it is receiving much use in muskmelon breeding programs (16).

Subsequent to its release, we have used line MR-1 as a resistant check in disease resistance evaluations on muskmelon accessions. *A. cucumerina* was the challenge pathogen in some of these tests. We found that MR-1 showed the same resistant reaction type against *A. cucumerina* as do other reported sources of resistance (12).

The mode of inheritance of resistance to *A. cucumerina* in muskmelon has been unclear. Sitterly (10) cites a personal communication that when the cultivar Hearts of Gold was the resistant parent, evidence indicated that resistance was dominant. Boyhan and Norton (2) reported that studies with AC-82-37-2, UF-G 511, and PI 164756 as the resistant parents "showed no clearly defined Mendelian ratio."

The objective of the research reported in this paper was to determine the mode of inheritance of resistance to *A. cucumerina* in muskmelon line MR-1.

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MATERIALS AND METHODS

Muskmelon line MR-1 was used as the resistant parent in crosses with the susceptible cultivars Perlita and PMR 6. The reaction of the resistant parent had been determined from numerous field and glasshouse studies and the reactions of the susceptible parents were based on similar and published studies (8,12). Parental lines were crossed in the glasshouse using standard techniques for muskmelon (17), except that MR-1, which is monoecious, did not require emasculation. Seeds of the parental, F_1, F_2, and BC_1 generations were produced. We experienced difficulty in obtaining seed of the F_1, F_2, and BC_1 generations with PMR 6 as the female parent because the fruit aborted soon after pollination. Therefore, no inheritance tests were performed on those populations.

The isolate of *A. cucumerina* used in these studies was collected by the first author in 1981 from infected muskmelon in Weslaco, Texas. It has been used in numerous artificial inoculation studies to evaluate resistance to *A. cucumerina* in muskmelon accessions. The isolate was maintained in petri dish culture on V-8 juice agar under fluorescent illumination in a 12-hr light/12-hr dark regime at a laboratory temperature of 24 ± 2 C.

Inocula for all studies were prepared by flooding the surface of 10- to 14-day-old cultures with sterile distilled water and scraping with a large glass coverslip to detach the conidia. The resultant suspension was then thoroughly mixed by a 15-sec treatment at high speed in the microcup of a blender. Because of their large size, the conidia of *A. cucumerina* tend to settle out of suspension rather quickly, and in suspensions of high concentration they tend to clump at the juncture of the slide and coverslip of a hemacytometer. To avoid these problems, suspensions were further diluted with sterile distilled water and were agitated by shaking before aliquots were transferred to a hemacytometer for counting to determine final dilutions.

Seeds of each generation were planted in Jiffy-7 (Jiffy Products Co. of America, West Chicago, IL) peat pellets, two seeds per pellet, in the glasshouse. Before inoculation, each plant was labeled with a string marking tag and pellets were completely randomized among and within planting trays. Symptoms of *A. cucumerina* are more severe on muskmelon leaves when young plants are in poor condition because of excessive crowding or nutrient deficiency (C. E. Thomas, *unpublished*). Therefore, every other row in the planting trays was left empty, and from planting until 10 days after inoculation, the daily watering regime of the plants was supplemented with the application of a dilute (1 g/L) solution of Peters 20-20-20 (N-P-K) (W. R. Grace & Co., Fogelsville, PA) every 7 days. Plants were handled carefully in all experimental manipulations because mechanical injury to the leaves can increase the severity of the reaction of known resistant lines to *A. cucumerina*. The presence of leafminer puncture wounds significantly increases the incidence of *A. cucumerina* lesions on muskmelon leaves (L. D. Chandler and C. E. Thomas, *unpublished*). To decrease the chance that leafminer or other insect wounds might confound the results of our tests, the glasshouse was fumigated weekly with either nicotine sulfate or acephate.

At the two-expanded-leaf stage, plants were inoculated with a suspension of 5×10^3 conidia of *A. cucumerina* per milliliter. The adaxial surfaces of leaves one and two were sprayed to incipient runoff with a Paasche Type-H airbrush (Paasche Airbrush Co., Chicago, IL) at 275 kPa. Inoculated plants were placed in the dark in a dew chamber at 20 C for 16 hr. Germ tubes from conidia of *A. cucumerina* often ramify over the surface of muskmelon leaves for up to 4 days after inoculation when plants are placed in the glasshouse immediately after removal from the dew chamber (C. E. Thomas, *unpublished*). In this study, therefore, when plants were removed from the dew chamber they were placed outside in full sunlight for exposure to ultraviolet light for 8 hr to inhibit the further growth of the germ tubes (9). Plants were subsequently placed in the dew chamber for 16 hr each night and on the glasshouse bench for 8 hr of light each day.

Because we did not know what interaction phenotypes might be encountered in segregating populations, we performed some preliminary tests on small populations of the parental and other generations using the protocols described above. Inoculated leaves were observed at 6 through 10 days postinoculation for lesion type, size, and number. On the basis of our observations, we evaluated the large test populations at 10 days postinoculation and classified the segregates into either of two interaction phenotypes, resistant or susceptible. Plants were classified as resistant if the lesions on leaves one and two remained small (≤1.0 mm) and restricted and as susceptible if the lesions on leaves one and two expanded (≥3.0 mm).

Chi-square tests were used to determine goodness of fit of observed to hypothetical segregation ratios in the F_2 and BC populations. Because the dew chamber could not hold all of the plants necessary for genetic analysis, the data represent the compilation of three tests with Perlita as the susceptible parent. The data for populations with PMR 6 as the susceptible parent were obtained from a single test. All generations were included in each test.

RESULTS

The parents reacted as expected; all plants of Perlita and PMR 6 were

Table 1. Segregation for resistance to *Alternaria cucumerina* in parental, F_1, F_2, and BC_1 generations for crosses of susceptible cultivars Perlita and PMR 6 with resistant line MR-1[a]

Generation	Observed		Expected ratio (R:S)[b]	χ^2		
	Resistant	Susceptible		Value	df	*P*
Perlita as susceptible parent						
P_1 (Perlita)	···	56	All S	···	···	···
P_2 (MR-1)	58	···	All R	···	···	···
F_1 ($P_1 \times P_2$)	57	···	All R	···	···	···
F_1 ($P_2 \times P_1$)	60	···	All R	···	···	···
F_2 ($P_1 \times P_2$)	190	72	3:1	0.860	1	0.37
F_2 ($P_2 \times P_1$)	146	60	3:1	1.870	1	0.18
F_2 Combined	336	132	3:1	2.564	1	0.11
F_2 Homogeneity	···	···	···	0.166	1	0.69
BC_{P1} ($P_1 \times P_2$)P_1	35	43	1:1	0.820	1	0.38
BC_{P1} ($P_2 \times P_1$)P_1	41	48	1:1	0.550	1	0.47
BC_{P1} Combined	76	91	1:1	1.347	1	0.21
BC_{P1} Homogeneity	···	···	···	0.023	1	0.88
BC_{P2} ($P_1 \times P_2$)P_2	73	···	All R	···	···	···
BC_{P2} ($P_2 \times P_1$)P_2	64	···	All R	···	···	···
PMR 6 as susceptible parent						
P_1 (PMR 6)	···	30	All S	···	···	···
P_2 (MR-1)	25	···	All R	···	···	···
F_1 ($P_2 \times P_1$)	29	···	All R	···	···	···
F_2 ($P_2 \times P_1$)	128	57	3:1	3.330	1	0.072
BC_{P1} ($P_2 \times P_1$)P_1	24	32	1:1	1.143	1	0.290
BC_{P2} P_2($P_2 \times P_1$)	42	···	All R	···	···	···
BC_{P2} ($P_2 \times P_1$)P_2	49	···	All R	···	···	···

[a] Plants were inoculated at the two-expanded-leaf stage with 5.0×10^3 conidia per milliliter and placed in a dew chamber at 20 C in the dark for 16 hr. Plants were removed from the dew chamber and placed in full sunlight for 8 hr, then returned to the dew chamber for 16 hr. Subsequently, plants were alternated between 8 hr of light in the glasshouse and 16 hr in the dew chamber. Plants were classified 10 days after inoculation as resistant (restricted, nonexpanding lesions on leaves) or susceptible (expanding lesions on leaves).

[b] R = resistant, S = susceptible.

susceptible and all plants of MR-1 were resistant (Table 1). The three F_1 families from the two crosses were homogeneous for resistance. No maternal effect was seen in the reactions of the F_1 families from reciprocal crosses of Perlita with MR-1.

The F_2 data from the Perlita families fit reasonably well to a 3 resistant:1 susceptible ratio expected if resistance is conditioned by a single dominant gene. In contrast, the F_2 data from the PMR 6 cross barely fit a 3:1 ratio.

The backcross families segregated as expected if a single dominant gene conditions resistance. The data from BC of F_1 individuals to Perlita and PMR 6 segregated reasonably well to a 1 resistant:1 susceptible ratio (Table 1; combined $\chi^2 = 2.372$, $P = 0.13$; homogeneity $\chi^2 = 0.142$, $P = 0.93$). The BC of F_1 individuals to MR-1 were homogeneous for resistance.

DISCUSSION

We propose that the single dominant gene that conditions resistance to *A. cucumerina* in muskmelon line MR-1 be designated *Ac*. Therefore, the genotype for resistance in MR-1 would be *Ac*/*Ac* and the genotype for susceptible cultivars Perlita and PMR 6 would be *ac*/*ac*.

This conclusion is consistent with the personal communication reported by Sitterly (10). Because their study was inconclusive, we cannot compare our results with those of Boyhan and Norton (2).

Our observations indicate that the full phenotypic expression of this single dominant gene for resistance can occasionally be masked by the presence of any condition, such as mechanical injury, necrotic tissue, or leaf senescence, that enhances infection by the pathogen. We noted that occasionally on plants that were expected to show a resistant reaction, one to three lesions did not remain restricted but expanded at a slower rate than those on susceptible plants. Plants with such lesions were rare (<5%) and the lesions were usually associated with the presence of necrotic tissue or mechanical damage to the leaf that existed before inoculation. For instance, the lesions would expand on that part of the leaf distal to a broken leaf vein or on tissue necrotic because of guttation salt injury at the leaf margin. Such injury or damage likewise resulted in a more severe reaction on susceptible plants. Those lesions that developed in association with some mechanical injury to the leaf or on preexisting necrotic tissue expanded faster than did those that developed on otherwise healthy tissue. We also noted that if a leaf had begun to senesce prematurely, as indicated by a general chlorosis at the time of inoculation, then the expansion of lesions was faster than usual. This situation was observed most often on leaf one of the PMR 6 cultivar. This phenomenon does not negate the value of the resistance, because even though lesions can expand when they develop on injured, necrotic, or senescent tissue of resistant plants, this expansion is still at a slower rate than on susceptible plants under similar conditions or circumstances.

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