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Powdery Mildew Resistance Genes in Muskmelon

James D. McCreight
U.S. Agricultural Research Station, Agricultural Research Service, U.S. Department of Agriculture, 1636 E. Alisal Street, Salinas, CA 93905

Michel Pitrat
Institut National de la Recherche Agronomique, Station d'Amélioration des Plantes Maraîchères, Domaine Saint-Maurice, B. P. 94, 84140 Montfavet, France

Claude E. Thomas
U.S. Vegetable Laboratory, Agricultural Research Service, U.S. Department of Agriculture, 2875 Savannah Highway, Charleston, SC 29407

Albert N. Kishaba
Boyden Entomology Laboratory, Agricultural Research Service, U.S. Department of Agriculture, University of California, Riverside, CA 92521

G. Weston Bohn[1]
Irrigated Desert Research Station, Agricultural Research Service, U.S. Department of Agriculture, 4151 Highway 86, Brawley, CA 92227

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Abstract. **Seven previously undescribed genes in muskmelon (*Cucumis melo* L.) for resistance to powdery mildew [*Sphaerotheca fuliginea* (Schlecht. ex Fr.) Poll., races 1 and 2] are reported. Progeny 92417 has a recessive gene for resistance to race 1, which is nonallelic to *Pm-1*. Breeding line WMR 29 has a gene for resistance to race 1 that is allelic to the recessive gene in 92417, but it is not known whether the two genes are identical alleles. Plant introduction 414723, 92417, and WMR 29 differentiated isolates of *S. fuliginea* race 2 at Montfavet, France, and Riverside, Calif. Comparative responses of F_1, F_2, and BC progenies from crosses involving 92417, PI 414723, and WMR 29 revealed six new genes for resistance to race 2. Genetic relationships among these seven genes are not fully known. Allelic and linkage relationships of these seven genes with the five previously known genes for powdery mildew resistance are also unknown.**

Powdery mildew is a limiting factor to muskmelon production throughout the world (23). *Erisyphe cichoracearum* DC ex Mecat and *Sphaerotheca fuliginea* have been the most frequently reported powdery mildew pathogens (1, 23). Based on three criteria (conidia borne in chains, fibrosin bodies in conidia, forked germ tubes) in the conidial stage, *S. fuliginea* is apparently more widespread than *E. cichoracearum* (1, 5–7). During 1964–67 and 1981–86, we have encountered only *S. fuliginea* in California, Arizona, Texas, and South Carolina in the United States, and in France.

The occurrence of races of powdery mildew confounds selection for resistance because races can change within and among seasons (9, 24). Three races of powdery mildew have been reported in the United States, and five genes for resistance to these races have been reported (4, 8, 22, 25). Reactions of the resistance genes to the three races along with the names of muskmelon lines carrying the genes are presented in Table 1.

Muskmelon breeding line WMR 29 (2) is resistant to papaya ringspot virus (PRV), synonomous to watermelon mosaic virus 1 (20). Resistance to PRV is from PI 180280. Observations made during the breeding of WMR 29 indicated that PI 180280 was resistant to *S. fuliginea* race 2. Breeding line PMR 29, the western shipping type muskmelon parent of WMR 29, was selected for resistance to *S. fuliginea* race 2. WMR 29 was known to be segregating for *S. fuliginea* resistance when it was released.

Muskmelon breeding lines AR 5, AR Hale's Best Jumbo, and AR Topmark (14) are resistant to the melon aphid (*Aphis gossypii* Glover). Aphid resistance was derived from progeny 90234, an aphid-resistant selection of PI 371795. Muskmelon progeny 92417 and PI 414723 were derived from 90234 after further selection for aphid resistance. Observations made during

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[1]Collaborator. Present address: 1094 Klish Way, Del Mar, CA 92014.

Table 1. Summary of reactions of the known genes in muskmelon for resistance to powdery mildew caused by *Sphaerotheca fuliginea*.[z]

Muskmelon resistance genes	Differential host	*S. fuliginea* race 1	2	3
0	Delicious 51	S	S	S
	Topmark	S	S	S
	Vedrantais	S	S	S
Pm-1	PMR 45	R	S	S
	PMR 450	R	S	S
Pm-1, *Pm-2*	PMR 6	R	R	S
	Perlita	R	R	S
Pm-3	PI 124111	R	R	R
Pm-4, *Pm-5*	PI 124112	R	R	R
	Seminole	R	R	R

[z]R = resistant; S = susceptible.

the breeding of the three aphid-resistant breeding lines suggested that 90234, 92417, and PI 414723 had a recessive gene for resistance to *S. fuliginea* race 2.

In 1984, McCreight (12) reported evidence of a recessive gene in 92417 for resistance to *S. fuliginea* race 1 at Salinas, Calif. Simultaneously, Pitrat (16) reported an unidentified dominant gene in PI 414723 for resistance to *S. fuliginea* race 2 at Montfavet, France. The objective of this research was to study the genetics of resistance to *S. fuliginea* in WMR 29, 92417, and PI 414723. In this work we report seven previously undescribed genes in muskmelon for resistance to *S. fuliginea* race 1 and race 2.

Materials and Methods

Crosses. The F_1, F_2, and BC generations from crosses of 92417 with 'Topmark', 'PMR 45', and WMR 29 were tested for resistance to race 1 at Salinas, Calif. They were tested for resistance to race 2 at Riverside, Calif.

The F_1, F_2, and BC generations from crosses of PI 414723 with 'Vedrantais' were tested for resistance to race 2 at Montfavet, France. The F_1 and F_2 generations from crosses of WMR 29 with 'Vedrantais' and PI 414723 were tested for resistance to race 2 at Montfavet. The parents were included in each test.

Experimental design. The different crosses that were tested at each location were not always tested simultaneously. The data from the differential hosts and parents were, however, consistent among the tests at each location. Thus, the data from each location are presented together.

The entries were randomly assigned to each replication. The number of replications and size of each replication varied among tests in accordance with the number of crosses included and the number of plants of each entry that were available.

Plant culture. Plant culture was different at each location. At Riverside and Salinas, the tests were done in greenhouses. The seeds were germinated in sand and watered with standard Hewitt nutrient solution modified as described by McCreight and Bohn (13). Five to 10 seeds were planted in 100-mm plastic pots and thinned to four to five seedlings per pot prior to inoculation. All of the tests at Riverside and some of the tests at Salinas were shaded with Saran cloth, which provided 40% shade.

At Montfavet, seeds were sown in a peatmoss–potting soil mixture in 85-mm pots, two seeds per pot, and thinned to one seedling per pot. Plants were grown in a plant growth chamber with a 12-hr photoperiod and day/night temperatures of 25°/18°C.

Inoculation procedures. The cultures used in each test were determined to be *S. fuliginea* based on presence of fibrosin bodies in the conidia (7) and forked germ tubes (5, 6). Each test included some or all of the 10 differential hosts (Table 1) in order to verify the racial identity of the *S. fuliginea* culture. The tests were artificially inoculated by dusting spores over the plants two or more times beginning at the cotyledon or first true leaf stages of growth.

The race 1 culture at Salinas was collected from a squash (*Cucurbita* spp.) seed production field near Saticoy, Calif. in Sept. 1981. The culture was maintained simultaneously on plants of 'Hale's Best Jumbo' and 'Topmark', grown as described previously.

The race 2 culture at Riverside originated from natural infection of 'PMR 45' in the greenhouse. The culture was maintained on 'PMR 45' grown as described above for more than 15 years. Progeny 90234 was resistant to powdery mildew race 2 at Riverside as well as at La Jolla, Irvine, Brawley, Texas, and South Carolina in the 1960 and early 1970 periods. Therefore, the present race 2 at Riverside is not identified with the race 2 that was there earlier.

The race 2 culture at Montfavet was collected in Spring 1984 from a muskmelon field near Montfavet. The culture was maintained on 'Vedrantais' in an isolation chamber with filtered air under positive pressure.

Powdery mildew evaluation. Plants were evaluated for resistance when the susceptible control plants ('Delicious 51', 'Hale's Best Jumbo', 'Topmark', 'Vedrantais') were covered with powdery mildew. The 1 to 5 scale of Markarian and Harwood (10) was used for evaluating resistance, where 1 corresponds to no growth and 5 shows heavy growth and sporulation on true leaves. The ratings were grouped into two categories for χ^2 (11) analysis. These were resistant (class 1) and susceptible (classes 2, 3, 4, and 5).

Results

Race 1. The differential powdery mildew hosts indicated race 1 at Salinas. 'PMR 45', 92417, and WMR 29 were resistant to race 1 (Table 2), whereas 'Topmark' was susceptible. The F_1 (92417 x Topmark) was susceptible (Table 2). Two F_2 families segregated in a 1 resistant : 3 susceptible ratio expected if 92417 carried a single recessive gene for resistance to race 1. Three BC (92417 x Topmark) 92417 families segregated in a 1 resistant : 1 susceptible ratio expected if 92417 carried one recessive gene for resistance to race 1.

The F_1 (92417 x PMR 45) was resistant to race 1 (Table 2). Two F_2 (92417 x PMR 45) families segregated in a 13 resistant : 3 susceptible ratio. In contrast, 3 BC (92417 x PMR 45) 92417 families segregated in a 3 resistant : 1 susceptible ratio. This ratio was expected if 92417 possessed one recessive gene and 'PMR 45' possessed one dominant gene (*Pm-1*) for resistance to race 1.

The F_1 and F_2 (92417 x WMR 29), and BC (92417 x WMR 29) 92417 were resistant to race 1, which was expected if 92417 was homozygous for a recessive gene that conditioned resistance, and WMR 29 was homozygous for a dominant or recessive gene that conditioned resistance at the same locus.

Race 2. The differential hosts indicated that the cultures at Montfavet and Riverside were race 2. 'PMR 45' was susceptible at Riverside (Table 3) and Montfavet (Table 4). The reactions of PI 414723 and WMR 29 to race 2 differed between the two

Table 2. Segregation for resistance to powdery mildew caused by *Sphaerotheca fuliginea* race 1 from crosses of muskmelon progeny 92417 with 'Topmark', 'PMR 45', and WMR 29 at Salinas, Calif.

Differential parent, or cross	Observed		Expected ratio	χ^2		
	Resistant	Susceptible		Value	df	*P*
92417	83	0	83:0			
WMR 29	168	0	168:0			
PMR 45	45	0	45:0			
Topmark	0	38	0:38			
92417 x Topmark						
F_1	0	98	0:98			
F_2	20	53	1:3	0.2237	1	0.65
	12	78	1:3	6.5333	1	0.011
F_2 combined	32	131	1:3	2.5051	1	0.12
F_2 homogeneity				3.363	1	0.07
BC(F_1 x 92417)	36	40	1:1	0.2105	1	0.66
	12	22	1:1	2.9412	1	0.09
	19	24	1:1	0.5814	1	0.46
BC Combined	67	86	1:1	2.3595	1	0.13
BC Homogeneity				1.3736	2	0.50
92417 x PMR 45						
F_1	60	0	60:0			
F_2	119	10	13:3	10.2423	1	0.002
	78	18	13:3	0	1	0.99
F_2 combined	197	28	13:3	5.8722	1	0.016
F_2 homogeneity				4.3700	1	0.040
BC (F_1 x 92417)	31	9	3:1	0.1333	1	0.72
	30	10	3:1	0	1	0.99
	27	14	3:1	1.8293	1	0.18
BC combined	88	33	3:1	0.3333	1	0.58
BC homogeneity				1.6293	2	0.45
92417 x WMR 29						
F_1	35	0	35:1			
F_2	173	0	173:0			
BC (F_1 x 92417)	45	0	45:0			

Table 3. Segregation for resistance to powdery mildew caused by *Sphaerotheca fuliginea* race 2 from crosses of muskmelon progeny 92417 with 'Topmark', 'PMR 45', and breeding line WMR 29 at Riverside, Calif. PI 414723, which is closely related to progeny 92417, was included for comparison.

Differential, parent, or cross	Observed		Expected ratio	χ^2		
	Resistant	Susceptible		Value	df	*P*
92417	0	67	0:67			
PI 414723	0	40	0:40			
WMR 29	29	111	1:3	1.4952	1	0.22
PMR 45	0	45	0:45			
92417 x Topmark						
F_1	0	35	0:35			
F_2	0	97	0:97			
BC (F_1 x 92417)	0	48	0:48			
92417 x PMR 45						
F_1	0	42	0:42			
F_2	0	69	0:69			
BC (F_1 x 92417)	0	43	0:43			
92417 x WMR 29						
F_1	0	33	0:33			
F_2	4	83	1:15	0.4054	1	0.53
BC (F_1 x 92417)	0	41	0:41			

cultures. PI 414723 was susceptible at Riverside (Table 3), but it was resistant at Montfavet (Table 4). WMR 29 was resistant at Montfavet (Table 4). WMR 29 segregated at Riverside, however, in a 1 resistant : 3 susceptible ratio (Table 3), which was expected if it was heterozygous for a single recessive gene that conditions resistance at Riverside race 2.

The F_1 and F_2 (92417 x Topmark), and the BC (92417 x Topmark) 92417 families were susceptible to Riverside race 2 (Table 3). The F_1 (92417 x PMR 45), F_2 (92417 x PMR 45) and BC (92417 x PMR 45) 92417 families also were susceptible to Riverside race 2 (Table 3). This susceptibility was expected if neither PI 414723 nor 'PMR 45' carried genes for resistance to Riverside race 2.

The F_1 (PI 414723 x Vedrantais) was susceptible to Mont-

Table 4. Segregation for resistance to powdery mildew caused by *Sphaerotheca fuliginea* race 2 in crosses among muskmelon PI 414723, WMR 29, and 'Vedrantais' at Montfavet, France.

Differential, parent, or cross	Observed		Expected ratio	Chi-square		
	Resistant	Susceptible		Value	df	*P*
PI 414723	40	0	40:0			
WMR 29	20	0	20:0			
Vedrantais	0	40	0:40			
PMR 45	0	20	0:20			
PI 414723 x Vedrantais						
F_1	0	20	0:20			
F_2	7	193	1:15	2.5813	1	0.11
BC (F_1 x PI 414723)	49	151	1:3	0.0267	1	0.88
WMR 29 x Vedrantais						
F_1	10	0	10:0			
F_2	136	64	3:1	5.2267	1	0.024
PI 414723 x WMR 29						
F_1	10	0	10:0			
F_2	46	154	1:3	0.4267	1	0.52
			19:45	4.285	1	0.042

favet race 2. The F_2 segregated 1 resistant : 15 susceptible, and the BC (PI 414723 x Vedrantais) PI 414723 segregated 1 resistant : 3 susceptible at Montfavet (Table 4). These results were expected if PI 414723 carried two recessive genes for resistance to Montfavet race 2.

The F_1 (WMR 29 x Vedrantais) was resistant to Montfavet race 2 (Table 4). The F_2 segregated in a 3 resistant : 1 susceptible ratio, which was expected if WMR 29 carried a single dominant gene for resistance to Montfavet race 2.

The F_1 (92417 x WMR 29) was susceptible to Riverside race 2 (Table 3). The F_2 segregated in 1 resistant : 15 susceptible ratio, but the BC (92417 x WMR 29) 92417 was susceptible (Table 3). These results were expected if the particular WMR 29 plant used in the cross was homozygous for two recessive genes for resistance to Riverside race 2, and if 92417 was homozygous dominant at these two loci for susceptibility to Riverside race 2. The recessive alleles in WMR 29 were epistatic to each other; only the double recessive was resistant.

The F_1 (PI 414723 x WMR 29) was resistant to Montfavet race 2 (Table 4). The F_2 segregation pattern did not, however, show a good fit to a 49 resistant : 15 susceptible ratio (Table 4). The ratio was, in fact, reversed. This ratio was expected if PI 414723 carried two recessive genes and WMR 29 carried one dominant gene at different loci for resistance to Montfavet race 2. The data fit a 15 : 49 ratio ($\chi^2 = 0.0213$), but one cannot reconcile this ratio with the individual reactions of PI 414723, WMR 29, and their F_1. The data fit a 19 : 45 ratio, which was expected if the three loci interacted in such a manner that a dominant gene at either locus in PI 414723 (recessive genes for resistance) conditioned susceptibility when the dominant gene for resistance in WMR 29 was heterozygous.

Discussion

Based on these results and those of Pitrat (16), we hypothesize seven previously undescribed genes for resistance to *S. fuliginea* race 1 and race 2. Progeny 92417 and WMR 29 have genes for resistance to race 1 at hypothetical locus A that is distinct from *Pm-1* (Table 5). It is not known whether the gene in WMR 29 is identical to the recessive gene in 92417, or whether it is a dominant allele.

There are six hypothetical loci (designated B, C, D, E, F, and G) for resistance to race 2 (Table 5). A dominant gene at

Table 5. Hypothetical genes for resistance to powdery mildew caused by *Sphaerotheca fuliginea* races 1 and 2 in muskmelon lines 92417, PI 414723, and WMR 29 (R = resistant).

Gene	Line	Race 1	Race 2	
			Montfavet, France	Riverside, Calif.
a	92417	R		
B	PI 414723		R	
c^z	PI 414723		R	
d^z	PI 414723		R	
E^y	WMR 29		R	
f^x	WMR 29			R
g^x	WMR 29			R

[z]Genes c and d are epistatic to each other; only the double recessive is resistant.

[y]Gene E is hypostatic to genes c and d.

[x]Genes f and g are epistatic to each other; only the double recessive is resistant.

locus B (Table 5) was reported by Pitrat (16) in the F_1, F_2, and BC families of PI 414723 x 'Vendrantais' inoculated with the Montfavet race 2 in 1983. This gene is distinct from *Pm-2* because it was expressed against the Montfavet race 2 isolate used in 1983, but not in 1984. 'PMR 6', which carried *Pm-1* and *Pm-2*, was resistant to both Montfavet race 2 isolates.

Recessive genes at loci C and D (Table 5) were expressed in the test of F_1, F_2, and BC families of PI 414723 x 'Vedrantais' against Montfavet race 2 in 1984. Loci C and D were epistatic to one another; a dominant allele at either locus conditioned susceptibility. These genes are carried by PI 414723. They were expressed against the Montfavet race 2 in 1984, but not to Riverside race 2.

A dominant gene at locus E (Table 5) was expressed in 1985 at Montfavet (same culture as in 1984) in a cross of WMR 29 x 'Vedrantais'. This gene is distinct from *Pm-2*, *Pm-3*, *Pm-4*, and *Pm-5* because it was expressed against Montfavet race 2, but not against Riverside race 2. Differentials carrying *Pm-2*, *Pm-3*, *Pm-4*, or *Pm-5* were resistant to these race 2 cultures. The heterozygote at this locus was hypostatic to the recessive genes at loci C and D.

Recessive genes at loci F and G (Table 5) expressed at Riverside were epistatic to one another. A dominant allele at either

locus conditioned susceptibility. The F_1 (92417 x WMR 29) and BC (92417 x WMR 29) 92417 were susceptible; only the double recessive was resistant in the F_2. WMR 29 segregated at locus F and was homozygous at locus G. These genes are carried by WMR 29. They are distinct from the recessive genes carried by PI 414723 at loci C and D because they were not expressed against Montfavet race 2.

The genetic relationships among these seven genes are not fully understood. The allelic relationships of these seven genes with the five known powdery mildew resistance genes are unknown. Additional data are needed before names and symbols can be assigned.

Race-specific (vertical) resistance occurs where there are differential interactions between races and host lines or cultivars (21) and results from concomitant genetic variation for pathogenicity in the pathogen and for resistance in the host. A number of pathogens are known to carry many genes for pathogenicity and recombine them in new combinations to overcome resistance genes in new host cultivars (26). The results presented herein indicate new virulence factors in *S. fuliginea* and corresponding resistance genes in muskmelon. The differential powdery mildew hosts gave identical response to the cultures at Montfavet and Riverside. The differential reactions of 92417, PI 414723, and WMR 29 to these two cultures revealed, however, that race 2 is highly heterogeneous.

These results have implications for the development of powdery mildew-resistant muskmelon varieties. Breeders now have additional genes for powdery mildew resistance to meet the occurrence of these new virulence factors in muskmelon production. The *Pm-1*, *Pm-2* combination in 'PMR 5' and 'PMR 6' is still effective for preventing powdery mildew caused by race 2, largely because cultivars with the *Pm-1*, *Pm-2* combination have not been widely grown on a commercial scale. 'Topmark' and 'PMR 45', the two major cultivars in California, are not resistant to race 2. Indeed, 'Topmark' does not have any known genes for powdery mildew resistance.

These results also provide additional evidence of the adverse effects of unnecessary virulence factors on the fitness of a pathogen. Investigators in Israel have observed that race 1 causes more damage to muskmelon production than does race 2 (Z. Karchi, personal communication). These data and the observations in Israel suggest, in the absence of controlled studies, that the newly revealed virulence factors in race 2 have made it less fit than race 1 on muskmelon (i.e., race 2 causes less damage than race 1).

WMR 29 has received interest from commercial breeders because it has the gene *Prv* (3) for resistance to PRV, the vines hold up well in a variety of growing conditions, and the fruits are very firm. PI 414723 is also of interest to breeders, pathologists, and entomologists because it has genes conditioning resistance to several important pathogens (15, 17–19). Should breeders attempt to pyramid genes for powdery mildew resistance, race 2 theoretically could become more damaging to muskmelon production because breeders may inadvertently introduce susceptibility to race 2 during the transfer of a disease-resistant trait from WMR 29, 92417, or PI 414723.

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