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Genetic analysis of resistance of five melon lines to powdery mildews

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Summary

Inheritance of resistance of five melon lines to two strains of *Sphaerotheca fuliginea* belonging to races 1 (Sf1) and 2 (Sf2) and to one strain of *Erysiphe cichoracearum* (Ec) have been studied. 'PMR 45' possesses one dominant gene controlling only Sf1. 'WMR 29' has one dominant gene for resistance to Sf1 and another for Sf2 and these genes seem to be linked. In line 'PMR 5', one dominant gene (or a group of three closely linked genes) is involved in the control of the three strains with one complementary gene for Sf1 and another one for Ec. 'PI 124112' has one dominant gene or two closely linked loci controlling Sf1 and Sf2 and two complementary different genes controlling Ec. 'Nantais Oblong' has one dominant gene controlling only Ec. A nomenclature of the genes described is proposed.

Introduction

Powdery mildew is an important limiting factor of melon (*Cucumis melo* L.) production in all producing countries and conditions (Sitterly, 1978). The disease can be provoked by two fungal species, *Sphaerotheca fuliginea* (Schlecht ex Fr.) Poll. and *Erysiphe cichoracearum* DC ex Merat, that produce identical symptoms. These fungi can be distinguished by examination under the microscope of the conidia (fibrosin bodies (Zopf, 1887 in Ballantyne, 1975) and germ tubes) or the perithecia (number of asci in the cleistothecia) (Hirata, 1942). Based on a survey made in 1987–1988 in France, *S. fuliginea* appears more widespread during the summer in the open-air than *E. cichoracearum* which is generally found earlier in the growing season in greenhouses (Bertrand, 1991).

Melon resistance to powdery mildew has been studied for a long time (Jagger & Scott, 1937) and many sources of resistance, usually from India, have been described. But literature on the genetics of the resistance remains somewhat confusing: (i) The identification of the fungus has not always been made very accurately and in some cases the two species have even been considered as synonymous (McCreight et al., 1987). For instance the cause of powdery mildew in California was considered to be *E. cichoracearum* by Paulus et al. (1967) and *S. fuliginea* by Paulus et al. (1968 and 1969). (ii) In trials, populations of powdery mildew have been used rather than pure monospore cultures. (iii) The action of the described genes on different strains of powdery mildew has not been studied and so the spectrum of action of the genes is not known. (iv) No allelism tests have been made between the different genes. Nevertheless resistant cultivars have been selected in different melon types.

Two races of *S. fuliginea* (races 1 and 2) and one race of *E. cichoracearum* have been observed in

France on melon. The recent development of efficient *in vitro* methods for isolation, cloning, preservation and multiplication of inoculum (Bertrand, 1991) have permitted the screening of melon germplasm for mildew resistance. Five genotypes representative of different types of resistance observed in the collection have been used in this study: 'PMR 45', 'WMR 29'. 'PMR 5', 'PI 124112' and 'Nantais oblong'. 'PMR 45' is resistant to *S. fuliginea* race 1 only, 'WMR 29' is resistant to the two races of *S. fuliginea*, 'PMR 5' and 'PI 124112' are resistant towards the three strains and 'Nantais Oblong' is resistant to *E. cichoracearum* only. Their resistances originate from India except for 'Nantais oblong' issued from an old French population of Charentais type. Previous studies have described the genetics of the resistance of these lines (except for cultivar 'Nantais oblong') towards *E. cichoracearum* or *S. fuliginea* (e.g. Bohn & Whitaker, 1964; Harwood & Markarian, 1968a, b; McCreight et al., 1987). Until now, no genetic study of resistance to powdery mildew has included the two species together. So, the spectrum of action of the resistance genes in a melon line has not been studied. Allelism tests between the five studied varieties have never been made either. The objective of this paper is to study the genetics of resistance of these lines towards two *S. fuliginea* and one *E. cichoracearum* isolates. The relations between genes that control different strains in a plant or genes coming from different cultivars will be examined.

Material and methods

Crosses

The F_1, F_2 and backcross generations (BCs= $F_1\times$ Susceptible parent and BCr= $F_1\times$ Resistant parent) from the crosses between each resistant line and the susceptible control 'Védrantais' were obtained. Two hundred plants of the F_2 progeny, 100 plants of each backcross and 30 plants of parental and F_1 generations were tested with *S. fuliginea* races 1 and 2 and *E. cichoracearum* race 1. For allelism tests, 200 plants from the testcrosses (Resistant line #1× Resistant line #2)× 'Védrantais' were tested; crosses with 'Nantais Oblong' for allelism test are lacking. Some F_3 families were obtained by selfing particular F_2 plants from (Resistant line× Védrantais) crosses presenting a recombinating pattern (i.e. plants resistant to one strain and susceptible to another). Twenty plants per F_3 family were tested.

Powdery mildew cultures

Three monoconidial isolates of mildew from melon were used: S88–133 is a clone of *S. fuliginea* race 1 isolated in Tunisia (Sf1). S87–7 a clone of *S. fuliginea* race 2 isolated in south eastern France (Sf2) and E87-3 a clone of *E. cichoracearum* isolated in south western France (Ec). The genus (*Sphaerotheca* or *Erysiphe*) was determined on conidia and perithecia examination. *Sphaerotheca* species produced conidia that contained fibrosin bodies and germinated laterally and cleistothecia that contained a single ascus with 8 ascospores while *Erysiphe* produced conidia that contained no fibrosin bodies and germinated apically. No cleistothecia were observed when *Erysiphe* isolates were crossed with testers belonging to *S. fuliginea* or *E. cichoracearum*. The distinction between race 1 and race 2 of *S. fuliginea* was based on the reaction of the melon cultivar 'PMR 45' which is resistant to race 1 and susceptible to race 2. Inoculum was produced in axenic conditions on 'Védrantais' cotyledons in Petri dishes according to the method developed by Bertrand (1991).

Plant culture and inoculation procedure

All plants were grown in 8.5 cm pots in the greenhouse. At the 2–3 leaf stage, disks (20 mm diameter) were cut from the two last expanded leaves and placed face upwards on moistened filter paper in clear polystyrene boxes. Each box was placed under a settling tower composed of a plastic tube (1 m high and 25 cm of diameter) and a cover with a centered hole of 5 cm of diameter. Inoculation was performed by blowing (with a Pasteur pipet), through the hole, the conidia present on the surface of one infected cotyledon, obtained in axenic conditions.

The inoculum, about 100–500 conidia per cm^2, was uniformly scattered over the leaf disks. After two minutes of sedimentation, the box was removed from the tower. Twenty ml of a solution of benzimidazole ($30 g \cdot ml^{-1}$) and mannitol ($10 g \cdot l^{-1}$) was added in each box to preserve disks from senescence and osmotic pressure stress (Bertrand, 1991). Boxes were incubated in growth chambers at 24° C for 12 hours of light and 18° C for 12 hours of dark.

Disease rating

After ten days, sporulation was recorded on a scale of 0 (= no sporulation) to 9 (= entire disk covered with heavy sporulation). The score attributed to a plant is the maxima of the ratings recorded on the two leaves. The plants were grouped in three categories for χ^2 analysis, based on disease ratings: Resistant (scores 0 and 1), Intermediate (scores 3 and 5) and Susceptible (scores 7 and 9).

Results

1. Inheritance of resistance to the three strains of mildew

– *Race 1 of* S. fuliginea *(Sf1)* (Table 1)
The data produced evidence for a dominant monogenic control in 'PMR 45', 'WMR 29' and 'PI 124112' as all the plants in the F_1 and BCr were re-

Table 1. Segregation for resistance to powdery mildew caused by *Sphaerotheca fuliginea* race 1 from (Resistant× Védrantais) crosses

Differential parent or cross	Observed number			Tested segregation ratio	χ^2	
	Res[a]	Int[a]	Sus[a]		Value	Proba
PMR 45	30	:	0			
WMR 29	30	:	0			
PMR 5	30	:	0			
PI 124112	30	:	0			
Védrantais	0	:	30			
PMR 45× Védrantais						
F_1	30	:	0			
F_2	145	:	55	3:1	0.67	41%
BCs	61	:	39	1:1	4.84	5%
BCr	100	:	0			
WMR 29× Védrantais						
F_1	30	:	0			
F_2	156	:	39	3:1	2.60	11%
BCs	55	:	45	1:1	1.00	32%
BCr	100	:	0			
PMR 5× Védrantais						
F_1	30 :	0 :	0			
F_2	109 :	71 :	20	9:6:1	4.82	9%
BCs	32 :	43 :	25	1:2:1	2.94	23%
BCr	100 :	0 :	0			
PI 124112× Védrantais						
F_1	30	:	0			
F_2	140	:	60	3:1	2.67	10%
BCs	58	:	42	1:1	2.56	11%
BCr	100	:	0			

[a]Res= Resistant, Int= Intermediate, Sus= Susceptible.

sistant and the observed segregations fitted well with a 3:1 resistant to susceptible ratio in the F_2 and a 1:1 ratio in the BCs. The disjunctions observed in 'PMR 5' correspond to a dominant digenic control with all the F_1 plants resistant, ratios of 9 resistant: 6 intermediate: 1 susceptible in F_2, 1:2:1 in the BCs and all the plants resistant in the BCr.

– *Race 2 of* S. fuliginea *(Sf2)* (Table 2)
From the F_1 results, we can conclude that resistance to this strain is highly dominant in 'WMR 29', 'PMR 5' and 'PI 124112'.

Segregations of 3:1 resistant to susceptible in F_2 and 1:1 in BCs support a monogenic dominant control in the three lines.

– E. cichoracearum *(Ec)* (Table 3)
Considering the F_1 ratings, 'PMR 5' and 'PI 124112' possess partially dominant resistance. The three resistance classes were represented among plants of the F_2 and BCs generations and the fit to a digenic partially dominant segregation was satisfactory for the two lines. The high proportions of susceptible and intermediate plants in the BCs can be attributed to partial dominance and caused high χ^2 values.

As opposed to these lines, 'Nantais Oblong' presents a monogenic dominant resistance toward *E. cichoracearum* (ratios of 3:1 resistant to susceptible in F_2 and 1:1 in BCs).

2. Relations between genes controlling resistance to the three strains in a single line

Two-way contingency tables of all segregation types present in (Resistant parent× 'Védrantais') progenies are presented by pairs of strains (F_2 progenies in Table 4 and BCs progenies in Table 5).

– *'WMR 29'× 'Védrantais' cross*
The respective distributions for Sf1 and Sf2 are not independent. Plants showing resistance to one race and susceptibility to the other race are in too low numbers (Tables 4.a and 5.a). Genetic distances can

Table 2. Segregation for resistance to powdery mildew caused by *Sphaerotheca fuliginea* race 2 from (Resistant× Védrantais) crosses

Differential parent or cross	Observed number			Tested segregation ratio	χ^2	
	Res[a]	:	Sus[a]		Value	Proba
WMR 29	30	:	0			
PMR 5	30	:	0			
PI 124112	30	:	0			
Védrantais	0	:	30			
WMR 29× Védrantais						
F_1	30	:	0			
F_2	146	:	39	3:1	1.52	22%
BCs	48	:	52	1:1	0.16	69%
BCr	100	:	0			
PMR 5× Védrantais						
F_1	30	:	0			
F_2	149	:	51	3:1	0.03	87%
BCs	54	:	46	1:1	0.64	42%
BCr	100	:	0			
PI 124112× Védrantais						
F_1	30	:	0			
F_2	155	:	45	3:1	0.67	41%
BCs	54	:	46	1:1	0.64	42%
BCr	100	:	0			

[a] Res= Resistant, Sus= Susceptible.

be estimated at 22± 3cM (value± standard error) from F_2 progeny and 25± 4cM from BCs progeny according to the maximum likelihood method (Allard, 1956). F_3 families were grown from F_2 plants showing either resistance to Sf1 and susceptibility to Sf2 or the reverse. Among them, we have observed F_3 families with resistance to *S. fuliginea* race 1 and susceptibility to race 2 but no families with susceptibility to race 1 and resistance to race 2.

– *'PMR 5'× 'Védrantais' cross*

No distributions between two strains show independence under the genetic assumptions (all χ^2 probabilities are inferior to 0.01%). Very few plants showing resistance to one race and susceptibility to the other race have been observed (Tables 4.b and 5.b). F_3 families were obtained from these few F_2 plants. Among all F_3 families tested, we found no families with resistance to *S. fuliginea* race 2 and susceptibility to *S. fuliginea* race 1 or to *E. cichoracearum*. But F_3 families resistant to Sf1 and susceptible to Sf2 or Ec have been observed.

– *'PI 124112'× 'Védrantais' cross*

Plants resistant to one strain and susceptible to another are in too low numbers (Tables 4.c and 5.c) in the two generations. The distribution of F_3 families issued from these particular F_2 plants shows that there are no family with resistance to one race of *S. fuliginea* and susceptibility to the other of *S. fuliginea*. But we observe F_3 families with resistance (resp. susceptibility) to one of the *S. fuliginea* races and susceptibility (resp. resistance) to *E. cichoracearum*.

3. Allelism relationships between genes of different resistant lines (Table 6)

The existence of susceptible plants in significant proportion in the testcross progenies indicates that different loci are concerned. Only in few crosses (between 'PI 124112' and 'PMR 5' for Sf1 and Sf2 and between 'PMR 45' and 'WMR 29' for Sf1) no susceptible plants have been observed. This may be

Table 3. Segregation for resistance to powdery mildew caused by *Erysiphe cichoracearum* from (Resistant× Védrantais) crosses

Differential parent or cross	Observed number			Tested expected ratio	χ^2	
	Res[a]	Int[a]	Sus[a]		Value	Proba
Nantais Oblong	30	:	0			
PMR 5	30	:	0			
PI 124112	30	:	0			
Védrantais	0	:	30			
Nantais Oblong× Védrantais						
F_1	30	:	0			
F_2	150	:	49	3:1	0.02	90%
BCs	48	:	52	1:1	0.16	69%
BCr	100	:	0			
PMR 5× Védrantais						
F_1	16 :	14 :	0			
F_2	105 :	80 :	15	9:6:1	1.33	51%
BCs	20 :	32 :	48	1:2:1	28.60	<0.01%
BCr	100 :	0 :	0			
PI 124112× Védrantais						
F_1	22 :	4 :	0			
F_2	115 :	75 :	10	9:6:1	0.55	76%
BCs	26 :	60 :	14	1:2:1	6.88	3%
BCr	100 :	0 :	0			

[a] Res= Resistant, Int= Intermediate, Sus= Susceptible.

explained by a common locus for the corresponding resistance genes.

In the cross between 'PI 124112' and 'WMR 29' there are less susceptible plants than expected after inoculation with Sf1 and Sf2. This may indicate a linkage between the resistance genes.

Discussion

Resistance of 'PMR 45' to *S. fuliginea* race 1 is governed by one dominant gene that we call *Pm-A*. 'PMR 45' was first described as resistant to *E. cichoracearum* by one dominant gene (Jagger et al., 1938). 'PMR 45' has always been found susceptible to strains of *E. cichoracearum* isolated in France (Bertrand, 1991). A first explanation of the differ-

Table 4. Contingency tables of (Resistant× Védrantais) crosses representing the types of response of plants for each pair of races. Plain figures represent the observed data of F_2 population and figures in brackets the theoretical distribution for a total number of plants of 200. The χ^2 values of independency (with corresponding degrees of freedom, df, in brackets) are given for each table

4.a. WMR 29× Védrantais

		Sf1[a]	
		R[b]	S[b]
Sf2[a]	R	131 (112.5)	12 (37.5)
	S	25 (37.5)	27 (12.5)
χ^2 (df)		42 (3)	

4.b. PMR 5× Védrantais

		Sf1					Sf1					Sf2	
		R	I	S			R	I	S			R	S
Sf2	R	104 (84.5)	20 (56)	0 (9.5)	Ec[a]	R	88 (63)	17 (42)	0 (7)	Ec	R	100 (84.5)	5 (28)
	I					I	21 (42)	47 (28)	12 (5)		I	24 (56)	56 (19)
	S	5 (28)	51 (19)	20 (3)		S	0 (7)	7 (5)	8 (1)		S	0 (9.5)	15 (3)
χ^2 (df)		174 (5)					141 (8)					169 (5)	

4.c. PI 124112× Védrantais

		Sf1				Sf1				Sf2	
		R	S			R	S			R	S
Sf2	R	136 (112.5)	19 (37.5)	Ec	R	105 (84.5)	13 (28)	Ec	R	107 (84.5)	8 (28)
	I				I	35 (56)	40 (19)		I	45 (56)	30 (19)
	S	4 (37.5)	41 (12.5)		S	3 (9.5)	7 (3)		S	3 (9.5)	7 (3)
χ^2 (df)		109 (3)				53 (5)				39 (5)	

[a] Sf1 = *S. fuliginea* race 1, Sf2 = *S. fuliginea* race 2, Ec = *E. cichoracearum*

[b] R = Resistant, I = Intermediate and S = Susceptible

All χ^2 probabilities are significant at 0.01%.

ences may be that American strains of *E. cichoracearum* do not belong to the same pathotype as the French ones. A second hypothesis would be that the fungus responsible for powdery mildew was misidentified as *E. cichoracearum* and was in fact *S. fuliginea*. In this case the gene symbolized *Pm-1* by Jagger et al. (1938) would be identical to *Pm-A*.

Resistance of line 'WMR 29' to *S. fuliginea* race 1 is a monogenic dominant character and appears to be closely linked or identical to *Pm-A* in 'PMR 45' (Table 6). We can hypothesize that the *Pm-A* gene is controlling *S. fuliginea* race 1 in 'WMR 29'. Resistance to *S. fuliginea* race 2 is also controlled by one dominant gene (*Pm-B*). So, resistance to *S. fuliginea* race 2 appears to be controlled by one dominant gene with no action on race 1 but linked with *Pm-A* locus (distance 22± 3 cM estimated on the F_2 progeny and 25± 4 cM on the BCs progeny).

Table 5. Contingency tables of (Resistant× Védrantais) crosses representing the types of response of plants for each pair of races. Plain figures represent the observed data of BCs population and figures in brackets the theoretical distributions for a total number of plants of 100. The χ^2 values of independency (with corresponding degrees of freedom, df. in brackets) are given for each table

5.a. WMR 29× Védrantais

		Sf1[a]	
		R[b]	S[b]
Sf2[a]	R	39 (25)	9 (25)
	S	16 (25)	36 (25)
χ^2 (df)		26 (3)	

5.b. PMR 5× Védrantais

		Sf1					Sf1					Sf2	
		R	I	S			R	I	S			R	S
Sf2	R	32 (12.5)	18 (25)	4 (12.5)	Ec[a]	R	13 (6.25)	6 (12.5)	1 (6.25)	Ec	R	20 (12.5)	0 (12.5)
	I					I	17 (12.5)	12 (25)	3 (12.5)		I	30 (25)	2 (25)
	S	0 (12.5)	25 (25)	21 (12.5)		S	2 (6.25)	25 (12.5)	21 (6.25)		S	4 (12.5)	44 (12.5)
χ^2 (df)		56 (5)					81 (8)					124 (5)	

5.c. PI 124112× Védrantais

		Sf1				Sf1				Sf2	
		R	S			R	S			R	S
Sf2	R	54 (25)	0 (25)	Ec	R	24 (6.25)	2 (12.5)	Ec	R	22 (12.5)	4 (12.5)
	I				I	28 (25)	32 (25)		I	28 (25)	32 (25)
	S	4 (25)	42 (25)		S	6 (12.5)	8 (12.5)		S	4 (12.5)	10 (12.5)
χ^2 (df)		88 (3)				27 (5)				22 (5)	

[a] Sf1 = *S. fuliginea* race 1, Sf2 = *S. fuliginea* race 2, Ec = *E. cichoracearum*

[b] R = Resistant, I = Intermediate and S = Susceptible

All χ^2 probabilities are significant at 0.01%.

This result is not in total agreement with the behaviour of F_3 families as we have not found families with susceptibility to race 1 and resistance to race 2. May be not enough F_3 families have been observed? This must be verified.

Resistance of 'PMR 5' to *S. fuliginea* race 1 is conferred by the simultaneous presence of two dominant and independent genes, resistance to *S. fuliginea* race 2 by one dominant gene and resistance to *E. cichoracearum* by two partially dominant genes. The very low number of plants resistant to one strain and susceptible to the other and the behaviour of the F_3 families observed support the hypothesis that at least one gene (or a group of three closely linked genes) has an effect on the three strains. We propose that the gene controlling Sf2 in 'PMR 5' (*Pm-C*) is involved in the control of Sf1 (with *Pm-D*) and Ec (with *Pm-E*).

Bohn & Whitaker (1964) and Harwood & Markarian (1968b) stated that resistance of 'PMR 5' to *E. cichoracearum* race 2 was controlled by one dominant gene (symbol *Pm-2*) with modifiers or interaction with *Pm-1*. As in the case of 'PMR 45' it is possible that *E. cichoracearum* was misidentified and that in fact *S. fuliginea* race 2 was used by both authors. If this hypothesis is right, the locus involved in the common control of *S. fuliginea* races 1 and 2 and *E. cichoracearum* may be *Pm-2* (*Pm-C*).

Allelism tests with *S. fuliginea* race 1 show that 'PMR 5' and 'PMR 45' (or 'WMR 29') resistances are controlled by distinct loci. So we can assume that 'PMR 5' does not include *Pm-A* in its genome. As compared to the literature, this appears surprising and must be verified by allelism tests between 'PMR 45' and F_3 families (from F_2 plants 'Védrantais' × 'PMR 5') with resistance to *S. fuliginea* race 1 and susceptibility to *S. fuliginea* race 2 and to *E. cichoracearum*.

Inheritance of resistance of 'PI 124112' to *S. fuliginea* races 1 and 2 was studied by Kenigsbuch & Cohen (1992). They found that two genes, the dominant gene *Pm-5* and the partially dominant gene *Pm-4* confer resistance to races 1 and 2 respectively.

We found that resistance of 'PI 125112' to *S. fuliginea* race 1 is controlled by one dominant gene and to race 2 also by one dominant gene. Very few plants susceptible to race 1 and resistant to race 2 or the opposite have been observed. No F_3 families (issued from the F_2 'PI 124112' × 'Védrantais' cross) show resistance to one race of *S. fuliginea* and susceptibility to the other. So we conclude that one major gene or

Table 6. Allelism tests between resistant lines for the three strains. Number of susceptible versus resistant plants in each cross are given and deviation to the independence hypothesis (expected ratio) is calculated (χ^2 and associated probability)

	Sf1			Sf2			Ec		
	Number observed	Ratio expected	χ^2 (P)	Number observed	Ratio expected	χ^2 (P)	Number observed	Ratio expected	χ^2 (P)
(PMR 45× WMR 29)× Védrantais	0:200	1:3	–						
(PMR 45× PMR 5)× Védrantais	15:185	1:7	4.57 (3.3%)						
(PI 124112× PMR 45)× Védrantais	52:148	1:3	0.11 (74%)						
(PMR 5× WMR 29)× Védrantais	34:166	1:7	3.70 (5.4%)	60:140	1:3	2.67 (10%)			
(PI 124112× WMR 29)× Védrantais	36:164	1:3	5.22 (2.2%)	27:173	1:3	14.1 (0.02%)			
(PI 124112× PMR 5)× Védrantais	0:200	1:7	–	0:200	1:3	–	19:181	1:7	1.65 (20%)

two closely linked genes may control resistance to both *S. fuliginea* races 1 and 2.

Resistance to *E. cichoracearum* is controlled by two partially dominant genes. There are some plants resistant to *S. fuliginea* and susceptible to *E. cichoracearum* and the opposite. The independence of the genetic controls towards *S. fuliginea* and *E. cichoracearum* in 'PI 124112' is supported by two facts:

- the presence of F_3 families with resistance (resp. susceptibility) to *S. fuliginea* races and susceptibility (resp. resistance) to *E. cichoracearum*,
- 'Edisto 47' (which has 'PI 124112' as a genitor) is resistant to *S. fuliginea* races 1 and 2 and susceptible to *E. cichoracearum* (Bertrand & Pitrat, 1989).

Moreover, the low number of plants resistant to *S. fuliginea* and susceptible to *E. cichoracearum* and the opposite (Table 4.c) indicates a linkage between the two genetic controls. Genetic distances from F_2 values are 14± 4cM between genes controlling resistance to *S. fuliginea* race 1 and *E. cichoracearum* and 12.8± 3.5cM between *S. fuliginea* race 2 and *E. cichoracearum* and from BCs are 20± 5.6cM for both *S. fuliginea* race 1 or race 2 and *E. cichoracearum*. The estimated values of the distances between the genes involved in the control of *S. fuliginea* races and *E. cichoracearum* are very similar and this confirms the hypothesis of a common control for *S. fuliginea* race 1 and race 2.

The resistance gene of 'PI 124112' to *S. fuliginea* race 1 is different from *Pm-A* in 'PMR 45' and 'WMR 29'. It is also different from the gene which controls *S. fuliginea* race 2 resistance in 'WMR 29' but the two genes appear to be linked (27cM) as there are less susceptible plants than expected. On the opposite, there are no susceptible plants in the testcross of 'PI 124112' by 'PMR 5' with *S. fuliginea* races 1 and 2. One common locus in the two lines seems to be involved in the control of each race. Conversely the genetic control of *E. cichoracearum* resistance seems to be different in 'PI 124112' and 'PMR 5'. An hypothesis could be that 'PMR 5' has the allele *Pm-C* (= *Pm-2*?) which is involved in the control of the three strains while 'PI 124112' possesses the allele *Pm-C*^{2} which controls only *S. fuliginea* races 1 and 2 but not *E. cichoracearum*. The two genes of 'PI 124112' controlling *E. cichoracearum* are called *Pm-F* and *Pm-G*.

'Nantais Oblong' whose origin is different from the other lines presents a monogenic dominant resistance to *E. cichoracearum* but a complete susceptibility to *S. fuliginea*. The allelism relationships with the loci previously described have not been studied. The loci involved are probably different since the genetic control of 'PMR 5' or 'PI 124112' resistance to *E. cichoracearum* is digenic and that of 'Nantais Oblong' monogenic. We proposed the name *Pm-H* for this locus.

The loci described are summed up in Table 7. We assume that a common locus controls the three races in 'PMR 5' and both *S. fuliginea* in 'PI 124112' (locus named *Pm-C*). However, the hypothesis of two or three loci closely linked is not rejected. Indeed more F_3 families need to be observed.

From the results of the testcross ('PI 124112'× 'WMR 29')× 'Védrantais', linkage between *Pm-B* and *Pm-C* is probable (27cM but the standard error is not available). But the segregation of the testcross ('PMR 5'× 'WMR 29')× 'Védrantais' implies that *Pm-B* and *Pm-C* are independent. These opposite results can be attributed either to the lack of

Table 7. Hypothesis for the genetic control of resistance to powdery mildew in five melon lines

Line	Sf1	Sf2	Ec
PMR 45	*Pm-A*		
WMR 29	*Pm-A*	*Pm-B*	
PMR 5	*Pm-D* and *Pm-C*^{1}	*Pm-C*^{1}	*Pm-C*^{1} and *Pm-E*
PI 124112	*Pm-C*^{2}	*Pm-C*^{2}	*Pm-F* and *Pm-G*
Nantais Oblong			*Pm-H*

with *Pm-A* = *Pm-1*?, *Pm-C* in 'PMR 5' = *Pm-2*?.

precision of the allelism test (possible infection escapes or size of the sample not sufficient) either to the absence of standard error for the distance estimation.

These results point out the fact that resistance genes towards powdery mildew have different ranges of action. Some genes appear to be specific of the pathogen species: for instance *Pm-A* in 'PMR 45' and 'WMR 29' or *Pm-B* in 'WMR 29' control resistance to one race of *S. fuliginea* and the allele in 'Nantais Oblong' controls only *E. cichoracearum*. On the opposite allele *Pm-C*^{1} in 'PMR-5' controls partial or complete resistance to *S. fuliginea* races 1 and 2 and to *E. cichoracearum*.

Another point is to be emphasized: we have not yet isolated F_3 lines from 'WMR 29', 'PMR 5' or 'PI 124112' which exhibit resistance to *S. fuliginea* race 2 and susceptibility to race 1. Similarly we have not observed on a collection of more than 400 melon lines such a behaviour (Bertrand & Pitrat, 1989). In 'PI 124111', resistance to *S. fuliginea* race 1 is under control of the gene *Pm-3* (Harwood & Markarian, 1968b) and resistance to *S. fuliginea* race 2 under control of the independent gene *Pm-6* (Kenigsbuch & Cohen, 1989). In this case it would be possible to obtain F_3 families with susceptibility to race 1 and resistance to race 2, but this has not yet been published.

The contradictory results obtained for example by Kenigsbuch & Cohen (1992) concerning variety 'PI 124112' are certainly due to the pathotype used in both analysis. Race 2 is defined by its ability to overcome *Pm-1* (= *Pm-A*?) gene in 'PMR 45' melon line. But strains able to grow and multiply on 'PMR 45' may belong to different pathotypes which will be shown by lines with other genes of resistance. Anyway, to have a more complete and precise table of the resistance genes and powdery mildew races, lines with only one resistance gene must be used i.e. F_3–F_4 lines from crosses between a resistant and a susceptible line instead of varieties 'PMR 5', 'PI 124111' or 'PI 124112' which apparently possess several genes.

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