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ARTICLE

Mapping of resistance genes to races 1, 3 and 5 of *Podosphaera xanthii* in melon PI 414723

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Abstract – *The fungus* Podosphaera xanthii *affects melon crops and presents several races controlled by race-specific resistance genes. The accession PI 414723 is resistant to races 1, 3 and 5 and it is a suitable source of resistance genes. The inheritance of resistance to these races was analyzed on 87 F_2 plants from the cross of PI 414723 × Védrantais, and resistance to all three races could be explained by the segregation of a single dominant gene, although a digenic model could also be accepted. A genetic map was assembled with 206 markers, and co-segregation analysis of resistance phenotypes indicated the existence of two linked loci in linkage group II, one conferring resistance to races 1 and 5 (denominated Pm-x1,5), and the second to race 3 (denominated Pm-x3), located 5.1 cM apart. This study reports for the first time the existence of Pm-x3 and the genetic locations of these resistance genes from PI 414723.*

Key words: *Disease resistance, molecular marker, genetic mapping, powdery mildew.*

INTRODUCTION

Melon (*Cucumis melo* L., 2n=2x=24) is an economically important export crop in Brazil. Between 2001 and 2008, melon production in the Northeast region accounted for 95% of the national total production, and in 2009, it resulted in the export of 184 thousand tons of fresh fruits, generating an income of U$ 122 million (Agra FNP 2011). Powdery mildew is a foliar disease caused by the fungus *Podosphaera xanthii* (Castagne) U. Braun & N. Shishkoff (Shishkoff 2000), which limits the production of melon crops worldwide. According to McCreight (2006), 28 probable races of *P. xanthii* can be identified based on the reactions of 31 melon genotypes. In Brazil, the presence of races 1 and 2-French has been reported (Reifschneider et al. 1985, Kobori et al. 2004, Reis and Buso 2004).

Resistance to *P. xanthii* is predominantly controlled by major genes with dominant effects (Kenigsbuch and Cohen 1992, Epinat et al. 1993), although there are reports of recessive genes and modifiers (McCreight et al. 1987, Yuste-Lisbona et al. 2010). To date, 12 major genes have been described (Pitrat 2006, Liu et al. 2010), but only four have been mapped (Pitrat 1991, Périn et al. 2002, Teixeira et al. 2008).

The accession PI 414723 (*Cucumis melo* subsp. *agrestis*) is resistant to insects and fungal diseases, including powdery mildew. Besides resistance to race 2-French, conferred by *Pm-x*, PI 414723 also carries *Pm-7* for resistance to race 1 (Anagnostou et al. 2000, Pitrat 2006) and is resistant to races 3 and 5 (Pitrat et al. 1998). However, the genetic location of *Pm-7* and the mode of inheritance of resistance to races 3 and 5 are not known. Thus, the aim of this study was to define the mode of inheritance of resistance to races 3 and 5 of *P. xanthii* in the cross PI 414723 × Védrantais, and to locate the resistance genes to these races, and to race 1 in a linkage map using referenced microsatellite, as well as AFLP and TRAP markers.

MATERIAL AND METHODS

Plant material

A population of 87 F_2 plants was generated from a cross between PI 414723 and Védrantais. The former was used as male parent, belonging to the Group Momordica (*Cucumis melo* subsp. *agrestis*), and it is resistant to races 1, 3, and 5 of *P. xanthii*, while the latter is a French commercial line belonging to Cantaloupensis Charentais Group (*Cucumis melo* subsp. *melo*), and it is susceptible to these races (Jahn et al. 2002).

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Isolates and scoring of resistance to *P. xanthii*

Isolates Px04 (race 1), Px08 (race 3) and Px02 (race 5) of *P. xanthii* were maintained by regular inoculations on cotyledons of Safira cucumber cultivar, under controlled conditions, using an eyelash brush (Nicot et al. 2002). After inoculation, cotyledons were incubated in a growth chamber with a photoperiod of 12 h light/12 h dark, at 21±2ºC, for about 15 days. Isolates were classified into races based on the reaction of seven differential melon genotypes (Védrantais, PMR 45, WMR 29, Edisto 47, PMR 5, PI 414723 and PI 124112), according to Pitrat et al. (1998), with the addition of Hale's Best Jumbo as susceptible line (Thomas 1978). F_2 plants plus three plants from each of the parental lines, and F_1 hybrid were inoculated with races 1, 3 and 5 of *P. xanthii*, using an eyelash brush in the third, fourth and fifth fully expanded leaves, by placing the inoculum of each race at two equidistant spots (in relation to the midrib), on each leaf (totaling six inoculation spots per race), according to Yuste-Lisbona et al. (2010). Leaves were monitored from eight to sixteen days after inoculation (42 to 50 days after sowing) when they were visually scored to the naked eye, for the presence of fungal structures. Plants with no visible sporulation or low level of sporulation were considered to be resistant, whereas those with moderate level of sporulation, or profuse sporulation were considered to be susceptible (Yuste-Lisbona et al. 2010).

DNA extraction

DNA was extracted from leaves, according to Teixeira and Camargo (2006), except that the DNA solution was extracted with phenol: chloroform: isoamyl alcohol (25:24:1), and, after precipitation, DNA pellet was re-suspended with 1 M NaCl, and re-precipitated with ethanol (Barker 2005). DNA was suspended in 100 µl of TE, and its concentration was determined by spectrometry.

Molecular markers

Forty seven microsatellite markers polymorphic between PI 414723 and Védrantais were selected from the linkage maps described by Fukino et al. (2008) and Gonzalo et al. (2005), in order to represent all linkage groups and provide a map framework. The PCR reactions consisted of 20 ng of DNA, 0.6 µM of each primer, 1X PCR Master Mix (Promega - M7505) and ultrapure sterile water to complete 12 µl. The amplification conditions were according to Gonzalo et al. (2005), with the exception that 25 amplification cycles were used and the annealing temperature was optimized for each marker.

The amplification of AFLP fragments was based on an adaptation of the protocol of Vos et al. (1995) using the PCR Master Mix (Promega - M7505). Six and 23 combinations of primers were used, respectively, in the pre and selective amplifications. Genomic DNA was digested with *Eco*RI/*Mse*I or *Hind*III/*Mse*I (Teixeira and Camargo 2006). AFLP markers were identified according to the Keygene AFLP primer nomenclature system followed by their size in base pairs (http://wheat.pw.usda.gov/ggpages/keygeneAFLPs.html).

Target region amplification polymorphism (TRAP) markers were generated according to Hu and Vick (2003). Fixed primers were designed based on nucleotide binding site-leucine-rich-repeat (NBS-LRR) sequences of melon reported by Brotman et al. (2002), and deposited in GenBank under accession numbers AF354505-07 and AF354515-17 (Table 1). Arbitrary primers were those reported by Li and Quiros (2001) and Hu and Vick (2003). Nine combinations

Table 1. Sequences of arbitrary and fixed primers and combinations of primers used to amplify TRAP markers

Arbitrary primers	Fixed primers (*GenBank accession number)*	Arbitrary/Fixed combinations
		em1/ NBS-5
Ga3-800[1]	NBS-2	em2/ NBS-2
TCATCTCAAACCATATACAC	CACCAATGCCTGTAGTTG	em3/ NBS-5
Ga5-800[1]	(AF354505)	em3/ NBS45-8
GGAACCAAACACATGAAGA	NBS-3	em3/ NBS46-7
em1[2]	TCCGACGAAGGAAGTAAT	Ga3-800/ NRT-A4
GACTGCGTACGAATTAAT	(AF354506)	Ga3-800/ NBS-3
em2[2]	NBS-5	Ga5-800/ NBS-2
GACTGCGTACGAATTTGC	AAACCTTAGCCAAAGTCG	Ga5-800/ NBS-5
em3[2]	(AF354507)	
GACTGCGTACGAATTGAC	NBS45-8	
	TTGTTCAGACACCCAAAC	
	(AF354515)	
	NBS46-7	
	GCTAGCCAATTTGAAGGT	
	(AF354516)	
	NRT-A4	
	GGAAACCAGATTTGAAGC	
	(AF354517)	

[1] Hu and Vick (2003);
[2] Li and Quiros (2001).

of fixed/arbitrary primers were used (Table 1), and amplification conditions followed Hu and Vick (2003), except that 12 cycles were used in the first amplification round. PCR reaction consisted of 100 ng of DNA, 1X of PCR Master Mix (Promega - M7505), 0.5 µM of arbitrary primer, 0.8 µM of fixed primer and ultrapure sterile water to complete 12 µl. Markers were identified by the primer combination, followed by the fragment size (bp).

Microsatellite markers were subjected to electrophoresis on 7.5% polyacrylamide gel, and AFLP and TRAP markers on 6.0% gel, according to Teixeira and Camargo (2006). Gels were stained with silver nitrate according to Creste et al. (2001).

Segregation and linkage analyses

χ^2 test was used to analyze the segregation ratio of resistant:susceptible F_2 plants for each race and to test the hypothesis of independent segregation of the resistance genes (p=0.01). The test was also used to identify any marker with segregation distortion by testing the conformity of the observed frequencies of marker-genotype classes for each marker locus to the expected ratios of 3:1 (dominant, in the case of AFLP and TRAP markers) or 1:2:1 (co-dominant, in the case of microsatellite markers), using the Bonferroni correction (α-value = 0,00020). Segregation distortion was inferred when p-value <α. A linkage map was constructed with MAPMAKER version 3.0 (Lander et al. 1987). Markers were associated with the *group* and ordered with the *order* commands with LOD ≥ 3.5. Microsatellite markers were used for defining the linkage groups which were named according to Périn et al. (2002). Distances were calculated with the Kosambi´s mapping function. MapChart program version 2.2 was used to draw the linkage groups (Voorrips 2002). Resistance genes were mapped by coding the reactions of F_2 plants as a dominant marker.

RESULTS AND DISCUSSION

Scoring of resistance to *P. xanthii* and segregation analysis

Structures of powdery mildew became visible at the inoculation points approximately 8 days after inoculation, and leaves were scored 16 days after inoculation. For all three races, plants of Védrantais displayed lesions typical of a susceptible reaction with abundant conidia and conidiophores, whereas those of PI 414723 and of F_1 plants were resistant, with none or very sparse production of these fungal structures. Most of F_2 plants showed the same reaction to all three races, except for four plants that were resistant to races 1 and 5, but susceptible to race 3, indicating that they were recombinants. Therefore, the lesions of these plants were again analyzed to the naked eye and the possibility that they represented inoculation escapes was ruled out. The number of resistant and susceptible F_2 plants to races 1 and 5 were 75 and 12, respectively. For race 3, 71 plants were resistant, and 16 were susceptible. In both cases, segregation ratios conformed both to 3:1 and 13:3 at different probability levels (Table 2). However, the hypothesis of independent segregation of the genes for resistance to races 1 and 5, and to race 3 was rejected regardless of the segregation ratio considered (data not shown).

The most parsimonious interpretation of the segregation ratios of resistant and susceptible plants indicated that resistance to races 1, 3 and 5 of PI 414723 can be explained by the segregation of a single dominant resistance gene (3:1 segregation ratio). However, data also fit a digenic epistatic model (13:3 segregation ratio) due to the high frequency of resistant plants**.** It is possible that some of these plants represent inoculation escapes. However, the frequency of such cases is expected to be low since each plant was inoculated six times with the same isolate. Therefore, under the digenic epistatic model, resistance to a given race would be controlled by one dominant and one recessive gene (designated as A and b, respectively). The genotypes of resistant plants would be either *A___* or *__bb*, and the susceptible's would be *aaB_*. Epistasis would occur in the case of the double recessive genotype *aabb*, where *bb* combination would be epistatic over *aa,* and would condition resistance. Most likely, population size and qualitative nature of the present phenotypic data did not allow a clear distinction between monogenic and digenic models. Notwithstanding, co-segregation analysis of disease reaction phenotypes indicated that the dominant gene that confers resistance to races 1 and 5 is distinct from the one that confers resistance to race 3, although they are closely linked. These genes are hereby designated *Pm-x1,5*

Table 2. Segregation of resistance to *Podosphaera xanthii* races 1, 5 and 3 in progeny of the cross between PI 414723 and Védrantais

	Races 1 and 5					Race 3				
Progeny	observed R:S	χ^2 (3:1)	p-value	χ^2 (13:3)	p-value	observed R:S	χ^2 (3:1)	p-value	χ^2 (13:3)	p-value
PI 414723	3:0					3:0				
Védrantais	0:3					0:3				
F_1	3:0					3:0				
F_2	75:12	5.8	0.016	1.4	0.236	71:16	2.0	0.154	0.0	0.932

and *Pm-x3*. The first two letters follow the conventional abbreviation used for resistance genes to *P. xanthii* in melon (Pitrat 2006) and the third follows the previous *Pm-x* gene identified in PI414723, which confers resistance to race 2-French (Pitrat 1991). The relation of *Pm-x1,5* to the previously described *Pm-7,* also from PI414723, and which confers resistance to race 1 (Anagnostou et al. 2000), remains to be determined once the map position of *Pm-7* was not defined. Monogenic control of resistance to *P. xanthii* has been extensively described in melon (Pitrat 2006, Liu et al. 2010), whereas a digenic control following the same dominant/recessive epistatic model mentioned above was recently proposed by Yuste-Lisbona et al. (2010) for resistance to races 1, 2, and 5 in TGR-1551 melon genotype. More interestingly, these data initially indicated that resistance to all three races was conferred by the same genes; however, in a following study using quantitative rather than qualitative phenotypic data (Yuste-Lisbona et al. 2011a), it was concluded, like in the present study, that the dominant locus consisted of two tightly linked genes located on LG V, one controlling resistance to races 1 and 2 (*Pm-R1-2*), and the other to race 5 (*Pm-R5*).

Linkage map and gene mapping

The primer combinations used to amplify AFLP and TRAP fragments resulted in the amplification of 1.173 AFLP and 213 TRAP loci, of which 139 (12%) and 18 (8%), respectively, were polymorphic and mapped. In addition, 47 microsatellite markers and the genes for resistance to races 1 and 5, and to race 3 were also mapped, resulting in the assembly of a linkage map with 206 markers distributed in 12 linkage groups (LG), and spanning 1,469 cM (Figure 1). Among the mapped loci, only one (AFLP) marker presented segregation distortion. 12 LG were named in accordance to Périn et al. (2002), based on SSR markers mapped by Gonzalo et al. (2005) and Fukino et al. (2008).

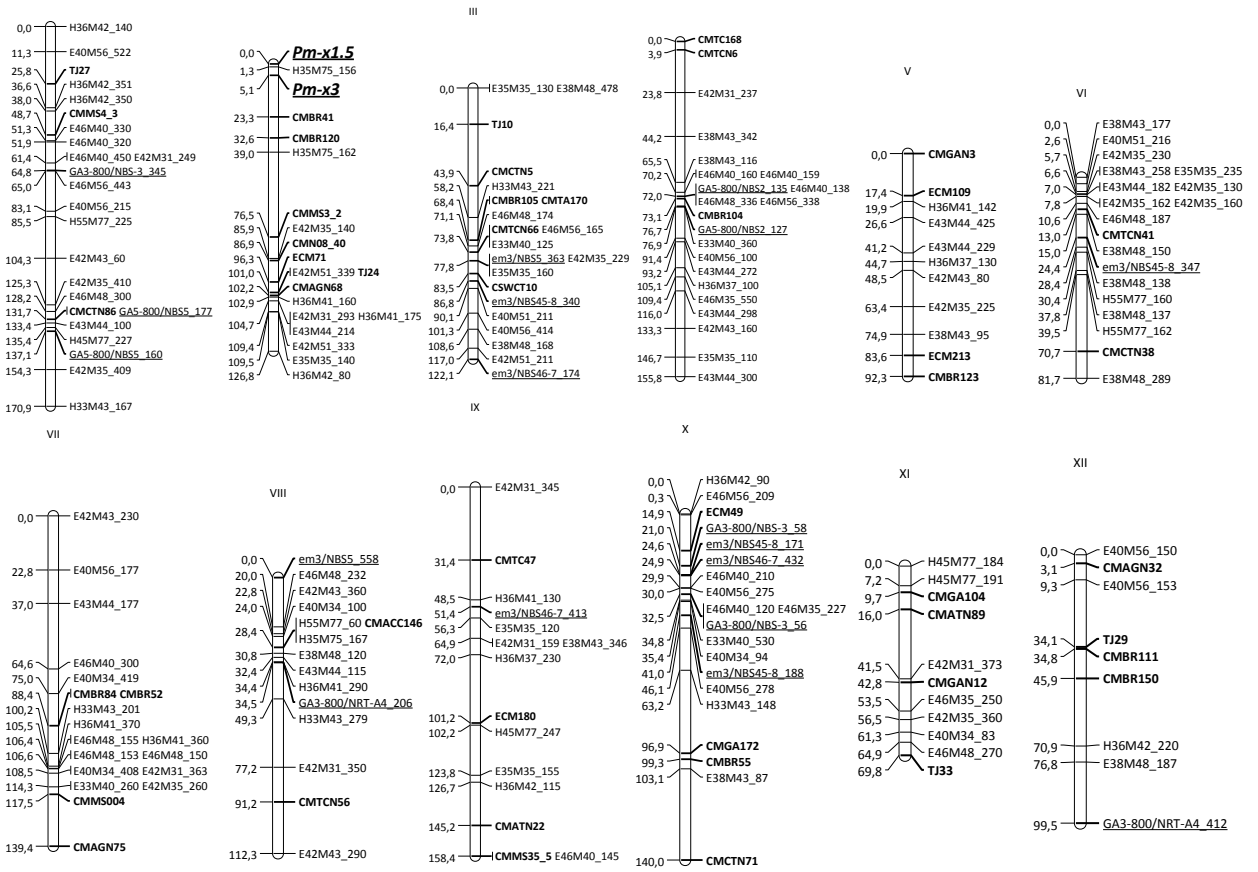

Figure 1. Linkage map of F_2 population (PI 414723 × Védrantais). Linkage groups are named according to Périn et al. (2002). Distances are indicated in centiMorgans (cM) on the left side, and markers are shown to the right of each linkage group. Markers for resistance genes (*Pmx-1,5* and *Pm-x3*) are presented on LG II in *italic bold underlined*. SSR markers are shown in *bold*. RGA markers are *underlined*.

The number of markers per group ranged from nine (group XII) to 24 (group I). The mean LG length was 122.4 cM, ranging from 69.8 cM (group IX) to 170.9 cM (group I). The average distance between markers per LG ranged from 4.3 cM (group VI) to 11.0 cM (group XII), and the largest interval between markers was 37.5 cM (group II). AFLP markers were mapped in all LG, whereas TRAP loci were mapped in eight LG.

The reaction of F_2 plants to *P. xanthii*, when scored as a phenotypic marker, co-segregated with molecular markers from LG II, indicating the likely position of resistance genes to races 1, 3, and 5. This LG comprises 11 AFLP, seven microsatellites and no TRAP markers. Genes were located at the extremity of LG, with the one conferring resistance to races 1 and 5, being the most distal, and 5.1 cM apart from the one, conferring resistance to race 3. Dominant AFLP marker H35M75_156, which amplified a fragment from the susceptible parent Védrantais in all four recombinant plants, was located between the resistance genes (Figure 1).

Regardless of the mode of inheritance of resistance to the three races in PI 414723, co-segregation analysis of the qualitative disease phenotypes of F_2 plants with molecular markers mapped both *Pm-x1,5* and *Pm-x3* in LG II. The number of LGs of the map corresponds to the basic chromosome number of melon (x = 12), and the average distance between markers (7.4 cM) is within the values reported in other maps, which ranged from 2.5 cM (Périn et al. 2002) to 17.7 cM (Baudracco-Arnas and Pitrat 1996). If the digenic model is assumed, then these mapped genes correspond to the dominant loci since during linkage analysis with markers most of F_2 plants classified as resistant (12/13) would present the marker genotype *A_*, whereas the susceptible would present the marker genotype *aa*. The exception would be the double recessive plants for the resistance genes (*aabb*) which would present *aa* susceptible marker genotype despite being resistant, due to the epistatic interaction with *bb* alleles. The misclassification of these individuals, of which expected frequency is low (1/13), would be interpreted as a recombination between markers and genes, thus resulting in an overestimation of genetic distances.

Besides *Pm-x1,5* and *Pm-x3*, other disease resistance genes from PI414723 have been located directly or indirectly in LGII. These include *Zym* (also *Zym-1*) for resistance to pathotype 0 of *Zucchini yellow mosaic virus, Pm-x* for resistance to race 2-French of *P. xanthii* (Périn et al. 2002), and *Wmr* for resistance to *Watermelon mosaic virus-2* (Gilbert et al. 1994). While *Zym* was directly mapped in the terminal portion of LGII by segregation analysis of a recombinant inbred population derived from the same cross used in this study, the location of *Pm-x* was presumed based on an earlier report of its linkage (7 cM) to *Zym* (Pitrat 1991). By the same reasoning, since *Wmr* was reported to be linked (7.5 cM) to *Zym* (Anagnostou et al. 2000) it must also be located in this linkage group. Taken together, these data suggest a cluster of resistance genes involving *Zym*, *Pm-x* and *Wmr*. However, due to a lack of common markers between the maps of Périn et al. (2002), and the one reported in this study, it is difficult to ascertain at this point whether *Pm-x1,5* and *Pm-x3* are part of this gene cluster or not. For this, a saturation of this linkage group with additional markers would be necessary. It is interesting to note that a resistance QTL to races 1 and N1 of *P. xanthii* from a different resistance source (AR-5) was found closely linked to the microsatellite loci CMBR8 and CMBR120 from LGII (Fukino et al. 2008). The first marker was not mapped in the present study since it was monomorphic between the parents, but the second was located 32.6 cM from *Pm-x1.5,* and 27.5 cM from *Pm-x3,* initially suggesting that QTL is located in a distinct portion of LG and, therefore, do not correspond to neither of these two genes. However, this conclusion needs further testing, given that the accurate location of QTLs depends largely on the density of markers and that LGII assembled by Fukino et al. (2008) is comprised of only eight markers distributed in two unlinked sub-groups.

P. xanthii resistance genes from other melon genotypes were located in linkage groups distinct from the one where the resistance genes from PI 414723 have been located in this study. These include *Pm-1* from PI 78374 (race 1) in linkage group IX (Teixeira et al. 2008), *Pm-w* from WMR-29 (race 2-French) in LG V, and *Pm-y* from VA-435 (race 2-French) in LG XII (Pitrat 1991, Périn et al. 2002). Besides, resistance QTL were also mapped in LGs II (races 1, 3, and N1), V (1, 2, and 5), and XII (races 1, 5, and N1) (Perchepied et al. 2005, Fukino et al. 2008, Yuste-Lisbona et al. 2011b). Thus, the diversity of resistance genes to different races of *P. xanthii* opens the possibility of combining them in single melon genotypes in order to achieve resistance to multiple races. For this, precise knowledge of their genetic location combined with linked molecular markers comprises indispensable information in the breeding of these so called gene pyramids.

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Mapeamento de genes de resistência às raças 1, 3 e 5 de *Podosphaera xanthii* em melão PI 414723

Resumo – *O fungo* Podosphaera xanthii *afeta a cultura do meloeiro, apresenta diversas raças e é controlado por genes de resistência raça-específica. O acesso PI414723 é resistente às raças 1, 3 e 5 e é uma fonte adequada de genes de resistência. A herança da resistência a essas raças foi analisada em 87 plantas F_2 do cruzamento PI414723 × Védrantais. A resistência às três raças pode ser explicada pela segregação de um gene dominante, embora um modelo digênico também possa ser aceito. Um mapa genético foi obtido com 206 marcadores e análise de co-segregação de fenótipos resistentes indicou a existência de dois locos ligados, posicionados no grupo de ligação II, um conferindo resistência às raças 1 e 5 (denominado Pm-x1,5) e o segundo à raça 3 (denominado Pm-x3), localizado a 5,1cM. Este estudo relata pela primeira vez a existência de Pm-x3 e as localizações genéticas desses genes de resistência de PI414723.*

Palavras-chave: *Resistência à doença, marcador molecular, mapeamento genético, oídio.*

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