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Inheritances and location of powdery mildew resistance gene in melon Edisto47

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Abstract Powdery mildew caused by *Podosphaera xanthii* is a major disease in melon. Here we report two *Px* race 1 strains named Px1A and Px1B in Xinjiang, which have different pathogenicities. The more pathogenic Px1B made some powdery mildew resistant genes on linkage group V (LGV) lose their resistant traits. The inheritances of resistance to Px1A and Px1B in melon Edisto47 were studied using a BC_1 population derived from a cross between the resistant genotype Edisto47 and the susceptible cultivar Queen. The resistance/susceptibility segregation ratios observed in the Px1A-inoculated BC_1 population and the loci of polymorphic markers indicated that resistance to Px1A was controlled by two dominant genes. Quantitative trait locus analysis identified two loci mapped on LGII and LGV, respectively, for powdery mildew resistance. However, for resistance to Px1B, Edisto47 was found to bear one dominant gene. A genetic linkage map was constructed using the Px1B-inoculated BC_1 population to map the resistant gene. Comparative genomic analyses revealed that the linkage map of *Pm-Edisto47-1* was collinear with the corresponding genomic region of the melon chromosome 2. Genetic analysis showed that *Pm-Edisto47-1* was located between simple sequence repeat (SSR) markers CMGA36 and SSR252089, at a genetic distance of 2.1 cM to both markers. Synteny analysis showed that two genes named MELO3C015353 and MELO3C015354 were predicted as candidates for *Edisto47-1* in this region.

Keywords *Podosphaera xanthii* race 1 · Resistance gene inheritance · Genetic map · *Cucumis melo* L.

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Introduction

Melon is a major economic fruit crop widely cultivated in China. Xinjiang melon, in particular, enjoys great reputation worldwide because of its unique flavor and high sugar content. However, frequent occurrence of powdery mildew causes serious wilt or death to melon plants, and thus greatly limits the production of melons (Zitter et al. 1996).

Powdery mildew caused by *Podosphaera xanthii* (Shishkoff 2000) or *Golovinomyces cichoracearum* (Vakalounakis and Klironomou 2001) is a devastating disease to melon worldwide (Yuste-Lisbona et al. 2010). Since the optimum temperature for conidial germination is 15–25 °C for *Gc* and 25–30 °C for *Px* (Krístková et al. 2009), *Px* occurs more frequently in subtropical and tropical areas, while *Gc* is common in temperate and cooler areas (Lebeda et al. 2011). In China, the major pathogen of melon powdery mildew is *Px* (Cheng et al. 2011; Liu et al. 2010), and the

predominant race in the melon-growing areas in Xinjiang is *Px* race 1 (Lin 2011).

Chemical treatments are most commonly used by melon growers to prevent the disease from spreading (Yuste-Lisbona et al. 2011b). However, long-term use of fungicides has led the pathogens to resist against many chemical fungicides, which have therefore lost their functions gradually (Hollomon and Wheeler 2002). Also most of these chemical fungicides are environmentally unfriendly, and thus the growing concern for the environment and public health has motivated melon breeders to seek other strategies to control powdery mildew (Yuste-Lisbona et al. 2011a). Thus, there is an urgent need for highly efficient, low cost and eco-compatible approaches against local predominant pathogen. The development of new resistant cultivars is such a promising way to control powdery mildew (Cheng et al. 2012).

The traditional breeding approach of phenotypic selection is laborious and time-consuming. The genetic variability of the pathogen population also makes it difficult to breed durably resistant cultivars (Cohen et al. 2004). Fortunately, marker-assisted selection (MAS) is promising in efficient and rapid selection of certain phenotypes, and it is not hampered by pathogen unavailability, which saves the time and expense of field work. However, MAS and transgenic technology have several prerequisites: development of linkage maps, identification of specific molecular markers linked to genes, and well understanding of the information about different resistance genes and quantitative trait loci (QTLs).

To date, many linkage maps have been constructed and reported in melons. Among them, the integrated melon map constructed by Diaz et al. (2011) using a variety of molecular markers provides some valuable information for the construction of saturated genetic linkage maps and the location of genes. However, its molecular marker resolution is insufficient to locate some powdery mildew resistant genes.

In melon, 17 powdery mildew resistance genes (Pitrat 2002; Wang et al. 2011; Yuste-Lisbona et al. 2011b; Zhang et al. 2013) and 4 QTLs associated with the resistant traits (Perchepied et al. 2005; Fukino et al. 2008) have been reported. Several genes and QTLs have been mapped: *Pm-x* (Perin et al. 2002), *Pm-2F* (Zhang et al. 2013) and QTL (AR 5) (Fukino et al. 2008) on LGII; *Pm-w* (Pitrat 1991), *Pm-R* (Yuste-Lisbona et al. 2011b), *Pm-AN* (Wang et al. 2011), and QTL *PmV.1* (PI 124112) (Perchepied et al. 2005) *on* LGV; *Pm-y* (Perin et al. 2002), QTL *PmXII.1* (PI 124112) (Perchepied et al. 2005) and QTL (AR 5) (Fukino et al. 2008) on LGXII. However, the development of cultivars from those resistant genotypes is hampered by low saturation (Yuste-Lisbona et al. 2011b) and lack of tightly linked markers (Zhang et al. 2013). In addition, some resistant genes of resistant melons, among 30 reported resistant cultivars (Zhang et al. 2013), have yet to be published.

With high polymorphism, codominance and transferability, simple sequence repeats (SSRs) can be used to construct high resolution linkage maps (Fukino et al. 2008; Noguera et al. 2005; Cuevas et al. 2009). In addition, with the completion of melon genome sequencing (Garcia-Mas et al. 2012), a growing number of SSR markers will be designed to construct dense linkage maps, which helps to locate resistant genes and discover tightly linked markers.

The melon Edisto47 was bred in the US in 1965, with orange flesh, dark green fruit skin and spotted and reticulated skin (Lin 2011). This genotype is resistant to races 0, 1, 2F, 3, 4, 5N1, and N2 of *Px* (Lebeda and Sedláková 2010; Kuzuya et al. 2006; Lebeda et al. 2011; Wang et al. 2006). However, the genes controlling its resistant traits are poorly understood.

In this context, two *Px* race 1 strains Px1A and Px1B were reported. Px1A belongs to the same *Px* race 1 as that in the experiment of Wang et al. (2011). Px1B has stronger pathogenicity which makes the resistant genes located on LGV lose their resistant traits. We report different inheritances of Edisto47 to these two strains, the detection of QTLs conferring Px1A resistance, and the resistant gene conferring Px1B resistance. The closely linked markers were identified by linkage mapping. Furthermore, candidate genes conferring powdery mildew resistance were reported and discussed through sequence analysis and comparative genomic analysis using melon genome sequencing. Knowledge about different inheritances of Edisto47 to the powdery mildew strains provides opportunities for understanding the interaction between resistant genes and fungal strains. Markers linked with *Pm-Edisto47-1* can be used to locate resistant gene on melon chromosome, which is helpful for resistant gene cloning and allows the development of MAS in new resistant melon cultivars.

Table 1 Reaction of melon powdery mildew race differential lines to the powdery mildew isolates

Genotype	Reaction	
	Px1A (2011 summer)	Px1B (2012 summer)
PMR5	R	R
PMR6	R	R
PMR45	R	R
PI414723	R	R
PI124111	R	R
PI124112	R	R
MR1	R	R
WMR29	R	R
IranH	S	S
Vedrantais	S	S
Edisto47	R	R
Topmark	S	S
Nantais Oblong	S	S
Ano2	R	S
HXC	R	S

R resistant, *S* susceptible

Materials and methods

Plant materials and DNA extraction

The population used in this study consisted of 632 BC_1 plants derived from a cross between Edisto47 and Queen. Edisto47, whose ancestry has 'PI124112' (Perchepied et al. 2005), is highly resistant to *Px* race 1, whereas Queen, which is a breeding line derived from the offspring of Golden beauty (Wang et al. 2011), is susceptible to powdery mildew. Fifteen accessions were used as host plants to identify powdery mildew races (Table 1). Thirteen melon lines, Queen and Ano2 were provided by National Melon Engineering and Technology Research Center, Xinjiang, China (NMETRC). HXC was supplied by Professor Mingzu Wu (Center of Hami Melon, Xinjiang Academy of Agricultural Science). Genomic DNA was isolated from young leaves according to the method from Porebski et al. (1997). The DNA was diluted to 100 ng/μl.

Powdery mildew strains

The powdery mildew fungi were collected at NMETRC in 2011 and 2012. Powdery mildew races can be assigned by comparing the reactions of 13 genotypes (Bardin et al. 1999; Lebeda et al. 2011; Hosoya et al. 2000; McCreight 2006), Ano2 and HXC to powdery mildew isolates.

Powdery mildew resistance test

The BC1 population was evaluated in three experiments conducted at different seasons and locations (Table 2). Two greenhouse experiments were performed at 22–23 °C under a 16 h photoperiod in Xinjiang University (experiments A and C). One experiment was done in the field of NMETRC [the same field as that of Wang et al. (2011)]. Powdery mildew resistance tests were performed under artificial inoculation using the methodology from Wang et al. (2011). Artificial inoculations were carried out on two parent plants, F_1 generation plants and all the BC_1 plants. Plants without visible sporulation on the leaf were resistant, while plants with typical mildew colonization were susceptible. The results were recorded until the symptom appeared on most third true leaves after 10–15 days. Data were analyzed for each experiment. The goodness-of-fit of resistance to powdery mildew was checked by χ^2 test.

Identification of SSR markers linked to powdery mildew resistance

For bulked segregant analysis (BSA), one resistant and one susceptible DNA bulks (each with 20 plants) were prepared from the BC_1 population in experiment A. Then 392 SSRs from melon and cucumber (Diaz et al. 2011) were screened in the resistant and susceptible DNA bulks. A total of 44 unpublished melon SSR primers, designed from the melon genome sequence (Garcia-Mas et al. 2012), were also used to amplify the blended DNA bulks. DNA was amplified in a BIO-RAD C1000™ Thermal Cycler under the following protocol: 94 °C for 3 min; 35 cycles at 94 °C for 30 s, 54 °C for 30 s, 72 °C for 30 s; and 72 °C for 10 min. Finally, after electrophoresis in 2 % agarose gels (3.0 μl/lane) with 1× tris–acetate-EDTA (TAE), the PCR products were visualized with ultraviolet (UV), stained with ethidium bromide, and then separated on 32 × 38 × 0.04 cm 6 % denaturing polyacrylamide gels (4.5 μl/lane) containing 7 M urea and 1× tris-boric acid-EDTA (TBE).

Table 2 Segregation of resistance in BC1 population after inoculation with two strains of *Px* race 1 (Px1A and Px1B) separately in 2011 winter, 2012 summer and 2012 winter

Experiment	Season	Condition	Strain	*n*	Observed R:S	Expected R:S	χ^2 test	
							χ^2	*P*
A	2011 winter	Glasshouse	Px1A	98	70:28	49:49	18	<0.05
B	2012 summer	Open field	Px1B	248	121:127	124:124	0.145	0.7
C	2012 winter	Glasshouse	Px1B	288	158:130	144:144	2.72	0.1

Px1A was isolated in 2011; Px1B was isolated in 2012; n is number of evaluated plants; R (resistant plants) is the sum of plants without visible sporulation on the leaf; S (susceptible plants) is the sum of plants with typical mildew colonization; $P > 0.05$

QTL analysis

Based on the resistance/susceptibility segregation ratios of the Px1A-inoculated BC_1 population (experiment A), two QTLs were located by testing the genotype of each BC_1 plant using the polymorphic markers (Table 1). The data were analyzed by Mapmaker 3.0 and the marker data were assigned to LGs using a logarithm of odds (LOD) threshold of 2.5 (Wang et al. 2011).

Identification of association between QTL and resistant strains

The association between strains and QTL was identified by bulked DNA analysis. Two resistant and two susceptible DNA bulks (each with 20 plants) were generated from the BC1 population in experiment A and C. SSR markers (SSR 310365, 37253, 433600) linked with the QTL *Pm-Edisto47-2* on LGV were tested on the resistant and susceptible bulks. From the PCR amplification results between experiments A and C, it can be inferred whether the resistant QTL on LGV lost its function in the Px1B-inoculated plants.

Mapping of *Pm-Edisto47-1* in the Px1B-inoculated BC1 population

Polymorphic markers on LGII were tested on the Px1B-inoculated BC_1 populations to locate *Pm-Edisto47-1*. A linkage map was constructed with QTL Cartographer v2.5 on windows (Wang et al. 2007). The marker data were analyzed by Mapmaker 3.0. Gene annotation and analysis were conducted according to the melon genome automated annotation database (https://melonomics.net).

Results

Determination of physiological race

Pathotypes of *Px* race have been recognized by their interactions with 13 melons. The susceptible reactions of IranH, Vedrantais, Topmark, and Nantais Oblong demonstrate that the strains in 2011 and 2012 both belong to *Px* race 1, whereas the susceptible reactions of Ano2 and HXC indicate that race 1 denoted as Px1B in 2012 has a stronger pathogenicity than Px1A (Table 1). Subsequently, powdery mildew Px1A and Px1B were maintained on Queen and Ano2 respectively.

Evaluation of powdery mildew resistance

Fifteen days after artificial inoculation with Px1A or Px1B, no melon plants of Edisto47 or F_1 progeny showed susceptible symptoms, whereas Queen showed whitish, talcum-like powder on the surface of inoculated leaves, suggesting that the resistant traits of Edisto 47 to Px1A and Px1B were both dominant.

In experiment A, 70 plants are resistant and 28 are susceptible (Table 2). This segregation ratio does not fit the Mendelian segregation ratio of 1:1 ($P < 0.05$), indicating that the inheritances of Edisto47 to Px1A do not correspond to the single dominant gene model.

In experiment C, 130 out of 288 plants are susceptible, indicating a ratio consistent with the Mendelian segregation ratio of 1:1 ($\chi2 = 2.72$, $P = 0.1$). Resistance to Px1B in experiment B also shows a significant resistance/susceptibility segregation ratio of 1:1 ($\chi2 = 0.145$, $P = 0.7$) (Table 2). The above results suggest that the resistance of Edisto47 to Px1B is controlled by a single dominant gene under a field environment as well as a controlled environment.

Table 3 The primer sequence of polymorphic SSR markers designed from the melon genome

Marker	Forward primer sequence	Reverse primer sequence	Location[a]
SSR203205(2-1)	ATGAACCCAAAGAAAACAAA	TAAGAAAGTTCTGCATGAGA	780,678
SSR219093(2-3)	CATTTTGCAGACAAGTTTTA	GATATGGTTTGGTTGGTCGT	842,062
SSR252089(2-19)	TCAAATCCTAAACCCTAAAC	ATGCACCTACTTGTTGTTGT	1,004,670
SSR268176(2-24)	CATGAAGTATAAATAGTTTG	CACAATCCTACACTTTGCTA	1,085,690
SSR283138(2-31)	TTTATTTGTAACATCGTGGC	TCTCAAACTTACTGCATACT	1,086,500
SSR310365(5-2)	GGCGATGGATGAAAGTTAGT	AGAAGGAGGATTATTGGGTT	
SSR37253(5-4)	AAAAGAGCTGAAATCGAACA	TATTGCCTTTCTCGTCTCTT	
SSR433600(5-6)	ATAGATGTAGTCGAGGTTGAA	AGAGGCAGAAATGGTGAAAT	

[a] The location of designed SSR markers in melon chromosome 2

Identification of SSR markers linked to powdery mildew resistance

Linked SSR markers in the BC_1 population of experiment A were identified. Among 436 SSR primers tested, 133 (30.5 %) markers were polymorphic and tested on the resistant and susceptible DNA bulks. Of the 133 SSR markers, 17 (12.8 %) SSRs were polymorphic between the resistant and susceptible bulks. They were ECM129, ECM92, GCM295, ECM142, DM0287, DE1469, GCM622, SSR433600, SSR310365 and SSR37253 on LGV; SSR203205, SSR219093, SSR268176, SSR283138, SSR252089, CMGA36 and CMBR008 on LGII. Segregation of all polymorphic SSR markers on LGII or LGV both fitted the expected segregation ratio of 1:1 by marker data comparison using Chi square test. The primer sequence of polymorphic SSR markers designed from the melon genome is shown in Table 3.

QTL analysis

Seventeen co-dominant SSR markers were used to construct a map consisting of 2 LGs. Two QTLs for powdery mildew resistance were identified, which were located on LGII and LGV, respectively (Table 4). The QTL on LGII between markers SSR219093 and SSR252089 was designated as *Pm-Edisto47-1*, while the QTL on LGV between markers GCM295 and DE1469 was named *Pm-Edisto47-2*.

Validation of resistance-strain association in BC_1 population inoculated with two strains

Unlike Edisto47 in experiment A, the segregation ratio of the BC_1 population in experiment C indicated that the resistant trait of Edisto47 to Px1B was controlled by one dominant gene ($\chi^2 = 0.145$, $P = 0.7$). In addition, the powdery mildew resistance gene *Pm-AN* from Ano2 was susceptible to Px1B. Therefore, we suppose that Px1B can overcome the resistance of QTL from Edisto47 and *Pm-AN* from Ano2 which are both mapped on LGV. To further determine whether Px1B can lead to the lose of resistant trait efficacy, the polymorphic markers between experiments A and C were compared using bulked DNA analysis. The polymorphic markers (5–2, 5–4, 5–6) were polymorphic between the resistant and susceptible bulks for the Px1A-inoculated plants in experiment A (Fig. 1a), but not for the Px1B-inoculated plants in experiment C. This result indicates that the resistance gene of Edisto47 to Px1B is unlinked to markers on LGV (Fig. 1b), which agrees with the hypothesis that the QTL for powdery mildew resistance on LGV fails to resist infection of Px1B.

Mapping of powdery mildew resistant gene

Seven polymorphic markers (screened from experiment A) on LGII were used to screen the resistant and susceptible bulks of BC_1 plants in experiment C. All those markers were found polymorphic and used to construct a linkage map of *Pm-Edisto47-1*. The linkage map was collinear with the corresponding genomic region of the melon chromosome 2 (Fig. 2). The resistant gene *Pm-Edisto47-1* locus in the region was delimited by markers CMGA36 and SSR252089. The recombination frequency analyses indicated that the genetic distances between closely linked markers and *Pm-Edisto47-1* were both 2.1 cM. Sequence of this region in the melon genome CM3.5_scaffold00025 was

Table 4 QTLs detected for resistance to Px1A in BC1 population using linkage mapping

QTL	Linkage group	Flanking markers	Position[a]	LOD	Additive effect	R^2
Pm-Edisto47-1	LGV	GCM295–DE1469	32.9	6	0.5	0.56
Pm-Edisto47-2	LGII	SSR219093–SSR252089	7.6	6.7	0.4	0.92

[a] Expressed in Kosambi cM

Fig. 1 Comparison between polymorphic markers of BC_1 population inoculated with two strains which are different in pathogenicity. Inoculation segregating population mark detection using Px1A in experiment A **a** and using Px1B in experiment C **b** with polymorphic markers (5–2, 5–4, 5–6). Number is for markers on LGV; each marker is used to amplify resistant (*left*) and susceptible (*right*) pool of BC_1 population separately

analyzed according to the melon genome automated annotation database (https://melonomics.net). The results indicated two candidate genes in the *Pm-Edisto47-1* resistant region. One candidate (accession number MELO3C015353 from 984845 to 987279) is similar to disease resistance protein RGA2 (*Solanum bulbocastanum*), while the other candidate (MELO3C 015354 from 990646 to 994117) is similar to putative disease resistance protein RGA4 (*S. bulbocastanum*).

Discussion

Recently, new races and variants of powdery mildew have been reported (Lebeda et al. 2011). However, since the predominant race of powdery mildew will change, the crucial problem is race identification so as to introduce appropriate resistances when working with powdery mildew resistance (Zhang et al. 2013). Currently, identification and classification of powdery mildew race are based on the reactions of 13 differential hosts. In this context, a new melon powdery mildew *Px* race 1 (Px1B) was distinguished by the reactions of 13 differential cultivars, Ano2 and HXC melon. Px1B has a stronger pathogenicity, which leads some resistant genes on LGV to lose their resistant traits. Some new resistant cultivars bred from Ano2 and HXC cannot even be commercialized because of dissatisfactory resistance necessity, since Px1B replaced Px1A as the predominant race in 2012. This result confirmed that pyramiding breeding is perhaps the necessary strategy to control powdery mildew. In addition, we found that both Ano2 and HXC can be used as host plants to determinate *Px* races, which expand the range of differential cultivars carrying a race-specific resistance gene.

Different inheritances of powdery mildew resistance in melon have been proposed, which could be roughly divided into three types: (1) Single gene control, such as PMR45 (Lebeda et al. 2011), PI414723 (Cohen et al. 2012) and Ano2 (Wang et al. 2011), PI124112,TGR-1551; (2) Multiple gene control, such as MR1 (Wang et al. 1997; Monforte et al. 2003) and PI124111 (Zhang et al. 2013); (3) Resistant trait presents different performances among races, such as PI313970 (Sedlárová et al. 2011). Up to 30 powdery mildew resistant melon cultigens have been found (Zhang et al. 2013). However, resistant inheritances of some cultivars are not understood clearly. In this study, the resistance of Edisto47 to Px1A was controlled by two dominant genes while that to Px1B was controlled by one dominant gene. This is the first time that the resistant inheritance of Edisto47 has been reported, which is similar to the third genetic model.

Previously, three genes and one QTL in melon LGV (Pitrat 1991; Yuste-Lisbona et al. 2011b; Wang et al. 2011; Perchepied et al. 2005), and two genes and one QTL in melon LGII (Perin et al. 2002; Zhang et al.

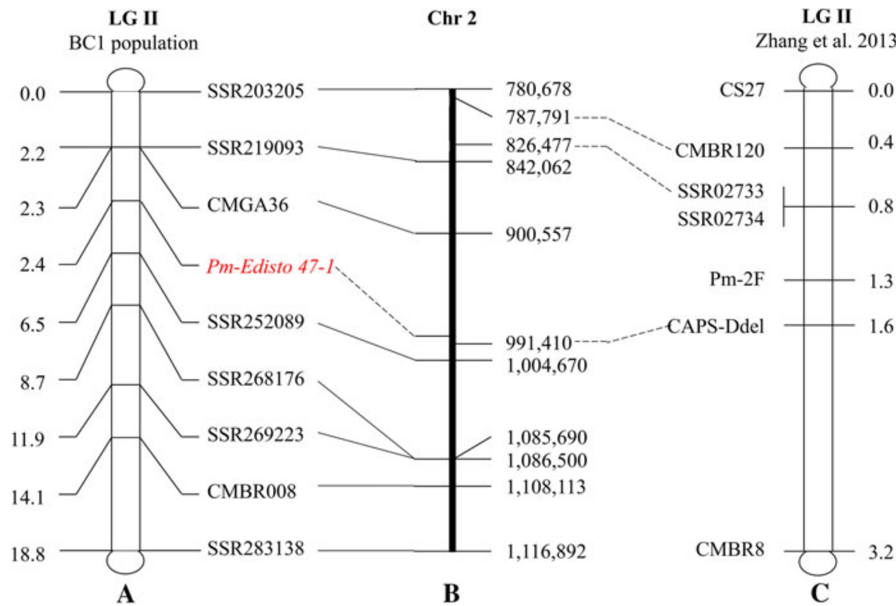

Fig. 2 The Linkage map of *Pm-Edisto47-1* is collinear with the corresponding genomic region of the melon chromosome 2. **a** Linkage map of *Pm-Edisto47-1* in the BC_1 population (experiment C). The markers are shown to the *right*, and map distances, to the *left*, expressed in centimorgan (cM). **b** The corresponding genomic region of melon chromosome 2. **c** Linkage map of the *Pm-2F* mapping powdery mildew resistance gene *Pm-2F* to races 2F using RILs of K 7-1 and K 7-2. The markers are shown to the *left*, and map distances, to the *right*, expressed in centimorgan (cM). *Solid lines* between **a** and **b** indicate the location of anchor markers of linkage map on melon chromosome 2. *Dashed lines* indicate that the location of marker of comparable linkage map of the *Pm-2F* on melon chromosome 2

2013; Fukino et al. 2008) have been mapped. In this context, two new independent QTLs named *Pm-Edisto47-1* and *Pm-Edisto47-2* were detected in Edisto47, which were located on LGII and LGV respectively. Compared with the reported resistant genes, *Pm-Edisto47-2* and resistant gene *Pm-AN* were found resistant to Px1A, while those two powdery mildew traits lost their resistant efficacy when Edisto47 and Ano2 were inoculated with Px1B. Therefore, QTL mapped on LGV of Edisto47 is thought to be in agreement with that of Ano2. In addition, SSR markers linked to *Pm-AN* were located near *Pm-Edisto47-2*. These results are consistent with the hypothesis that *Pm-Edisto47-2* of Edisto47 can be related or linked with *Pm-AN* from Ano2. On the other hand, although the ancestry of Edisto47 has ‘PI124112’, the reactions of these two lines to Px1B were quite different. In addition, the reaction of WMR 29 to Px1B was inconsistent with that of Edisto47. These results indicate that the *Pm-Edisto47-2* mapped on LGV carried by Edisto47 can be unallelic or unrelated to *Pm-w* or *PmV.1* detected in WMR29 and PI124112, respectively.

It is usually postulated that in resistant gene mapping experiments, a single gene is relatively easy to position. The resistant genes of multi-gene-controlled materials need to be broken down into simple genetic factors before study. In this paper, the segregation ratios of BC_1 population confirms that resistance of Edisto47 to Px1B is controlled by a single dominant gene. Therefore, this BC_1 population can be used to locate the resistant gene.

Currently, two powdery mildew resistance genes *Pm-x* (Perin et al. 2002), *Pm-2F* (Zhang et al. 2013) and one QTL (AR 5) (Fukino et al. 2008) have been mapped in melon LGII. In this context, one dominant gene *Pm-Edisto47*-1 was mapped on LGII in Edisto47. We found two SSR markers on LGII (CMGA36 and SSR252089) linked with *Pm-Edisto47*-1. Such SSR markers provide anchor points for comparison with available melon genome (https://melonomics.net) and other melon maps (Yuste-Lisbona et al. 2011b; Fukino et al. 2008; Zhang et al. 2013). Comparative mapping results indicated that the collinear region between those two markers (from 900,557 to 1004,670) were closer than SSR02733-CAPS-Ddel makers of *Pm-2F*

(from 826,477 to 991,410) (Zhang et al. 2013) and CMBR120-CMBR008 markers of the linkage map of AR 5×Harukei 3 RILS (Fukino et al. 2008).

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