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QTL Analysis for Resistance to *Phytophthora capsici* in Pepper Using a High Density SSR-based Map

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A segregating doubled haploid (DH) population (n = 96) was developed by anther culture of an F_1 plant crossed between susceptible ('Manganji') and resistant ('Criollo de Morelos 334') lines of pepper (*Capsicum annuum* L.) to conduct a genetic analysis of resistance to Phytophthora rot caused by *Phytophthora capsici*. In order to perform a quantitative trait locus (QTL) analysis, we constructed a high density simple sequence repeat (SSR)-based map with a total length of 878 cM. Sixteen linkage groups (LGs) and 118 SSR markers were located using the 626 SSR markers that we previously developed. Resistance was evaluated in two root inoculation tests. Interval mapping for the resistance to *P. capsici* detected a common major QTL in the duplicate tests and a minor QTL specific to the first test. The major QTL was located on LG15 and flanked with an SSR marker, CAMS420. In addition, seven SSR markers were located within 21 cM intervals from the peak of this QTL. In contrast, the QTL on LG3 was detected with small effects in the first test, the nearest marker was a dominant amplified fragment length polymorphism (AFLP) marker, and the QTL was surrounded by eight SSR markers within a distance of 10 cM. Since some of the linkage markers for agriculturally valuable traits cannot detect polymorphism within breeding populations in *C. annuum*, the present linkage markers may widen the choice in marker-assisted selection in breeding programs for Phytophthora rot resistant pepper cultivars.

Key Words: *Capsicum annuum* L., *Phytophthora capsici*, disease resistance, QTL, SSR, marker-assisted selection.

Introduction

An oomycete parasite, *Phytophthora capsici* Leon., has been reported to attack many crops, including cucumber, pumpkin and watermelon, as well as Solanaceae (Barksdale *et al*. 1984, Ristaino and Johnston 1999). Root rot and foliar blight caused by this pathogen are major diseases in pepper worldwide. *P. capsici* can infect virtually every part of the pepper plant, including roots, stems, leaves and fruits, and causes serious crop losses, especially during periods of heavy rainfall. It is a soil-borne pathogen that can survive on host residuals in soil for months (Oelke *et al*. 2003). Control of the disease mainly relies on fungicide application, which increases agricultural inputs as well as environmental and health risks. Therefore, the development of resistant pepper cultivars would be important. However, no modern commercial cultivars of pepper (*Capsicum annuum* L.) have adequate resistance to *P. capsici*, because of the complex mode of inheritance for resistance.

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Presently, the advent of molecular marker technology has provided information that enables to determine the number, positions and individual effects of resistance loci for quantitatively inherited traits. Several studies have been conducted to screen resistant lines from *Capsicum* genetic resources, and some lines resistant to *P. capsici* have been found (Barksdale *et al*. 1984, Ortega *et al*. 1992, Reifschneider *et al*. 1992). From these resistant resources, a local cultivar from Morelos in Mexico, 'Criollo de Morelos 334' ('CM334'), was reported to be highly resistant to *P. capsici* (Ortega *et al*. 1991, Walker and Bosland 1999, Oelke *et al*. 2003). However, inheritance of the resistance trait seems complex (Lefebvre and Palloix 1996). In a more recent study, a quantitative trait locus (QTL) analysis of the resistance of 'CM334' was performed using a genetic map based on an F_2 progeny, and six major chromosomal regions were reported to be involved in the resistance to *P. capsici* (Thabuis *et al*. 2003). The authors used restriction fragment length polymorphism (RFLP) and amplified fragment length polymorphism (AFLP) as a marker system. However, these marker techniques cannot be routinely used for marker-assisted selection (MAS) in a practical breeding scheme. Moreover, there are no reports about fine mapping and

individual effects of the respective resistance QTLs to *P. capsici*.

We developed approximately 600 simple sequence repeat (SSR) markers from genomic DNA and constructed a linkage map of *C. annuum* that contains 106 new SSR markers distributed across all the linkage groups (LGs) (Minamiyama *et al*. 2006). The objective of the present study was to detect some polymerase chain reaction (PCR)-based markers around QTLs for resistance to Phytophthora rot. The information about marker sequence and QTLs may enable breeders to select resistant individuals during breeding.

Materials and Methods

Plant materials and DNA extraction

One of the parents was the pepper cultivar 'Manganji' (*C. annuum*), which is locally grown in Kyoto, Japan. This cultivar has an occasionally pungent and long conical-shaped fruit, and is susceptible to *P. capsici*. The response to the pathogen is similar to that of susceptible bell pepper lines. The other parent was 'Criollo de Morelos 334' ('CM334', *C. annuum*), a landrace from Morelos, Mexico. This cultivar bears pungent and small fruits, and is highly resistant to the pathogen. A segregating doubled haploid (DH) population (n=96) which was developed by anther culture of an F_1 plant of this parentage, was used for the genetic analysis. DNA was extracted from young leaves using a DNA extraction kit (Amersham Biosciences, Buckinghamshire, UK).

Test for resistance to P. capsici

The *P. capsici* isolate 'P-5' used in the present study was isolated from infected roots of a pepper landrace growing in a field in Kyoto and maintained at the Kyoto Prefectural Institute of Agriculture. The isolate was cultured by shaking in V8 liquid medium at 25°C in an incubator for one week. To prepare inocula, a mass of plectenchyma was homogenized with 1 liter of V8 liquid medium and diluted 10 times. Inoculum (20 ml per cell) was injected via autobullet into the soil in each cell in which a four-week-old seedling was grown. The inoculated plants were grown for a further 16 days in a greenhouse maintained at 25°C. Symptoms were scored visually on a scale of 0–4 (0=no symptoms; 1=leaf chlorosis; 2=leaf chlorosis and slightly necrotic crown; 3=necrotic crown and severe wilting; 4=almost dead). This resistance test was performed twice using nine plants per line per replication. The resistance to *P. capsici* for each DH line was estimated as the mean of the symptom score.

Scoring of DNA polymorphisms

AFLP analysis was carried out according to the method of Vos *et al*. (1995) with some modifications. The total DNA of each plant was digested with the restriction enzymes *Eco*RI and *Mse*I, ligated to the two adapters for *Eco*RI and *Mse*I cutting sites, and then preamplified with a pair of preselective primers for *Eco*RI and *Mse*I. The selective amplifications were performed using six *Eco*RI primers and eight *Mse*I primers, each with three additional nucleotides at the 3′ ends. The 5′ end of the *Eco*RI primer was labeled with D2-, D3- or D4-fluorescent dye (Proligo Japan KK, Kyoto, Japan). A mixture of the labeled PCR products and a molecular marker was loaded onto a Beckman CEQ 2000XL sequencer equipped with 33 cm capillaries (Beckman Coulter, Fullerton, CA). The resulting electrophoregram was analyzed using the CEQ 8000 genetic analysis system (Beckman Coulter).

Both the 626 SSR primer pairs previously reported by Minamiyama *et al*. (2006) and 36 of the 42 SSR primer pairs from the SNU2 map (Lee *et al*. 2004) were tested for polymorphisms between the parents 'Manganji' and 'CM334' using the modified post-PCR fluorescence-labeling method (Inazuka *et al*. 1996). Only the 120 primer pairs that detected reproducible polymorphisms were then analyzed using dye-labeled primers. The 5′ end of the forward primers was labeled with D2-, D3- or D4-fluorescent dye and then used to score polymorphisms in the segregating population. Electrophoresis of the PCR products and fragment analyses were carried out using the same method as that for the AFLP analysis.

Linkage map construction and QTL analysis

Linkage analysis was performed using JoinMap 3.0 software with a population type code, DH1 (Van Ooijen and Voorrips 2001). LOD scores less than 5.5 resulted in the loss of many markers and there was an insufficient number of linkage markers in the calculated map after grouping. Therefore, the LGs were separated using a LOD score of 6.0. Recombination values were converted to genetic distances using the Kosambi mapping function (Kosambi 1944). Interval QTL mapping was carried out with the software MapQTL 4.0 (Van Ooijen *et al*. 2002). The significance LOD thresholds for QTLs computed by permutation test (1000 permutations) were 2.8 in the first test and 2.7 in the second test.

Results

Phenotyping

Wilting symptoms were observed in the susceptible parent and a few DH lines at 5 days after inoculation (DAI). The symptoms developed gradually by 14 DAI, and then reached a plateau. Therefore the inoculated plants were scored based on the disease reaction at 14 DAI. The phenotypic distribution using the means of the symptom score for each test is shown in Fig. 1. The susceptible parent was severely infected and almost died in both tests. In contrast, in the case of 'CM334' the symptoms were very mild (first test) or absent (second test). Continuous distribution of the DH lines was observed in both tests. Among 87 DH lines, 10 lines showed a symptom score of more than 3.5 and 26 showed no symptoms in the first test. As a result, the distribution appeared to be a bimodal curve, indicating that a major genetic factor, as well as a few minor factors, controlled this resistance. On the other hand, symptoms in the second

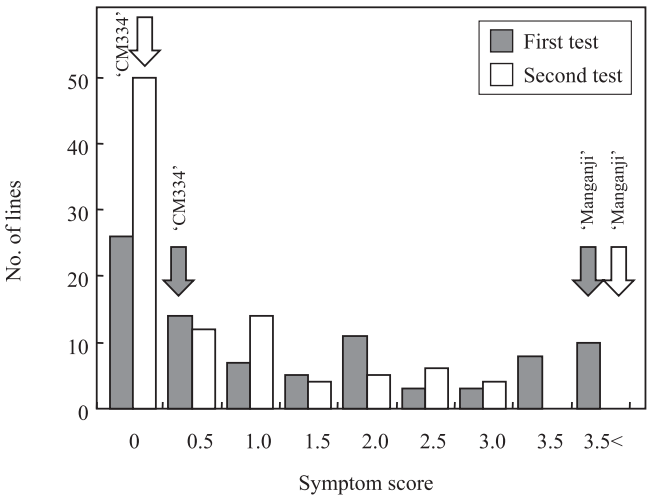

Fig. 1. Frequency distribution of symptom scores for *P. capsici* in the DH population derived from the F_1 of 'Manganji' × 'Criollo de Morelos 334'. The scores of the parental lines were 4.0 and 3.7 for 'Manganji' (n = 9) in the first and second tests, respectively, and 0.4 and 0 for 'CM334' (n = 9).

test were somewhat mild, and the DH lines did not show a high susceptibility (the mean score was 3.5–4.0). The susceptible parent showed an average value of 3.7.

Genetic map

Among the 99 SSR markers previously used for mapping, 59 primer pairs detected polymorphism in the parentage and were used for mapping in the present study. In addition, 48 primer pairs were newly selected as polymorphic primer pairs from the SSR-enriched libraries, while 13 SSRs from the SNU2 map (Lee *et al*. 2004) were also mapped. Among a total of 120 primer pairs, 116 pairs amplified single loci and four pairs detected more than two loci. As a result, 120 primer pairs detected a total of 126 loci.

Consequently, we analyzed 126 SSR loci along with 243 AFLP markers and constructed a linkage map. The total map length was 878 cM including 16 LGs with a LOD score of 6.0. The average distance between the markers in our map was 2.4 cM. We mapped 118 of the 126 SSR markers scored, and the remaining eight were independent.

QTL analysis

Two QTLs were detected on LG3 and LG15 in the first test. The QTL on LG15 showed the largest effect and explained 58.1% of the phenotypic variation in the resistance to *P. capsici* (Table 1 and Fig. 2). The flanking marker of this QTL was an SSR marker, CAMS420. This QTL was also detected as a major QTL in the second test. LOD score profiles in both tests were very similar. In addition, seven SSR markers were located within 21 cM intervals from the peak of this QTL. In contrast, the QTL on LG3 was detected with small effects in the first test, and accounted for 16.8% of the total variance. The LOD score of this QTL was 2.28, which was below the threshold of 2.7, in the second test. However, the LOD profiles in this region were similar in both tests. The nearest marker was a dominant AFLP marker, but the QTL was surrounded by eight SSR markers within a distance of 10 cM. The combination of these two QTLs on LG3 and LG15 explained 74.9% and 50.7% of the total variation in the first and second tests, respectively. The sequence of the SSR primers mapped on LG3 and LG15 is shown in Table 2.

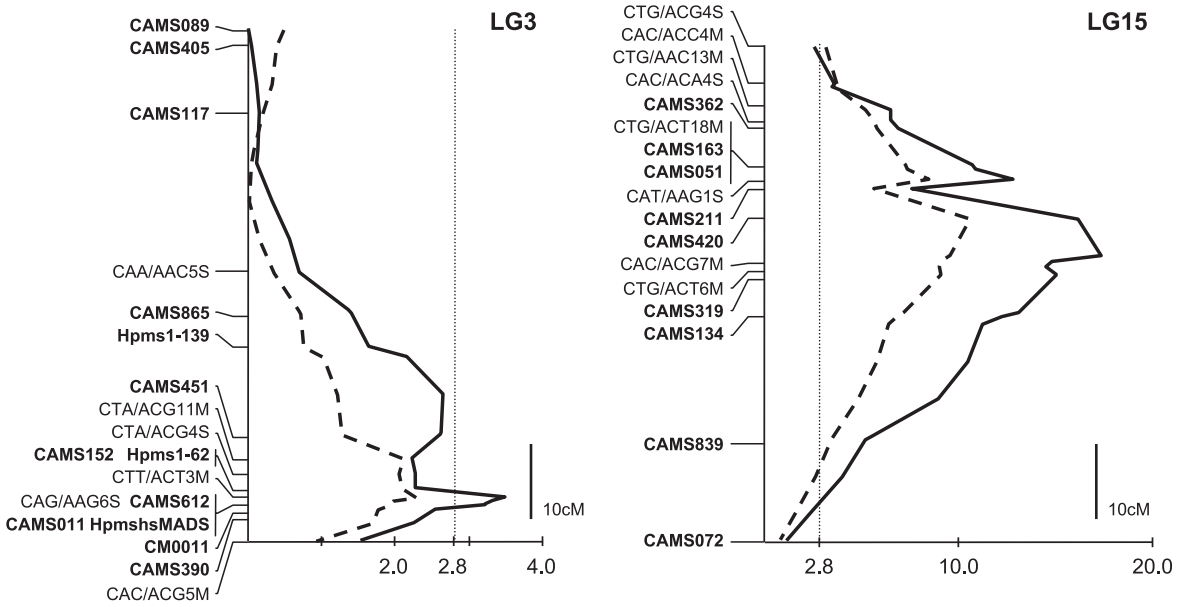

Fig. 2. LOD score plot on LG3 and LG15 for the QTLs of the resistance to *P. capsici* in the DH population derived from anther culture of F_1 of 'Manganji' × 'Criollo de Morelos 334'. The *solid* and *broken lines* indicate the first and second tests, respectively. The SSR markers are indicated in bold characters on the *y*-axis. The *vertical dotted line* indicates the LOD threshold.

Table 1. QTLs detected for the resistance to *P. capsici* in the DH population

Marker[1)]	LG	Position[2)]	LOD		R[2,3)]		Additive[4)]	
			1st[5)]	2nd	1st	2nd	1st	2nd
CAMS420	15	23.1	16.13	10.59	58.1	40.2	1.05	0.52
CTT/ACT3M	3	62.0	3.48	2.28	16.8	10.5	0.56	0.26

[1)] The marker on or in the vicinity of the LOD score peak.
[2)] Position of the marker in the linkage group (in cM).
[3)] Percentage of phenotypic variation explained.
[4)] Additive effect of QTLs of the 'CM334' allele.
[5)] Number of inoculation tests.

Table 2. Primer sequences of pepper SSR markers mapped around QTLs for the resistance to *P. capsici*

LG	Marker	Position[1)]	Forward primer (5′ to 3′)	Reverse primer (5′ to 3′)	Repeat motif	Source
3	CAMS089	0.0	aacagcgctgatcctttacc	caacatcacagtggcagaaga	(tc)19	Minamiyama *et al*. (2006)
	CAMS405	2.2	ttcttgggtcccacactttc	aggttgaaaggagggcaata	(tc)18	Minamiyama *et al*. (2006)
	CAMS117	11.0	ttgtggaggaaacaagcaaa	cctcagcccaggagacataa	(tg)21(ta)3	Minamiyama *et al*. (2006)
	CAMS865	37.6	agaaatcgtggttgggtgag	cactttggcacattttgctg	(gaa)7	Minamiyama *et al*. (2006)
	Hpms1-139	42.0	ccaacagtaggacccgaaaatcc	atgaaggctactgctgcgatcc	(ct)2(ag)15	Lee *et al*. (2004)
	CAMS451	53.6	tgcattggtgggctaacata	gctcttgacacaaccccaat	(tc)21t(ac)3	Minamiyama *et al*. (2006)
	Hpms1-62	60.7	catgaggtctcgcatgatttcac	ggagaaggaccatgtactgcagag	(tg)23(ag)9	Lee *et al*. (2004)
	CAMS152	60.8	cggattagagggtgaatgct	aattccctttcccgttatgg	(ac)15a(ta)8	Present study
	CAMS612	62.5	tccaccatgaatcgaagaca	agtcgcatcctgtccaaagt	(taa)16	Present study
	CAMS011	62.9	gggttatcaaatggccgata	attccctttcccagcattta	(ca)3(ac)11a(ta)3	Present study
	HpmshsMADS	62.9	tgctttcaaaacaatttgcatgg	gcgtctaatgcaaaacacacattac	(at)17	Lee *et al*. (2004)
	CM0011	63.6	tctgctttaaaaacacatacat	cattctaactgaaattgcatg	(ac)5(ta)8	Lee *et al*. (2004)
	CAMS390	65.4	ctgttctcctccctccctct	tgaagcaagaaactgaacaatca	(ag)19	Present study
15	CAMS362	10.9	ccccttctgaccttgattga	tatgcccctcctgtgatagc	(tc)9	Present study
	CAMS051	16.4	acccagttccctttcttggt	gaaggttagcggaatgaacg	(gt)3a(ta)4(tg)11	Minamiyama *et al*. (2006)
	CAMS163	16.4	tccatatagcccgtgtgtga	gcgtgggaatacaatgctaga	(at)7(gt)14	Minamiyama *et al*. (2006)
	CAMS211	19.0	cgtgggtgccttcttatgtt	atcgtccggacatggttagt	(tg)7	Present study
	CAMS420	23.1	cagcgttctatcgtctcaaatg	ttgacaaaccagaaattgatcg	(tc)5ca(tc)4	Minamiyama *et al*. (2006)
	CAMS319	30.7	tcaccttccacagcatcaag	caaacgcaaacaccaatcag	(tc)20	Present study
	CAMS134	36.3	atatggttcggcttcgttct	catcaatttggggcatctct	(ac)18	Present study
	CAMS839	52.8	gcaagcacatcatgctgaat	cgagcgcattattgaagtga	(tct)16	Present study
	CAMS072	66.3	cccgcgaaatcaaggtaat	aaagctattgctactgggttcg	(ac)13	Minamiyama *et al*. (2006)

[1)] Position of the marker in the linkage group (in cM).

Discussion

In the present study, the results of the root inoculation test indicated the existence of QTL (s) with a large effect on LG15. The LOD profile was somewhat broad. Although the profile was largely affected by the limited number of families used in the present study, the possibility of the presence of two tightly linked genes could not be ruled out. The present results were in agreement with those in previous reports, in which the resistance of 'CM334' to *P. capsici* showed the presence of one QTL or of two QTLs with large effects on the same LG. Thabuis *et al*. (2003) reported the existence of two closely linked QTLs, *rri5.1* and *rri5.2*, on a LG on chromosome P5. Phenotypic variation explained by the QTL (s) exceeded 60%. These QTLs were renamed *Phyto5.1* and *Phyto5.2*, respectively (Thabuis *et al*. 2004). Ogundiwin *et al*. (2005) detected a QTL, *Mr-5*, in LG JC5 on chromosome 5 and stated that this QTL corresponded to *Phyto5.1* and *Phyto5.2*. Direct correspondence of the present QTL to those described in previous studies is difficult to confirm because of the lack of common linkage markers. However, Quirin *et al*. (2005) have recently reported a sequence-characterized amplified region (SCAR) marker for *Phyto5.2*. The SCAR marker was mapped at nearly the same locus as that indicated by the CAMS051 and CAMS163 markers in LG5 in our previous map based on another population (Minamiyama *et al*. 2006) (data not shown). The locus should be located between CAMS211 and CAMS362 in the present LG15, because of the common markers. Therefore, the present largest QTL in LG15 may correspond to *Phyto5.2*, and may possibly include *Phyto5.1*.

In previous studies, the presence of additional QTL (s) with minor effects was reported (Thabuis *et al*. 2003, Ogundiwin *et al*. 2005). The QTL detected in LG3 in the first test, which exerted a minor effect, was not detected in the second test. However, the LOD score profile in LG3 was similar in the two tests, where a peak occurred. The expression of resistance to *P. capsici* is markedly influenced by

environmental factors, such as the age of the inoculated plant seedlings, temperature and soil moisture level (Thabuis *et al*. 2003, Ogundiwin *et al*. 2005). The DH lines did not show severe symptoms in the second test (Fig. 1). Since some of the environmental factors could have affected the results, it is possible that the expression of this QTL may be largely affected by environmental conditions. As no other regions showed conspicuous peaks in the QTL analysis, this region may harbor a QTL for Phytophthora rot resistance. However, the correspondence of this locus to those descriptions in previous studies should be studied in detail.

One of the major difficulties in the application of MAS by breeders is the use of complex techniques for DNA analysis. Since the RFLP technique is very fundamental, but requires a large amount of DNA and complex procedures, it is not readily accepted by breeders. However, in most of the mapping studies on *Capsicum*, RFLP markers that originated from tomato were used as common markers (Lefebvre *et al*. 1995, Livingstone *et al*. 1999, Ben Chaim *et al*. 2001, Kang *et al*. 2001, Thabuis *et al*. 2003). The other difficulty in pepper breeding is the low polymorphism among the lines of *C. annuum* (Minamiyama *et al*. 2006). The reported polymorphism is sometimes not detected in the breeding lines (Minamiyama *et al*. 2005). Although SSR is considered to be a highly polymorphic marker in general (Goldstein and Schlötterer 1999), the average polymorphism information content value of SSR was not so high in the *C. annuum* lines (Minamiyama *et al*. 2006).

In the previous studies, the information about the QTLs for resistance to *P. capsici* was supplied by RFLP or AFLP markers. Recently in a few studies, PCR-based common markers have been used (Lee *et al*. 2004, Minamiyama *et al*. 2006). However, mapping of the SSR markers by Lee *et al.* (2004) was based on an interspecific cross between *C. annuum* and *C. chinense*. The level of polymorphism may considerably decrease in crosses between lines of *C. annuum*. Based on an intraspecific cross, Ogundiwin *et al*. (2005) tried to map QTLs for resistance to *P. capsici* using the SSR markers developed by Lee *et al.* (2004). However, they could not detect a sufficient number of SSR markers linked to the QTLs. Moreover, the SCAR marker reported by Quirin *et al*. (2005) did not show polymorphism in our parentage.

In the present study, we obtained a number of SSR markers linked to the two QTLs detected for resistance to *P. capsici* based on an intraspecific crossing in *C. annuum*. The linkage markers reported here may widen the choice in breeding programs for Phytophthora rot-resistant pepper cultivars.

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