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Genetic mapping of quantitative resistance to race 5 of *Pseudomonas syringae* pv. *phaseolicola* in common bean

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Abstract Halo-blight is an important worldwide bacterial disease of common bean (*Phaseolus vulgaris* L.) caused by *Pseudomonas syringae* pv. *phaseolicola*. Nine races of the pathogen and five race-specific resistance genes have been previously described. However, a quantitative response to this pathogen has also been described. The objective of this study was to identify halo-blight resistance loci linked to molecular markers that could be used in resistance breeding. Chromosomal regions related to race 5 halo-blight resistance were localized on a genetic map of RAPD and AFLP molecular markers and constructed by the analysis of a ''Jules'' × ''Canela'' F_2 progeny. ''Jules'' shows quantitative resistance to halo-blight and ''Canela'' is a very appreciated but susceptible Spanish bean landrace. Two QTL for resistance to halo-blight were mapped in two linkage groups. There were four large groups, with 14–22 molecular markers each, five with 4–8 markers each, and three with 2 or 3 markers each.

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Abreviations
MAS Marker-assisted selection
Psp Pseudomonas syringae pv. phaseolicola

Introduction

Halo-blight is a seed-borne bacterial disease that infects common bean worldwide. It is caused by *Pseudomonas syringae* pv. *phaseolicola* (hereafter *Psp*) and causes significant reduction in yield and seed quality. In the regions where *Psp* is common and when the weather conditions are favourable to the development of the bacteria, pod and seed production can be almost completely lost. Genetic variability of *Psp* has been observed in different parts of the world where beans are grown, and nine races of the pathogen have been characterized based on their interaction with eight differential bean cultivars (Taylor et al. 1996a).

A gene for gene interaction drives the bean hypersensitive reaction when bean plants are

infected by *Psp*, and a set of five resistance genes are combined in the differential cultivars (Taylor et al. 1996a, 1996b). Nonetheless, the existence of isolates have been reported whose race could not be established (Abi et al. 2000; López et al. 2003). Frequently several races can be present simultaneously (López et al. 2003), making inefficient the use of varieties carrying individual race-specific genes. Likewise, the predominant races in a particular locality can change from year to year (Asensio 1995). Due to this variability and the plant-pathogen co-evolution, genetic resistance may not be as efficient as desired and it has been suggested that a combination of race specific and race non-specific resistance could provide an effective strategy for establishing durable resistance (Taylor et al. 1996b). In addition to the qualitative resistance to this pathogen, quantitative resistance has also been described, depending on the source of resistance used.

Race-specific resistance genes to halo-blight have been mapped on chromosomes 4 and 10 (Fourie et al. 2004; Freyre et al. 1998; Pedrosa et al. 2003). To date few data have been published regarding the chromosomal location of the quantitative loci for halo-blight resistance (Ariyarathne et al. 1999; Fourie et al. 2004). Therefore, the identification of quantitative trait loci (QTL) linked to molecular markers is of great importance, not only for bean, but for other related crops, since a conservation of linkage groups has been clearly proved (Jung et al. 1999; Kelly et al. 2003). The development of molecular markers that help to saturate genetic maps is a prerequisite to marker-assisted selection (MAS) and to clone genes of agronomic interest, such as resistance genes. Thus, every effort in this research line can be useful.

The aims of this work were to identify RAPD and AFLP markers useful for MAS, and genetic mapping of halo-blight resistance related QTL in a ''Jules'' × ''Canela'' F_2 population. The work is a part of a larger research project aimed at introducing halo-blight resistance in several susceptible Spanish landraces that are appreciated for their culinary quality and high economic value.

Materials and methods

Plant materials

A total of 103 *Phaseolus vulgaris* L. individuals from the F_2 progeny of the cross ''Jules'' × ''Canela'' were used for studying resistance to *Psp*. Only 86 progeny individuals were available for QTL mapping. ''Jules'' is a resistant white cultivar of Mesoamerican origin obtained from ''Great Northern'' #1 sel.27 (Coyne and Schuster 1970), which in turn was derived from an interspecific cross between *P. vulgaris* and *P. acutifolius*. This last species was source of *Psp* resistance. ''Canela'' is a traditional Spanish variety of Andean origin with creamy beans. Although it is highly appreciated in Spain, it is susceptible to *Psp*.

Seeds were surface sterilized and then grown in a chamber under a photoperiod of 16 h light and 21 ± 1°C in pots filed with a sterilized mixture of sand and commercial substrate (50% v/v).

Halo-blight

This study used an isolate of *Psp* race 5 (isolate 1375) kindly donated by Dr. J.D. Taylor (Horticultural Research International, Warwick, UK). Cultures were grown in liquid King's B medium (King et al. 1954) for 3 days at 25°C in an orbital shaker (250 rpm). Aliquots standardized at 10^8 cfu ml^{-1} were used to inoculate primary leaves and immature pods. The two primary leaves of the seedlings and at least two immature pods per plant were inoculated.

Seedlings of 7–10 days old with fully expanded primary leaves were inoculated on both sides of the midrib by spraying the bacteria suspension on the lower epidermis with the aid of an aerograph (De Vilbiss N°15) at a distance of 10 cm from the surface. This covered an area of between 5 and 10 mm^2. After the inoculation the plants were kept at 100% humidity for 2 days and then at 80% of humidity until the infection degree was scored.

Pods were detached when they reached between 1/2 and 3/4 of their final size, and immediately inoculated and stored in a humid container until symptoms evaluation. Pods were

inoculated by puncturing the epidermis with a toothpick previously soaked in the bacteria suspension. Since pod inoculation allows more than one puncturing on each pod, sterile distilled water was used for control inoculation.

Symptoms were observed 10 days after the inoculation in both leaves and pods. The susceptibility level was evaluated on a five-level scale as by Innes et al. (1984), as follows: (1) red-brown necrotic reaction in the area of maximum inoculation (highly resistant); (2) red-brown necrotic reaction with a trace of water-soaking (resistant); (3) some necrosis but more extensive water-soaking largely confined to the area of maximum inoculation (slightly susceptible); (4) small water-soaked lesions (<1 mm diameter) distributed over the leaf surface (susceptible); (5) larger water-soaked lesions (>1 mm diameter) distributed over the leaf surface (highly susceptible).

Molecular markers

Genomic DNA was prepared from fresh trifoliate leaves of 2-week old plants using the method described by Dellaporta et al. (1983) with minor modifications.

Randomly amplified polymorphic DNA (RAPD) were obtained using 10-mer primers from Operon (Operon Technologies, Alameda USA). Amplification reactions contained 10 ng/µl DNA, 5 pmol/µl of primer, 2.5 mM dNTPs (Amersham), 2 U *Taq* polimerasa (PROMEGA Co. Ltd.), 10 mM Tris–HCl pH 8.3, 25 mM $MgCl_2$ and 50 mM KCl in a final volume of 25 µl. PCR reactions were performed by denaturing the DNA for 1 min at 94°C, followed by 45 cycles of 1 min at 94°C, 1 min at 36°C, and 2 min at 72°C, in a Perkin Elmer 2400 GeneAmp PCR System. PCR products were separated in 1.5% p/v agarose gels in TAE buffer and visualized by ethidium bromide staining. RAPD markers were named with the OP letters followed by their primer serial identification and the size of the amplified polymorphic band.

The amplified fragment length polymorphism (AFLP) analysis was performed as described in the AFLP Plant Mapping Kit Protocol (Applied Biosystems). Fluorescence labelled primers for *Eco*RI/*Mse*I restriction enzymes were used, along with three selective nucleotides. The products of the amplifications were separated by capillary electrophoresis on an ABI Prism 310 Genetic Analyser (Applied Biosystems) and the fluorescence was analyzed with the manufacturer software. The AFLP marker nomenclature followed a general rule: the first three letters represent the *Eco*RI + 3 selective nucleotides; the second three letters represent the *Mse*I + 3 selective nucleotides; and the number identifies the size of the polymorphic amplified fragment for a given primer pair.

Data analysis and QTL mapping

For every RAPD and AFLP marker band, presence versus absence was scored, thus all markers were dominant. In order to test the 3:1 goodness-of-fit the chi-square test was used. Only markers fitting the 3:1 segregation (not distorted markers) were included in the analysis. This analysis includes 27 RAPD and 114 AFLP markers. The genetic linkage map was constructed using MAPMAKER v3.0 (Lander et al. 1987) based on a LOD score > 3.0 and a recombination threshold of 0.33. The Kosambi function (Kosambi 1944) was used to estimate the map distance from recombination frequencies. Linkage groups were determined by the ''group'' command and numbered with Roman numbers. The order within each linkage group was resolved by the ''compare'' command, and additional markers were subsequently added by the ''try'' command. The best order of the markers was then verified by the ''ripple'' command. The location of QTL for halo-blight-disease resistance and the association between molecular markers and QTL were carried out using the method of interval mapping using MAPMAKER/QTL v. 1.1 (Lincoln et al. 1993). The LOD score threshold was set at 3.0 and the chromosomal location of QTL was scanned at every 2 cM intervals. The confidence interval for each QTL was fixed around the maximum probability position including the region for a tenfold reduction of probability (Patterson et al. 1991).

Results and discussion

Resistance-susceptibility segregation analysis

The first analysis was conducted to check the genetic control of the resistance-susceptibility to race 5 of *Psp* in the 103 individuals of the ''Jules'' × ''Canela'' F_2 population. Race 5 was selected since it is one of the most frequent in Leon (Spain) and surrounding areas (González et al. 2003). No differences were observed between the responses obtained from leaves and pods from the same individual. The distribution among the five susceptibility categories described in materials and methods was: category 1 (highly resistant), 8 individuals; category 2 (resistant) 22 individuals; 3 (slightly susceptible), 55; 4 (susceptible), 14; 5 (highly susceptible), 4.

The data from the ''Jules'' × ''Canela'' F_2 showed a phenotype distribution among the five classes, with many individuals in the intermediate classes, supporting the suitability of the QTL analysis. Taylor et al. (1996b) suggested that ''Jules'' has quantitative resistance to halo-blight without race specificity. Alternatively resistance to *Psp* has been described as monogenic dominant or recessive depending upon the maternal parent in crosses where the paternal parent was ''Jules'' or ''Great Northern'' #1 sel. 27 (origin of ''Jules'') (Coyne and Schuster 1970; Hill et al. 1972; Asensio et al. 1993). These paradoxical results could rely on the analytical procedure and/or the material used. Since sometimes data have been pooled into resistant versus susceptible classes only one gene for resistance have been described (Asensio et al. 1993; Fourie et al. 2004). This would be the case if our data are pooled into the same two classes. In another study, genes responsible for hypersensitive reaction as well as genes for partial resistance have been suggested (Ariyarathne et al. 1999).

Linkage analysis and QTL detection

A total of 141 RAPD and AFLP markers which fitted the 3:1 segregation were used to construct the genetic map. Among them, 103 were included in 12 linkage groups (with LOD score >3) while 38 were not grouped. The distribution of markers in the linkage groups was uneven ranging from 22 in group III to only 2 in groups XI and XII (Table 1 and Fig. 1). There were four large groups, with 14–22 molecular markers each, five with 4–8 markers each, and three with 2 or 3 markers each. The average distance between adjacent markers was 21.60 cM and the total map length was 1,983.6 cM, which was higher than the 1,226 cM of the core map (Freyre et al. 1998), and the 1,720 cM of the microsatellite map of Blair et al. (2003). When the LOD score threshold was reduced to 2.9 in order to look for group aggregation, groups XI and XII joined together in a group with 4 molecular markers. This changed the number of linkage groups to 11, consistent with the *P. vulgaris* haploid chromosome number. When the LOD score was reduced to 2.5, none of the other groups were joined nor new markers were assigned. Some clustering of markers from the same parent were observed in the linkage groups (Fig. 1).

Vallejos et al. (1992) previously observed non-uniform marker distribution among linkage groups and a general tendency to cluster for markers coming from each parental cultivar. These disparities in the distribution of markers have been attributed to different factors, in particular to the use of populations from crosses between Mesoamerican and Andean genotypes. As these authors suggest, differences in the recombination frequencies throughout the genome may account for different marker density among several chromosome segments. Additionally they also propose that densely populated chromosome segments could correspond to genome regions that are variable between the two gene pools of beans, whereas sparsely populated segments may carry more conserved sequences.

Another general event between maps is the close vicinity of markers of the same type, i.e., RAPD, AFLP, etc. In the genetic map described in this work, there is a tendency for RAPDs to form clusters (see Fig. 1, linkage group I) even though they are much less abundant than AFLP markers. Freyre et al. (1998) argue that there is a tendency for genes coding for proteins to cluster in some regions and unspecific sequences to cluster in another regions. Our markers also followed a clustering pattern despite all of them being unspecific.

Table 1 Molecular marker distribution on the linkage groups

Linkage group	No. of markers		Genetic distance (cM)
	RAPDs	AFLPs	
I	8	10	314.4
II	–	4	61.9
III	2	20	525.7
IV	3	11	214.9
V	–	6	102.3
VI	–	4	60.7
VII	5	9	369.4
VIII	1	5	121.0
IX	–	3	55.9
X	2	6	114.4
XI[a]	–	2	25.0
XII[a]	–	2	18.0
Unlinked	6	32	
Total	27	114	1,983.6

[a]Indicate groups that join at LOD = 2.9

Several types of molecular markers, such as RAPD, AFLP, SSR, ISSR and RFLP, have been used to obtain linkage maps in *Phaseolus vulgaris* (Adam-Blondon et al. 1994; Ariyarathne et al. 1999; Blair et al. 2003; Freyre et al. 1998; Jung et al. 1996, 1997; Nodari et al. 1993; Pedrosa et al. 2003; Santos et al. 2003; Tar'an et al. 2002; Vallejos et al. 1992). RAPD are the most commonly employed markers in this set of papers, probably due to their low cost and easy handling. We used several RAPD and AFLP primers in common with other maps but unfortunately markers which were located previously did not segregate in our F_2. This circumstance only depends on the parental genotypes. Although a number of common primers were selected from previous papers for working with, the same polymorphic bands were not segregating, and therefore, no common markers could be included, which meant that the alignment of our map with others was not possible.

Breeding for resistance to halo-blight is an imperative in many parts of the world. Until now five resistance genes have been identified (Taylor 1996a, 1996b) based on response to infection in differential bean lines. Additionally, a quantitative and race non-specific response to halo-blight has been described in the common bean (Taylor 1996b). Therefore, it has been argued that both major and minor genes are conditioning halo-blight resistance in beans. Thus, the identification of QTL related to this disease resistance is one of the goals for breeding programs. In this work, two QTL were located in groups VII (HB-QTL1) and IX (HB-QTL2), respectively (Fig. 1). HB-QTL1 behaved recessively and was located in the region between ACT/CAA-397 and OPG12-1000 markers, and HB-QTL2 behaved as additive and was located in between AGG/CAT-130 and AGG/CAT-178. Thus, both QTL were linked to at least one AFLP marker which could have a good reproducibility in any laboratory. The amount of phenotypic variation simultaneously explained by the two QTL determined using the multiple QTL model was 69%.

Since ''Jules'' has been described to show a quantitative race non-specific resistance to halo-blight (Taylor 1996b), it was possible that two QTL were implicated. There were other chromosomal regions for which some association with resistance may be attributed, however, only the two principal peaks were considered. This was done to avoid any noise that could be related to the low density of the map. It is known that even with densely saturated maps it is difficult to accurately resolve QTL located on the same linkage group (Young 1996).

Fourie et al. (2004) have described in the B4 linkage group the gene *Pse*-4 which confers resistance to race 5 and a gene cluster that contain three genes responsible for resistance to races 1, 7 and 9 of the halo-blight. Since these authors grouped phenotypes into two classes (resistant versus susceptible) perhaps this locus *Pse*-4 could

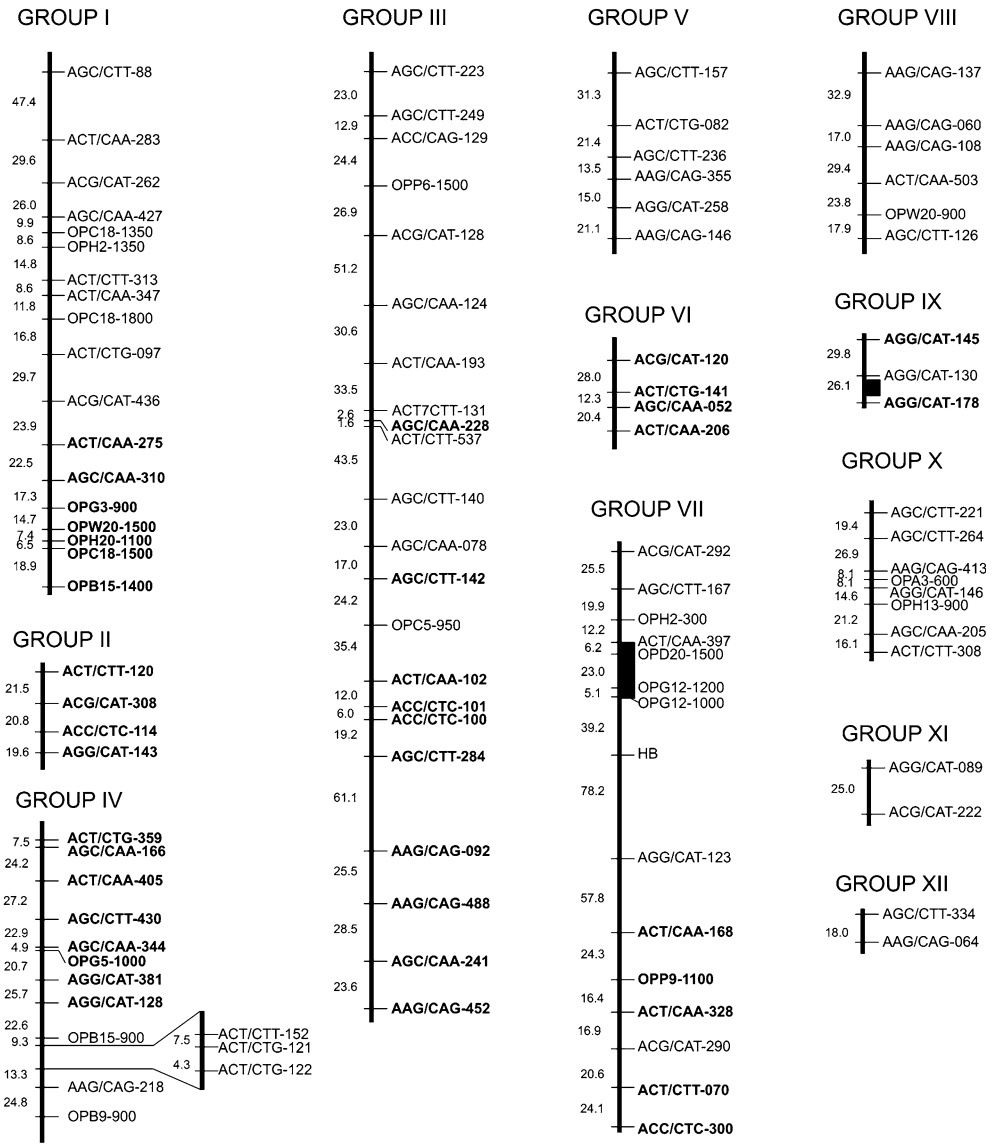

Fig. 1 Genetic map of *Phaseolus vulgaris* L. obtained by using RAPD and AFLP markers. Linkage group numbers are arbitrary, without known relation to common-bean chromosomes. Marker names are indicated on the right and the genetic distance in cM between them on the left. The most likely locations of the QTL related to resistance are indicated by the boxes to the right of groups VII and IX. RAPD markers are named beginning with OP followed by their primer serial identification and the size of the amplified polymorphic band, and AFLP markers are named with six letters and a number: the first three letters represent the *Eco*RI + 3 selective nucleotides; the second three letters represent the *Mse*I + 3 selective nucleotides; and the number identifies the size of the polymorphic band. Markers from ''Jules'' are written in bold letters, the others are from ''Canela''

be equivalent to the locus that we would described if the same grouping were made from the present data. This would result in a locus assigned to the VII linkage group (*HB* in Fig. 1). In the map this locus would be included within the LOD 2.0 interval confidence of the QTL, so it is likely that both approaches would indicate the same locus, in a similar way to the results of Ariyarathne et al. (1999) for the locus *Hb*-7 and the QTL HB 83 located in the quoted B4 linkage group. Therefore, it could be possible that group B4 in these articles corresponds to our VII group.

Although the present data are not enough to prove it, if this correspondence were to be true, this would concur with the observation of Fourie et al. (2004) that certain chromosomal regions accumulate resistance genes and QTL, and/or some genes could confer race-specific resistance to one race and quantitative resistance to another. Putative clusters of disease and pest resistance genes and QTL have been shown in several bean chromosomes (Kelly et al. 2003). It is interesting that genes conditioning for a hypersensitive response to different races of *P. syringae* are in the same linkage group, and linked to resistance genes for other diseases such as the *I* gene for BCMNV as noted Fourie et al. (2004).

In summary, we have developed a bean map based on RAPD and AFLP markers. The map includes two QTL controlling the resistance to halo-blight. However, more markers must be added to connect this map with others previously developed. Since associations between molecular markers and QTL controlling traits in beans have been proven to be maintained among different populations, the identification of these QTL can help in MAS for resistance to this pathogen in the future.

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