Inheritance of Partial Resistance Against *Colletotrichum lindemuthianum* in *Phaseolus vulgaris* and Co-localization of Quantitative Trait Loci with Genes Involved in Specific Resistance

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Anthracnose, one of the most important diseases of common bean (*Phaseolus vulgaris*), is caused by the fungus *Colletotrichum lindemuthianum*. A "candidate gene" approach was used to map anthracnose resistance quantitative trait loci (QTL). Candidate genes included genes for both pathogen recognition (resistance genes and resistance gene analogs [RGAs]) and general plant defense (defense response genes). Two strains of *C. lindemuthianum*, identified in a world collection of 177 strains, displayed a reproducible and differential aggressiveness toward BAT93 and JaloEEP558, two parental lines of *P. vulgaris* representing the two major gene pools of this crop. A reliable test was developed to score partial resistance in aerial organs of the plant (stem, leaf, petiole) under controlled growth chamber conditions. BAT93 was more resistant than Jalo-EEP558 regardless of the organ or strain tested. With a recombinant inbred line (RIL) population derived from a cross between these two parental lines, 10 QTL were located on a genetic map harboring 143 markers, including known defense response genes, anthracnose-specific resistance genes, and RGAs. Eight of the QTL displayed isolate specificity. Two were co-localized with known defense genes (phenylalanine ammonia-lyase and hydroxyproline-rich glycoprotein) and three with anthracnose-specific resistance genes and/or RGAs. Interestingly, two QTL, with different allelic contribution, mapped on linkage group B4 in a 5.0 cM interval containing Andean and Mesoamerican specific resistance genes against *C. lindemuthianum* and 11 polymorphic fragments revealed with a RGA probe. The possible relationship between genes underlying specific and partial resistance is discussed.

Additional keywords: residual effect.

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Anthracnose, caused by the specialized hemibiotrophic fungus *Colletotrichum lindemuthianum,* is one of the most important diseases of common bean (*Phaseolus vulgaris* L.) throughout the world (Pastor-Corrales and Tu 1989). Disease outbreaks generally originate from contaminated seeds or infected plant debris (Dillard and Cobb 1993; Tu 1981). Up to now, breeding for anthracnose resistance has focused on monogenic resistance systems. Several dominant anthracnose resistance genes, recently renamed *Co-1* to *Co-9,* have been described in common bean (Alzate-Marin et al. 1997; Kelly and Young 1996; Young et al. 1998; Geffroy et al. 1999). However, the use of these specific resistance genes has not provided durable resistance, especially in Latin America, the area of origin of common bean (reviewed by Gepts 1998). An explanation for the rapid breakdown of specific resistance genes relies on the extensive pathogenic diversity displayed by *C. lindemuthianum,* as evidenced by the numerous races described in the literature (Kelly et al. 1994; Pastor-Corrales and Tu 1989). Therefore, a different management of specific resistance genes and/or a different type of resistance need to be considered, mainly when farmers cannot afford the purchase of pathogen-free seeds or fungicides.

Two major gene pools, known as the Andean (South America) and Mesoamerican (Mexico and Central America) gene pools, have been identified for cultivated common bean (reviewed by Gepts 1998). Pyramiding of Andean and Mesoamerican specific resistance genes in a single cultivar has been proposed as a way to achieve durable resistance in this crop (Kelly 1995). Alternatively, genes providing partial resistance have been shown in other crops to play an important role in durable resistance, as defined by Johnson (1984). For instance, in rice, the durable resistance of the cultivar Moroberekan against rice blast was shown to result from a combination of both partial and complete resistance genes (Wang et al. 1994). However, for anthracnose in common bean, the implementation of this strategy has been limited by the lack of information concerning the existence of partial resistance.

In this paper, we present evidence for partial resistance against anthracnose and investigate its genetic basis in three different organs (stem, leaf, petiole). After selection of two appropriate strains and development of the methodology required to efficiently measure partial resistance, genetic factors involved in anthracnose quantitative resistance were mapped with the BAT93 × JaloEEP558 recombinant inbred line (RIL) population, previously used to set up an integrated linkage map of common bean (Freyre et al. 1998). A comparison of the position of anthracnose quantitative trait loci (QTL) involved in the partial control of the two different strains reveals that the QTL are mainly isolate specific and that some colocalize with cloned defense genes, resistance gene analogs (RGAs), and anthracnose-specific resistance genes.

RESULTS

Disease resistance tests.

Because partial resistance against anthracnose in *P. vulgaris* had never been scored before, a search for appropriate strains was carried out that did not reveal any specific resistance genes in both BAT93 and JaloEEP558, the two parental lines of the RIL population used to set the integrated genetic map of common bean (Freyre et al. 1998). In a collection of 177 strains, two strains of *C. lindemuthianum* (45, A7) displayed measurable and differential symptoms on the two parental

Fig. 1. Quantitative severity scale used to score percentage of stem presenting symptoms. A score of 1 = no observed symptoms; 6 = 100% of organ covered by brown typical lesions of anthracnose.

lines. Quantitative resistance against these two strains was measured on stem, leaf, and petiole, on a 1 to 6 scale severity, under controlled growth chamber conditions (Fig. 1). The parental line BAT93 was partially resistant, displaying an average score of 1.44 to 2.86, while JaloEEP558, with scores between 3.56 and 5.33, was highly susceptible to both strains 45 and A7 on stem, leaf, and petiole (Table 1). The reaction of the RILs revealed transgressive segregation in both directions when compared with the parents, regardless of the organ or strain analyzed (data not shown). Resistance scores for strain 45 showed a continuous but bimodal distribution from resistant (1) to highly susceptible (6) regardless of the organ tested. The involvement of a major genetic factor for partial resistance to strain 45 would explain such behavior. Inoculation with strain A7 led to an almost normal distribution of resistance scores, indicating a more complex genetic determinism. The analysis of variance among the RIL progeny showed highly significant genetic variation for all traits ($P < 0.0001$), demonstrating a genetic origin for the different levels of resistance in the RIL population. The genetic variation for all traits was highly heritable, with heritabilities (h^2) ranging from 0.90 to 0.98 (Table 1).

Phenotypic and genetic correlations were calculated to investigate relationships between characters (Table 2). For a given strain, there was a strong correlation ($P < 0.001$) between the different organs, especially stem and petiole (0.96 for strain 45, 0.84 for strain A7; Table 2). This was in sharp contrast to the absence of correlation (phenotypic and genetic) when the data regarding the two strains were compared (Table 2). These observations supported the hypothesis that the genes responsible for the resistance against each strain were distinct but that reactions against the same strain in different organs were conditioned by the same genes.

Table 2. Phenotypic (above diagonal) and genetic (below diagonal) correlation coefficients among resistance to strain 45 on stem (S45), leaf (L45), petiole (P45) and resistance to strain A7 on stem (SA7), leaf (LA7), petiole (PA7)[a]

	Traits					
Traits	**S45**	**L45**	**P45**	**SA7**	**LA7**	**PA7**
S45		0.89***	0.96***	0.16	–0.06	0.11
L45	0.90		0.90***	0.04	–0.10	0.01
P45	0.97	0.91		0.06	–0.14	0.05
SA7	0.16	0.00	0.06		0.74***	0.84***
LA7	–0.07	–0.12	–0.14	0.76		0.81***
PA7	0.12	–0.03	0.05	0.88	0.85	

[a] *** = $P < 0.001$.

Table 1. Estimates of means and heritabilities from parent lines (BAT93 and JaloEEP558) and RILs lines for resistance to the strains 45 and A7 on stem, leaf, and petiole

	Strain 45			Strain A7		
Means and heritabilities	**Stem**	**Leaf**	**Petiole**	**Stem**	**Leaf**	**Petiole**
Means[a]						
BAT93	1.56 ± 0.18	2.86 ± 0.23	1.60 ± 0.16	1.44 ± 0.21	2.77 ± 0.34	1.55 ± 0.21
JaloEEP558	5.33 ± 0.23	4.15 ± 0.18	4.97 ± 0.24	4.37 ± 0.25	3.56 ± 0.37	4.05 ± 0.35
Recombinant inbred lines (RILs)	3.42 ± 0.12	4.02 ± 0.12	3.52 ± 0.13	2.37 ± 0.09	3.22 ± 0.09	2.62 ± 0.09
Heritability						
h^2	0.98	0.98	0.98	0.90	0.96	0.91
90% C.I.[b]	(0.97, 0.99)	(0.97, 0.99)	(0.97, 0.99)	(0.87, 0.92)	(0.95, 0.97)	(0.89, 0.94)

[a] Standard errors are attached.

[b] Confidence intervals (C.I.) of h^2 were calculated by the method of Knapp et al. (1985).

Linkage map.

The integrated linkage map established in the BAT93 × JaloEEP558 RIL population has been presented in detail elsewhere (Freyre et al. 1998). The present BAT93 × JaloEEP558 RIL map was established with a subset of the available markers (Freyre et al. 1998) and 35 additional random amplified polymorphic DNA (RAPD) markers significantly ordered at an LOD threshold of 2. One hundred forty-three markers were mapped on 11 linkage groups spanning a map distance of 894 centimorgans (cM) Kosambi, which represented one marker every 6 cM on average (Fig. 2). Of the 143 markers, 17 loci corresponding to genes of known function were spread among linkage groups B2 (*PvPR-2*, *Pgip*, *ChS-3*, *Cel*, *Grp1.8-1*, *ChS-1*), B3 (*Grp1.8-3*, *PvPR-1*), B5 (*Lox*), B6 (*Hsp70*), B7 (*Hrgp36*, *ChI-2*, *ChI-1*, *Per*), B9 (*Gluc*, *Ch*), and B10 (*Vpe-3*). Four markers corresponding to RGAs were also mapped on linkage groups B4 (*PRLJ1ha.a*, *PRLJ1hn.b*) (Geffroy et al. 1999) and B11 (*PvH20ST*, *PvH20NSa*) (Geffroy et al. 1998). The latter region is known by comparative mapping to contain the *Co-2* specific resistance gene, present neither in BAT93 nor in JaloEEP558 (Freyre et al. 1998; Geffroy et al. 1998). Finally, three anthracnose-specific resistance genes present in either the JaloEEP558 Andean parent (*Co-y* and *Co-z* on B4) or the BAT93 Mesoamerican parent (*Co-9* on B4), but overcome by strains A7 and 45, were also included (Fig. 2). *Co-y* and *Co-9* were previously identified as alleles of the same locus (Geffroy et al. 1999).

Mapping QTL for anthracnose resistance.

At the LOD = 2.0 threshold level, regression analysis with PLABQTL (Utz and Melchinger 1996) revealed 10 putative QTL involved in anthracnose resistance toward both strains (Tables 3 and 4, Fig. 2). All these putative QTL were confirmed by one-way analysis of variance ($P < 0.005$) and nonparametric rank sum test (Wilcoxon test) ($P < 0.01$). This is fully consistent with the fact that a type I error = 0.005 used in single factor analysis corresponds to an LOD = 1.77, according to the formula given by Champoux et al. (1995). Because the three methods of analysis provided similar results, we will focus on the results obtained with PLABQTL, which provides a more accurate estimation of the location of the putative QTL.

Strain 45: Six putative QTL were identified for resistance to strain 45 on linkage groups B2, B3, B4 (two QTL), B7, and B11. A QTL with major effect associated with resistance in stem, leaf, and petiole was detected at the end of linkage group B4 (Table 3, Fig. 2). In this genomic region, the LOD score value reached a maximum on the *Co-y/Co-9* anthracnose-specific resistance genes (Fig. 3). The phenotypic variation explained by this QTL (R^2) was 72% for stem, 71% for leaf, and 76% for petiole, and the resistance-increasing allele came from BAT93 (Table 3). The large effect of this QTL is consistent with the bimodal distribution of resistance scores among the RILs of the BAT93 × JaloEEP558 population. Five QTL explaining a smaller fraction of the phenotypic variation
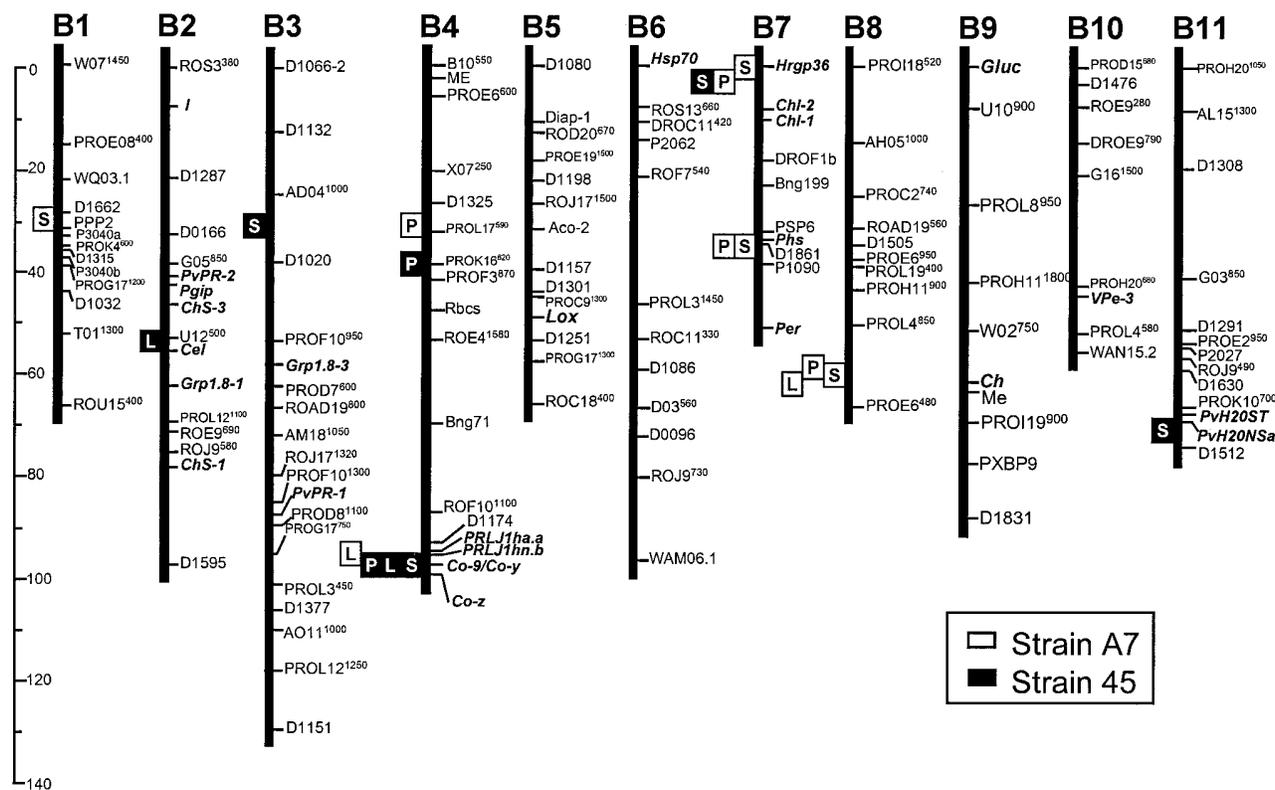

Fig. 2. BAT93 × JaloEEP558 recombinant inbred lines (RILs) linkage map showing locations of markers used. Vertical lines: bean linkage groups (from B1 to B11); designations to the right represent names of markers. A scale in centimorgans is shown on left. Filled and empty rectangles: maximum LOD score locations of quantitative trait loci (QTL) associated with resistance to strain 45 and A7, respectively. Letter inside rectangle indicates the organ in which the QTL is effective: S (stem), P (petiole), L (leaf). Anthracnose-specific resistance genes and markers revealed with probes corresponding to defense genes or resistance gene analogs (RGAs) are indicated in bold italics.

(R^2 values ranging from 11 to 16%) were also identified (Table 3). These QTL were organ specific (Table 3). Three of them, positioned on linkage groups B3, B7, and B11, were found to have significant effects on stem resistance, one QTL on B2 was associated with leaf resistance and the remaining QTL on the middle of B4 was involved in petiole resistance. For the QTL on B4, B7, and B11, the resistant alleles were contributed by BAT93, whereas for the two QTL located on B2 and B3, the alleles for increased resistance were inherited from the susceptible parent JaloEEP558. The total variation explained by the QTL detected varied from 76% for leaf reaction to 82% for stem reaction.

Strain A7: Six genomic regions were found to be associated with resistance to strain A7 with R^2 values ranging from 12 to 32% (Table 4). The absence of any major QTL is consistent with the unimodal distribution of resistance scores among RILs in the BAT93 × JaloEEP558 population. A QTL on B8 was found to have significant effects on resistance in all three plant organs (Table 4, Fig. 2). Two different genomic regions on B7 (close to *Hrgp36* and close to *D1861*) were associated with both stem and petiole resistance. Finally, organ-specific

QTL were identified on linkage group B1 (stem resistance), in the middle of B4 (petiole resistance), and at the end of B4 (leaf resistance). BAT93 alleles were associated with resistance for all these QTL with two exceptions on B8 and at the end of B4, for which the favorable alleles were contributed by JaloEEP558. Collectively, the QTL detected for stem, leaf, and petiole resistance accounted for 46, 48, and 45% of the phenotypic variation, respectively (Table 4).

The locations of the QTL for resistance against the two different strains of *C. lindemuthianum* tested were different in most cases (Tables 3 and 4, Fig. 2), in agreement with the absence of correlation between the resistance reactions against the two strains (Table 2). Only two QTL located in the middle of B4 and on B7 (close to *Hrgp36*) were associated with resistance against both strains (Tables 3 and 4, Fig. 2). At the end of linkage group B4, two tightly linked intervals were involved in leaf resistance against strain A7 and in stem, leaf, and petiole resistance against strain 45 (Figs. 2 and 3). For the QTL involved in leaf resistance against strain A7, the LOD score value reached a maximum on the *PRLJ1hn.b* RGA marker (Fig. 3). These two regions had opposite allele effects

Table 3. Biometrical parameters for regions of the bean genome associated with resistance to strain 45, on stem, leaf, and petiole, in the recombinant inbred lines (RILs) progeny derived from the cross between BAT93 and JaloEEP558

| | | Strain 45 | | | | | | | | | | | |
| | | Stem | | | | Leaf | | | | Petiole | | | |
LG[a]	Marker interval	Position[b]	LOD	R^{2c}	Allele[d]	Position	LOD	R^2	Allele	Position	LOD	R^2	Allele
B2	U12[500]–*Cel*	–[e]	–	–	–	47-53-55	2.44	0.13	J	–	–	–	–
B3	AD04[1000]–D1020	21-31-49	3.03	0.16	J	–	–	–	–	–	–	–	–
B4	PROL17[590]–PROK16[620]	–	–	–	–	–	–	–	–	35-39-42	2.07	0.11	B
	Co-y–Co-z	96-97-98	41.85	0.72	B	96-97-98	39.75	0.71	B	96-97-98	52.81	0.76	B
B7	*Hrgp36–ChI-2*	0-3-30	2.48	0.13	B	–	–	–	–	–	–	–	–
B11	PvH20NSa–D1512	67-72-74	2.11	0.12	B	–	–	–	–	–	–	–	–
Total[c]				0.82				0.76				0.78	

[a] Linkage group.

[b] Positions of likelihood peak (maximum LOD) with underscored numbers plus support interval in Kosambi centimorgans relative to the first marker on linkage group (Fig. 2). The support interval corresponds to the region in which the LOD score remains within 1.0 unit of the peak. Results obtained with PLABQTL (Utz and Melchinger 1996).

[c] R^2 or the proportion of the phenotypic variance explained by individual quantitative trait loci (QTL), or by all multiple QTL acting together (Total), results obtained with PLAPQTL (Utz and Melchinger 1996).

[d] B or J indicates that the BAT93 or the JaloEEP558 allele increase the resistance, respectively.

[e] Not statistically significant.

Table 4. Biometrical parameters for regions of the bean genome associated with resistance to strain A7, on stem, leaf, and petiole, in the recombinant inbred lines (RILs) progeny derived from the cross between BAT93 and JaloEEP558

| | | Strain A7 | | | | | | | | | | | |
| | | Stem | | | | Leaf | | | | Petiole | | | |
LG[a]	Marker interval	Position[b]	LOD	R^{2c}	Allele[d]	Position	LOD	R^2	Allele	Position	LOD	R^2	Allele
B1	D1662–PPP2	20-27-40	5.35	0.25	B	–	–	–	–	–	–	–	–
B4	D1325–PROL17[590]	–[e]	–	–	–	–	–	–	–	23-32-37	2.18	0.12	B
	PRLJ1hn.b–*Co-y*	–	–	–	–	92-95-97	4.01	0.20	J	–	–	–	–
B7	*Hrgp36–ChI-2*	0-0-4	4.25	0.21	B	–	–	–	–	0-3-9	3.38	0.17	B
	D1861–P1090	27-35-39	2.32	0.13	B	–	–	–	–	32-35-38	5.06	0.24	B
B8	PROL4[850]–PROE6[480]	48-60-66	2.17	0.12	J	57-62-66	7.55	0.32	J	49-58-66	3.16	0.16	J
Total[c]				0.46				0.48				0.45	

[a] Linkage group.

[b] Positions of likelihood peak (maximum LOD) with underscored numbers plus support interval in Kosambi centimorgans relative to the first marker on linkage group (Fig. 2). The support interval corresponds to the region in which the LOD score remains within 1.0 unit of the peak. Results obtained with PLABQTL (Utz and Melchinger 1996).

[c] R^2 or the proportion of the phenotypic variance explained by individual QTL, or by all multiple QTL acting together (Total), results obtained with PLAPQTL (Utz and Melchinger 1996).

[d] B or J indicates that the BAT93 or the JaloEEP558 allele increase the resistance, respectively.

[e] Not statistically significant.

(Tables 3 and 4), suggesting the existence of at least two different linked QTL.

DISCUSSION

Our findings raise issues regarding the inheritance of quantitative resistance to anthracnose and its relationship with durable resistance, the co-localization of QTL with resistance and defense genes, and the molecular basis of partial resistance.

Of the 177 strains tested, only four were virulent on the two parental lines. Two of these (strains 45 and A7) showed differential aggressiveness toward the two parents. Double susceptibility is therefore quite rare; this may be explained by co-evolution between host and pathogens taking place in common bean (Geffroy et al. 1999; Gepts and Bliss 1985; Guzmán et al. 1995) and the large genetic distance between the two host genotypes, each of which represents a different gene pool of *P. vulgaris* (Nodari et al. 1992). If we assume that this interaction is controlled by a gene-for-gene system, the limited number of strains that are virulent toward the two parents can be explained by multiple anthracnose resistance specificities distributed differentially between the two host gene pools. This was already suggested from pathogen population studies (Sicard et al. 1997b) and was recently confirmed by cross-inoculation experiments (Geffroy et al. 1999).

Genotype BAT93 was much less susceptible, regardless of the organ or strain tested, than JaloEEP558. However, positive QTL alleles for anthracnose resistance were identified in the two parental lines, explaining the transgressive segregations observed in the RIL progeny. Unfortunately, the limited number of these RILs has certainly prevented the detection of minor QTL. The overall level of BAT93 resistance against strain A7 was equivalent to BAT93 resistance against strain 45. Nevertheless, the data showed that distinct QTL were generally involved in the control of each of these two strains (Fig. 2). Of 10 putative QTL (Tables 3 and 4), only two, located near the *Hrgp36* defense gene (B7) and near the marker *PROL17* (B4), appeared to be involved in the interactions with both strains. The eight remaining QTL displayed isolate specificity. At one end of linkage group B4, two closely linked intervals displayed different allelic reactions toward each strain, and were therefore considered to be distinct QTL. Similarly, isolate-specific QTL were described for resistance against *Phytophthora infestans* in potato (Leonards-Schippers et al. 1994), *Pseudomonas solanacearum* and *Heterodera glycines* in tomato (Young 1996), potyviruses in pepper (Caranta et al. 1997), and *Puccinia hordei* in barley (Qi et al. 1999), and to a lesser extent for resistance against *Xanthomonas oryzae* pv. *oryzae* in rice (Li et al. 1999). In the present study, only two *C. lindemuthianum* strains were evaluated; therefore, further QTL mapping involving more strains of *C. lindemuthianum* will be necessary to see to what extent these QTL are isolate specific. Furthermore, a major-effect QTL against strain 45 explaining around 70% of the phenotypic variance was detected at one end of B4 (Table 3). It is questionable to call such a major-effect QTL a "QTL." However, involvement of a major QTL is a common feature when mapping QTL for disease resistance (reviewed by Young 1996; Keller et al. 1999). As previously noticed by Nelson (1981), this result shows that the distinction between polygenic and monogenic resistance is

not a definitive issue of black and white, but rather a *continuum* of shades of gray.

Although the genetic basis of durable resistance in plants is not understood (Johnson 1981), it is frequently presumed that quantitative resistance, conditioned by "minor" genes and supposed to act in a race-nonspecific manner, would provide durable resistance (Vanderplank 1968). Taken together, our data (major-effect QTL and strain-specific QTL) raised some important questions in relation to the durability of a resistance governed by such genetic factors. First, the existence of isolate-specific QTL will certainly complicate the definition and the marker-assisted construction of an ideotype capable of controlling the complete spectrum of pathogenic diversity. Second, deployment of cultivars protected by partial resistance amounts to selection for aggressiveness (Burdon 1993). If resistance mechanisms involve multiple genetic factors, each of which explains a limited fraction of the phenotype, a slow erosion of partial resistance should be linked to a step-wise increase in aggressiveness toward each component (Burdon 1993). However, the existence of major-effect QTL challenges this idea. Whether these major effects resulted from a single gene or from a battery of closely linked genes will not be solved until the appropriate genes have been cloned and their precise phenotype obtained by complementation.

The present data seem to suggest the existence of specialized QTL acting in specific organs. For example, on linkage groups B3 and B11, significant QTL against strain 45 were only found for the stem. However, graphical representations of the LOD value along linkage group B3 displayed strictly parallel curves for the three organs when infected by strain 45 (Fig. 4). This was in agreement with their strong genetic cor-
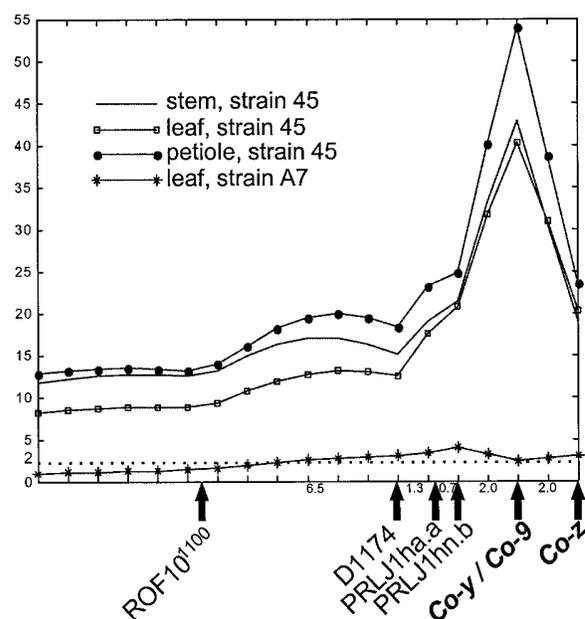

Fig. 3. PlabQTL scans of one end of linkage group B4 for resistance in stem, leaf, and petiole against strain 45 and for resistance in leaf against strain A7. Vertical axis: LOD score values; horizontal axis: position of markers and their map distances in Kosambi centimorgans (cM). Distance between tick marks is 1 cM on horizontal axis. Horizontal dotted line at LOD = 2 indicates significance level for declaration of quantitative trait locus (QTL).

relation. Nevertheless, only the stem resistance reached the LOD threshold of 2, necessary to accept the existence of a QTL (Fig. 4). As emphasized by Freymark et al. (1993), non-significance in statistical terms might not denote insignificance in biological terms. When looking at each graph the same observation was true except for the QTL against strain 45 on B7 (close to *Hrgp36*), which appears to have no effect on leaf resistance (data not shown).

With regard to co-localization of these QTL with previously mapped resistance or defense genes, markers *D1020* (B3), *D1861* (B7), and *D1512* (B11), shown in this study to be linked to partial anthracnose resistance, were also associated with common bacterial blight (CBB) resistance detected in a previous study of CBB QTL (Nodari et al. 1993b). Pleiotropic or closely linked genes might thus explain resistance against both anthracnose and CBB. Interestingly, this region on B11 is known by comparative mapping to contain the *Co-2* anthracnose-specific resistance gene expressed neither in BAT93 nor in JaloEEP558 (Freyre et al. 1998; Geffroy et al. 1998). Furthermore, a family of 9 leucine-rich repeat (LRR) sequences has been located in this region on the BAT93 × JaloEEP558 RIL map (Geffroy et al. 1998). In fact, one of the significant markers associated with this QTL was *PvH20NSa,* a member of this LRR family. The presence of both quantitative and qualitative resistance genes on the same genomic region opens the possibility that QTL might correspond to allelic versions of qualitative resistance genes with intermediate phenotypes. This hypothesis is coherent with arguments relating to quantitative traits where mutant phenotypes are thought to be extreme alleles of a QTL (Beavis et al. 1991; Robertson 1989).

The end of linkage group B4 merits particular comment for its high density of resistance genes and RGAs. In a region spannin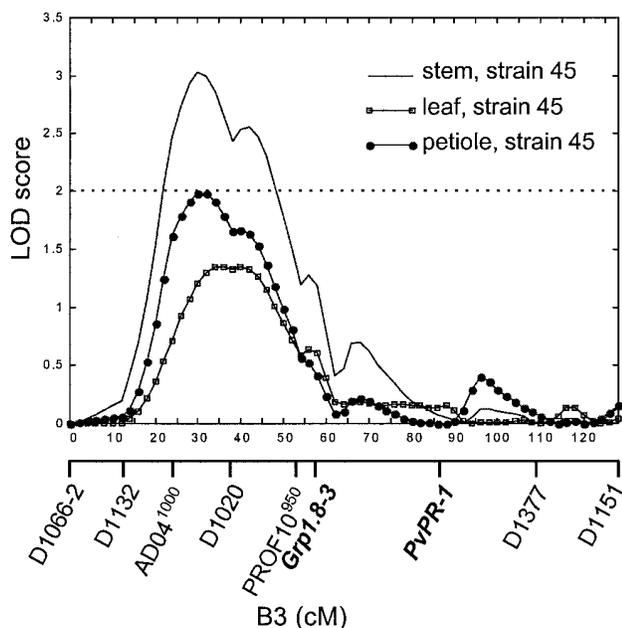g less than 5 cM, three specific resistance genes for anthracnose were clustered, which originated either from the Mesoamerican BAT93 parent (*Co-9*) or the Andean Jalo-EEP558 parent (*Co-x, Co-y*). In addition, 11 restriction fragment length polymorphisms (RFLPs), revealed with an RGA clone, which displays homology with the nucleotide binding site (NBS) domain of recently cloned specific resistance genes, also mapped to this region (Geffroy et al. 1999) (Fig. 5). Interestingly, a major-effect QTL against strain 45 (for leaf, stem, and petiole resistance) and a reverse-effect QTL (i.e., coming from the susceptible Andean JaloEEP558 parent) for leaf resistance against strain A7 were also located in this region (Fig. 3). For the QTL against strain 45, the LOD score value reached a maximum on the *Co-y/Co-9* anthracnose-specific resistance genes, while for the QTL against strain A7 the maximum was observed on the *PRLJ1hn.b* RGA marker (Figs. 3 and 5). It was proposed many years ago that major resistance genes, once overcome by a strain of the pathogen, might conserve some residual effect (Martin and Ellingboe 1976; Nass et al. 1981; Nelson 1978; Pedersen and Leath 1988). The co-localization between QTL and the "defeated" specific resistance genes localized on linkage group B4 is in agreement with this hypothesis.

Specific resistance genes cloned from several plant species share striking structural similarities at the molecular level despite their interaction with a diversity of pathogen species (Hammond-Kosack and Jones 1997). Genetic and molecular
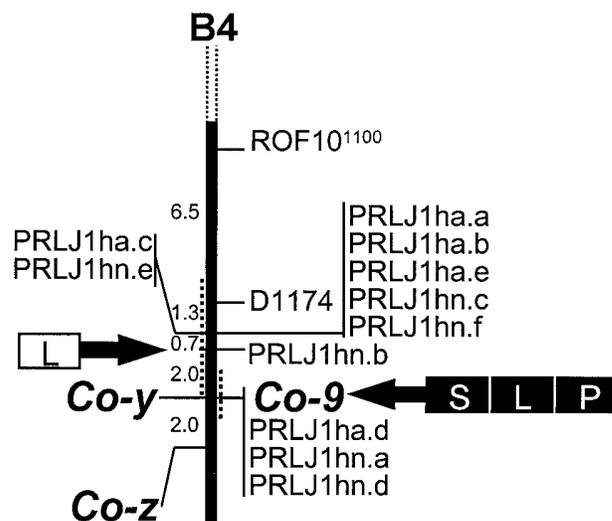

Fig. 5. Complexity of linkage group B4 in terms of anthracnose resistance. PRLJ1 is a DNA clone from JaloEEP558 displaying homology with the nucleotide binding site (NBS) of recently cloned specific resistance genes (Hammond-Kosack and Jones 1997). PRLJ1hn.a to f and PRLJ1ha.a to e correspond to polymorphic bands revealed in Southern blot analysis with PRLJ1 as a radiolabeled probe on DNA digested with either *Hin*dIII (PRLJ1hn) or *Hae*III (PRLJ1ha) (Geffroy et al. 1999). PRLJ1 polymorphic bands revealed in BAT93, the *Co-9* Mesoamerican anthracnose specificity and quantitative trait loci (QTL) for which BAT93 allele increase the resistance are mentioned on the right of linkage group B4. Reciprocally, PRLJ1 polymorphic bands revealed in Jalo-EEP558, the Andean anthracnose specificities (*Co-y* and *Co-z*) and QTL for which JaloEEP558 allele increase the resistance are on the left of linkage group B4. Arrows: most probable locations of QTL on linkage group B4 defined with PLABQTL. Motifs to represent QTL: identical to Figure 2. Support interval of each QTL (region in which LOD score remains within 1.0 unit of the peak) is represented by a dotted line beside linkage group. Distance in Kosambi centimorgans (cM) is shown to left.

Fig. 4. PlabQTL scans of linkage group B3 for resistance in stem, leaf, and petiole against strain 45. Vertical axis: LOD score values; horizontal axis: position of markers and their map distances in Kosambi centimorgans (cM) on linkage group B3. Horizontal dotted line at LOD = 2 indicates significance level for declaration of quantitative trait locus (QTL).

studies have shown that major genes are often members of large, multigene families located at complex loci composed of linked and evolutionarily related resistance specificities (Crute and Pink 1996; Parniske et al. 1997; Pryor and Ellis 1993; Ronald 1998). In rice, the *Xa21* resistance gene against *Xanthomonas oryzae* pv. *oryzae* (Song et al. 1995), encoding a putative receptor kinase-like protein carrying an LRR motif in its presumed extracellular domain, was reported as member of a multigene family including at least seven members within a 230-kb genomic region (Song et al. 1997). Transposition, recombination, and duplication were shown to contribute to the genetic diversity of the *Xa21* resistance gene family (Song et al. 1997). *Xa21* conferred complete resistance; however, another family member, designated *Xa21D,* encoding the extracellular LRR domain alone because of the insertion of a retrotransposon between the kinase and LRR domain, was found to confer only partial resistance with the same spectrum as *Xa21* (Wang et al. 1998). Similarly, insertion of the tranposable element *Ac* in the promoter region of the *M* rust resistance gene from flax resulted in partial resistance (Anderson et al. 1997). Furthermore, partial resistance to *Fusarium oxysporum* f. sp. *lycopersici* was observed in tomato plants transformed with a member of the multigene *I2C* family, referred to as *I2C-1,* cloned from the *I-2* locus that normally confers complete resistance (Ori et al. 1997). The *I2C-1* gene was shown to encode a protein containing an NBS motif and an LRR domain, characteristic of recently cloned specific resistance genes (Hammond-Kosack and Jones 1997). These three examples, in rice and tomato, showed that modifications of a complete resistance gene could give rise to a gene conferring partial resistance. Therefore, an alternative explanation to allelism relationship between specific and QTL resistance genes (Robertson 1989) or to the residual effect of "defeated" specific resistance genes against virulent strains (Li et al. 1999; Martin and Ellingboe 1976; Nass et al. 1981; Nelson 1978) is that major resistance genes and QTL located in the same genomic regions may correspond to different members within clusters of resistance gene families. This hypothesis is in agreement with the many examples of co-localization between QTL and specific resistance genes that have now been reported (Caranta et al. 1997; Freymark et al. 1993; Keller et al. 1999; Leonards-Schippers et al. 1994; Li et al. 1999; Roupe van der Voort et al. 1998; Wang et al. 1994). This suggests a potential structural and functional relationship between quantitative and qualitative resistance genes. Therefore, QTL located in genomic regions containing specific resistance genes might be involved in pathogen recognition. It is therefore plausible that allelic variants or different linked copies of the PRLJ1 RGA family located at the end of B4 confer either complete or partial resistance against anthracnose.

However, co-localization of QTL and specific resistance genes cannot be interpreted as a general rule. In our study, QTL were also detected in genomic regions containing functional genes involved in the defense response. A phenylalanine ammonia-lyase locus (*Pal-2*) has been located 3 cM under the D1662 marker on B1, in a BAT93 × JaloEEP558 F_2 map (Freyre et al. 1998; Nodari et al. 1993a). In our study, a QTL for stem resistance against strain A7 was mapped in this genomic region (Fig. 2). Another association between defense genes and QTL was detected on B7 near a hydroxyproline-rich glycoprotein locus (*Hrgp36*). PAL is an enzyme of the general phenylpropanoid metabolism and controls a key branch point in the biosynthetic pathways of flavonoid phytoalexins, which are antimicrobial compounds (Bowles 1990). Hydroxyproline-rich glycoproteins (HRGPs) are thought to play a key role in the organization of primary cell wall architecture and may act as the foci for the initiation of lignin polymerization and consequently may contribute to the formation of a structural barrier to pathogen invasion (Bowles 1990). Based on these biological properties, it is possible that allelic variants of *Pal-2* and *Hrgp36* might cause differences in quantitative resistance response to *C. lindemuthianum*. Other examples of co-localization between QTL and defense genes have been reported in various pathosystems (Ferreira et al. 1995; Giese et al. 1993; Leonards-Schippers et al. 1994). Therefore, there might be at least two types of resistance QTL, those located near specific resistance genes, which might be involved in pathogen recognition, and those located near defense genes, which might be involved in a general defense mechanism. This hypothesis is supported by the fact that all the QTL detected near RGA or specific resistance genes are isolate specific, while one of two QTL efficient against both isolates tested has been localized near the *Hrgp36* defense gene.

In plants, no QTL for disease resistance has yet been cloned. Gene sequences underlying QTL and sharing homologies with cloned specific resistance genes would represent a major insight in this field. As already mentioned in a companion paper, comparison of the complex locus on the end of B4 together in BAT93 and JaloEEP558 is currently underway in our laboratory to understand the evolutionary mechanisms by which this complex resistance locus has evolved in the geographically independent bean gene pools (Geffroy et al. 1999). The molecular analysis of this complex locus also offers prospects for understanding the potential structural relationship between genes underlying complete and partial specific resistance.

MATERIALS AND METHODS

Plant and fungal material.

An F_9 RIL population was derived from the cross between Andean landrace JaloEEP558 and Mesoamerican breeding line BAT93 at the University of California, Davis (Nodari et al. 1993a). These 77 RILs have been used to set up an integrated linkage map of common bean (Freyre et al. 1998).

A collection of 177 *C. lindemuthianum* strains, from the Orsay fungal library, was inoculated to the two parents BAT93 and JaloEEP558 (Geffroy et al. 1999). All these strains were known to attack the multi-susceptible cultivar La Victoire. Symptoms were scored on the aerial parts of the plants. Only four strains were pathogenic on both parents. Of these four strains, strain A7, isolated from wild beans in Argentina, and strain 45, isolated from cultivated beans in France, were chosen for the present study. Although the two strains attack both BAT93 and JaloEEP558, severity of symptoms on all tested organs was higher on JaloEEP558. Thus, BAT93 was considered to be displaying partial resistance against these two strains, compared with JaloEEP558.

Molecular data analysis.

The available BAT93 × JaloEEP558 RIL linkage map was used in this study (Freyre et al. 1998). Seventeen RFLP and

35 RAPD markers were added to the available markers. Plant genomic DNA extraction and RFLP analysis were carried out as described in Geffroy et al. (1998). Probes corresponding to either genes of known function or RGAs were analyzed with RFLP to investigate their potential association with partial resistance. Probes corresponding to genes of known function were a cellulase (*Cel;* Tucker and Milligan 1991), chalcone isomerase (*ChI;* Mehdy and Lamb 1987), chalcone synthase (*ChS;* Ryder et al. 1987), chitinase (*Ch;* Broglie et al. 1986), glucanase (*Gluc;* Edington et al. 1991), glycine-rich protein (*Grp1.8;* Keller et al. 1988), heat-shock protein (*Hsp70;* Vidal et al. 1993), lipoxygenase (*Lox;* Eiben and Slusarenko 1994), hydroxyproline-rich glycoprotein (*Hrgp36;* Corbin et al. 1987), two pathogenesis-related proteins (*PvPR-1* and *PvPR-2;* Walter et al. 1990), pectin esterase (*Vpe-3;* Ebbelaar et al. 1996), peroxidase (*Per;* J. C. F. De Oliveira, *personal communication*), polygalacturonase-inhibitor protein (*Pgip;* Toubart et al. 1992). Probes corresponding to RGAs were PvH20, a clone containing six imperfect LRRs (Geffroy et al. 1998), and PRLJ1, a DNA clone from JaloEEP558 displaying homology with the NBS of recently cloned specific resistance genes (Geffroy et al. 1999). RAPD assays were carried out as described in Adam-Blondon et al. (1994) with the following modifications: 3 ng of genomic DNA was used in a 25-µl reaction. Amplifications were performed in a GeneAmp PCR System 9600 (Perkin Elmer, Norwalk, CT) programmed for 40 cycles of 30 s at 92°C, 1 min at 42°C, and 1 min at 72°C. Loci corresponding to clear and segregating bands for each primer were named as follows: P(aris)R(APD)O(peron) followed by the primer serial name and the size of the band in base pairs (bp) in exponent: e.g., PROE8^{400}. Segregation data also included three anthracnose-specific resistance genes present in either BAT93 (*Co-9*) or JaloEEP558 (*Co-y*, *Co-z*) (Geffroy et al. 1999). The map was constructed with Mapmaker/exp v3.0 software (Lander et al. 1987). Linkage groups were established with an LOD threshold of 3.0 and a recombination fraction of 0.3. Marker order was estimated with an LOD threshold of 2.0 based on multipoint "Compare," "Order," and "Ripple" analyses. Genetic distances between markers were estimated with the Kosambi mapping function (Kosambi 1944).

Pathogenicity tests and disease scoring.

The experiment was set up as a randomized complete block design with four replicates. Each RIL genotype was represented by one plant in each block. The seeds were planted in individuals pots filled with moist vermiculite. Three independent replicates of this four-block experiment were carried out for each strain. In each block, two replicates of the parental lines BAT93 and JaloEEP558 and of the highly susceptible cultivar La Victoire were included as controls.

Seedlings were grown for 8 days at 23°C, 75% relative humidity, with 8 h dark and 16 h light photoperiods under fluorescent tubes (166 µE s^{-1} m^{-2}). The seedlings were infected by spray inoculation 8 days after germination and incubated at 19°C under the same light conditions as described above with 90% relative humidity in a control growth chamber. The preparation of inoculum was as described previously (Sicard et al. 1997a). The spore concentration was measured with a hemacytometer and adjusted to 10^6 spores per ml. Assessment of lesion development was performed 7 days after inoculation on leaves, stems, and petioles. This duration was shown to correspond to a little more than one life cycle of the pathogen. A 1 to 6 severity scale, based on visual appreciation of the percentage of the organ presenting symptoms, was used. A score of 1 represented no observed symptoms while 6 corresponded to 100% of the organ covered by brown typical lesions of anthracnose (Fig. 1). Acervuli, from which conidiospores were released, were observed for scores of 3 and above.

Statistical analysis.

Heritabilities were estimated as $\sigma_g^2 / (\sigma_g^2 + 1/_n \sigma_e^2)$ where σ_g^2 and σ_e^2 were the estimated genetic and residual variance, and n the number of individuals per line. The Pearson coefficient was calculated to determine phenotypic correlations among traits. Within one strain, genetic correlations between traits were assessed with the following formula:

$$\rho_{GxGy} = cov\ xy / \sqrt{cov\ xx . cov\ yy}$$

where x and y were the two traits under consideration, *cov xy* was the covariance of the two traits, *cov xx* and *cov yy* were the variance of each trait. For two traits measured with different strains, genetic correlations were estimated as follows:

$\rho_{GxGy} = \rho_{PxPy} / \sqrt{h_x^2 \cdot h_y^2}$, where ρ_{PxPy} was the phenotypic correlation coefficient between the two traits x and y, h_x^2 and h_y^2 were the heritabilities of each trait. The mean values of all replications for each of the six characters measured (two strains, three organs) were used in QTL analysis. To identify QTL, single factor analysis (one-way analysis of variance) was conducted with Proc GLM (Statistical Analysis System [SAS], 1989 SAS/STAT User's Guide, vers. 6, 4th ed.; SAS Institute, Cary, NC). A significance level of $P < 0.005$ was used to uncover putative association between a DNA marker and anthracnose resistance. Whenever significant marker-trait associations were detected at several linked marker loci, all of these associations were assumed to be detecting the presence of a single QTL. The proportion of the phenotypic variance explained by marker segregation was determined by the R^2 value. In order to estimate more accurately the chromosomal position of QTL, data were also analyzed with PLABQTL (Utz and Melchinger 1996), which employs the interval mapping approach (Lander and Botstein 1989). A putative QTL was inferred when the LOD exceeded 2.0. For each trait studied, this LOD threshold corresponds to a type I error = 25%, for the entire genome (Churchill and Doerge 1994). Finally, the detected QTL were confirmed with nonparametric Wilcoxon rank sum test ($P < 0.01$), with the NPAR1WAY procedure of SAS. If two significant genomic region affecting two different traits have overlapping support intervals (i.e., the region in which the LOD score remains within 1.0 unit of the peak) we declare that a single QTL was common to the two traits under consideration.

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