Mapping of Quantitative Trait Loci Controlling Resistance of *Brassica oleracea* to *Xanthomonas campestris* pv. *campestris* in the Field and Greenhouse

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ABSTRACT

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Despite the existence of resistant cultivars, little is known about the genetic control of resistance of *Brassica oleracea* to *Xanthomonas campestris* pv. *campestris*. Quantitative trait loci (QTL) controlling juvenile and adult plant reactions of *B. oleracea* to *X. campestris* pv. *campestris* were mapped using restriction fragment length polymorphism loci and disease ratings of F_3 families from a resistant cabbage × susceptible broccoli cross. In the greenhouse, 2.5-week-old, plants were screened by inoculating the pathogen directly into the veins and measuring the diseased leaf area. In the field, 11-week-old plants were screened by inoculating intact plants and assessing the symptoms using an interaction phenotype scale. Two genomic regions on linkage groups 1 and 9 were associated with both young and adult plant resistance. These regions also were associated with variation for petiole length. Two additional QTL on linkage group 2 that were associated only with young plant resistance were found. For one of these, alleles from the resistant parent contributed to greater susceptibility. These results suggest that plants selected for resistance based on screening young plants in the greenhouse should exhibit adult plant resistance.

Black rot of crucifers, caused by the vascular bacterium *Xanthomonas campestris* pv. *campestris*, is a worldwide economically important disease of crucifers (21). Control practices include the production of pathogen-free seed in regions less favorable for the spread of the pathogen, disinfestation of seeds with hot water, and the development of resistant cultivars. Disease resistance has been a main goal in breeding programs, especially for cultivars grown in the humid tropics and subtropics (33). However, little is known about the genetic control of host resistance. Resistance to black rot in *Brassica oleracea* L. has been reported as quantitative in nature, with varying numbers of genes involved (1,24,25,35).

Molecular markers can be used to study the genetic control of quantitative traits by establishing linkage associations between markers and quantitative trait loci (QTL). The identification of markers linked to QTL could be used to study the consistency of QTL effects across different environments and genetic backgrounds (2,3,13,23,28,32) and increase the frequency of favorable alleles during selection (7,19,29). Restriction fragment length polymorphisms (RFLPs) have been used as markers for identifying QTL controlling resistance to pathogens in various crops, such as maize (3), *B. oleracea* (9,17), *B. napus* (8), barley (10,27), common bean (22), mungbean (36), and potato (15). In addition, recent studies have investigated the consistency of marker-trait associations between greenhouse and field screenings and the allelic relationships of resistance genes (8,26,27,37).

In *B. oleracea*, seedlings or young plants with 2 to 3 primary leaves are often screened in greenhouses to select genotypes resistant to black rot. Correlation between adult plant and seedling resistance has been reported (1,12,25), but it is not known if the same genes control resistance at different growth stages. In this study, the number, map position, and magnitude of effects of QTL controlling adult plant and seedling reactions of *B. oleracea* to black rot were compared. Since our previous observations (*unpublished data*) suggested a correlation between leaf morphology and resistance, linkage relationships between genes for resistance to black rot and genes controlling two morphological traits and petiole length and leaf area also were investigated. The results are discussed in relation to breeding for black rot resistance.

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MATERIALS AND METHODS

Plant materials. The black rot-resistant cabbage line Badger Inbred-16 (BI-16; *B. oleracea* subsp. *capitata*) was crossed as the female parent to the susceptible inbred broccoli line OSU Cr-7 (*B. oleracea* subsp. *italica*). A single F_1 hybrid plant was bud self-pollinated after vernalization for 8 weeks at 4°C. Randomly selected F_2 plants were vernalized and bud self-pollinated to generate seed of F_3 families that was used to assess disease resistance in greenhouse and field trials. Selfed progeny of the parents, F_1 hybrid, and black rot-susceptible cabbage cultivars Titanic 90 (hybrid) and Golden Acre (open-pollinated) were included in both trials as controls.

Greenhouse trial. A total of 83 F_3 families and controls were screened in a greenhouse for black rot resistance using a randomized complete block design consisting of three blocks. Two blocks were evaluated simultaneously during March 1993 and the third block during April 1993. Plots consisted of 12 plants from each family, and the plot mean was the experimental unit. Seeds were sown in No. 812 Com-Pack trays (T. O. Plastics, Minneapolis, MN) filled with Jiffy mix (Jiffy Products of America, Chicago)

and germinated under continuous, very high output, fluorescent lights at 19 to 25°C. Plants were watered daily with H_2O, and fertilized every 2 days with half-strength Hoagland's solution. Inoculum of *X. campestris* pv. *campestris* isolate PHW 1205 from the Crucifer Genetics Cooperative (University of Wisconsin, Madison) was cultured in 100 ml of potato-dextrose broth (PDB) under constant agitation (250 rpm) for 48 h at 28°C and adjusted to a concentration of 10^8 cells per ml (34). Fully expanded primary leaves were inoculated 18 days after planting by clipping the tip of the leaf and rubbing a cotton swab saturated with bacterial suspension over the wounded site. All leaves other than the inoculated ones were removed prior to and 1 week after inoculation to promote leaf retention. Lesion area (square centimeters) was evaluated 14 days after inoculation by tracing the edges of individual lesions on an acetate transparency sheet and measuring the traced area with a leaf area meter (Li-Cor, Lincoln, NE). The mean diseased leaf area (DLA) for the three replicates was used for QTL analysis.

Field trial. A total of 92 F_3 families and controls (entries) were screened for black rot resistance in the field (including 76 of the F_3 families used in the greenhouse trial). Seedlings were grown as described above, and 5-week-old plants were hardened outdoors 2 weeks prior to transplanting. Two experiments were conducted during the summer of 1993 at the West Madison Agricultural Research Station of the University of Wisconsin. In each experiment, plots consisted of 12 plants from each entry arranged in rows with ~0.9-m spacing between plants within and between rows, and the plot mean was the experimental unit. One plot was grown for each entry. One experiment was inoculated as described below and was used to assess reactions to black rot. The second experiment was not inoculated and was used to measure petiole length and leaf area. Normal cultural practices were used throughout the trial. Inoculum was produced as described above, except that 1.8 liters of PDB was inoculated with 1 ml of a 12-h liquid culture of the pathogen and was incubated for 72 h under the same conditions. Plants were inoculated twice on consecutive days 1 month after transplanting by spraying the bacterial suspension using a backpack pesticide sprayer (Solo, Indianapolis, IN). Plants were inoculated between 6:00 and 7:00 a.m. when hydathodes were congested with water and gutation was extensive. Symptoms were scored 22 days after inoculation using an interaction phenotype (IP) rating scale (34) of 0 to 9 in increments of 1 (0 = no symptoms and 9 = rapidly progressing marginal lesions frequently coalescing to give scorched leaves, systemic invasion of the plant frequently accompanied by soft rot, and severe stunting or death of plant). Two infected leaves per plant, totaling 24 leaves per plot, were rated independently by two persons, and the mean IP of the two ratings was used to calculate the plot mean. For morphological measurements, one mature leaf from each plant of all families was collected 35 days after transplanting, and petiole length (centimeters) and leaf area (square centimeters) were measured. Plot means of IP scores, petiole length, and leaf area were used for QTL analysis.

RFLP and data analysis. A RFLP linkage map was constructed based on segregation data of 112 marker loci obtained from 124 F_2 plants (4). The map covered 1,002 centimorgans (cM), with an average marker interval of 9.9 cM. Prior to QTL analysis, the phenotypic distributions were tested for normality using the Shapiro-Wilk correlation test for normality, and the F_3 family and block effects for the black rot ratings in the greenhouse trial were tested for significance by analyses of variance (20). The trait means of the parents and controls were compared for all traits using standard errors calculated from individual plant values. Pearson product moment correlation coefficients were calculated among all traits analyzed.

QTL were mapped using the maximum likelihood method based on interval mapping (16). A $\log_{10}$ of the odds ratio (LOD) higher than 2.5 was considered evidence of the presence of a QTL within a marker interval. The computer program MAPMAKER/QTL 1.1 (18) was used for interval mapping analysis and also for investigating the mode of gene action as described by Paterson et al. (23). MAPMAKER/QTL 1.1 was used to calculate the LOD score of the unconstrained (free) genetic model for a particular QTL and the LOD scores of constrained (fixed) genetic models in which a particular type of gene action was assumed for the QTL (additive, dominant, or recessive in the case of an F_2 population). A $\Delta LOD \leq -1.0$ was used as a threshold to reject the type of gene action specified by the constrained model. However, estimations of gene action of QTL based on F_3 family means rather than on the phenotypic means of F_2 plants are less accurate due to an additional cycle of recombination and consequent decrease in overall heterozygosity, which reduces the ability to detect dominance deviations (23). Therefore, inferences of gene action reported here should be interpreted with caution.

RESULTS

Greenhouse evaluations. Inoculation of seedlings via wounding was highly effective in establishing the disease, judging by symptom expression on the susceptible parent and controls. Symptoms began appearing 5 days after inoculation as a water-soaked lesion around the inoculation site. On susceptible plants, lesions enlarged as they progressed toward the midrib, resulting in typical chlorotic, V-shaped lesions. On resistant plants, lesions were restricted in size and were often associated with a small necrotic edge surrounding the point of inoculation.

The mean DLA of the susceptible control Titanic 90 differed significantly from all other controls. Also, the overall mean DLA of the two parents differed significantly, but no significant difference was found among the susceptible parent, the F_1 hybrid, and the susceptible control Golden Acre (Table 1). The mean DLA of F_3 families was distributed normally($P < 0.05$; Fig. 1), with some

TABLE 1. Means and standard errors for *Brassica oleracea* parents (Badger Inbred-16 [BI-16] cabbage and OSU Cr-7 broccoli), F_1 plants, and susceptible controls (cabbage cvs. Golden Acre and Titanic 90) and range of F_3 family means for diseased leaf area (DLA) measured in the greenhouse and interaction phenotype (IP) scores, based on inoculation with *Xanthomonas campestris* pv. *campestris*, petiole length, and leaf area measured in the field

	Genotype[a]					
Trait	B1-16	Cr-7	F_1	Golden Acre	Titanic 90	F_3 (range)
DLA (cm^2)	1.1 ± 0.2 (n = 35)	3.4 ± 0.3 (n = 31)	3.1 ± 0.3 (n = 35)	3.2 ± 0.3 (n = 35)	4.3 ± 0.3 (n = 35)	0.5–5.6
IP (0–9)	1.1 ± 0.1 (n = 12)	7.1 ± 0.2 (n = 10)	4.6 ± 0.1 (n = 11)	7.2 ± 0.1 (n = 11)	8.3 ± 0.1 (n = 11)	1.3–6.8
Petiole length (cm)	0 ± 0.0 (n = 12)	6.2 ± 0.3 (n = 12)	8.8 ± 0.7 (n = 12)	0.0 ± 0.0 (n = 12)	0.0 ± 0.0 (n = 12)	0.9–7.9
Leaf area (cm^2)	165.8 ± 15.8 (n = 12)	97.1 ± 8.5 (n = 12)	409.0 ± 35.4 (n = 12)	367.7 ± 31.0 (n = 12)	558.2 ± 17.8 (n = 12)	109.3–400.3

[a] n = the number of plants included in the estimate.

F_3 families having values 1.5- to 2.0-fold higher or lower than the parents, suggesting transgressive segregation. F_3 families were a significant source of variation ($P < 0.001$), but the block effect was not significant ($P > 0.10$).

Interval mapping analysis using the mean DLA over all three replicates revealed four regions containing putative QTL located on three linkage groups (LGs) (Table 2). A major locus on LG 1 explained 34.7% of the variation in DLA. Alleles from BI-16 at this interval acted additively and contributed to resistance, as indicated by the negative value of the additive gene action (Table 2). Two markers on LG 2 were significantly associated with DLA and explained smaller fractions of the phenotypic variation (Table 2). For interval *ec5e12-ec2h2*, BI-16 alleles contributed to resistance in an additive or dominant fashion, whereas BI-16 alleles contributed to susceptibility in an additive or recessive fashion for interval *wg6h1-tg4d2b*. Segregation ratios of marker loci flanking the latter interval were severely distorted toward Cr-7 (4). A fourth interval on LG 9 also was significantly associated with DLA. Alleles from BI-16 contributed to resistance and acted in an additive or recessive fashion. The four loci explained 73.3% of the phenotypic variation when combined in a multilocus model.

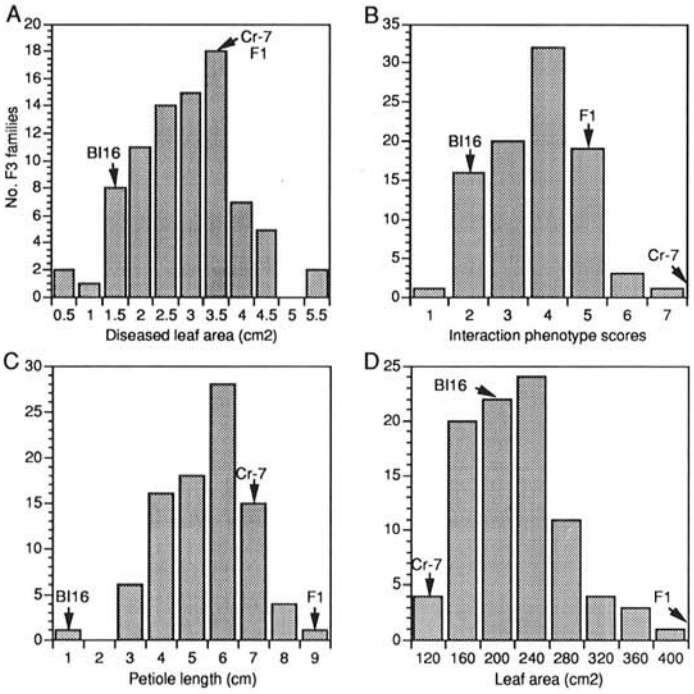

Fig. 1. Phenotypic distributions of *Brassica oleracea* F_3 family means for resistance to *Xanthomonas campestris* pv. *campestris* **A,** in the greenhouse and **B,** for resistance, **C,** petiole length, and **D,** leaf area in the field. Positions of the parents (Badger Inbred-16 and OSU Cr-7) and F_1 generation are shown.

Field evaluations. The parents were clearly differentiated by IP scores, whereas the susceptible control Titanic 90 had ratings slightly, but significantly, higher than Cr-7 (Table 1). All BI-16 plants had minute necrotic spots at the hydathodes, with no lesions progressing past the leaf lamina. The broccoli parent showed typical V-shaped lesions and systemic infection. Also, Cr-7 plants became defoliated as a result of infection. This could have interfered with the visual ratings of symptoms, leading to an underestimation of susceptibility. Mean IP scores of F_3 families were distributed normally (Fig. 1), and no transgressive segregants were detected. Two marker intervals containing putative QTL were identified on LGs 1 and 9 adjacent to the intervals identified in the greenhouse trial, with peak LOD scores separated only by 2 cM on LG 1 and 10 cM on LG 9 (Fig. 2). BI-16 alleles contributed to resistance and displayed the same modes of gene action as in the greenhouse. When combined in a multilocus model, the two loci explained 46.6% of the phenotypic variation.

BI-16 and Cr-7 differed greatly for petiole length. The F_1 hybrid had significantly greater petiole length than Cr-7, indicating heterosis for this trait. The mean petiole length of F_3 families was distributed normally ($P < 0.05$; Fig. 1), and transgressive segregation was apparent in the direction of the broccoli parent. Three putative QTL controlled petiole length on three LGs, with BI-16 alleles contributing to a shorter petiole for all QTL (Table 2). The F_3 family displaying the highest mean petiole length was derived from an F_2 plant heterozygous for each of the marker loci flanking the three putative QTL. The peak LOD scores of the putative QTL on LGs 1 and 9 affecting petiole length were adjacent to the ones associated with black rot resistance in the greenhouse and field (Fig. 2). The mode of gene action of BI-16 alleles was similar to QTL controlling resistance on LGs 1 and 9 and was recessive for the QTL on LG 6. When combined on a multilocus model, the three QTL explained 46% of the phenotypic variation.

The parents differed significantly for leaf area and a clear heterotic response was detected in F_1 plants that had a mean leaf area

TABLE 2. Marker intervals significantly associated ($\log_{10}$ of the odds ratio [LOD] ≥ 2.5) with *Brassica oleracea* resistance to black rot in the greenhouse (diseased leaf area [DLA]) and with resistance (interaction phenotype [IP] scores), petiole length, and leaf area in the field, based on F_3 lines of a Badger Inbred-16 (B1-16) cabbage × OSU Cr-7 broccoli cross

Marker interval	Linkage group[a]	LOD (peak)	% Variance[b]	Gene action[c]		
				Dominant	Additive	Mode
DLA (cm^2)						
wg2g11-wg6g5	1	7.1	34.7	−0.08	−0.98	A
ec5e12-ec2h2	2	3.1	17.8	0.17	−0.66	A/D
wg6h1-tg4d2b	2	3.5	21.5	0.49	0.99	A/R
wg8a9b-wg4d7	9	3.2	20.4	−0.88	−0.15	A/R
IP scores (0–9)						
wg6g5-wg1e3b	1	4.2	19.5	−0.03	−0.78	A
ec2d9-wg8a9b	9	6.3	43.3	−0.44	−1.17	A/R
Petiole length (cm)						
wg2g11-wg6g5	1	7.8	36.9	−0.43	−1.28	A
wg2h1-wg6f10	6	2.7	15.6	−0.88	−0.47	R
wg8a9b-wg4d7	9	2.6	13.1	−0.18	0.97	A/R
Leaf area (cm^2)						
wg1g5-wg7f8	3	7.4	35.2	−0.10	0.22	A/D

[a] Linkage groups are as in Figure 1.

[b] Percentage of variance explained by quantitative trait loci.

[c] Dominant and additive values of BI-16 alleles are expressed in trait units, as indicated by the unconstrained genetics model, and possible modes of gene action are based on comparison between LOD scores of constrained and unconstrained models. A = additive, D = dominant, and R = recessive for BI-16 alleles.

more than twice that of the high parent mean (Table 1). The distribution of F_3 family means was skewed and included transgressive segregant F_3 families (Fig. 1). A $\log_{10}$ transformation of the data was distributed normally, and the transformed values were used for interval analysis. Only one marker interval on LG 3 was significantly associated with leaf area (Table 2) and was unlinked to any other trait loci. The putative locus accounted for 35.2% of the phenotypic variance, with BI-16 alleles contributing to a larger leaf area. F_3 families with extreme mean leaf area (1- to 1.5-fold higher than the broccoli parent) were derived from F_2 plants heterozygous for at least one marker locus flanking this putative QTL.

Trait correlations. DLA, IP scores, and petiole length were significantly correlated ($P < 0.01$; Table 3). However, correlations between these traits and leaf area were nonsignificant ($P > 0.05$). Also, there was no significant correlation ($P > 0.05$) between the overall level of heterozygosity of individual F_2 plants (expressed as the percentage of heterozygous marker loci over all codominant marker loci) and trait means of their derived F_3 families (Table 3).

DISCUSSION

Marker interval analysis revealed two genomic regions on LGs 1 and 9 associated with resistance to black rot for young plants screened in the greenhouse and for adult plants screened in the field. The absence of a clear recombinant F_3 family that was resistant in the greenhouse and susceptible in the field suggests that the same genes were detected under these two different screening conditions. However, the hypothesis of two or more loci in tight linkage cannot be ruled out. In either case, the results support the conclusion that resistant plants selected based on greenhouse screenings should exhibit field resistance.

Analysis of greenhouse data revealed two additional loci that were not detected under field conditions, perhaps due to a greater experimental error in the field experiment and to the lower resolution of visual rating scales used to score symptoms in the field (11,31). However, the hypothesis that these loci are only involved in young plant reactions under greenhouse screening conditions cannot be discarded. Alleles from the resistant parent were associated with susceptibility at one locus on LG 2. This cryptic gene effect could explain the transgressive segregation of some F_3 families for DLA. Such families could be derived from F_2 plants combining complementary alleles from both parents. In fact, the four most resistant F_3 families were derived from F_2 plants either homozygous for broccoli alleles (three cases) or heterozygous at this locus on LG 2, whereas the four most susceptible were either homozygous for cabbage alleles (one case) or heterozygous. Another explanation of the apparent transgressive segregation observed in the seedling plants might be the influence of heterosis for leaf area exhibited among F_3 families when compared with those of the parents (Table 1; Fig. 1). Leaves of F_3 plants were considerably larger than those of the inbred parents, possibly contributing to the proportion of F_3 families exceeding the susceptible Cr-7 parent in diseased leaf area.

A small number of QTL with large effects were associated with petiole length (three QTL) and leaf area (one QTL). This agreed with previous results from analysis of morphological variation in a cabbage × broccoli cross (14) in which most traits associated with leaf dimensions, including petiole length, were controlled by a few genes with large effects. Two QTL controlling petiole length were found on LGs 1 and 9 near QTL controlling resistance to black rot. The presence of a small number of recombinant susceptible and petioleless plants within some F_3 families suggest tight linkage between resistance and shorter petioles.

We previously proposed a single recessive gene model controlling resistance in this cross based on Wright's formula (6) for estimating the number of genes controlling a quantitative trait (5). However, this formula can underestimate the effective number of genes (38), and results should be interpreted loosely. A single dominant gene controlling resistance to black rot in *B. oleracea* and *Arabidopsis thaliana* has been reported (1,3,30). Our results do not conform to either of these models, but they agree with reports of cabbage and cauliflower in which resistance is proposed to be controlled by more than one gene (24,35).

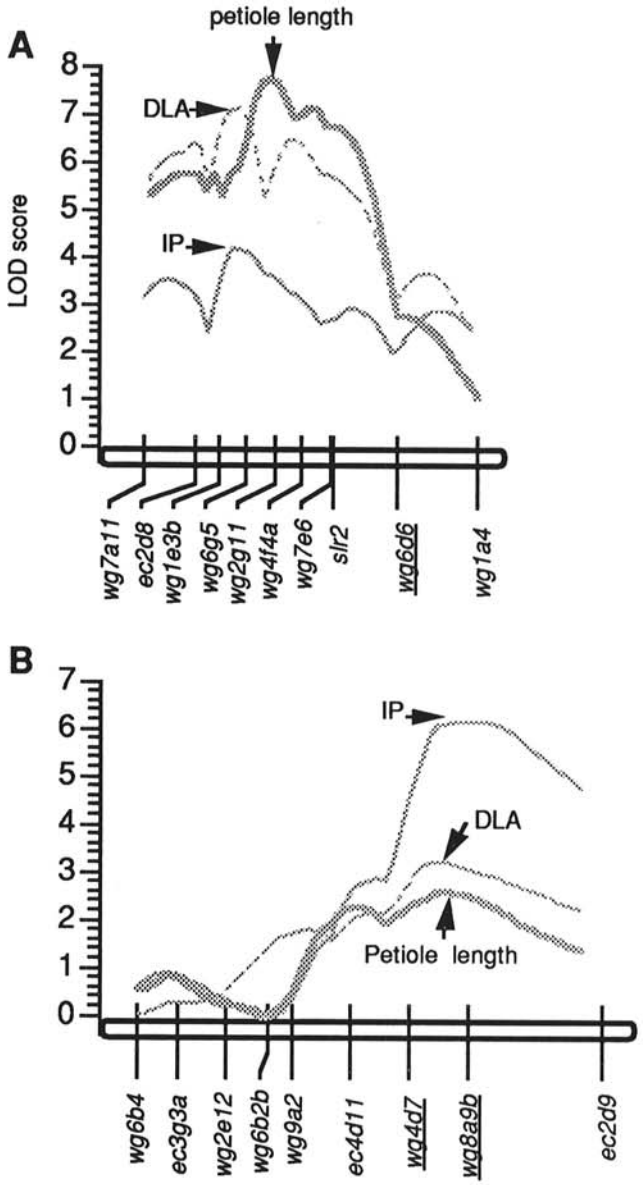

Fig. 2. Log_{10} of the odds ratio (LOD) scores for *Brassica oleracea* resistance to black rot in the greenhouse (DLA = diseased leaf area) and resistance (IP = interaction phenotype) and petiole length in the field on **A and B,** linkage groups 1 and 9, respectively. Peak LOD scores for each trait are indicated by arrows. Marker loci are indicated on the x-axis. Marker loci with significant segregation distortion are underlined.

Table 3. Correlation coefficients among *Brassica oleracea* resistance to black rot in the greenhouse (diseased leaf area [DLA]), resistance (interaction phenotype [IP] scores), petiole length, and leaf area in the field and percentage of heterozygosity from F_3 lines of a Badger Inbred-16 cabbage × OSU Cr-7 broccoli cross

	DLA[a]	IP score[a]	Petiole length	Leaf area
IP score	0.557**			
Petiole length	0.497**	0.518**		
Leaf area	0.170	0.126	0.194	
% heterozygosity[b]	0.048	0.045	0.224	0.248

[a] ** indicates significant correlation coefficient at $P < 0.01$.

[b] The percentage of heterozygous marker loci over all codominant marker loci.

Williams et al. (35) postulated the existence of a major gene '*f*' controlling field resistance to black rot based on segregation data from crosses involving the highly resistant Japanese cabbage cultivar Early Fuji and the susceptible Badger Inbreds-3, -5, -8, and -13. Resistance was inherited either as a dominant or recessive trait depending on the cross. Selection of resistant plants from these crosses and subsequent inbreeding led to the Badger Inbred series 14 through 20. Thus, it seems likely that BI-16 carries the *f* gene both because of its pedigree relationship with Early Fuji and because it displays the same highly conspicuous hypersensitive-like reaction at the hydathodes. It is hypothesized that the QTL detected on LG 9, which accounted for a major portion of the phenotypic variation in IP scores, is the *f* gene. Moreover, Williams et al. (35) also reported that all the F_1 hybrid cultivars produced in the United States were highly susceptible. This could be explained by the recessive or additive mode of gene action of resistant alleles at virtually all QTL controlling resistance. Therefore, resistant hybrids could be produced only if both parental inbred lines used for hybrid production carry resistance alleles. This could be accomplished by incorporating resistance into the existing parental lines through marker-assisted backcrossing in addition to phenotypic selection.

Based on this first report of mapping of QTL associated with resistance to black rot in a cruciferous host, additional studies could be conducted with the objectives of (i) confirming the marker-disease resistance associations reported here in crosses involving the resistant cabbage BI-16 and other susceptible *B. oleracea* accessions, (ii) introgressing resistance from cabbage into broccoli using both of the molecular markers associated with resistance in this study, and (iii) verifying whether these markers also are associated with resistance to black rot in different cruciferous species. For example, the region on LG 2 associated with resistance to black rot has RFLP marker loci in common with a region on LG 6 of *B. napus* that is associated with resistance to *Leptosphaeria maculans* (8). Thus, it is possible to test whether these conserved genomic regions are associated with resistance to different pathogens in *B. oleracea*. These results may suggest the presence of a cluster of resistance genes and should add to our understanding of the function of disease-resistance genes and to the genetics and evolution of host-pathogen interactions.

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