Analysis of Quantitative Trait Loci for Resistance to Southern Leaf Blight in Juvenile Maize

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ABSTRACT

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A set of 192 maize recombinant inbred lines (RILs), derived from a cross between the inbred lines Mo17 and B73, were evaluated as 3-week-old seedlings in the greenhouse for resistance to southern leaf blight, caused by *Cochliobolus heterostrophus* race O. Six significant (LOD >3.1) quantitative trait loci (QTL) were identified for disease resistance, located on chromosomes 1, 2, 3, 6, 7, and 8. Results were compared with a previous study that had used the same RIL population and pathogen isolate, but had examined resistance in mature rather than juvenile plants. There was a very weak but significant correlation between the overall resistance phenotypes of the RILs scored as mature and juvenile plants. Two QTL were found in similar positions on chromosomes 1 and 3 at both growth stages. Other QTL were specific to one growth stage or the other. Twenty-three of these RILs, together with the parental lines, were inoculated in the greenhouse with four *C. heterostrophus* isolates. Results indicated that the quantitative resistance observed was largely isolate nonspecific.

Additional keyword: corn.

Cochliobolus heterostrophus (Drechs.) Drechs. (anamorph = *Bipolaris maydis* (Nisikado) Shoemaker; synonym = *Helminthosporium maydis* Nisikado) is a necrotrophic plant pathogen and the causal agent of southern leaf blight (SLB). This disease is widely found in hot, humid maize-growing areas but was not considered an important pathogen until 1970 when *C. heterostrophus* race T became prevalent in the U.S. corn belt. Race T was highly pathogenic on Texas male-sterile cytoplasm (cms-T) and caused a major epidemic in 1970 and 1971 (37). Since that time, cms-T has been eliminated from elite germ plasm and effective polygenic resistance has been introduced. Most of this resistance is quantitative and additive or recessive in effect (5,24,27, 34,36), although one qualitative recessive gene, *rhm*, which primarily conditions resistance in pre-anthesis growth stages, has been mapped to the distal end of the short arm of chromosome six (bin 6.00) (40). The disease, predominantly caused by race O, is still a problem in sweet corn and seed production and in the southern Atlantic coast area of the United States on field maize. It can cause grain yield losses of 40% or more (6,18,22,37).

Most U.S. maize hybrids rely primarily on some form of quantitative resistance to control a variety of diseases. Many qualitative plant resistance genes have been cloned (23) and their downstream pathways characterized (23,32), but little is known about the molecular genetic basis or mechanisms of action of quantitative plant disease resistance genes. Numerous studies have been published on the mapping of disease resistance quantitative trait loci (QTL) to particular genomic regions (3,4,13,14). While knowledge of QTL is useful for, among other things, marker assisted breeding, QTL can generally be located with confidence within, at minimum, a 20 cM window, which gives little clue as to the molecular identity of the genes involved. Scoring for any quantitative trait on an individual plant basis is difficult and is often inconsistent across environments (4,31), making gene cloning techniques such as transposon tagging and map-based cloning challenging. Most cases in which a quantitative gene has been successfully identified (15,17,19,21) involved some combination of relatively easily scorable phenotype, defined environmental conditions, and the construction and use of near-isogenic lines, facilitating the phenotypic scoring of the QTL in a segregating population.

Field evaluation of quantitative resistance can be considered the ultimate reference against which other evaluation techniques are measured. In the field, mature plants can be assessed under natural disease conditions and multiple infection cycles. However, a laboratory or greenhouse-based assay can be a useful additional tool as it allows for greater environmental and inoculum standardization (both in terms of isolate identity and amount) and should enable a more reproducible assessment of resistance phenotype. Multiple experiments a year can be performed in the greenhouse, whereas in the field, there is usually only one opportunity annually. In addition, a greenhouse inoculation technique could be used in conjunction with other experimental approaches such as expression profiling. However, it is not clear that greenhouse studies with maize, which use juvenile plants, will detect resistance mechanisms that function in mature plants in the field. Since field resistance is the goal of our research, it was important to answer this question.

The main objective of this study was to determine whether, at a gross level, resistance in juvenile and mature plants was related and, at a finer level, whether loci that are important for quantitative resistance in mature plants in the field might also be important for resistance in juvenile plants. If loci are identified that function in both environments, one can then proceed with the analysis of these loci in the greenhouse, in the expectation that

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any findings will be relevant to the more practically important situation of mature plants in the field.

MATERIALS AND METHODS

Plant materials. Phenotypic data were collected from a mapping population comprised of 192 $F_{6:7}$ recombinant inbred lines (RILs) that were derived from the cross of maize inbred lines B73 (relatively susceptible parent) and Mo17 (relatively resistant parent) and from the two parental lines. Genotypic information for this population had previously been generated at 234 restriction fragment length polymorphisms, isozymes, and simple sequence repeat marker loci (35).

Plant growth conditions. Experimental units consisted of two plants of a particular line grown in a single 15-cm-diameter clay pot filled with a 50:50 mixture of Metromix (Scott's Inc., Marysville, OH) and sterilized soil. Supplemental lighting was used during winter months to maintain a ≈16 h day length, approximately 14 h/day during January and 10 h/day in February. The plants were maintained at 26°C during the day and 22°C during the night. The actual temperature rarely deviated more than 4°C from these target temperatures.

Fungal growth, inoculation, and rating techniques. *C. heterostrophus* isolate 2-16Bm was used for the QTL studies. Three additional isolates (Hm28, 9-31Bm, and NI1011) were used for the investigation of line–isolate interactions. Isolates were grown on potato dextrose agar plates at 25°C under a 12 h light/dark cycle. Spores were collected by submerging 1- to 2-week-old cultures in water and brushing the spores free with a camel-hair brush.

Plants were inoculated using a technique based on one reported in Zhu et al. (41). Water containing 0.05% agar and 0.25% Tween 20 was used to make a 5×10^3 ml conidial suspension. The suspension was sprayed as a fine mist until runoff onto both surfaces of the youngest fully expanded leaf of 21-day-old (i.e., 21 days from planting) seedlings at the five-leaf stage. Inoculated plants were placed in a humidity tent for 12 to 16 h overnight and then replaced on the greenhouse bench. Leaves were rated for percent necrotic leaf area 6 days after inoculation.

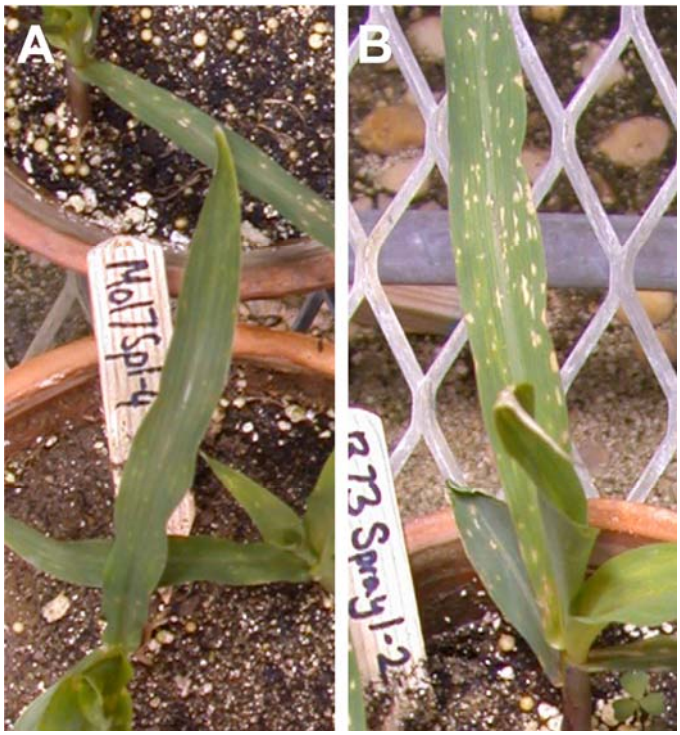

Fig. 1. A, Mo17 and **B,** B73 maize lines inoculated at 3 weeks after planting using spray inoculation, 6 days postinoculation.

Experimental design. Four replicates of the mapping population plus the parents were screened. Replicates 1 and 2 were screened in January 2004, replicates 3 and 4 in February 2004. A randomized complete block design was used in all cases.

The inoculations for the isolate–line interaction experiment took place in May 2004. A set of 25 lines were inoculated with four different isolates of *C. heterostrophus* race O (Hm28, 9-31Bm, 2-16Bm, and NI1011), all isolated from samples collected in North Carolina. The lines consisted of the parent lines (B73 and Mo17) and 23 RILs, selected from among lines for which significant amounts of seed were available, to include lines displaying high, moderate, and low resistance based on the previous greenhouse assays. The *C. heterostrophus* isolates were chosen from among those examined in a previous study (8). Ten isolates from this study had been maintained frozen as glycerol stocks at –80°C. Initial experiments were performed to identify suitably aggressive isolates (lesions produced within 4 days after inoculation). From among these relatively aggressive isolates, four were chosen to represent the two different mating types. The number of 25 maize lines was chosen for logistical reasons. It was considered important that all the isolates were inoculated on the same day within a relatively short period of time, to minimize any possible environmental interactions. This would not have been possible if more than 25 lines had been used. Each isolate was used to inoculate all 25 lines, arranged as a randomized complete block. Two randomized complete blocks were inoculated with each isolate. In this case, lesion lengths were measured (eight measurements per line per replication), so that the results could be better compared with those reported previously for these isolates (8).

Statistical analysis. Due to poor seed germination or growth, approximately 5% of the lines were not suitable for inoculation in each replicate. To account for missing data in QTL calculations, a least square means average for each line was calculated using the PROC GLM procedure of SAS (SAS Institute, Cary, NC), based on these four replications. All correlation calculations were made using the PROC CORR procedure of SAS.

The QTL cartographer software package (38) was used to detect the QTL. Composite interval mapping (CIM) was used. Several analyses were performed using simple interval mapping and CIM under Model 6 (the standard model), varying values for the walk speed (the genome scan interval), window size, and numbers of control markers. A CIM analysis using a walk speed of 2 cM, a window size of 2 cM, and 30 control markers, with a threshold value of 14.11 (determined by permutation analysis with a significance level of 0.05 and corresponding to a log of odds [LOD] of 3.1), is reported here. These conditions resulted in the detection of a relatively small number of distinct QTL peaks with relatively high LOD likelihood scores. All the QTL detected under these parameters were also consistently detected using a range of different analysis parameters.

RESULTS

Reproducibility of disease symptoms after inoculation in the greenhouse. Clear differences were observed between the resistant and susceptible parents after inoculation in the greenhouse (Fig. 1). Correlations between the four replications performed were high, with the six pair-wise Pearson correlation coefficients ranging between 0.71 and 0.79.

Comparison of field and greenhouse resistance. The phenotypic range of least square means of necrotic leaf area observed in the recombinant inbred population was 1.2 to 79.5% (Fig. 2). Some transgressive segregation was observed with Mo17 (resistant parent) averaging 6.9% necrotic leaf area and B73 (susceptible parent) averaging 61.7% necrotic leaf area.

The correlation between the least square means found in this study with the area under disease progress curve values obtained

previously in the field for the same lines assayed with the same *C. heterostrophus* isolate (9) was calculated. The Pearson correlation coefficient (r) between these two sets of data was 0.21 (P = 0.012) and Spearman rank correlation coefficient was 0.22 (P = 0.007), indicating a significant but very weak correlation between the resistance phenotypes of juvenile and mature plants.

QTL identification. Significant QTL (LOD score >3.1) were found in six chromosomal bins: 1.08/09, 2.09, 3.04, 6.00, 7.02, and 8.03 (Tables 1 and 2). The resistant alleles were derived from Mo17 in four of the six cases, while the resistant alleles in bins 2.09 and 7.02 were derived from B73. These QTL together accounted for over 40% of the phenotypic variation observed. Other less significant QTL (LOD scores from 2.0 to 3.1) were detected in bins 1.06, 3.06, 5.07, and 10.06/07. In each of these cases, the resistant allele was derived from Mo17. No epistatic interactions were found between any of the QTL detected.

Six major QTL were also detected in the companion field study using mature plants of the same population and inoculating with the same pathogen isolate (9). Of these QTL, only two, in bins 3.04 and 1.08/09, could be considered to have been detected in both studies (Table 2). For both QTL the resistant allele was derived from Mo17 at both growth stages.

Investigation of isolate–line interactions. The resistance phenotypes of 25 lines consisting of the RIL parents (B73 and Mo17) and 23 RILs against four different race O *C. heterostrophus* isolates (Table 3) were assessed. Analysis of variance, using lines and isolates as independent variables (Table 4), showed that differences between lines were highly significant. Spearman rank correlations of lines ranked by resistance to each isolate (Table 5) ranged from 0.92 to 0.72 for the six pair-wise interactions, i.e., the ranking of lines with each isolate was quite similar, and Pearson correlation coefficients for each line–isolate combination ranged from 0.88 to 0.64 (Table 5). Although the isolates chosen showed consistent differences in the size of the lesions they formed (Table 3), they did not vary enough in aggressiveness in this limited study for significant differences between them to be observed ($P > F$ = 0.154). Isolate–line interactions were nonsignificant at the 95% level ($P > F$ = 0.054) (Table 4).

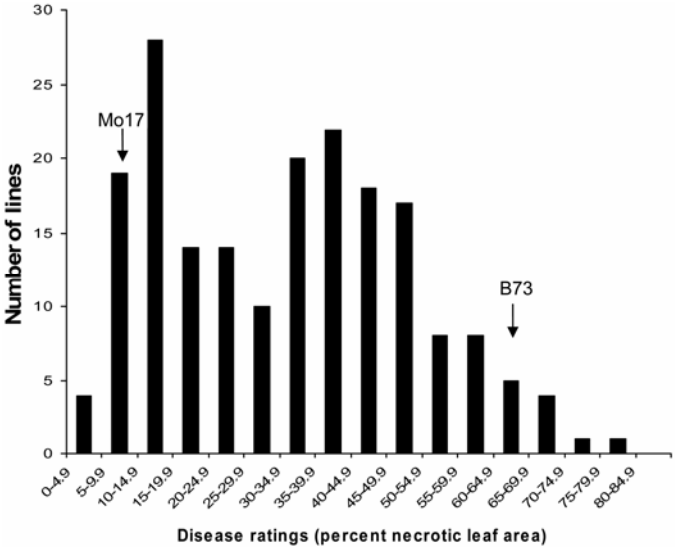

Fig. 2. The distribution of average scores for percent necrotic leaf area in the B73/Mo17 maize recombinant inbred line population. The position of the average scores of the parental types, B73 and Mo17, are indicated.

TABLE 2. The major quantitative trait loci (QTL) for resistance in maize to southern leaf blight (SLB) and associated parameters found in this study using juvenile plants (LOD score > 3.1) compared with the major QTL detected for resistance to SLB in the same population scored as mature plants in the field by Carson et al. (9)[a]

	Associated parameters	
Chromosome bin	Juvenile plants	Mature plants
1.02	...	R^2 = 3.2, a = –31.4
1.08/1.09	R^2 = 5.1, a = 4.2	...
1.09/1.10	...	R^2 = 24.9, a = –81.3
2.04	...	R^2 = 19.8, a = –71.5
2.09	R^2 = 3.5, a = –3.3	...
3.03/3.04	...	R^2 = 11.2, a = –49.1
3.04	R^2 = 12.3, a = 6.5	...
4.09	...	R^2 = 4.3, a = –28.2
6.00	R^2 = 7.2, a = 5.1	...
7.02	R^2 = 3.2, a = –3.3	...
8.03	R^2 = 10.2, a = 5.9	...
10.04	...	R^2 = 7.7, a = –44.0

[a] Both studies used the same pathogen isolate. The additive effect is denoted by 'a'. Additive values cannot be compared between studies as they were measured on different scales. This study used necrotic leaf area while Carson et al. (9) used area under disease progress curve.

TABLE 1. Location and parameters associated with major quantitative trait loci (QTL) for resistance to southern leaf blight of maize, caused by *Cochliobolus heterostrophus* race O, in a B73 × Mo17 recombinant inbred (RI) line population comprising 192 lines

Chromosome	Bin[a]	QTL peak flanking markers[b]	One LOD interval flanking markers[c]	LOD score[d]	Additive effect[e]	R^{2f}
1	1.06	csu3-umc133c	csu3-bnl5.59	2.2	2.8	1.9
1	1.08/1.09	csu164a-phi055	phi002-phi055	4.9	4.2	5.1
2	2.09	umc49a-php20581b	DHFR-TS2-php20581b	3.1	–3.3	3.5
3	3.04	asg48-phi036	asg48-umc10a	12.6	6.5	12.3
3	3.06	bnl5.37-umc60	bnl5.37-bnl6.16	2.5	2.8	2.3
5	5.07	umc108-phi048	phi101-phi085	2.6	3.0	2.2
6	6.00	bnlg161-Rxo	bnlg161-umc163b	7.4	5.1	7.3
7	7.02	umc005b-bnlg657	bnlg398-umc116a	3.7	–3.3	3.2
8	8.03	umc124a-umc120a	phi119-umc120a	10.6	5.9	10.2
10	10.06/07	phi035-bnl7.49	umc44a-bnl7.49	2.4	3.1	2.8

[a] Chromosome bin location of QTL peak on 1 of the 10 chromosomes of the maize genome. Bins divide the genetic map into 100 approximately equal segments. The segments are designated with the chromosome number followed by a two-digit decimal (e.g., 1.00, 1.01, 1.02 and so on). The marker order determined for the population used in this experiment largely follows the marker order shown in the standard maize genetic map (the IBM map). However, there are some discrepancies.

[b] The markers that flank the locus with the maximum log of odds (LOD) likelihood score for each QTL as determined by composite interval mapping. The left marker is the marker nearest the distal end of the short arm of the chromosome.

[c] The molecular markers flanking the area within one LOD value of the maximum LOD likelihood value.

[d] The maximum LOD score associated with each QTL.

[e] The additive effect in terms of percent necrotic leaf area associated with each QTL.

[f] R^2 estimates the proportion of phenotypic variance (%) explained by the detected QTL.

DISCUSSION

The analysis of quantitative disease resistance genes presents particular challenges, as the phenotype involves interaction with another organism and is, as such, inherently more variable than those based purely on the physiology of the organism under study. In the field, other sources of variation are introduced such as a naturally occurring, mixed population of inoculum, varying in aggressiveness (8), and unpredictable environmental conditions. If detailed characterization, mapping, and cloning of quantitative disease resistance genes is to be successful, it is important to minimize the environmental variation under which data is collected. One option is to use juvenile plants in a greenhouse environment, where the isolate used for inoculation, the inoculum level, and the light and temperature levels can all be controlled.

The main objective of this work was to determine whether one can justifiably extrapolate results obtained in the greenhouse with seedlings to phenotypes expected in the field with mature plants. This is a particular concern with foliar diseases because juvenile and mature maize leaves differ for a number of characteristics such as epicuticular wax, leaf hairs, and cell structure (20). The low correlation ($r = 0.21$) between the lines scored as juvenile plants in this study and as mature plants in Carson et al. (9) indicates that the gross level of SLB resistance in juvenile plants is not highly related to the resistance level observed in the same line in the field.

At a more detailed level, of the six major QTL (LOD >3.1) detected in this study, two (in bins 1.08/9 and 3.04) coincided with or were very near two of the six significant QTL detected previously using this same population and pathogen isolate in the field study (Table 2) (9). The low but significant correlation between the lines scored as juvenile and mature plants could therefore be ascribed to these common QTL segregating with eight maturity-specific QTL. An important caveat is that even though two QTL regions do coincide in the two studies, we cannot say at this stage whether resistance is conferred by the same mechanisms or genes at the two growth stages, or whether two or more linked genes are involved, one or more at each growth stage.

TABLE 3. *Cochliobolus heterostrophus* isolates used for the isolate–maize line interaction studies

		Lesion length (mm)[a]		
Isolate	Mating type	Greenhouse[b]	Field[c]	Greenhouse (this study)[d]
Hm28	2	5.8	4.0	5.4
9-31Bm	1	5.7	5.9	3.7
2-16Bm	2	6.5	4.8	7.1
NI1011	1	6.5	5.8	6.2

[a] Lesion lengths are averages of all measurements taken from greenhouse and field studies. Except for last column, all details taken from Carson (8).
[b] Average of measurements on the susceptible maize host Pioneer brand hybrid 3184. Details taken from Carson (8).
[c] Average of measurements on the maize hosts Pioneer brand hybrids 3184 and 3162. Details taken from Carson (8).
[d] Average of measurements on 25 maize lines: B73, Mo17, and 23 recombinant inbred lines of varying resistance. Details taken from this study.

TABLE 4. Analysis of variance of lesion length from inoculations of four *Cochliobolus heterostrophus* isolates on 25 different maize lines[a]

Source	df	Mean square	*F* value	*P* > *F*
Isolate	3	87.0	3.07	0.154
Rep(isolate)	4	28.4	8.52	<0.0001
Line	24	62.5	18.42	<0.0001
Isolate–line	71	4.9	1.44	0.054

[a] Isolate and line were treated as independent variables. df = degrees of freedom.

A significant QTL was found in bin 7.02 in this study. A QTL was also detected in the same genomic location in one of the 2 years (1995) evaluated by Carson et al. (9). However, in the present study, the juvenile plant resistance allele was derived from B73, whereas the mature plant resistance allele was derived from Mo17. None of the weaker QTL (LOD between 2 and 3.1) in bins 1.06, 3.06, 5.07, and 10.06/7 detected in this study co-localize with any QTL detected in the field by Carson et al. (9).

A field study of maize QTL for resistance to northern corn leaf blight (39) identified QTL at different growth stages, including in juvenile plants at the four- to six-leaf stage. As in this study, several growth stage-specific QTL were identified while other QTL were effective at a range of growth stages.

The recessive SLB resistance gene *rhm* has been mapped to bin 6.00 (40). The effect of *rhm* is strongest during seedling stages though it does provide some level of resistance to mature plants post-anthesis (36,40). It has been proposed that a second locus, *rhm2*, is located at a linked site about 10 cM from *rhm1*, also in bin 6.00 (11). We found a major SLB QTL in bin 6.00, while no corresponding QTL was identified in the mature plant study (9). The coincidence of position and the fact that this resistance is detected in juvenile plants but not in mature plants suggests similarities to *rhm*. Although Mo17 is not thought to carry *rhm*, it may be that it has an alternate allele of the same gene which provides more resistance than the B73 allele.

McMullen and Simcox (28) previously identified two bins, 3.04 and 6.01, as regions where many resistance loci are tightly linked. SLB QTL were identified in both these regions in this study (albeit in an adjacent bin in the case of 6.01). Bin 3.04 is of particular interest as it was identified as an SLB resistance QTL in both juvenile (this study) and mature plants (9). In addition, a previous study using tropical germplasm also identified a QTL for SLB in the vicinity of bin 3.04 (26). Furthermore, in our unpublished, ongoing studies, we have identified strong SLB resistance QTL in bin 3.04 in three populations derived from the crosses ADENT × B73, NC250 × B73, and B104 × NC300 (M. M. Goodman, M. P. Jines, D. M. Bubeck, and P. J. Balint-Kurti, *unpublished data*).

The question of isolate specificity of disease resistance QTL has been examined in several recent studies. In some of these, no evidence was found of isolate-specific interactions (10,12,29,33), while others report finding significant isolate–line interactions or isolate-specific resistance QTL (1,2,7,16,30). A previous report (25) documented the interaction between 10 *C. heterostrophus* isolates and 10 maize inbred lines. Although isolate–line interactions were found within individual replications, no significant isolate–line interactions were found when the data were analyzed over the six replications performed. We have also shown here that the resistance we observed is generally isolate nonspecific with respect to the four isolates examined.

TABLE 5. Correlations between southern leaf blight disease scores (percent necrotic leaf area) obtained on 25 different maize lines using four different *Cochliobolus heterostrophus* race O isolates[a]

	2-16Bm	9-31	Hm28	NI1011
2-16Bm	1	...	...	...
	0.81	...	...	...
I9-31	0.76	1	...	...
	0.88	0.64*	...	...
Hm28	0.92	0.72	1	...
	0.86	0.77	0.83	...
NI1011	0.84	0.76	0.82	1

[a] Pearson correlation coefficient is in normal font, and the Spearman rank correlation coefficient is underlined below. * Indicates significant at $P = 0.0007$. All other correlations are significant at $P < 0.0001$.

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