Identification of Quantitative Trait Loci for Resistance to Southern Leaf Blight and Days to Anthesis in a Maize Recombinant Inbred Line Population

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Accepted for publication 24 April 2006.

ABSTRACT

Balint-Kurti, P. J., Krakowsky, M. D., Jines, M. P., Robertson, L. A., Molnár, T. L., Goodman, M. M., and Holland, J. B. 2006. Identification of quantitative trait loci for resistance to southern leaf blight and days to anthesis in a maize recombinant inbred line population. *Phytopathology* 96:1067-1071.

A recombinant inbred line population derived from a cross between the maize lines NC300 (resistant) and B104 (susceptible) was evaluated for resistance to southern leaf blight (SLB) disease caused by *Cochliobolus heterostrophus* race O and for days to anthesis in four environments (Clayton, NC, and Tifton, GA, in both 2004 and 2005). Entry mean and average genetic correlations between disease ratings in different environ-

ments were high (0.78 to 0.89 and 0.9, respectively) and the overall entry mean heritability for SLB resistance was 0.89. When weighted mean disease ratings were fitted to a model using multiple interval mapping, seven potential quantitative trait loci (QTL) were identified, the two strongest being on chromosomes 3 (bin 3.04) and 9 (bin 9.03-9.04). These QTL explained a combined 80% of the phenotypic variation for SLB resistance. Some time-point-specific SLB resistance QTL were also identified. There was no significant correlation between disease resistance and days to anthesis. Six putative QTL for time to anthesis were identified, none of which coincided with any SLB resistance QTL.

Additional keywords: flowering, *Helminthosporium*.

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), causing a major epidemic in 1970 and 1971 (23). Since that time, cms-T has been eliminated from elite germplasm and effective polygenic resistance has been introduced. The disease, predominantly caused by race O, is still a significant problem in the southern Atlantic coast area of the United States and parts of India, Africa, and Western Europe. It can cause grain yield losses of 40% or more (4,10,11).

Most of the SLB resistance that has been characterized is quantitative and additive or recessive in effect (3,13,16,21) although one qualitative recessive gene, *rh*m, 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) (22,28).

To date, only one comprehensive study has been published on mapping quantitative trait loci for field resistance to SLB in maize (6). In that paper, three major quantitative trait loci (QTL) for resistance were identified on chromosomes 1, 2, and 3 in a recombinant inbred (RI) line population derived from a cross

between the maize inbreds Mo17 and B73. A follow-up study, using the same RI line population and pathogen isolate, identified QTL for SLB resistance in juvenile plants (1). There it was shown that some QTL, including those on chromosomes 1 and 3, conferred SLB resistance in both juvenile and mature plants, while others were growth stage-specific. Another study identified an SLB resistance QTL on chromosome 3 from a single environment (14). The present study reports the identification of QTL for SLB resistance in an RI line population derived from a cross between B104, a Stiff Stalk Synthetic line developed at Iowa State University with low SLB resistance, and NC300, a line derived from tropical germplasm at North Carolina State University and known for its high SLB resistance. B104 is slightly earlier maturing than NC300, usually starting to shed pollen 2 to 4 days before NC300.

MATERIALS AND METHODS

Plant materials. Phenotypic data were collected from a mapping population comprised of 133 F₂-derived F₇ RI lines derived from the cross of maize inbred lines B104 (relatively susceptible parent) and NC300 (relatively resistant parent). Genotypic information for 130 of the 133 lines in this population has been generated at 113 simple sequence repeat marker loci (20) and a map constructed with an average distance between loci of 19.35 centimorgans (cM). The total length of the map was 1,993 cM. Segregation distortion was observed at 19% of the markers.

Field trials. The RI and parental lines were evaluated for resistance to SLB (*C. heterostrophus* race O) in field trials at North Carolina State University Central Crops Research Station located at Clayton, NC, and at the University of Georgia Coastal Plain Experiment Station in Tifton, GA, in the summers of 2004 and

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DOI: 10.1094/PHYTO-96-1067

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2005. Experimental units in each case consisted of single-row plots arranged in randomized complete blocks with two replications. Plots were 2 m in length with a 0.6 m alley at the end of each plot. Interrow spacing was 0.97 m. Twelve seeds per plot were planted and rows were not thinned. Two plots of border were planted on all sides of the experiment. Overhead irrigation was used as needed to ensure satisfactory plant growth. Standard fertilizer and herbicide regimes were used.

Fungal growth and inoculation. Techniques used for inoculum preparation are identical to those reported previously (6). Experimental plots were inoculated at the four- to six-leaf stage by placing ≈ 20 grains of a sorghum seed culture of *C. heterostrophus* isolate 2-16Bm (5) in the leaf whorl of every plant in every plot (including border plots).

Ratings. Plants were rated on a plot basis four times at each location. The first rating was taken approximately 5 days before anthesis and subsequent ratings were made at 10- to 16-day intervals. Plots were rated on a 1 to 9 scale, with 1 being a symptomless plant and 9 being a completely dead plant and each decreasing number representing an approximate 12% increase in disease severity. The number of days after planting when half the plants in the row were shedding pollen was noted as the time of anthesis. Weighted mean disease (WMD) rating values were calculated for each replication in each environment by taking the average value of two consecutive ratings and multiplying by the number of days between the ratings. Values were then summed over all the intervals and divided by the number of days of evaluation.

Statistical analyses. SLB resistance in four environments was evaluated in this study; Clayton 2004, Clayton 2005, Tifton 2004, and Tifton 2005. Due to poor seed germination or growth, approximately 2% of the lines were not scorable in each replicate. To account for these missing data for subsequent QTL analyses, least squares means were calculated using the PROC GLM procedure of SAS (SAS Institute, Cary, NC). The first, third, and fourth set of ratings from each environment (averaged over two replications) were chosen to represent early, middle, and late ratings, respectively.

All phenotypic correlation calculations were made using the PROC CORR procedure of SAS. Analysis of variance (ANOVA) calculations were made using the PROC GLM procedure of SAS. Heritability was estimated for each trait using the PROC MIXED procedure of SAS, as described previously (12). Genetic correlation calculations were made according to the technique described previously (8).

The Windows QTL cartographer version 2.5 software package (Department of Statistics, North Carolina State University, Raleigh) (4) was used to detect the QTL. Composite interval mapping (CIM), using the default parameters (Model 6), was used to supply the initial models for multiple interval mapping (MIM) of each trait. QTL were selected for initial MIM models if they were significant at at least the 0.01 significance level in CIM (the default parameter). MIM models were created and tested in an interactive, stepwise fashion, searching for new QTL to add to the current model, and testing their significance after each search cycle. New models were accepted if they decreased the Bayesian information criterion (BIC) (18). The BIC favors models with

higher likelihoods, but includes a penalty for each additional parameter added to the model, to help prevent overfitting the models. After no additional QTL could be added to a model according to the BIC, each pair of QTL in the model was tested for epistatic interactions. Epistatic interactions were chosen if they decreased the BIC. While deriving the model using MIM, we were mindful not to “over-fit” the model such that the proportion of total variation due to QTL exceeded the entry mean heritability. If this occurred, QTL were dropped in a backward regression fashion to obtain the best model according to the BIC that did not explain a greater proportion of the phenotypic variation than the phenotypic variation.

RESULTS

Disease and anthesis ratings. Phenotypic data were analyzed using ANOVA (Table 1). The contribution to the overall variation due to differences between RI lines for all traits (WMD, early-, mid-, and late-season ratings and anthesis) was substantially larger than the variation ascribed to the environment–line interaction. Variation due to environment was significant; however, CIM using WMD entry mean values for each environment detected the same major QTL on chromosomes 3 and 9 in every case (data not shown). Additionally, pairwise Pearson correlation coefficients between replications within an environment and between scores for each environment for both WMD and anthesis were high (0.78 to 0.89, $P < 0.0001$ in each case) (Table 2) and the average genetic correlation between WMD measurements in the four different environments was very high at 0.90. For these reasons, WMD averages of the four environments were used for all the MIM QTL analyses reported here.

The entry mean heritability for disease resistance over the four environments was 0.89 (standard error 0.013). The correlations between the overall averages of WMD and anthesis were not significant (Pearson correlation coefficient = 0.01, $P < 0.9$).

Average WMD scores followed an approximately normal distribution. There was no evidence for transgressive segregation (Fig. 1).

QTL analyses. MIM yielded models predicting significant QTL for all traits examined: WMD, early, middle, and late ratings

TABLE 2. Pearson correlation coefficients between weighted mean for disease resistance of southern leaf blight disease ratings (scored on a 1 to 9 scale) and days to anthesis for the maize B104/NC300 recombinant inbred line population obtained in four environments; Clayton, NC, 2004 and 2005 and Tifton, GA, 2004 and 2005^a

	Clayton 2005	Clayton 2004	Tifton 2005	Tifton 2004
Clayton 2004	0.89 <u>0.70</u>	1.00 ...	...	...
Tifton 2005	0.85 <u>0.76</u>	0.78 <u>0.72</u>	1.00 ...	...
Tifton 2004	0.84 <u>0.64</u>	0.79 <u>0.68</u>	0.82 <u>0.70</u>	1.00 ...

^a Disease rating correlations are upper values. Days to anthesis ratings are underlined lower values. All correlations are significant at $P < 0.0001$.

TABLE 1. Combined analysis of variance of southern leaf blight resistance scores from a population consisting of 133 recombinant inbred maize lines from a NC300 $\times$ B104 cross and four inbred line checks (B73, Mo17, NC300, and B104), scored across four environments (Tifton, GA, and Clayton, NC, in 2004 and 2005)^a

Source	df	MS WMD	MS early	MS mid	MS late	MS anthesis
Line	136	4.83	2.58	7.44	10.34	32.77
Environment $\times$ line	403	0.23	0.35	0.46	0.64	3.27
Error	531	0.12	0.26	0.25	0.25	2.05

^a In each environment, two replications, arranged in a complete randomized block design, were performed. Mean sum of squares (MS) is shown for the line, line $\times$ environment, and error terms for five traits: overall weighted mean for disease resistance (WMD), early-season disease rating (early), mid-season disease rating (mid), late-season disease rating (late), and time to anthesis (anthesis).

and days to anthesis (Table 3). Major QTL (arbitrarily defined as $R^2 \geq 0.05$) for WMD were found on chromosomes 1 (bin 1.08-1.09), 2 (bin 2.06-2.07), 3 (bins 3.04, 3.07-3.08, and 3.08-3.09), 6 (bin 6.06), and 9 (bin 9.03-9.04). Major QTL for days to anthesis were found on chromosomes 1 (bins 1.02 and 1.05-1.06), 4 (bin 4.08), 5 (bin 5.03-5.04), 7 (bin 7.03-7.04), 8 (bin 8.05), and 9 (bin 9.01-9.02). In no case did QTL for days to anthesis and WMD coincide. QTL identified for the early, middle, and late disease ratings generally coincided with the QTL identified for WMD (Table 3). The major exceptions were a QTL on chromosome 8 (bin 8.03-8.04) for the early rating and QTL on chromosomes 4 (bin 4.03-4.04) and 5 (bin 5.04) specific to middle and late ratings. Among these major QTL, all alleles for resistance were contributed by NC300, with the exception of the WMD QTL in bin 3.07-3.08. No major epistatic effects between QTL were identified for any of the disease parameters or for days to anthesis. A small additive epistatic interaction (partial $R^2 = 0.036$) was detected between the QTL identified for the early ratings in bins 2.06-2.07 and 8.03-8.04.

DISCUSSION

This study represents the second comprehensive report of identification of QTL for SLB resistance in field evaluations in a segregating maize population. The results reported are similar in several respects to the previous study by Carson et al. (6), which used a B73 $\times$ Mo17 RI line population, and are consistent with most previous studies on the inheritance of SLB resistance (3, 13, 16, 21), in that heritability was high and a relatively small number of loci controlled most of the variation (six loci had individual partial R^2 values of greater than 0.05 for WMD) and epistatic interactions were of minor importance.

However, few of the QTL identified in this study co-localize with QTL found in Carson et al. (6). Only bin 3.04 was identified as an SLB resistance QTL location in both studies. Jiang et al. (14) also detected an SLB QTL from tropical germplasm in this region. McMullen and Simcox (17) previously identified two bins, 3.04 and 6.01, as regions where many resistance loci to a variety of diseases are tightly linked. Bin 3.04 is of particular interest as it has been identified as containing an SLB resistance QTL in both juvenile (1) and mature plants. Furthermore, in our unpublished, ongoing studies, we have identified strong SLB resistance QTL in bin 3.04 in two populations derived from the crosses ADENT $\times$ B73 and NC250 $\times$ B73 (D. M. Bubeck, P. J. Balint-Kurti, and M. M. Goodman, *unpublished data*). A more recent synthesis of 50 maize disease QTL studies (27) identifies the region around bin 3.04 as a hotspot for SLB, viral and possibly common rust and ear and stalk rot resistance QTLs, though, when adjustments are made to account for gene density, it is not one of the main disease QTL hotspots in the genome.

The QTL with the greatest effect on overall disease severity identified in this study was on chromosome 9 (bin 9.03-9.04). An SLB QTL has been detected in this region in an $F_{2:3}$ population derived from the NC250 $\times$ B73 cross (D. M. Bubeck, P. J. Balint-Kurti, and M. M. Goodman, *unpublished data*) but very few other disease QTL have been reported in this region (27).

MIM is a powerful tool in estimating the genetic interactions governing a quantitative trait. It is, however, a somewhat subjective process. In some cases, MIM may allow the detection of two tightly linked QTL with opposite effects that might not be detected by other QTL analysis procedures such as simple interval mapping or CIM, but the detection of QTL of this type must be treated with some caution, as they may represent cases of multicollinearity (18, 19). The closely linked WMD QTL in bins 3.07-3.08 and 3.08-3.09 are cases in point. Their inclusion in the MIM model results in a better fit to the data, but the fact that major peaks are not evident in the CIM likelihood plot for this region (data not shown), and the fact that QTL were not found in this

region for the other disease parameters, suggests that they may be analytical artifacts. Analysis of a larger population would help to resolve this point. Similar observations regarding MIM have been made elsewhere (9). All other predicted QTL shown in Table 3 correspond to peaks in the CIM log likelihood plot (data not shown).

A previous study (1) showed that many maize QTL for SLB resistance act only at the juvenile or at the mature plant stage. In this study, all the ratings were taken on adult plants around or after the time of anthesis. There is some evidence for differential QTL efficacy at different time points within this period. The resistance QTL on chromosome 4 (bin 4.03-4.04) and chromosome 5 (bin 5.04) observed at the middle and late time points are not observed at the early time point, and the QTL on chromosome 2 (bin 2.06-2.07) is observed in the early and middle but not the late time points. But for the most part, major QTL are detected across all time points for WMD, sometimes with slight changes in position (bins 6.05-6.06 and 9.03-9.05 in Table 3). Some evidence for time-point-specific disease resistance QTL efficacy in maize has been reported previously (6, 25).

SLB, in common with other necrotrophic foliar diseases of maize such as gray leaf spot and anthracnose (caused by *Cercospora zeae-maydis* and *Colletotrichum graminicola*, respectively), is generally a late season disease, with most disease development occurring postanthesis (26). Thus, there is concern that disease ratings might be affected by variations between lines in time to maturity. Several mapping studies have examined both maturity-related and disease-related traits for maize necrotrophic diseases in the same populations and none showed a strong correlation between the two traits (2, 6, 7, 14, 15), although some co-localization of disease QTL and maturity-related QTL and/or significant correlations between disease resistance and time to anthesis was observed in some studies, especially for studies on gray leaf spot resistance. When a diverse 300-line panel of maize germplasm was evaluated for both SLB resistance and flowering time, 23% of the variance for resistance was explained by variation in flowering time (P. J. Balint-Kurti, *unpublished data*). In this study, however, no significant correlation was observed between disease rating and time to anthesis, nor were there any shared QTL for flowering and disease traits (Table 3). The identification of SLB resistance QTL derived from the highly SLB-resistant maize line NC300 should allow more efficient breeding of SLB-resistant maize inbred lines.

Fig. 1. The distribution of weighted mean ratings for resistance to southern leaf blight of maize, caused by *Cochliobolus heterostrophus* race O, rated on a 1-to-9 scale, where 1 represents a symptomless plant and 9 represents a dead plant, in the B104/NC300 maize recombinant inbred line population. The position of the average scores of the parental types, B104 and NC300, are indicated.

TABLE 3. Chromosomal location in centimorgans (cM) 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 B104 × NC300 recombinant inbred line population comprising 133 lines^a

Bin ^b	Flanking markers ^c	Early	Mid	Late	WMD	Anthesis
1.02	bnlg1803-bnlg147					42.6 cM ^d a = 0.80 ^e R ² = 11.8 ^f
1.05-1.06	bnlg1884-umc1335		132.2 cM a = -0.18 R ² = 4.8			111.2 cM a = -0.77 R ² = 8.4
1.08-1.09	bnlg2228-umc2047		209.5 cM a = 0.23 R ² = 5.4		206.5 cM a = 0.19 R ² = 5.6	
2.06-2.07	bnlg1036-umc1637	132.3 cM a = 0.13 R ² = 6.6	130.3 cM a = 0.20 R ² = 6.3		130 cM a = 0.18 R ² = 6.8	
3.04	phi036-bnlg602	19.8 cM a = 0.36 R ² = 34.7	19.8 cM a = 0.31 R ² = 12.9	19.8 cM a = 0.33 R ² = 11.4	19.8 cM a = 0.28 R ² = 15.5	
3.07-3.08	umc1498-umc1844				117.8 cM a = -0.41 R ² = 12.6	
3.08-3.09	umc1844-bnlg1496				131.2 cM a = 0.32 R ² = 6.1	
4.03-4.04	umc2082-umc1171		35.4 cM a = 0.25 R ² = 8.5	33.4 cM a = 0.40 R ² = 13.4		
4.08	umc1051-umc1808					139.2 cM a = 0.83 R ² = 17.6
5.03-5.04	bnlg1046-bnlg1208					62.3 cM a = -0.5 R ² = 5.7
5.04	bnlg1208-umc1221		99.4 cM a = 0.16 R ² = 2.6	98.4 cM a = 0.26 R ² = 5.0		
6.05-6.06	bnlg1702-umc1859	48.6 cM a = 0.13 R ² = 4.5				
6.06	umc1859-umc1520		65.5 cM a = 0.32 R ² = 10.5	59.5 cM a = 0.34 R ² = 8.8	66.5 cM a = 0.22 R ² = 7.6	
8.03-8.04	umc1034-umc1172	98.3 cM a = 0.16 R ² = 5.8				
8.05	umc1562-bnlg2181					119.7 cM a = 0.56 R ² = 8.7
9.01-9.02	bnlg1810-dupssr6					10.0 cM a = -0.62 R ² = 10.9
9.03-9.04	phi022-umc1571	58.1 cM a = 0.23 R ² = 14.5	63.1 cM a = 0.42 R ² = 21.7		63.1 cM a = 0.37 R ² = 23	
9.04-9.05	umc1571-umc1357			72.2 cM a = 0.51 R ² = 22.7		

^a QTL for early-, middle-, and late-season ratings are shown, together with QTL for overall weighted mean disease (WMD) ratings. QTL associated with days from planting to anthesis for this population are also shown. Early ratings were made about 5 days before anthesis (52 to 65 days after planting), mid-season ratings were made about 2 weeks after anthesis (73 to 83 days after planting), and late ratings about 4 weeks after anthesis (83 to 103 days after planting). All ratings were made on a 1 to 9 scale, where 1 represents a symptomless plant and 9 represents a dead plant. QTL present in the multiple interval mapping models where none of the R² values in an interval are above 5 are not reported in this table. These include an early QTL in bins 2.02, 5.07, and 8.02; anthesis QTLs in bins 7.03-7.04 and 1.10-1.11; and early, middle, and WMD QTL in bin 8.08-8.09.

^b 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).

^c The markers that flank the locus determined by multiple interval mapping. The left marker is the marker nearest the distal end of the short arm of the chromosome.

^d The chromosomal position of the predicted QTL in centimorgans (cM).

^e The additive effect of the QTL. For disease ratings, this is in terms of the 1 to 9 scale employed. For days to anthesis, this is terms of days. A positive number indicates that the allele for resistance (or late anthesis) was derived from NC300.

^f Partial R² estimates the proportion of phenotypic variance (%) explained by the detected QTL.

ACKNOWLEDGMENTS

We thank R. Baesman, V. Brewster, and J. Zwonitzer for assistance with various aspects of this work. This work was funded by the USDA-ARS and by a grant from the CGIAR Generation Challenge Program.

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