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Analysis of Digenic Epistatic and QTL × Environment Interactions for Resistance to Banded Leaf and Sheath Blight in Maize (*Zea mays*)

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ABSTRACT

Maize banded leaf and sheath blight (BLSB) due to Rhizoctonia solani Kühn, is an increasing problem in maize production areas, particularly in China and Southeast of Asian. A genetic map containing 146 simple sequence repeat (SSR) markers and 229 F2 plants derived from the cross R 15 (resistant) × Ye 478 (susceptible) were used in this study. QTL were characterized in a population of 229 F2:4 lines derived from selfing the F2 plants and were evaluated with two replications at two environments. QTL mapping analysis of disease resistance index of banded leaf and sheath blight (BLSB) in a F2: 4 population was performed with QTL Mapper 2.0 software. Twelve pairs of distinctly digenic epistatic QTL including a total of seventeen QTL were detected and distributed on seven chromosomes (2, 3, 4, 6, 7, 9 & 10). QTL main effects, epistatic effects, and QTL × environment (QE) interactions effects were predicted. Less than 20% of single effects, for identified QTL were significant at 5% level, most of which were dominance effects and additive × dominance epistatic interactions effects. There are few QTL with significantly QE interactions effects and they were mainly interactions between aa and environment. The information about QTL epistatic and QE interactions will facilitate marker-assisted (MAS) selection for BLSB resistance breeding programs in maize.

Key Words: Maize; QTL; QTL × environment interactions; BLSB
Abbreviations. AG, anastomosis groups; BLSB, banded leaf and sheath blight; cM, centimorgan; DI, disease index; GCA, general combining ability; GE, genotype × environment; LOD, log likelihood ratio; MAS, marker-assisted selection; PDA, potato dextrose agar; QE, QTL × environment; QTL, quantitative trait loci; SSR, simple sequence repeat.

INTRODUCTION

Maize banded leaf and sheath blight (BLSB), caused by Rhizoctonia solani Kühn is a highly devastating disease in most maize-growing areas of the world. R. solani is soil-borne, the sclerotia or mycelia present in the plant debris float to water surface during irrigation and flood and infect maize plants. The disease may also spread from one hill to another through leaf-to-leaf and leaf-to-sheath contacts. Infection often leads to extensive necrosis of leaf sheaths mostly in improved, semi dwarf and nitrogen-responsive maize cultivars. Although this is a regional maize disease mainly occurring in China and Southeast Asia, it is possible that this disease may spread to other parts of the world in the future (Bertus, 1927; Sharma & Saxena, 2002). Identification of QTL for resistance to this disease should facilitate the development of maize varieties (hybrids) resistance to the disease.

Most plant disease-resistant traits are quantitative in nature and are influenced by many genes or quantitative trait loci (QTL). Quantitative traits are also influenced by the environment and tend to show varied degrees of genotype × environment (GE) interactions. Epistasis, or interaction between non-allelic genes, is an important factor that affects phenotypic expression of genes and genetic variation in populations (Li *et al*., 1997). QTL × environment (QE) interaction is another important component for quantitative traits. Significant QE interactions have been reported (Zhuang *et al*., 1997; Yan *et al.*, 1998). QTL detected in one environment but not in another might indicate QE interaction (Veldboom & Lee, 1996b). But it is impossible to estimate the real QE interaction by simply comparing QTL detected in different environments. Wang *et al.* (1999) proposed a QTL mapping strategy that can estimate epistatic effects of QTL and predict their interactions with environment. However, so far estimations of dominance effects, epistatic effects related to dominance and predictions of their interactions with environment are lacking. Recently, Gao and Zhu (un-published results) extended the additive and additive × additive model by adding dominance effects,

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epistatic effects of additive × dominance and dominance × dominance as well as their interaction with environments, and updated the software QTL Mapper to version 2.0.

In the present study, a F2:4 population derived from a cross between R15 and Ye478 was used to conduct QTL study on BLSB resistance in maize at two environments, which could provide essential information to better understand the genetic control of resistance to BLSB and identify the potential target QTL to be manipulated by marker-assisted selection (MAS) in maize BLSB resistance breeding programs.

MATERIALS AND METHODS

Plant material. Two hundred and twenty nine F2:4 families were used as a mapping population coming from an elite cross between R15 (resistant) and Ye478 (susceptible), which are widely used in China. The resistant parent R15 has high tolerance to BLSB, with high general combining ability (GCA) for yield and elite yield components. The susceptible parent Ye478 is susceptible to BLSB in southwest China.

Inoculum preparation. The pathogenic fungus causing maize BLSB is Rhizoctonia solani kühn (Zhu, 1982; Yan 1984; Gao, 1987) in China and the preponderant fungus is anastomosis group AG1-IA, which has high pathogenicity and broad host spectrum (Xia & Li, 1993; Xiao, 2002). Anastomosis group AG1-IA isolates were provided by the Plant Pathology Institute of Sichuan Agricultural University, maintained on potato dextrose agar (PDA: potato, 200 g; dextrose, 20 g; agar, 10 g; H2O 1000 mL) and incubated at 26^{o}C for 3 to 5 days before use. Colonized wheat grains, for use in the field inoculations, were prepared by transferring the mycelium to sterilized wheat grain and incubating at 26^{o}C until mycelium covered the surface of the wheat grain.

Field trials. The phenotyping was carried out in field evaluations of 229 F2:4 families derived from the cross R15 and Ye478 at the Maize Research Institute farm in Ya'an city and the Institute of Agricultural Science farm in Chongqing city during 2003. The two locations represent two ecological types. The climate of Chongqing is characterized by high temperature and high humidity and that's Ya'an with much rain and low sunshine. A randomized complete block design with three replications was utilized at both locations, with plots consisting of single rows 3 m long and spaced 0.8 m apart. The plots were overplanted and thinned to 14 plants. At each location, the experimental plot size and management were in accordance with local practice. At the jointing stage, two colonized wheat grains were artificially inoculated into the third sheath of P1, P2 and all F2:4 population individual plants. All inoculations were finished in the same day.

Field data analysis. The disease resistance index was calculated according to the method of Zhao *et al.* (2005 & 2006) and the disease resistance values were used as the phenotypic values for QTL analysis. A high index value means low resistant to BLSB. Simple analysis of variance was presented to indicate whether genotypes, environments, or GE interaction were significant sources of variance or not. For each environment, the means of the disease resistance index, standard deviations, skewness and kurtosis of trait distribution were calculated. All these analyses were performed using SPSS software (2000).

Construction of linkage map. DNA samples of parents and F2 individuals were extracted as described by Saghai Maroof *et al.* (1984). Three hundred SSR markers (http://www.maizegdb.org/ssr.php) were selected for screening polymorphism between the two parents. SSR analysis followed the method described by Senior Lynn and Manfred (1993). The genetic linkage map was constructed using the software Mapmaker/EXP version 3.0 (Lander *et al*., 1987; Lincoln *et al*., 1993). Linkage groups were created with a log-10 likelihood ratio (LOD) score of 3.0 and a recombination fraction of 0.4 using the GROUP command. The THREE POINT and RIPPLE command was used to establish and verify the order of markers on each chromosome. Data quality was checked using the ERROR DETECTION command and unlikely double crossovers, due to possible genotyping errors, were corrected by rechecking the data. The map distance centi-Morgan (cM) was derived based on the Kosambi function.

QTL analysis. QTL analysis, including digenic epistatic QTL and QE interactions, was carried out using mixed linear model approaches conducted with QTL Maper V2.0 (http://www.cab.zju.edu.cn/ics/faculty/zhuj/software/QTLMaper1.6&QTLMaper2.0.zip). This program is based on mixed linear models and allows simultaneous mapping of both main effect and digenic epiststic QTL in a F2 population. The digenic epistatic loci were determined at a significance level of $p \leq 0.05$. Genetic parameters (effects & test statistics) associated with significant epistatic QTL were estimated at the positions of respective LOD peaks in individual putative QTL regions. MAPCHART software (Voorrips, 2002) was used to graphically position QTL with support interval (drop=1.0 LOD) on the linkage map. The QTL were designated according to the method introduced by McCouch *et al.* (1997).

RESULTS

Statistics of phenotypic variation. The statistical analysis showed that the disease resistant index at the two environments was not significantly different. The skewness and kurtosis were near zero at both sites (Table I), indicating the phenotypic values of the disease resistance index were normally distributed and segregated continuously, which indicate that the population was suitable for QTL mapping. Frequency distributions of disease resistance index were shown in Fig. 1 and Fig. 2. Simple analysis of variance was shown highly significant effects of genotype and genotype × environment interactions in Table II. This identified genotype and GE interactions as the major source of variation.

Map construction. One hundred and forty six SSR markers showing co-dominant segregation were employed for constructing a linkage map. The polymorphic markers were assigned into 10 linkage groups, which cover 10 chromosomes of maize. The linkage map had a total length of 1666 cM and an average interval of 11.4 cM, with approximately 80% of the genome within 20 cM of the nearest marker. Only ten markers were found that significantly deviated from the expected 1:2:1 genotypic segregation. They were distributed at chromosomes 1, 5, 6, 8 and 10.

Digenic epistatic QTL identification and location. LOD values of twelve epistatic loci were beyond the significance threshold, suggesting that these loci might be QTL controlling BLSB resistance in maize. The positions and designations for these QTL were summarized in Table III and Fig. 3. A total of seventeen QTL among twelve epistatic loci were detected and distributed on chromosomes 2, 3, 4, 6, 7, 9 and 10. However, these QTL need to be confirmed according to the significance test for their QTL main effects and QE interaction effects based on the null hypothesis for the genetic model of QTL mapping.

Estimations of genetic main effects of digenic epistatic QTL. Among 17 QTL main effects of 12 pairs of epistatic loci, less than 20% were significantly different from zero and most of these were dominance effects and additive × dominance epistatic interactions effects (Table IV). In the present study, the negative (or positive) additive effects denoted that the alleles of R15 could increase (or decrease) the resistant to BLSB, meanwhile alleles of Ye478 could decrease (or increase) the phenotype. The negative (or positive) epistatic effects among of additive × additive suggested that the two epistatic loci with homozygous alleles from the same parent could increase (or decrease) the resistant to BLSB, otherwise could decrease (or increase) the phenotype. The negative (or positive) additive × dominance (adij or adji) epistatic effects indicated that QiQi Qjqj or Qiqi QjQj could increase (or decrease) the resistant to BLSB, otherwise could decrease (or increase) the phenotype. In the present study, two pairs of epistatic loci were significantly with additive × additive interaction. Four among of six significant dominance effects were positive, three significant dominance × dominance epistatic loci were detected and its epistatic effects were all positive. Four pairs of additive × dominance epistatic interactions effects were all negative. Two pairs of epistatic QTL were not found have any significant genetic main effects.

Prediction of QE interaction effects. The advantage of QTL mapping approaches using mixed linear models is for simultaneously dealing with complicated epistatic and QE interactions, so that it provides a powerful tool for geneticists and breeders to further analyze the interaction between QTL and environment. Significant QE interaction effects were summarized in Table V. There were few QTL with significant QE interaction effects, most were the interactions between aa and environment. Two pairs of epistatic loci had not found any QE interaction effects.

DISCUSSION

Quantitative trait locus (QTL) mapping is a highly effective approach for studying genetically complex forms of plant disease resistance. With QTL mapping, the roles of specific resistance and interactions between resistance genes and the environment can be analyzed. These studies provide insights into the number of quantitative resistance loci involved in complex disease resistance and their epistatic and environmental interactions. QTL mapping also provides a framework for MAS of complex disease resistance characters and the positional cloning of partial resistance genes.

Quantitative geneticists have long recognized the importance of genotype by environment interaction and it has been documented for numerous crops and for various traits. Information about epistasis related to additive effects will be helpful to traditional breeding. In breeding practice, it is a risk to apply the superior genotype to various environments when it is predicted based on the QTL information obtained only in one environment. If the superior genotypes predicted in different environments differ greatly, their superiority may be dramatically reduced across environments. Hence, in order to develop broadly adaptable cultivars, we need to partition the QTL main effects and QE interaction effects. However, for a special environment, it is quite necessary to develop varieties specific for that environment by conducting QTL mapping study and genetic improvement on the traits of interest in that special environment (Yang & Zhu, 2005). The commonly used QTL mapping methods such as interval mapping (Lander & Botstein, 1989) or composite interval mapping (Zeng, 1994) can only detect the overall effect of single-locus QTL in separate environments. Epistatic effects between QTL are either neglected or separately analyzed using different analytical tools such as two-way ANOVA or multiple regression methods (Xiao *et al*., 1995; Li *et al.*, 1997; Yu *et al*., 1997). However, with such indirect calculation, it is difficult to evaluate the importance of additive and epistatic effects related to one QTL. Another disadvantage is that the estimation of epistasis by indirect way measures actually the effects between marker loci and the exact QTL effect is biased (Xing *et al*., 2002). QE interaction is another important component for quantitative traits. QTL detected in one environment but not in another might indicate QE interaction (Jansen *et al*., 1995; Veldboom & Lee, 1996a & b), while consistency in detection of QTL at different environments may not conclusively indicate the absence of QE interaction (Yan *et al.*, 1998). In the present study, we used a mixed model-based QTL mapping program that detects QTL with additive and epistatic effects as well as their QE interaction effects simultaneously. The information about QTL epistatic and QE interactions will facilitate marker-assisted (MAS) selection for BLSB resistance breeding programs in maize.

Fig. 1 Frequency distribution of phenotypic values of the BLSB disease resistance index in Ya'an environment

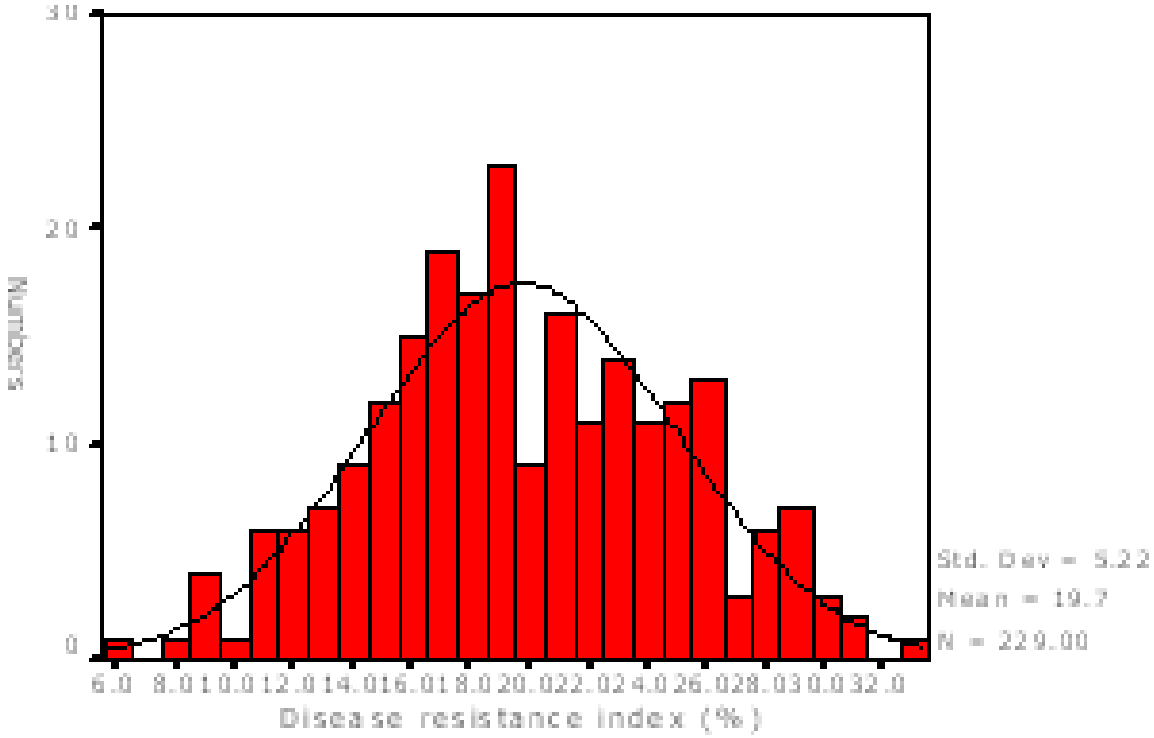

Fig. 2 Frequency distribution of phenotypic values of the BLSB disease resistance index in Chongqing environment

Table I. Summary statistics for phenotypic values of the disease resistance index

Resistance Index	*Mean*[a]	*Range*	*Coefficient of Variation (%)*	*Kurtosis*	*Skewness*
Ya'an (%)	19.73 ±5.22	5.79~33.33	0.24	-0.41	0.05
Chongqing (%)	19.81 ±5.22	7.14~32.64	0.32	-0.47	0.20

[a] With standard errors.

Table II. Simple analysis of variance of the BLSB disease resistance index in two environments

Source	*DF*	*SS*	*Mean Squares*	*F*	*P*
Environment(E)	1	1.26	1.26	0.04	NS
Genotype(G)	228	15156.21	66.47	1.94	P< 0.0001
G × *E*	228	9699.26	42.54	1.24	0.05 < P < 0.10
Error	458	15723.03	34.32		

Table III. Digenic epistatic QTL controlling BLSB resistant in maize

	QTL$_i$ [a]				*QTL* $_j$		
Chr.	**Marker interval**	**Site1(M)** [b]	**QTL designation**	**Chr.**	**Marker interval**	**Site2(M)**	**QTL designation**
2	Umc1285-nc003	0.12	*BLSB2-1*	9	umc1231-umc2343	0.00	*BLSB9-1*
2	umc2150-bnlg1721	0.00	*BLSB2-2*	7	bnlg2132-umc1016	0.00	*BLSB7-1*
2	bnlg1662-bnlg1606	0.06	*BLSB2-3*	3	bnlg1523-bnlg1447	0.00	*BLSB3-1*
2	bnlg1662-bnlg1606	0.00	*BLSB2-3*	7	umc1125-umc1154	0.00	*BLSB7-2*
2	bnlg1606-bnlg1940	0.00	*BLSB2-4*	3	umc1659-umc1052	0.00	*BLSB3-2*
2	bnlg1606-bnlg1940	0.00	*BLSB2-4*	4	bnlg2162-umc1051	0.08	*BLSB4-1*
3	umc1659-umc1052	0.38	*BLSB3-2*	4	umc2281-umc1662	0.00	*BLSB4-2*
3	umc1659-umc1052	0.38	*BLSB3-2*	4	bnlg1621-umc1299	0.00	*BLSB4-3*
6	bnlg1538-umc1818	0.00	*BLSB6-1*	10	phi118-umc1319	0.04	*BLSB10-1*
6	umc1818-umc1083	0.00	*BLSB6-2*	6	umc1723-umc1014	0.02	*BLSB6-3*
6	umc1818-umc1083	0.00	*BLSB6-2*	10	phi118-umc1319	0.08	*BLSB10-1*
6	umc1818-umc1083	0.00	*BLSB6-2*	10	mmc0501-phi054	0.14	*BLSB10-2*

[a] QTL $_i$ and QTL $_j$ are a pair of QTL detected by two-dimensional searching;
[b] The Site1(M) column is the genetic distance (in Morgan) of the testing points from the left end of the chromosomes on which the testing points are set. The Site2(M) column is the genetic distance (in Morgan) of the testing points from the left marker on the interval on which the testing points are set.

Table IV. Estimation of epistatic QTL for BLSB resistant in maize

QTL i	*QTL j*	*LOD*	a_i [a]	d_i	d_j	aa_j	ad_{ij}	da_{ij}	dd_{ij}
BLSB2-1	*BLSB9-1*	10.27							3.01*
BLSB2-2	*BLSB7-1*	6.97	-0.77*	1.40*					
BLSB2-3	*BLSB3-1*	10.93		1.42*					
BLSB2-3	*BLSB7-2*	7.42				1.33**			
BLSB2-4	*BLSB3-2*	11.29		1.63*					
BLSB2-4	***BLSB4-1***	6.02							
BLSB3-2	*BLSB4-2*	9.15			-1.98*				
BLSB3-2	*BLSB4-3*	6.02	-1.09*				-2.31**		
BLSB6-1	*BLSB10-1*	7.03		-1.85*		-0.86*		-2.39*	4.90***
BLSB6-2	***BLSB6-3***	6.22							
BLSB6-2	*BLSB10-1*	6.60					-1.40*	-2.53**	3.82**
BLSB6-2	*BLSB10-2*	7.81			1.70*				

[a] a_i and d_i are the additive and dominance effects of QTL $_i$, respectively; d_j are the dominance effects of QTL $_j$; aa_{ij}, ad_{ij} and dd_{ij} are the epistatic effects of additive × additive, additive × dominance, and dominance × dominance between QTL $_i$ and QTL $_j$, respectively;
*, ** and *** denote significance level at 0.05, 0.01 and 0.005, respectively.

Epistasis and QE interaction are challenge to plant breeders and has been shown to reduce the progress of the quantitative traits from selection. QE interactions are vital in expression of the QTL effect. In the present study, the most important result is the statistical characterization of the genetic components that control the expression of the traits, including main effects of the epistatic QTL and QTL by environmental interactions. In the present study, nine pairs of QTL had aae effects, while two other pairs had only aa effects and one pair had no any aa or aae effect. It was shown that aae effects were more often detected than aa effects. This indicated that environments could greatly affect the gene expression for epistatic effects on developmental traits. The composition of epistatic interactions was interesting on that all QTL with additive effects were not engaged in digenic epistatic interactions. The results might suggest that the epistatic interactions may be largely due to induction of the loci without detectable QTL additive effect, as signify

Table V. Prediction of *QE* interaction effects for BLSB resistant in maize

Environment	QTL_i	QTL_j	$e_{DiE\ h}$	$e_{AjE\ h}$	$e_{AAijE\ h}$
Ya'an	*BLSB2-1*	*BLSB9-1*			-1.26***
(h=1)	*BLSB2-2*	*BLSB7-1*			0.94**
	BLSB2-3	*BLSB3-1*			-1.35***
	BLSB2-3	***BLSB7-2***			
	BLSB2-4	*BLSB3-2*			-1.13***
	BLSB2-4	*BLSB4-1*			0.58*
	BLSB3-2	*BLSB4-2*			1.22***
	BLSB3-2	*BLSB4-3*			-0.95**
	BLSB6-1	***BLSB10-1***			
	BLSB6-2	*BLSB6-3*			0.99*
	BLSB6-2	*BLSB10-1*			0.93*
	BLSB6-2	*BLSB10-2*	-1.75***	0.79*	
Chongqing	*BLSB2-1*	*BLSB9-1*			1.26***
(h=2)	*BLSB2-2*	*BLSB7-1*			-0.94**
	BLSB2-3	*BLSB3-1*			1.35***
	BLSB2-3	***BLSB7-2***			
	BLSB2-4	*BLSB3-2*			1.13***
	BLSB2-4	*BLSB4-1*			-0.58*
	BLSB3-2	*BLSB4-2*			-1.22***
	BLSB3-2	*BLSB4-3*			0.95**
	BLSB6-1	***BLSB10-1***			
	BLSB6-2	*BLSB6-3*			-0.99*
	BLSB6-2	*BLSB10-1*			-0.93*
	BLSB6-2	*BLSB10-2*	1.75***	-0.79*	

[a] $e_{AjE\ h}$ is additive interactions of QTL_j with environment *h*; $e_{DiE\ h}$ is dominance interactions of QTL_i with environment *h*; $e_{AAijE\ h}$ is the interactions between AA_{ij} and environment *h*;
*, **, *** is significance level at 0.05, 0.01 and 0.005, respectively
The negative means the values mainly come from the small value parent

the importance of keeping the concept in mind that the loci without detectable QTL additive effect can also be putative QTL when doing QTL analysis. In the present study, one pair of epistatic interactions loci of qBLSB2-4 and qBLSB4-1 did not have any significant additive effects and additive × additive interaction effects, but had epistasis × environment interaction effects (aae). The other pair of epistatic interactions loci of qBLSB6-1 and qBLSB10-1 had significant negative dominance effects, additive × additive epistatic effects, additive × dominance epistatic effects, and positive dominance × dominance epistatic effects, but had no any QTL × environment interactions effects. Therefore pyramiding and manipulation of genes in selection programs should consider not only the additive effects of genes or QTL, but also the additive × additive epistatic effects, and epistatic × environment interaction effects among these genes and others.

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