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QTLs for Fusarium head blight response in a wheat DH population of Wangshuibai/Alondra's'

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Summary

A doubled haploid (DH) wheat population derived from the cross Wangshuibai/Alondra's' was developed through chromosome doubling of haploids generated by anther culture of hybrids. Fusarium head blight (FHB) was evaluated for three years from 2001 to 2003 in Jianyang, Fujian Province, China, where epidemics of FHB have been consistently severe. After 307 pairs of simple sequence repeat (SSR) primers were screened, 110 pairs were polymorphic between Wangshuibai and Alondra's', and used to construct a genetic linkage map for detection of quantitative trait loci (QTLs). A stable QTL for low FHB severity was detected on chromosomes 3B over all three years, and QTLs on chromosomes 5B, 2D, and 7A were detected over two years. Additional QTLs on chromosomes 3A, 3D, 4B, 5A, 5D, 6B and 7B showed marginal significance in only one year. Six QTLs were detected when phenotypic data from three years were combined. In addition, significant additive-by-additive epistasis was detected for a QTL on 6A although its additive effect was not significant. Additive effects (A) and additive-by-additive epistasis (AA) explained a major portion of the phenotypic variation (76.5%) for FHB response. *Xgwm533-3B* and *Xgwm335-5B* were the closest markers to QTLs, and have potential to be used as selectable markers for marker-assisted selection (MAS) in wheat breeding programs.

Introduction

Fusarium head blight (FHB), mainly caused by Fusarium graminearum Schw., can cause great losses in grain yield and grain quality. In recent years, FHB epidemics have become more frequent and severe in China and have extended to the northern wheat-growing areas where FHB epidemics were previously rare, due to warmer weather and changes in cultivation practice (Chen et al., 1997; Lu et al., 2001). Many reports showed that the inheritance of FHB resistance is a quantitative trait controlled by a few major genes plus several minor genes (Liao & Yu, 1985; Bai et al., 1989; Liu et al., 2005). Several molecular marker systems have been used to characterize FHB resistance (Bai et al., 1999; Zhou et al., 2002a; Guo et al., 2003; Sun et al., 2003; Xu & Ban, 2004). Quantitative trait loci (QTLs) for FHB resistance have been associated with most wheat chromosomes. Conclusions on the location and number of those QTLs were inconsistent among reports due to different sources of resistance, mapping populations, FHB evaluation methods and environments used for disease evaluation (Waldron et al., 1999; Otto et al., 2002; Buerstmayr et al., 2002; Gervais et al., 2003; Shen et al., 2003a, 2003b; Lin et al., 2004; Steiner et al., 2004; Zhang et al., 2004; Zhou et al., 2004).

Wheat cultivar Sumai 3 from China has been recognized worldwide as the best source of resistance and extensively used in wheat breeding programs. QTLs for FHB resistance in Sumai 3 have been extensively investigated and molecular markers for the major QTL on 3BS have been widely recommended for MAS (Bai et al., 1999; Waldron et al., 1999; Zhou et al., 2002b; Liu & Anderson, 2003; Del Blanco et al., 2003). However, Wangshuibai, a landrace originating from Jiangsu Province, China, also showed a high level of resistance to FHB in field conditions (Chen et al., 1997). Mapping indicated that a QTL on 3BS was also important for FHB resistance in Wangshuibai, but its contribution was different from that of Sumai 3 (Zhou et al., 2002a, 2004; Guo et al., 2003; Lin et al., 2004; Zhang et al., 2004). Therefore, exploration of the interactions among QTLs and between QTL and environment may provide further understanding of the genetic mechanisms of FHB resistance in Wangshuibai and information for the efficient use of Wangshuibai as an alternative source of resistance in breeding programs.

In this study, we developed a doubled haploid (DH) mapping population using Wangshuibai as the resistant parent and Alondra's' as the susceptible parent. This population was evaluated for FHB response for three years under FHB epidemic conditions in the field. QTLs for FHB resistance were identified and the interactions between these QTLs and between QTLs and environments were elucidated.

Materials and methods

Production of DH population

A mapping population with 134 DH lines was developed from the cross Wangshuibai/Alondra's' following anther culture of F_1 hybrids based on the method of Bajaj (1983). Wangshuibai is a Chinese landrace with a high level of FHB resistance, whereas Alondra's' is a highly susceptible cultivar from CIMMYT, Mexico.

FHB evaluation

FHB responses of DH population were evaluated for three years from 2001 to 2003 in Jianyang, Fujian Province, where natural infection of FHB has been consistently severe for several decades. The DH population and its parents were planted in the field following a complete random block design with two replications. For each replication, 30 seeds per DH line were spaceplanted in a 1.5 m-row. About 15 and 20 days after flowering, 15 spikes per row were randomly selected for FHB evaluation. FHB severity was assessed as number of scabbed spikelets and percentage of scabbed spikelets in a spike for each DH line. The normal distribution of DH population was determined by the Kolmogorov-Smirnov statistical method (Boes et al., 1974).

Marker analysis

Genomic DNA was extracted from leaf tissue collected from the wheat seedlings grown in the field at NAU in 2001 according to the method of Gill et al. (1991). A total of 155 pairs of GWM SSR primers (Röder et al., 1998) were synthesized by Shanghai Genebase Genetech. Corp., Ltd. (Shanghai, China), and 152 pairs of BARC SSR primers were kindly provided by Dr. P.B. Cregan and Dr. Q.J. Song (Song et al., 2005).

All PCRs were performed in PE-9600 thermocyclers. For SSR, a 20 μl volume of reaction mixture contained 250 nM of each primer, 0.2 mM dNTPs, 1.2 mM $MgCl_2$, 1 $\times$ PCR buffer, 1U *Taq* polymerase, and about 80 ng of template DNA. PCR amplification started at 94 °C for 3 min, followed by 35 cycles of 1 min at 94 °C, 40 s at either 50, 55, or 60 °C depending on the annealing temperatures of individual SSR primers, 45 s at 72 °C, with a final extension step of 10 min at 72 °C. PCR products were separated in a 6% polyacrylamide gel and visualized by silver staining (Sourdille et al., 1998).

QTLs analysis

Marker data were scored from gels by visual inspection. A DH line with the same DNA banding pattern as that from Wangshuibai was assigned as 1, and that from Alondra's' was assigned as 3. A genetic linkage map was constructed using MAPMAKER version 3.0 (Lincoln et al., 1992). Genetic distance (cM) was calculated based on Haldane mapping function (Lander et al., 1987) and LOD threshold was set at 3.0. QTLMapper V2.0 was used for QTL detection (Wang et al., 1999; Gao et al., 2004) and for calculation of QTL main effects, epistatic effects and interaction effects between QTLs and environments using a mixed linear model (Wang et al., 1999; Zhu, 1999; Gao et al., 2004). In this model, the phenotypic value y_{hk} of a DH line in environment h can be partitioned as the

following (Zhu & Weir, 1998; Zhu, 1999):

$$y_{hk} = \mu + a_i x_{A_{ik}} + a_j x_{A_{jk}} + aa_{ij} x_{AA_{ijk}} + \mu_{E_{hk}} e_{E_h} + \mu_{A_i E_{hk}} e_{A_i E_h} + \mu_{A_j E_{hk}} e_{A_j E_h} + \mu_{AA_{ij} E_{hk}} e_{AA_{ij} E_h} + \sum_{f(h)} u_{M_{fk(h)}} e_{M_{f(h)}} + \sum_{l(h)} u_{MM_{lk(h)}} e_{MM_{l(h)}} + \varepsilon_{hk}$$

where μ is the population mean; a_i and a_j are the additive effects (fixed effects) of two putative QTLs *Qi* and *Qj*, respectively; aa_{ij} is the additive-by-additive epistatic effect (fixed effect) between the two QTL; $x_{A_{ik}}$, $x_{A_{jk}}$ and $x_{AA_{ijk}}$ are the coefficients of these main effects; e_{E_h} is the random effect of environment h with a coefficient $\mu_{E_{hk}}$; $e_{A_i E_h}$ (or $e_{A_j E_h}$) is the random effect of additive-by-environment interaction with a coefficient $\mu_{A_i E_{hk}}$ (or $\mu_{A_j E_{hk}}$) for *Qi* (or *Qj*); $e_{AA_{ij} E_{hk}}$ is the random epistasis-by-environment interaction effect with a coefficient $\mu_{AA_{ij} E_{hk}}$; $e_{M_{f(h)}}$ is the random effect of marker f nested within the h environment with a coefficient $\mu_{M_{fk(h)}}$, $e_{MM_{l(h)}}$ is the random effect of the l marker-by-marker interaction nested within the h environment with a coefficient $\mu_{MM_{lk(h)}}$; ε_{hk} residual effect. The marker factors $e_{M_{f(h)}}$ and $e_{MM_{l(h)}}$ in the model are used to absorb additive and epistatic effects of background QTLs to control the background noise.

Background genetic variation (BGV) was used as a control to calculate main effect of a marker and interaction between markers. The LOD threshold was set at $\alpha = 0.005$ to claim significance of QTLs.

Results

Variation in FHB response

The mean value of infected spikelets was 6.7 with a standard deviation of 3.5 from 2001 to 2003 (Table 1). The most resistant lines had 2.8% infected spikelets per spike, whereas the most susceptible lines had 96.1% infected spikelets per spike. Different genotypes in the population were well expressed and separated based on their levels of resistance. On the other hand, variances for FHB severities were significant among the three years ($F = 7.94$, $P < 0.01$). The most severe FHB was observed in 2001 with a long period of rainy weather from flowering to kernel filling stage, whereas FHB was the least severe in 2003. Though FHB response was affected by the weather conditions, highly positive correlations were still observed for severity ratings among years. Correlation coefficients were 0.61, 0.38 and 0.34 between 2001 and 2002, 2001 and 2003, 2002 and 2003, respectively ($P < 0.01$). The level of FHB response for each DH line was relatively consistent among years. The results suggested that genetic factors play a major role in FHB response. In addition, a normal distribution was observed for plant height in the DH population with a Kolmogorw-Smimov's statistic value of 0.048 (P 0.20); the correlation between plant height and FHB severity was not significant among the three years ($r = -0.095$). The frequency distribution of FHB severities was continuous with two peaks in all three years (Figure 1) and therefore, FHB response was characterized as a quantitative trait controlled by a few major QTLs and some minor QTLs.

SSR markers linked to FHB resistance

Of the 307 SSR primers screened, 110 (35.8%) were polymorphic between the parents, and were therefore used to screen the DH population. Allelic frequencies were analyzed for all polymorphic SSR markers. One-hundred-and-four markers exhibited 1:1 ratios of random segregation, and were mapped on 18 chromosomes with at least two markers in each chromosome, based on published information from SSR maps (Röder et al., 1998; Song et al., 2005). Three chromosomes had only one marker. Overall, these markers covered about

Table 1. Statistics of FHB severity ratings for the DH lines derived from Wangshuibai/Alondra's' and their parents evaluated at Jianyang, Fujian, 2001–2003

		Parental response		DHLs			
Item	Year	Wangshuibai	Alondra's'	Min.	Max.	Mean	Standard deviation
No. of scabbed spikelets/spike	2001	0.8	16.0	0.6	18.9	8.8	4.4
	2002	1.0	8.0	0.7	18.3	6.9	3.7
	2003	0.3	6.8	0.5	10.5	4.9	2.7
	Mean	0.7	10.3	0.5	18.9	6.7	3.5

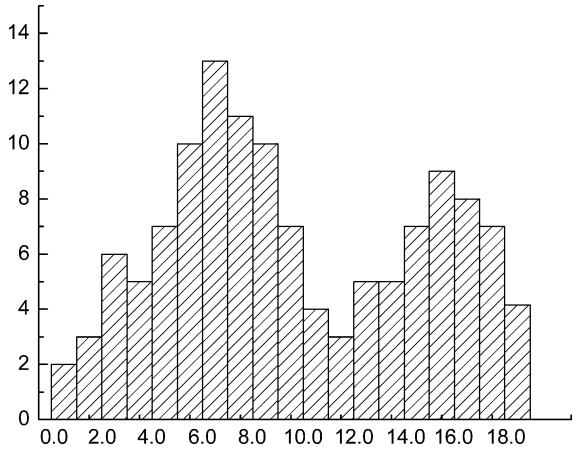

Figure 1. Frequency distribution of number of scabbed spikelets per infected spike for the DH population derived from Wangshuibai/Alondra's' evaluated in Jianyang, Fujian, China, 2001. X axis: No. of Scabbed spikelets, Y axis: No. of lines.

1170 cM. More markers mapped on chromosomes 3B (12), 7B (11) and 5B (9).

Single marker analysis of variance (ANOVA) was conducted to associate SSR markers with FHB resistance in the DH population. Markers *Xgwm533-3B*, *Xgwm335-5B*, *Xgwm282-7A*, and *Xgwm611-7B* showed significant associations with FHB resistance in three consecutive years. Markers *Xgwm644-7B*, *Xgwm341-3D* showed significant associations with FHB resistance in two years, and markers *Xgwm68-7B*, *Xgwm337-1D* and *Xgwm111-7D* showed marginal significance for association with FHB resistance in one year. Thus chromosomes 3B, 3D, 5B, 7A, and 7B may carry QTLs for FHB resistance.

Interval mapping of QTLs for FHB resistance in Wangshuibai

QTLMapper v2.0 was used to detect QTLs through composite interval mapping (CIM). Seven, five and six putative QTLs were detected in 2001, 2002 and 2003, respectively (Table 2). A QTL on the chromosome 3B was consistently significant over three years. This QTL played an important role in FHB resistance and was least affected by environments. Interval mapping also detected QTLs on chromosomes 5B, 2D and 7A from two years of data. Among them, the QTLs on 5B made the largest mean contribution to reduced FHB severity whereas the mean contribution of the QTL on 2D was the smallest. One QTL on each of chromosomes 3A, 3D, 4B, 5A, 5D and 6B as well as two on 7B were detected only in one year. Two QTLs on chromosomes 5D and 7B demonstrated increased FHB severity in 2002 and 2003, respectively. QTLs on 1D and 7D detected by single marker analysis were not significant in CIM.

Since the effects of QTLs varied with years due to the variation in weather conditions after anthesis, averaged disease data over three years for each DHL may provide more accurate estimates for QTL effect and location. Using these data, six QTLs were identified on chromosomes 3B, 5B (two QTLs), 2D, 4B and 7A. Four of them were previously detected based on two-years' phenotypic data and two were detected from one-year's data. New QTL was not found using the combined data. Genetic distances between markers and peaks of putative QTLs derived from the combined data were shorter than those derived from single years (Table 2, Figure 2). For example, the genetic distance between the QTL on 3BS and the closest marker was only 5cM based on analysis of averaged data, but 8cM, 10cM and 6cM based on the data from 2001, 2002 and 2003, respectively.

Interactions between QTLs and between QTL and environment

Besides additive effects, additive epistatic interactions between QTLs were also significant between some QTLs detected by interval analysis (Table 3). Significant interactions between QTLs on chromosomes 3A and 6A and between QTLs on 4B and 5B resulted in increased FHB resistance. However, interaction between QTLs on 3A and 4B and between 4B and 5D led to increases of 1.3 and 1.4 scabbed spikelets per spike, respectively. The interaction between the QTL on 2D and three other QTLs also resulted in slightly increased numbers of scabbed spikelets. Although no QTL was detected on chromosome 6A in CIM, a significant epistatic effect was observed between QTLs on 6A and 3A, and the epistatic interaction resulted in a decrease of 2.8 scabbed spikelets per spike.

Interactions between QTLs on chromosomes 3A, 4B, 5B, 5D, and 6A and environment were evident. The additive-by-environment contribution ranged from 0.7% to 4.1% with the largest interaction effect of QTLs on 5B and 2D (4.1 and 3.9%). Significant interaction also occurred between environment and additive-by-additive epistasis of the QTLs on 6A and 3A (4.0%). The interaction between the additive epistasis and environment was trivial for other QTLs in this study (less than 1.0%). In summary, additive effects and additive-by-additive epistasis explained about 50.0%

Table 2. Additive effects of QTLs for FHB resistance of the DH population derived from Wangshuibai/Alondra's' evaluated in Jianyang, Fujian, 2001–2003

Year	Chromosome	Marker interval	Genetic distance (cM)[a]	LOD[b]	R^2 (%)[c]	Additive effect (A)	Additive contribution (%)[d]
2001	2D	Xgwm261-Xgwm484	11.0	2.15	8.2	−0.91	4.0
	3A	Xgwm369-Xbarc045	14.0	2.59	12.3	−1.05	4.7
	3B	Xgwm533-Xgwm493	8.0	2.80	14.4	−1.35	8.0
	4B	Xgwm368-Xgwm149	16.0	2.70	10.5	−1.78	10.8
	5B	Xgwm335-Xgwm371	13.0	3.15	15.7	−2.90	15.5
	6B	Xgwm133-Xgwm191	8.0	2.31	8.8	−0.75	3.7
	7A	Xgwm276-Xgwm282	17.0	2.84	12.0	−1.53	8.8
2002	2D	Xgwm261-Xgwm484	14.0	2.90	8.0	−1.01	8.5
	3B	Xgwm533-Xgwm493	10.0	2.33	12.7	−0.85	7.8
	5B	Xgwm443-Xbarc32	10.0	2.70	13.1	−1.13	12.4
	5D	Xgwm190-Xgwm358	15.0	2.60	11.6	0.82	6.7
	7B	Xgwm297-Xgwm644	10.0	2.67	7.9	−1.08	9.0
2003	3B	Xgwm533-Xgwm493	6.0	2.55	13.0	−1.04	10.2
	3D	Xgwm645-Xgwm383	18.0	2.66	11.6	−1.10	10.7
	5A	Xgwm129-Xgwm156	20.0	3.54	14.5	−1.32	11.9
	5B	Xgwm335-Xgwm371	16.0	2.18	12.2	−0.80	6.6
	7A	Xgwm276-Xgwm282	15.0	2.40	7.7	−0.91	7.1
	7B	Xgwm146-Xgwm611	12.0	2.35	11.4	0.75	5.0
2001–2003	2D	Xgwm261-Xgwm484	10.0	2.46	10.6	−0.75	4.7
	3B	Xgwm533-Xgwm493	5.0	2.54	11.0	−1.06	9.0
	4B	Xgwm368-Xgwm149	11.0	2.47	9.9	−0.85	5.2
	5B	Xgwm443-Xbarc32	8.0	2.86	13.3	−1.21	10.6
	5B	Xgwm335-Xgwm371	14.0	2.05	10.8	−1.19	9.8
	7A	Xgwm276-Xgwm282	15.0	2.75	12.6	−1.20	10.1

General contributions: additive effect (A): $h^2_{(A)} = 57.8\%$ (2001), 48.2% (2002), 53.6% (2003), 49.5% (2001–2003).
[a]Genetic distance (cM) between the left marker and the peak of the QTL.
[b]LOD scores were derived from the LR values where $1\ LOD = 0.217\ LR$, the likelihood ratio values for the QTL peak.
[c]R^2 is the determination coefficient generated by stepwise regression method based on mixed linear model.
[d]Additive contribution is a relative contribution (h^2) and estimates the proportion of additive variance to the total phenotypic variance using the formula $h^2 = \sigma^2_g/\sigma^2_g + s^2_e$.

Table 3. Epistatic effect of QTLs for FHB resistance of the DH population derived from Wangshuibai/Alondra's' evaluated in Jianyang, Fujian, 2001–2003

Chrom.	Marker	Distance (cM)[a]	Chrom.	Marker	Distance (cM)[a]	LOD	Additive epistatic effect (AA)[b]	R^2 (%)	Relative contribution (%)
2D	Xgwm261	8.0	4B	Xgwm368	16.0	2.21	0.46	6.4	1.5
2D	Xgwm261	14.0	5B	Xgwm335	18.0	2.81	0.27	5.9	3.5
2D	Xgwm261	16.0	5D	Xgwm190	13.0	2.68	0.37	7.0	3.4
3A	Xgwm369	14.0	4B	Xgwm368	20.0	3.38	1.42	9.5	4.3
3A	Xgwm369	16.0	6A	Xgwm169	4.0	3.56	−2.78	12.0	7.5
4B	Xgwm368	22.0	5B	Xgwm335	5.0	2.31	−0.85	8.9	1.6
4B	Xgwm368	12.0	5D	Xgwm190	13.0	2.89	1.30	9.0	3.3

General contributions: additive epistasis (AA): $h^2_{(AA)} = 26.5\%$.
[a]Distance (cM) between QTL peak and the closest marker.
[b]Additive epistasis effect (AA) means the interaction effect between two QTLs.

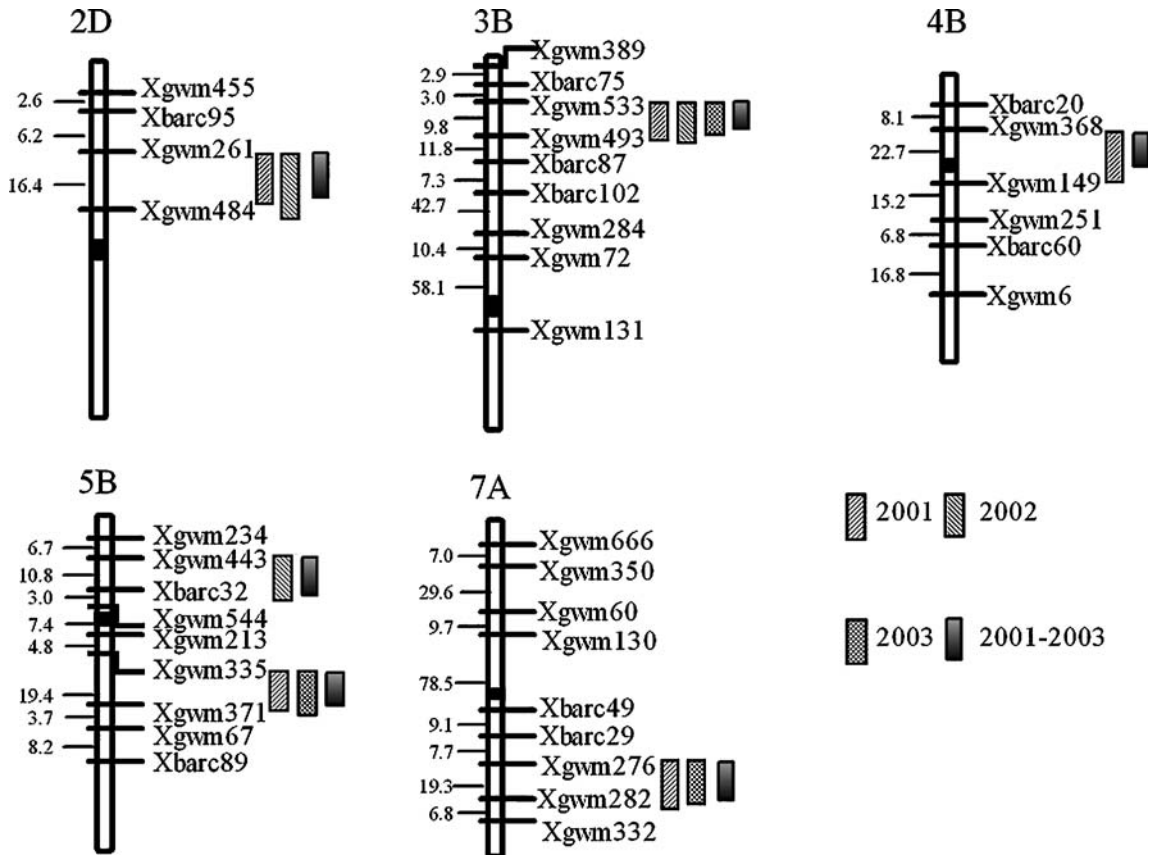

Figure 2. Map locations of QTLs for Fusarium head blight resistance in the DH population derived from Wangshuibai/Alondra's'.

(48.2–57.8%) and 26.5%, respectively, of phenotypic variances for FHB response. Environmental effects and G×E interaction were relatively low.

Potential markers for marker assistant-selection (MAS)

SSR markers *Xgwm493* and *Xgwm533* linked to the QTL on 3BS and *Xgwm335* and *Xgwm371* linked to QTLs on 5B were analyzed for effectiveness of using the SSRs as selectable markers. The DH lines in the population were divided into two groups based on marker alleles (Table 4). Average numbers of scabbed spikelets per spike were significantly different between allelic groups for markers *Xgwm493*, *Xgwm533* and *Xgwm335* over three years. Among them, *Xgwm533* showed the most significant effect in reducing FHB severity and is a suitable marker for MAS. *Xgwm335* is a good marker for MAS of the QTL on chromosome 5B.

Discussion

FHB resistance is a complex trait and its expression can be affected by genetic background, disease evaluation method, and the environment used for disease evaluation. Multiple tests of a permanent population are essential for accurate mapping of QTLs for FHB resistance. A DH population consists of genetically homozygous lines without allelic segregation within each line. This population can be repeatedly tested for FHB resistance in different locations for several years. In addition, it takes much less time to develop a DH population than that for development of a recombinant inbred population.

The DH population was evaluated for FHB response in the field at Jianyang, Fujian Province, where natural FHB epidemics have been severe and frequent (Chen et al., 1997). In that environment consistent results were obtained in the three successive years of experimentation. Disease symptoms did not spread from initial infection sites in resistant DH lines, whereas

Table 4. Effect of marker-assistant selection based on SSR markers

		2001		2002		2003	
Marker locus	Marker allele[a]	Mean scabbed spikelets	*t*-test	Mean scabbed Spikelets	*t*-test[b]	Mean scabbed spikelets	*t*-test
Xgwm493-3B	'1'	7.33	3.05**	5.61	2.15*	3.87	2.48*
	'3'	9.75		7.20		5.02	
Xgwm533-3B	'1'	6.12	7.05**	4.94	6.81**	3.41	5.84**
	'3'	10.64		8.11		5.63	
Xgwm335-5B	'1'	6.01	7.00**	4.90	4.29**	4.06	2.17*
	'3'	10.58		8.00		5.23	
Xgwm371-5B	'1'	7.09	6.87**	5.70	1.62	4.29	2.31*
	'3'	10.66		6.50		5.55	

[a]'1' indicates marker allele from the resistant parent, Wangshuibai, and '3' indicates marker allele from the susceptible parent Alondra's'.
[b](*,**) significant *t*-value at $p = 0.05$ and $p = 0.01$, respectively.

there was spread over all spikelets of infected spikes in some highly susceptible DH lines. Although the disease pressure in the third year was lower than the first two years, the correlations between the data from the third year and data from first two years remained highly significant. Therefore, the FHB data from this study were reliable for QTL analysis, especially from the first two years. Numbers of QTLs detected from three years of data ranged from five to seven, and four QTLs appeared at least twice in the same genomic regions in three years. Six QTLs were detected when three-year mean data were analyzed; these included the four QTLs that were significant for two years. Because combined data summarized FHB severities for each line from three experiments, they may provide more accurate information on number and location of QTLs, with better estimation of genotypic and environmental effects. Based on combined data, putative QTLs for FHB resistance in Wangshuibai mainly located on chromosomes 3B, 5B, 2D, 7A and 4B.

The DH population was subjected to natural infection in the field where there were opportunities for multiple initial infection sites. Therefore, the QTLs identified for FHB resistance in this study are most likely to include resistance to both initial infection (Type I resistance) and FHB spread within a spike (Type II resistance) (Schroeder & Christensen, 1963). The results should be highly applicable to breeding programs.

Many researchers have reported QTL mapping work on Sumai 3 and its derivatives. A major QTL on 3BS has explained up to 60% of the phenotypic variance for FHB response in several studies (Bai et al., 1999; Waldron et al., 1999; Buerstmayr et al., 2002; Guo et al., 2003). Other minor QTLs on several chromosomes were also reported, but the locations and effects of these were inconsistent across studies (Bai et al., 1999; Waldron et al., 1999; Buerstmayr et al., 2002). QTLs from non-Sumai 3 sources have also been detected on 1B, 3A, 3D and 5A from F201R/Patterson (Shen et al., 2003a), 2A, 2B, 3A, 3B, 5A, 5D and 6D from Renan/Récital (Gervais et al., 2003), and 1B, 2A, 2B, 3A, 3B, 4B, 5A and 6B from Frontana/Remus (Steiner et al., 2004). The Chinese landrace Wangshuibai has shown the highest resistance to FHB in field trials (Chen et al., 1997; Jia et al., 2005). Monosomic analysis located FHB-resistance genes on 4A, 4D, 5A, 7A and 7B (Liao & Yu, 1985). Zhou et al. (2004) located one QTL on 3BS, explaining 37.3% of the phenotypic variation for FHB response, and additional QTLs on 3BS, 7AL and 1BL explained 7.4, 9.8 and 11.9% of the phenotypic variation, respectively, in RILs derived from Wangshuibai/Wheaton. Zhang et al. (2004) found a QTL on 3BS, explaining 23.8% of the phenotypic variation among RILs derived from Wangshuibai/Alondra's'. An additional QTL was detected on 1B in the same study. In our study, six QTLs were detected on 3BS, 5B, 2D, 7A and 4B in the DH population of Wangshuibai/Alondra's' when combined data from three years were used. The QTL on 3BS was detected across all three years. Since the QTL on 3BS was highly significant in different populations derived from Wangshuibai, it is clearly a stable QTL. However, its effect was smaller than that from Sumai 3 although it mapped to the same location. When data for three years were analyzed separately, putative QTLs appeared on eleven chromosomes at least once. QTLs on 3B and 5B had relatively large effects and their linked markers *Xgwm533* and *Xgwm335* should be suitable for MAS.

QTLs on 2D and 7A were detected in two years and were also significant when the phenotypic data were combined from three years. These QTLs have smaller effects and might be minor QTLs for FHB resistance. Another minor QTL on 4B was significant when combined data were analyzed. This QTL showed significant interactions with other QTLs on 2D, 3A, 5B and 5D. Other minor QTLs were detected in only one year and had significant interaction with environmental conditions. Therefore, they are not stable and may not be reliable for use in MAS.

The phenotypic variance for response to FHB was separated into several components including additive (A), additive-by-additive (AA), additive-by-environment (AE) and epitasis-by-environment (AAE). In this study, additive effects made up a predominant component of the phenotypic variance (~50%), but additive-by-additive epitasis also played a major role and explained 26.5% of total phenotypic variance when combined data were analyzed. An additive effect for the QTL on chromosome 6A was not detected, but epistasis was significant when it was combined with other QTLs. Accumulation of several QTLs may enhance FHB resistance in a cultivar. However, pyramiding of different QTLs may not always result in increased FHB resistance. Additive epistasis in some cases in this study led to a decreased FHB resistance. For example, adding QTLs from 4B or 3A and 6A may significantly decrease FHB in some years. QTL on 3B and 5B did not show interaction with each other, therefore, pyramiding of these QTLs should increase resistance. Additive-by-environment and epistasis-by-environment accounted for a relatively small portion of the phenotypic variation. Finally, evaluation of FHB resistance in multiple years and locations may reduce environmental effects and provide more reliable phenotypic data for measuring QTL effects.

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